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Review

Assessing the Effects of Pesticides on Aquacultured Fish and Ecosystems: A Comprehensive Environmental Health Review

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Abstract: Aquaculture has been rapidly growing during the past decade to accommodate the increasing need for seafood as a vital source of nutrients for human beings. The nutritional benefits of incorporating fish into one's diet are paramount in promoting overall health, bolstering immunity and warding off diseases. Nonetheless, farm-raised aquatic species are frequently subjected to elevated contamination levels due to pesticides, antibiotics, and heavy metals in the marine environment. Pesticides affect fish differently based on species, class, dosage, and exposure duration. They can induce histological damage or neurobehavioral changes by inhibiting acetylcholinesterase production. This can promote liver dysfunction, metabolism deregulation, oxidative stress, and hematological imbalances, impair immune responses, and adversely affect fish reproduction. Furthermore, pesticides negatively affect the nutritional composition of fish by reducing the total protein levels in muscle, liver, gills, and kidney tissues. They disrupt lipid metabolism, resulting in lipid accumulation in the liver and a decrease in polyunsaturated fatty acids. Additionally, pesticides interfere with metabolism by altering carbohydrate levels in the gills, muscles, and kidneys while decreasing glycogen storage in the liver. Pesticide exposure has been linked to severe health impacts in humans, such as non-communicable diseases, reproductive issues, cognitive dysfunction, and cancer. The current review comprehensively emphasizes the harmful effects of pesticides on fish and human health, urging the establishment of environmental monitoring programs and biomonitoring studies. It accentuates the need for risk assessment models to evaluate pesticide impacts on marine ecosystems and advocates for stricter safety standards and lower pesticide residue limits in aquaculture products.

Keywords: aquaculture; carbamate; organophosphates; organochlorines; pyrethroid pesticides; fish health; nutrient density

Key Contribution: Aquaculture is a rapidly expanding field driven by a significant increase in global seafood demand. Fish and seafood are invaluable sources of essential nutrients, serving as critical sources of protein and polyunsaturated fatty acids. Pesticide contamination infiltrates marine ecosystems via various pathways, such as atmospheric drift, surface runoff, and subsurface leaching. Exposure to pesticides in the aquatic environment affects fish physiology and biology, impacts the nutritional composition of seafood, and

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consequently presents inevitable health risks for humans. The widespread utilization of pesticides underscores their beneficial impacts while at the same time warning against the potential negative implications arising from their improper use or disposal.

1. Introduction

Aquaculture, a technique that utilizes natural aquatic resources to breed and harvest fish and shellfish, has emerged as one of the most rapidly expanding sectors in food production as a result of the global increase in demand for seafood [1,2]. The exponential growth in the world population has driven fish farming towards an upsurge of continuous seafood production in nearly all regions worldwide to meet the demand for seafood as a provider of vital nutrients for the human body [3]. On a global level, the aquaculture sector accounts for almost half of the seafood produced annually. Specifically, fish contributes one-sixth of the world's protein consumption [4,5]. Fish and seafood greatly contribute to human health by providing us with protein, polyunsaturated fatty acids (PUFAs), and other vital nutrients [6,7].

Fish is considered the main source of nutrients for many nutritionally vulnerable individuals as it roughly contributes one-third of the total animal protein in 22 low-income and food-deficient countries [8]. Moreover, increasing seafood consumption to combat the rising incidence of non-communicable diseases has been one of the main public health initiatives in high-income countries [9]. Aquatic media, where fish are cultured, have shown the high levels of pesticides necessary for disease prevention and public safety. However, marine species are often exposed to an increased risk of contamination from natural and anthropogenic sources, which results in undesired adverse health outcomes in fish, consequently posing health threats to consumers [4]. The type of contaminants and the level of contamination depend on several factors, including the fish species, the geographical region, age, size, diet, and farming methods [9].

Pesticides permeate the aquatic environment through various routes, including drift, runoff, and leaching and build-up in the marine species, resulting in physiological and biological alterations that result in variation in enzyme activities and the disruption of hormonal levels within these species, making them vulnerable to disease by modifying their nutritional composition [10–12]. This review article examines the various classes of pesticides and categorizes their influence on fish composition and human health. It provides a comprehensive classification of ingested toxins in fish, their detrimental impact on seafood as a source of nutrition, and subsequent health risks for humans.

2. Pesticides

Pesticides are chemicals used to manage harmful pests that threaten cultivated plants, animals, or humans. This class of chemicals includes insecticides, herbicides, and fungicides, specifically formulated to control insects, weeds, and fungi, respectively [13]. Various pesticides can potentially eradicate many pests, while others are specifically formulated to target particular organisms and disrupt the normal physiological functions of their target organism, leading to physiological impairment and a decrease in the organism's viability [14].

Annually, the environment is exposed to many pesticides used in various industrial sectors, such as agriculture, forestry, aquaculture, food industry, processing, transportation, and the storage of biological materials [15,16]. Pesticides have many advantageous impacts on the ecosystem, agricultural output, and human well-being. Nonetheless, the improper application or disposal of pesticides may lead to adverse consequences for the

ecosystems and living organisms. Pesticide endurance is very high due to their tendency to bioaccumulate in living organisms, their long half-life, and their ability to dissolve in fats, capacities which render them resistant to decay and greatly contribute to their short-term and long-term endurance in the environment [14]. Aquatic ecosystems are particularly susceptible to pesticide exposure through multiple paths, including direct river discharge, runoff from agricultural areas, industrial effluents, sewage discharges, and spray and drift processes [17].

2.1. Classification of Pesticides

Pesticides can be categorized based on various factors, including the mechanism of action, organism, chemical composition, and entry route [13]. Insecticides, herbicides, fungicides, rodenticides, and bactericides are all examples of organism-based categorization. In addition, the route of entry-based categorization of pesticides includes the stomach, contact, fumigants, and systemic poisons. While chemical-based classification is considered the most common and helpful categorization method, it is classified into four major groups: carbamate, organophosphate (OP), organochlorine (OCPs), and pyrethroid. It is a complex system [13,18]. Synthetic pesticides can be divided into inorganic and organic. Conversely, biopesticides are usually derived from living organisms like bacteria, fungi, and plants and can be further classified into biochemical pesticides, microbial pesticides, and plant-incorporated protectants [13].

2.2. Organochlorines

Organochlorines (OCPs) are organic compounds synthesized from chlorinated hydrocarbons and are used across various sectors, including in the chemical industry and agricultural practices. They are known to be lipophilic, highly toxic, slow to degrade, and strongly associated with bioaccumulation. Therefore, they persist in the ecosystem for a long time and are classified as persistent organic pollutants (POPs) [19]. Even though developed countries have banned the use of many products containing OCPs, several developing countries still utilize these compounds due to their low cost and simple management during production [20]. Several pesticides have been successfully used to control diseases like malaria and typhus, including dichlorodiphenyltrichloroethane (DDT), dieldrin, dichlorodiphenyldichloroethylene (DDE), chlordane, aldrin, toxaphene, endosulfan, and methoxychlor; however, they are considered to be highly toxic substances, [14,21,22]. Large ecological distributions and high residue levels of OCPs were detected in various environmental matrices. For instance, high levels of OCPs have been found in surface water and groundwater in China, ranging from 12.3 to 77.5 ng/L and 21.0–61.4 ng/L, respectively, with hexachlorocyclohexane (HCHs) and heptachlor being the most predominant [23]. A study examining the level of OCPs in freshwater snails in Argentina found that concentrations ranged from 2.59 to 6.46 ng/g of dry weight [24]. Another study examined the level of OCPs in Pakistan, in both soil and air, and found the level range from 0.68 to 13.47 ng/g in soil and from 375.1 to 1975 pg/m^3 in air, respectively, with β-HCH and p,p′-DDE being the most common metabolites [25]. Moreover, biomonitoring studies in humans also emphasized the high level of OCPs. For instance, a study in Brazil demonstrated that p,p′-DDE, lindane, and heptachlor have a high level in humans [26]. Another study emphasized high p,p′-DDE, and hexachlorobenzene (HCB) [27] detection frequencies. Similarly, another study found that p,p′-DDE, p,p′-DDT, and dieldrin were highly abundant in Tanzania [28]. While predominantly known as endocrine and neuroendocrine disruptors, hindering hormone-signaling pathways in vertebrates and invertebrates, they also interfere with metabolism, mitochondrial oxidative respiration, and immune function (Figure 1) [29].

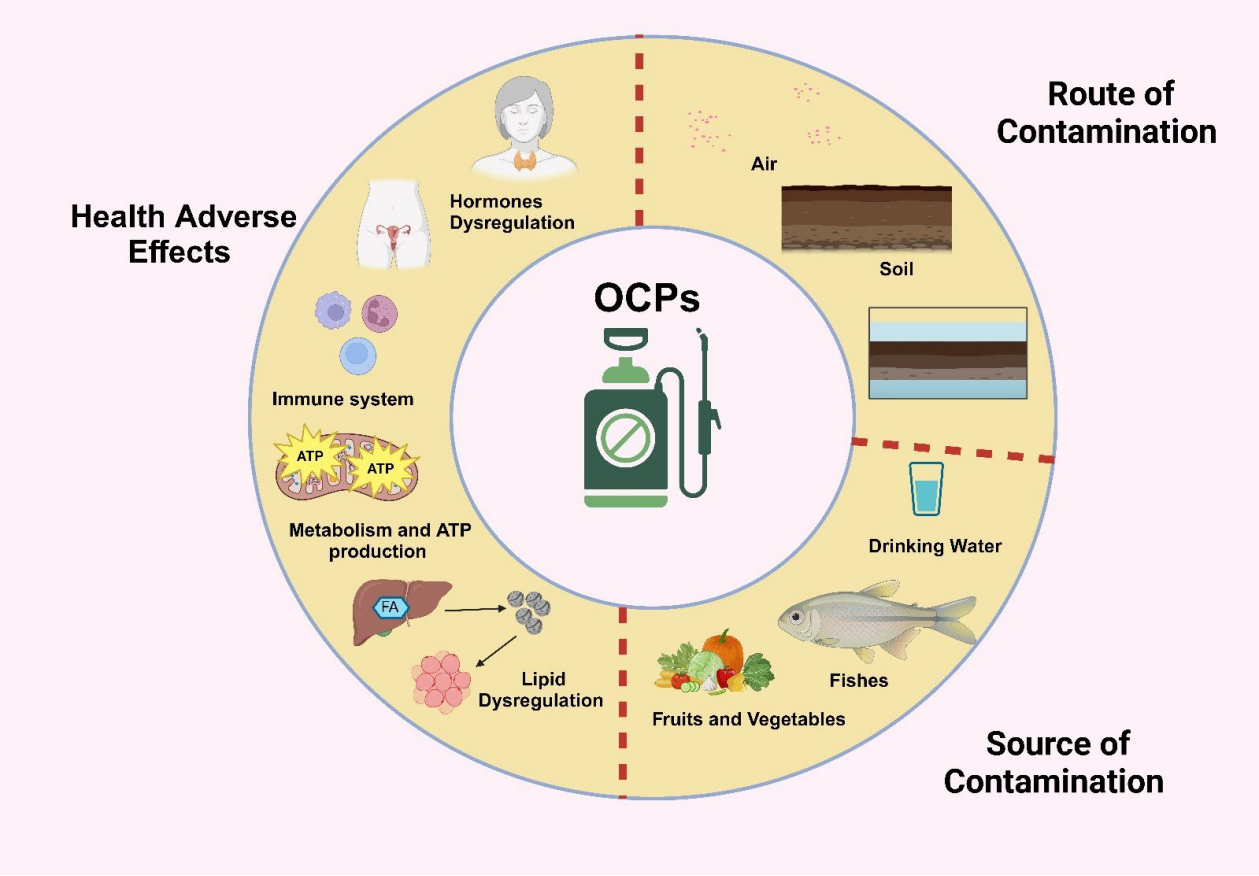

Figure 1. A summary of the influence of OCPs on fish and human health, the routes of contamination, and the primary sources of human exposure. OCPs can adversely affect fish and human health by mediating severe health consequences in fish, such as hormonal dysregulation, immune system impairment, metabolic disruptions affecting ATP production, and lipid dysregulation, which can contribute to liver dysfunction and other health complications that subsequently affect human health by bioaccumulation and biomagnification. It also illustrates the major routes of OCP contamination, including air, soil, and the runoff contamination of drinking water. Furthermore, it highlights the primary sources of human exposure, such as contaminated fish, fruits, and vegetables, as well as runoff, that can contaminate drinking water. Furthermore, it highlights the main sources of human exposure, such as the ingestion of contaminated fish, fruits, and vegetables. Figure 1 was created with BioRender.com (accessed on 6 July 2024).

2.3. Organophosphates

Organophosphates (OPs) are derived from phosphoric acid and are well known for having multiple functions that enable them to manage a broad spectrum of pests. A plethora of evidence shows that there are high levels of OPs in the environment. Higher OP residues have been found in water and sediments across several countries. For example, residues of OPs such as dimethoate, chlorpyrifos, and malathion were detected in Nigeria's soil, water, sediments, and banana crops, frequently exceeding the permissible limits [30]. In Indonesia, methidathion, malathion, chlorpyrifos, and parathion were found in high levels in surface water and Bilih fish [31]. Similarly, elevated detection residue rates were detected in agricultural soils in China [32]. Biomonitoring studies in humans have highlighted significant residue levels. A study in Japan found that children in diapers exhibited noticeable levels of six dialkyl phosphates, linked to exposure to household insecticides [32]. Interestingly, about 98% of schoolchildren tested for OPs levels in Thailand had at least one detectable OPs metabolite in their urine, with chlorpyrifos being the most common [33]. They function by exhibiting neurotoxic effects, promoting alterations in ion channels, and

inducing energy metabolism [34]. Nerve poisoning can occur through different entry modes, including stomach poisoning, contact poisoning, or fumigant poisoning. These pesticides are thought to be biodegradable and lead to minimal environmental pollution. OPs include chlorpyriphos, parathion, malathion Folidol 600® (methyl parathion), and diazinon. The effect of OPs is apparent through acetylcholinesterase (AChE) inhibition in fish, activating the active derivatives (oxons) and forming an enzyme–inhibitor complex, thus damaging nerve impulse transmission and negatively affecting the nervous system (Figure 2). The inhibition of AChE causes excessive stimulation of both nicotinic and muscarinic receptors. Moreover, their adverse effects are mainly linked with metabolic dysregulation, which is evident in alterations in oxygen consumption and ammonia excretion [35].

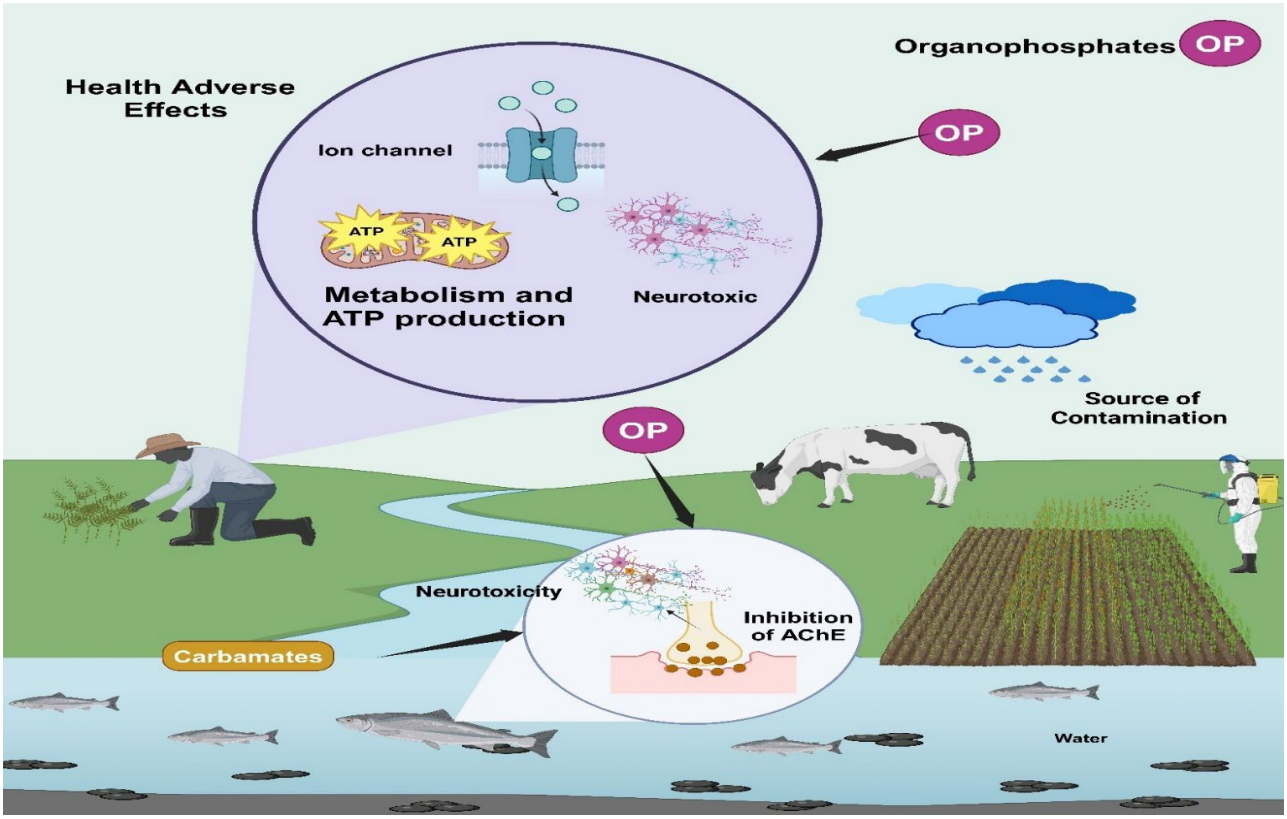

Figure 2. Depicting the major source of contamination and the negative impact of OPs and carbamate on fish health. The major pathways of OP contamination include agricultural practices, rainfall and runoff, and livestock exposure, resulting in bioaccumulation in fish and other species. Furthermore, it illustrates the adverse health consequences, including neurotoxicity through ion channel disruption and AChE inhibition, as well as mitochondrial dysfunction affecting ATP production, which can impact metabolism and overall health in both aquatic and terrestrial species. Figure 2 was created with BioRender.com (accessed on 6 July 2024).

2.4. Carbamates

Carbamates are carbamic acid derivatives that are similar to OPs, but they have different origins. This category of pesticide has a wide range of biocidal activities. Common members of this class are bendiocarb, carbofuran, carbaryl, and propoxur [18]. Several studies examined carbamate pesticides' environmental distribution and residue levels in different matrices. In Egypt, elevated levels of carbamate residues were detected in fish, sediment, and water samples [36]. Higher levels were also detected in Vietnam's commodities [37]. Biomonitoring studies in Thailand revealed higher levels of carbamate residues, exceeding health risk thresholds, in toddlers in agricultural regions [38].

These inhibit AChE and cause symptoms similar to those of OPs. Hence, they adversely affect the transmission of nerve signaling, resulting in death. Unlike OPs, carbamate pesticides are not inducers of neuropathy as they can reversibly inhibit the neuropathy target esterase [39] (Figure 2). These pesticides can exert their action via different entry routes. For instance, stomach poison infiltrates organisms, primarily through oral ingestion via the gastrointestinal tract, while contact poison enters the body through contact with the skin and mucous membranes. On the other hand, fumigant poison is mainly inhaled through the lungs and rapidly diffused into the bloodstream. They are quickly degraded in the environment, resulting in minimal environmental pollution [40,41].

2.5. Pyrethroids

Pyrethroids originate from the flowers of the pyrethrum. Synthetic pyrethroids were developed to enhance the specific chemical characteristics in those molecules and subsequently enhance the activity of the naturally occurring pyrethrum [39]. Pyrethroids elicit diverse ecological distributions and elevated residue levels in various environmental systems. Several studies reported higher residue levels of pyrethroids in soil and water samples in China [32,42–44]. In the United States, moderate levels were reported in dust samples [45]. Higher pyrethroid residues were also reported in food commodities in several countries, such as Benin, Vietnam, Ghana, and Spain, posing a higher risk to human health [46–49].

Pyrethroids are derived from keto-alcoholic esters of chrysanthemum and pyrethroid acids [14]. They induce the paralysis of an organism by affecting its sodium channels and causing the prolonged opening of the channel, which, at high concentrations, causes it to remain open for an extended period. High concentrations of pyrethroids lead to the full depolarization of the neural membrane and the subsequent loss of neuronal excitability (Figure 3). Repetitive excitatory activity will occur with low concentrations of pyrethroids. In general, pyrethroid intoxication is primarily caused by hyperexcitability. Alterations in 1% of sodium channels are sufficient to induce hyperexcitability [39]. Examples of pyrethroids include cypermethrin, permethrin, lambda-cyhalothrin, and deltamethrin.

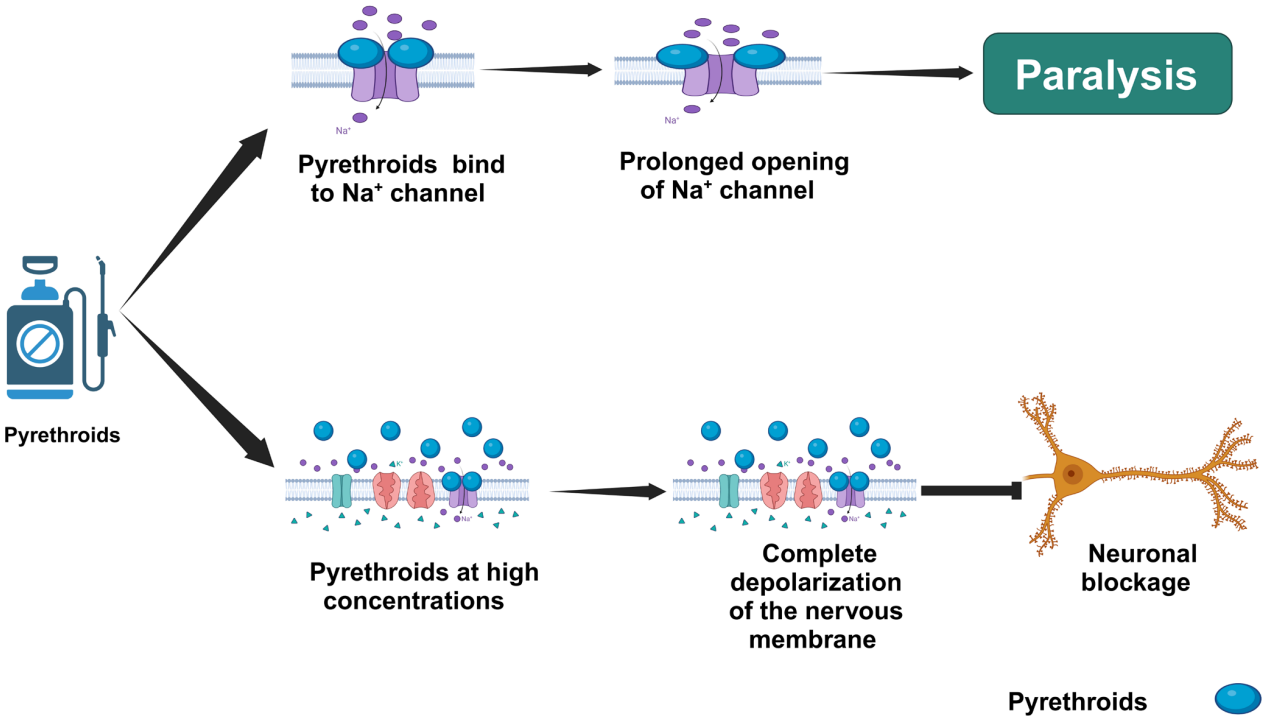

Figure 3. Summarizing the health impacts of pyrethroid. Figure 3 was created with BioRender.com (accessed on 6 July 2024).

3. Pesticides and Fish Health

Pesticides' presence in aquatic systems adversely affects the marine environment and human health [13]. Aquatic pollutants have direct contact with fish and other marine organisms within the water, making them particularly susceptible to contamination. A thin epithelial layer in fish gills further enhances susceptibility to aquatic pollutants, as gills represent more than fifty percent of their body's surface area [17]. Pesticides in the environment are inclined to bioaccumulation. As a result, there is an increase in pesticide concentrations in many aquatic species. Pesticide exposure alters fish quality, decreases fish growth, and increases mortality rates, especially in juvenile and small-sized fish [50]. The extent of pesticide influence corresponds to the size and age of the fish. Typically, smaller or younger fish are more susceptible to negative consequences associated with pesticide exposure. Hematological components, including red blood cells (RBCs), white blood cells, hemoglobin (HB), and others, are commonly employed as diagnostic markers to assess the health state of fish. Indeed, research in this area revealed a rise in the lymphocyte, leucocyte, and erythrocyte concentrations and hemoglobin levels in fish exposed to pesticides. Conversely, other studies reported a decline in HB, total RBCs, and packed cell volume, alongside an elevation in total leukocyte count [51,52]. Moreover, pesticide exposure inhibits fish's immune system, induces oxidative stress, and enhances the likelihood of acquiring a disease [51].

Various aquatic species exposed to pesticides experience organ disturbances. For instance, fish exposed to certain pesticides show histological pathological changes such as the congestion of afferent arterioles, the distortion of the cartilaginous support, the shedding of the epithelium of the intestinal villi, and the degeneration of hepatic and pancreatic cells, accompanied by the invasion of lymphatic cells [53–55]. Furthermore, a plethora of evidence emphasized the harmful effects of pesticides on AChE in multiple organs, including the brain, liver, and muscles, glucose metabolism, liver function, and inflammation, as delineated in Tables 1–3. Furthermore, pesticides induce oxidative stress by generating reactive oxygen species (ROS), culminating in lipid peroxidation, the diminished integrity of cellular membranes, and escalated risk of oxidative damage to DNA and proteins [56–58]. Consequent alterations in the metabolic systems of fish compromise nutrient density and have potential repercussions for the aquaculture industry [12]. The type of pesticide, the toxic dose, the exposure period, and the species influence the extent and nature of the impact on aquatic organisms.

3.1. Insecticides and Its Effect on Fish Species

Insecticides are a category of pesticides specifically designed to target and control insects. Insecticides have significantly transformed agriculture and human well-being by effectively managing insects that harm crops and transmit diseases to humans, such as Dengue fever, the Zika virus, and malaria, increasing food productivity. Over 80,000 insecticides are currently available for commercial use in agricultural and industrial practices worldwide, representing the highest percentage of all pesticides utilized worldwide [59]. The most commonly used insecticides for indoor and agricultural uses are carbamates, pyrethroid, and OPs [39]. The association between insecticide exposure and harmful effects on various fish species is well-established in the literature, including histopathological changes, neurological changes, endocrine and metabolism changes, behavior changes, immune changes, reproduction, and oxidative stress, as depicted in Table 1. Interestingly, insecticides impose a deleterious effect on fish's physiological status through various mechanisms, primarily neurotoxicity, oxidative stress, metabolic disruption, and histopathological damage, which consequently mediate several molecular effects, including AChE inhibition, oxidative damage, liver dysfunction, and hematological changes, leading to

changes in physiological and behavioral status. Furthermore, species-specific sensitivity and dose-dependent toxicity warrant further investigation to establish safe exposure levels for aquatic environments. In the following subsection, we will dissect each molecular effect in detail.

Table 1. Impact of insecticides on fish health.

Fish Species	Insecticide	Toxic Dose	Molecular Effect on Fish	Reference
Oncorhynchus mykiss	endosulfan	19.78 µg	↑lamellar edema in fish gills, ↑lamellar–epithelial separation, ↑lamellar fusion, ↑swelling and necrosis, ↑melanomacrophage centers	[60]
Clarias batrachus	phorate	0.27 ppm (1/3 LC50)	↑serum glucose, ↑ALP, and ↑ bilirubin levels	[61]
	carbaryl	15.3 ppm (1/3 LC30)	↑serum glucose, ↑ALP, and ↑bilirubin levels	[61]
Channa punctatus	chlorpyrifos	0.365 ppm	↑ALP, ↑acid phosphatase, and ↑cholesterol, and ↓glycogen in the liver	[62]
Rhamdia quelen	trichlorfon	11 mg trichlorfon/L water	↑cerebral ROS and ↑lipid peroxidation	[63]
Oreochromis niloticus	chlorpyrifos	42.0 µg/L (20% EC)	↓hepatic glycogen, ↓ALP, ↓AChE, ↓catalase activity in the liver, and ↑plasma glucose	[64]
	Folidol 600®	17.82 mg/L	↓ ChE, ↓AChE, ↓BChE, ↓oxygen consumption, and ↑ammonium excretion.	[35]
Brycon cephalus		2 ppm (1/3 of 96 h-LC50)	↓plasma and brain AChE activity, ↓liver ALT, ↑plasma ALT, and ↓hepatic glycogen and glucose	[65]
Mystus cavasius		5.9 ppm	↓oxygen consumption	[66]
Anguilla anguilla	dichlorvos	1.5 mg/L (the 96 h LC85)	↑GSH, ↑GSH/GSSG ratio, ↑hepatic glutathione reductase, ↑GST, ↑glutamate: cysteine ligase, ↑γ-glutamyl transferase activities, ↓GSH loss and oxidation, and ↑AChE and caspase-3 inactivation	[67]
Cyprinus carpio	dichlorvos	1 ppm	↓AChE and ↑oxidative stress	[68]

Table 1. *Cont.*

Fish Species	Insecticide	Toxic Dose	Molecular Effect on Fish	Reference
Ictalurus nebulosus	dichlorvos	1 ppm	↓AChE and ↑oxidative stress	[68]
	chlorpyrifos	I_{50} 28–33 nM	↓AChE	[69]
	parathion	I_{50} 446–578 nM	↓AChE	[69]
Anguilla anguilla	diazinon	0.042 mg/L (0.50 of the 96-h LC50)	↓ChE activity in the brain, plasma, and eye	[70]
	fenitrothion	Sublethal 0.02	↓brain AChE activity, ↓Na^+, K^+-ATPase activity in gill tissue, ↑blood glucose, and ↑blood lactate values	[71]
Tilapia guineensis	chlorpyrifos	0.002 mg/L	↓leucocyte and erythrocyte number	[72]
Oreochromis niloticus	diazinon	7.830 ppm	↓intracellular calcium flux, ↓ERK1/2, ↓pERK1/2, ↓apoptosis, ↓senescence, and ↓mitochondrial membrane potential in spleen mononuclear cells	[73]
			↑ROS production, ↓phagocytic active cells, ↓splenocyte proliferation, ↓phagocytic indices	[74]
			↓AChE activity, ↑ACh concentration	[75]
	atrazine	428 µg/L	↑AChE, ↓nicotinic AChR, ↓muscarinic AChR, and ↓AChE activity immune cells	[76]
			↑IL-1β, ↑IL-1RI expression in spleen and head kidney	[77]
Cyprinus carpio	chlorpyrifos	116 µg/L	↑IL-1β and ↑IL-1R1 expression significantly after exposure in the spleen and head kidney	[2]
	artazine	428 µg/L	↓GSTs	[78]
	chlorpyrifos	116 µg/L	↓GSTs	[78]
Channa striata	diazinon	500 g/ha	Long-term brain ChE inhibition	[79]
	diazinon	0.35 mg/L	Long-term inhibition of brain ChE activity and growth inhibition	[79]

Table 1. *Cont.*

Fish Species	Insecticide	Toxic Dose	Molecular Effect on Fish	Reference
Oncorhynchus mykiss	AzMe	0.007 (0.004–0.009) (LC_{50})	↓brain and muscular ChE	[80]
	carbamate carbaryl	5.40 (4.27–6.18) (LC_{50})	↓brain and muscular ChE	[80]
B. arenarum Larvae	AzMe	10.44 (LC_{50})	↓ChE	[80]
	carbamate carbaryl	24.64 (17.68–34.77) (LC_{50})	↓ChE	[80]
Oncorhynchus mykiss	AzMe	0.007 mg/L	↓brain ChE	[81]
	azinphos carbaryl		↓brain ChE	[81]
	methiocarb	5.43 ± 0.19 mg/L	↑lamellar edema, ↑lamellar–epithelial separation, ↑lamellar fusion, and ↑necrosis	[82]
Labeo rohita	profenofos	0.06 mg/L	↓the AChE in the brain, gills, muscle, blood, liver, and kidney, and ↓BuChE in the liver and blood	[83]
	carbofuran	0.28 mg/L	↓the AChE in the brain, gills, muscle, blood, liver, and kidney, and ↓BuChE in the liver and blood	[83]
Channa punctatus	sevin	Sublethal concentration 1.05 mg/L	↓glycogen in the liver and muscles, ↑lactic acid in the liver and muscles, ↑ LDH activity in the liver, muscles, brain, and gills, ↓LDH in the kidney and intestine, ↓PDH activity in the liver, muscles, brain, gills, kidney, and intestines, ↓succinate dehydrogenase in muscle, and ↑succinate dehydrogenase in kidney and intestine	[84]
Colossoma macropomum	dichlorvos	0.04 µmol/L	↓brain AChE	[85]
	chlorpyrifos	7.6 µmol/L	↓brain AChE	[85]
Colossoma macropomum	TEPP	3.7 µmol/L	↓brain AChE	[85]
	carbaryl	33.8 µmol/L	↓brain AChE	[85]
Carassius carassius L.	endosulfan	0.070 ppm	↑lipid peroxidation and ↓ GSH	[86]
Colossoma macropomum	carbofuran	0.92 µmol/L	↓brain AChE	[85]

Table 1. *Cont.*

Fish Species	Insecticide	Toxic Dose	Molecular Effect on Fish	Reference
Puntius conchonius	carbaryl	2.142 ppm	↑hyperglycemia and ↑glycogenesis in the liver, brain, and heart, and ↑hypercholesterolemia, Long-term exposure: ↑hypoglycemia, ↓liver glycogen, ↑glycogenesis in the heart, ↓glycogen levels in the brain, and ↓blood and liver cholesterol	[87]
Puntius conchonius	dimethoate	4.784 ppm	↑hyperglycemia and ↑glycogenesis in the liver, brain, and heart, and ↑hypercholesterolemia. Long-term exposure: ↑hypoglycemia, ↓liver glycogen, ↑glycogenesis in the heart, ↓glycogen levels in the brain, and ↓blood and liver cholesterol	[87]
Clarias batrachus	carbofuran	23 mg/L	Reduction in blood and liver cholesterol ↓total ATPase in the tissue of kidney, muscle, liver, and gills	[88]
Oncorhynchus kisutch	carbofuran	10.4 µg/L (EC 50)	↓brain and olfactory rosette AChE activity	[89]
Clarias batrachus	endosulfan	Sublethal concentration 0.06 mg/L	↓activity of citrate synthase, ↓G6PDH in the brain, liver, and skeletal muscle, ↓activity of lactase dehydrogenase in the brain, and ↓RNA and ↓protein content of the brain, liver, and skeletal muscle tissue	[90]
Clarias gariepinus	carbaryl	0.003 µM (IC 50)	↓brain AChE	[91]
	chlorfenvinphos	0.03 µM (IC 50)	↓the AChE in plasma and eye homogenate	[91]
	diazinon	0.15 µM (IC 50)	↓brain AChE	[91]
	dimethoate	190 µM (IC 50)	↓brain AChE	[91]
	fenitrothion	0.02 µM (IC 50)	↓brain AChE	[91]
	pirimiphosmethyl	0.003 µM (IC 50)	↓brain AChE	[91]
	profenofos	0.003 µM (IC 50)	↓brain AChE	[91]

Table 1. *Cont.*

Fish Species	Insecticide	Toxic Dose	Molecular Effect on Fish	Reference
Salmo salar L.	carbofuran	1.0 µg/L (Not LC50)	↓priming pheromonal system in mature males, and ↓the ability of the olfactory system to detect PGF2α	[92]
Cirrhinus mrigala	Cartap hydrochloride	0.339–0.436 mg/L	↓glycogen, ↓total protein, and ↓ nucleic acids in gill, liver, brain, kidney and muscle tissues	[93]
Chironomus riparius	carbofuran	27.2 µg/L	↓AChE activity	[94]
	pirimiphos-methyl	63.8 µg/L	↓AChE activity	[94]
	permethrin	16.6 µg/L	↓AChE activity	[94]
Rana perezi	ZZ-aphox®	0.02% and 0.14%	↑histological damage to gills, liver, gallbladder, heart and notochord	[55]
Monopterus albus	endosulfan	0.42 µg/L	Abnormal behavioral response, ↓erythrocyte and leukocyte count, ↑size of erythrocyte cell, ↓HB, and ↓hematocrit	[95]
Cichlasoma dimerus	endosulfan	2.6 µg/L	↓erythrocyte volume, ↓corpuscular hemoglobin, ↑hyperplasia if there is ↑ interlamellar epithelium, ↑blood congestion in secondary lamellae, ↑mucous cells hyperplasia, and ↑hypertrophy in gills, ↑pyknotic nuclei, ↑hydropic degeneration in the liver, and ↑testicular damage	[96]
Channa punctatus	endosulfan	5 ppb	↑glutathione peroxidase, ↑glutathione S-transferase activity and ↑GSH levels in all organs, ↓catalase activity, and ↑lipid peroxidation	[97]

Table 1. *Cont.*

Fish Species	Insecticide	Toxic Dose	Molecular Effect on Fish	Reference
Oncorhynchus mykiss	endosulfan	0.6 and 1.3 micro g/L	↑histological lesions in the gill, liver, spleen and trunk, kidney	[54]
Prochilodus lineatus	endosulfan	2.4 µg/L	↓HB, ↓mean cell hemoglobin, ↓total plasma protein, ↑white blood cell count, ↑plasma glucose, ↑lipid peroxidation in the intestine, liver, and brain	[98]
Lepomis macrochirus	endosulfan	1.2 µg/L (exposure dose)	↑damage to connective tissue and ↑seminiferous tubules in the testis	[99]
	endosulfan	1.2 µg/L	↓AChE activity	[100]
Chanos chanos	endosulfan	21.5 g/L	At a moderate dose: ↑the activity of catalase, ↑SOD, and ↑GST in the brain, gill, and liver; brain AChE, ↑LDH, and ↑MDH activity in the brain, liver, and gill; ↑activity of ↑ALT, ↑AST, and ↑G6PDH in the liver and gill; curling of secondary lamellae, ↑thickening of primary epithelium, ↑shorting of secondary lamellae, ↑epithelial hyperplasia, ↑a fusion of secondary lamellae, ↑aneurysm, ↑collapsed secondary lamellae in gills, ↑cloudy swelling, ↑necrosis with pyknotic nuclei in the liver,	[101]
Jordanella floridae	endosulfan	10.8 µg/L (LOEC)	At a high dose: ↑necrosis of hepatic cells in the liver ↑hyperactivity, ↑convulsions, ↑axis malformations, ↑adverse effects on growth, reproduction, and survivability	[102]

Table 1. *Cont.*

Fish Species	Insecticide	Toxic Dose	Molecular Effect on Fish	Reference
Oncorhynchus mykiss	maneb	1.19 mg/L	↑lamellar edema, ↑lamellar–epithelial separation, ↑lamellar fusion, ↑swelling, and ↑necrosis of epithelial cells, ↑focal lamellar hyperplasia in gills, ↑inflammation, ↑focal necrosis in the liver, trunk kidney, and spleen	[53]
	carbaryl	2.52 mg/L	↑lamellar edema, ↑lamellar–epithelial separation, ↑lamellar fusion, ↑swelling, and ↑necrosis of epithelial cells, ↑focal lamellar hyperplasia in gills, ↑inflammation, ↑focal necrosis in the liver, trunk kidney, and spleen	[53]

3.1.1. Effect of Insecticide Exposure on Histopathological Abnormalities

Histopathological changes are common in various species exposed to insecticides. Notably, the exposure of *Oncorhynchus mykiss* to endosulfan at a dose of 19.78 µg/L induces lamellar edema, fusion, and separation from the epithelium, epithelial cell swelling, the formation of melanomacrophage centers on various organs, and severe liver focal necrosis [60]. Similarly, administering endosulfan at a dose of 2.6 µg/L induces histological lesions, typified by interlamellar epithelium and mucous cell hyperplasia, secondary lamellae congestion, hypertrophy in gills, and pyknotic nuclei and hydropic degeneration in the liver in *Cichlasoma dimers* [96]. In another study, this one on the species *Chanos chanos*, an endosulfan dose of 21.5 g/L promoted several histopathological lesions, including the curling, shortening, fusion, and collapse of the secondary lamellae, inducing epithelium thickening, epithelial hyperplasia, and aneurysms in gills [101]. In the liver, the histological lesions consisted of cloudy swelling and necrosis, with pyknotic nuclei emerging at moderate doses and severe hepatic cell necrosis seen at high doses [101]. In addition, the administration of methiocarb at a dose of 5.43 ± 0.19 mg/L induced lamellar edema, fusion, and separation from the epithelium, epithelial cell swelling, the formation of melanomacrophage centers on various organs, and severe liver focal necrosis, as well as necrosis, between a molecular and granular layer of the cerebellum where Purkinje cells existed [82]. Similarly, the administration of maneb at a dose of 1.19 mg/L and carbaryl at a dose of 2.52 mg/L produced comparable histopathological conditions in addition to inflammation, focal necrosis, and focal lamellar hyperplasia [53]. The administration of endosulfan at doses of 0.6 and 1.3 µg/L caused more predominant histological lesions in various organs, including the gill, liver, spleen, and trunk kidney [54]. The exposure of *Chironomus riparius* to carbofuran at a dose of 27.2 µg/L, to pirimiphos-methyl at a dose of 63.8 µg/L, and to permethrin at a dose of 16.6 µg/L resulted in histological damage to the gills, liver, gallbladder, heart, and notochord. In addition, the same effect was observed in *Rana perezi* after exposure to 0.02%and 0.14% of ZZ-Aphox® [55].

The impact of insecticides on fish varies by species, insecticide class, dose, and exposure duration. Insecticide-mediated histological damage is typical in gills, liver, kidney, and muscle tissues (Table 1). Severe histological abnormalities are common in species with high metabolic rates that lead to higher insecticide uptake, such as *Oncorhynchus mykiss* and *Channa punctatus* [53,60,84]. Moreover, the duration and the concentration of insecticides can significantly affect the limit of histological damage. For instance, acute exposure to a high dose of endosulfan at 19.78 µg/L induces rapid gill necrosis and cellular damage [60]. Chronic exposure to a low dose of diazinon at 0.042 mg/L induces gradual metabolic tissue damage in liver and muscle tissue [70].

3.1.2. Effect of Insecticide Exposure on Neurobehavioral Abnormalities

Several fish species exhibit neurological changes following exposure to insecticides. For instance, exposure to azinphos methyl (AzMe) at a toxic dosage of 0.007 mg/L (0.004–0.009) (LC_{50}), or to carbamate carbaryl at a dose of 5.40 (4.27–6.18) (LC_{50}), causes neurological changes related to choline esterase (ChE) inhibition in the brain and muscle [80,81]. Furthermore, exposure to endosulfan at a dose of 1.2 µg/L in *Lepomis macrochirus* (Bluegill sunfish) decreased AChE activity [99,100]. In another study of *Chanos chanos,* endosulfan suppressed AChE activity in the brain [101]. Several neurological conditions were also observed when *Jordanella floridae* (American flagfish) was exposed to an endosulfan dose of 10.8 µg/L (LOEC), comprising hyperactivity, convulsions, and axis malformations [102]. Numerous insecticides were associated with the inhibition of AChE in the brain of *Clarias batrachus*, such as carbaryl (0.003 µM), diazinon (0.15 µM), dimethoate, (190 µM), fenitrothion (0.02 µM), pirimiphosmethyl (0.003 µM), and profenofos (0.003 µM) [91]. Likewise, the inhibition of brain AChE was also observed in *Colossoma macropomum* after exposure to different doses of dichlorvos (0.04 µmol/L), chlorpyrifos (7.6 µmol/L), tetraethyl pyrophosphate (TEPP) (3.7 µmol/L), carbaryl (33.8 µmol/L), and carbofuran (0.92 µmol/L) [85]. Furthermore, another study reported the inhibition of AChE in brain, plasma, and eye homogenate upon the exposure of *Clarias gariepinus* to the following pesticide doses: carbaryl (0.003 µM), diazinon (0.15 µM), dimethoate (190 µM), fenitrothion (0.02 µM), pirimiphosmethyl (0.003 µM); profenofos (0.003 µM), and chlorfenvinphos (0.03 µM) [91]. Folidol 600® administration at a 17.82 mg/L dose inhibited plasma ChE, AChE, and butyrylcholinesterase (BChE). Similarly, diazinon, at a concentration of 7.83 ppm, inhibited the activity of AChE, leading to an increase in acetylcholine (ACh) concentration and a decrease in nicotinic acetylcholine receptor (AChR) and muscarinic AChR concentrations in fish immune cells [73–75,103]. Two independent studies examined the effect of Folidol 600® at a dose of 2 ppm (1/3 of 96 h-LC50) on *Brycon cephalus*. They reported reduced AChE activity in the brain and plasma [65,76]. Also, Folidol 600® (17.82 mg/L) exposure to *Oreochromis niloticus* inhibits the levels of plasma ChE, AChE, and BChE [35]. Three different doses of diazinon in *Channa striata*, 0.016, 0.079 mg/L, and 0.35 mg/L, caused long-term inhibition of brain ChE activity [79]. The inhibition of ChE was observed in larvae of *B. arenarum* when exposed to 10.44 (LC_{50}) of AzMe or 24.64 (17.68–34.77) (LC_{50}) of carbamate carbaryl [80]. In *B. arenarum Labeo rohita*, similar effects of AChE inhibition in the brain, gills, muscle, blood, liver, and kidney, as well as inhibition of BuChE in liver and blood, were induced by profenofos (0.06 mg/L) and carbofuran (0.28 mg/L) [83]. *Oncorhynchus kisutch* was exposed to 10.4 µg/L (EC 50) of carbofuran, causing inhibition of brain and olfactory rosette AChE activity, and a dose of 2.05 mg/L (EC 50) of mancozeb, causing increased activity of AChE in the brain [89]. Diazinon administration at a dose of 0.042 mg/L (0.50 of the 96-h LC50) inhibits brain, plasma, and whole-eye ChE activity [70]. Likewise, a sublethal dose of 0.02 ppm of fenitrothion inhibits AChE activity in the brain [71]. In addition, the administration of dichlorvos at a dose of 1 ppm in both the *Ictalurus nebulosus* and *Cyprinus*

carpio species inhibits AChE [68]. A previous study reported the inhibition of AChE in the *Ictalurus punctatus* when administering I_{50} 28–33 nM of chlorpyrifos or I_{50} 446–578 nM of parathion [69]. Majumder and Kaviraj's study on *Oreochromis niloticus* showed that the administration of chlorpyrifos at 42.0 µg/L (20% EC) reduced AChE activity in the liver [64]. Only one study discussed the abnormal behavioral responses when *Monopterus albus* (Asian swamp eel) was exposed to a low dose of endosulfan (0.42 µg/L) [95]. Most insecticides induce neurobehavioral changes in fish via the modulation of cholinergic neurotransmission by inhibiting AChE, leading to neuromuscular blockade, convulsions, erratic swimming, and social behaviors (Table 1). Chlorpyrifos, dichlorvos, and diazinon are potent AChE inhibitors. Endosulfan causes neurobehavioral issues and a loss of reflex control. Likewise, carbaryl reduces social behaviors and responses to stimuli. Moreover, chronic exposure to insecticides at low doses can induce long-term cognitive dysfunction and learning impairment. Acute exposure to high doses results in neurotoxic effects such as neuromuscular dysfunction, convulsions, and paralysis.

3.1.3. Effect of Insecticide Exposure on Hepatic Dysfunctions, Metabolism Disorders, and Oxidative Stress Responses

Moving beyond neurological abnormalities, this part of the review will discuss the association between insecticide exposure and perturbations in liver function, metabolism, and systemic oxidative stress in different aquatic species. For instance, *Clarias batrachus*, after being exposed to carbaryl at 15.3 ppm (1/3 LC30) and phorate at 0.27 ppm (1/3 LC50), exhibited an upsurge in the serum glucose, alkaline phosphatase (ALP), and bilirubin levels, signifying hepatic distress [61]. Moreover, the administration of carbofuran at a dose of 23 mg/L was implicated in decreased total ATPase in the tissue of the kidney, muscle, liver, and gills, emphasizing the broad spectrum of physiological imbalances induced by insecticide exposure [88]. Endosulfan is one of the most studied insecticides, showing a wide range of pathological conditions in different organs of various species. A sublethal dose of endosulfan (0.06 mg/L) reduces citrate synthase and G6PDH activity in the brain, liver, and skeletal muscle. In *Carassius carassius* L., an endosulfan dose of 0.070 ppm caused the induction of lipid peroxidation and a reduction in glutathione (GSH) [86,90]. In another study on the species *Chanos chanos*, an endosulfan dose of 21.5 g/L promoted the activity of crucial enzymes, namely catalase, superoxide dismutase (SOD), glutathione S-transferase isoenzymes (GST), lactate dehydrogenase (LDH), and malate dehydrogenase (MDH) in the brain, gill, and liver. It also exhibits increased alanine aminotransferase (ALT), aspartate aminotransferase (AST), and glucose-6-phosphate dehydrogenase (G6PDH) activity, but only in the liver and gill [101]. Additionally, an endosulfan dose of 2.4 µg/L increases lipid peroxidation in the intestine, liver, and brain in the species *Prochilodus lineatus* [98]. Moreover, an endosulfan dose of 5 ppb increases glutathione peroxidase, GST, and GSH levels in all organs while decreasing catalase activity and increasing lipid peroxidation values in all organs [97].

Hepatic dysfunction is exemplified by increased ALP, acid phosphatase, and cholesterol activity, while there are decreased glycogen levels in the liver when *Channa punctatus* fish is exposed to a 0.366 ppm dose of chlorpyrifos [62]. In addition, a sublethal concentration of 1.05 mg/L of sevin in the same species caused an array of symptoms that differ from one organ to another. For instance, a decrease in glycogen and increased lactic acid levels were observed in the liver. Additionally, the activity of LDH increased, while the activity of pyruvate dehydrogenase (PDH) decreased [84]. In the muscle, glycogen and succinate dehydrogenase levels decreased while lactic acid levels increased. Also, LDH activity in the muscles increased, while pyruvate dehydrogenase (PDH) activity decreased [84]. In the kidneys and intestines, LDH was inhibited, and PDH activity decreased. Additionally,

succinate dehydrogenase levels increased [84]. In the brain and gills, the LDH activity increased, while PDH activity decreased [84].

The protein content also changes as *Channa punctatus* fish are exposed to malathion (0.1 mg/L), as shown by the increased free amino acid content in the muscles, kidney, heart, and stomach and by the decreased protein content in the muscle, gill, and liver [104]. Similarly, Bharti and Rasool reported that a dose of 0.4 mg/L, 1/20th of the 96 h LC50 value of malathion, increased the protein content while decreasing the serum glucose level in *Channa punctatus* [105]. In contrast, the liver showed decreased protein content and increased cholesterol when fish were exposed to 0.365 ppm of chlorpyrifos [62].

Studies showed that the exposure of the *Rhamdia quelen* species to trichlorfon (11 mg/L) induces an increase in cerebral levels of ROS and lipid peroxidation and a decrease in total PUFA content [63,106]. Furthermore, several studies were performed on the *Oreochromis niloticus* species. They revealed decreased liver hepatic glycogen, ALP, and catalase activity levels and elevated plasma glucose levels at 42.0 µg/L (20% EC) of chlorpyrifos [35,64]. Research on the eel species *Anguilla anguilla* has shown that the insecticide dichlorvos, at a concentration of 1.5 mg/L (the 96 h LC85), has multiple effects. These effects include increases in GSH content, GSH/GSSG ratio, hepatic glutathione reductase, GST, glutamate, cysteine ligase, and γ-glutamyl transferase activities with decreases in GSH loss and oxidation [67]. Several studies on *Oreochromis niloticus* showed that chlorpyrifos, 42.0 µg/L (20% EC), reduced levels of hepatic glycogen and reduced ALP and catalase activity in the liver [64]. A previous study found a series of effects on the *Puntius conchonius* species after exposure to carbaryl (2.142 ppm) or to dimethoate (4.784 ppm). These effects included perturbation in carbohydrate and cholesterol metabolism, hyperglycemia, and glycogenesis in the liver, brain, and heart; hypercholesterolemia, an increase in liver cholesterol, and long-term exposure led to hypoglycemia and the depletion of liver glycogen, inducing enhanced glycogenesis in the heart. Furthermore, it caused decreased glycogen levels in the brain and reduced blood and liver cholesterol [87].

The use of Cartap hydrochloride at 0.339–0.436 mg/L in *Cirrhinus mrigala* decreases glycogen, total protein, and nucleic acids in the gill, liver, brain, kidney, and muscle tissues [93,94]. The use of Folidol 600® at a dose of 2 ppm (1/3 of 96 h-LC50) on Brycon *cephalus* decreases liver enzyme ALT and lowers liver glycogen and glucose levels. Additionally, plasma ALT levels increased [65,76]. Likewise, a sublethal dose of 0.02 ppm of fenitrothion suppresses Na^+ and K^+-ATPase activity in gill tissue and elevates lactate and blood glucose levels in e thgill, liver, and blood [71].

Diazinon, at a concentration of 7.83 ppm, disrupts intracellular calcium flux, reduces the levels of extracellular signal-regulated kinase 1/2 (ERK1/2) and phospho extracellular signal-regulated kinase 1/2 (pERK1/2), induces apoptosis and senescence, and decreases the mitochondrial membrane potential in spleen mononuclear cells, which play a crucial role in intracytoplasmic signaling in immune cells. Furthermore, it increases the levels of ROS [73–75,103]. Formerly, Murty et al. illustrated that Folidol 600®, at a dose of 5.9 ppm, induces a decrease in oxygen consumption in the *Mystus cavasius* [66]. Conclusively, this body of evidence highlights the diverse impact of insecticide exposure on aquatic organisms. It reveals significant hepatic, metabolic, and oxidative stress-induced disruptions, indicating a need to reevaluate the widespread use of these chemical agents in the environment (Table 1). Furthermore, predatory fish such as *Cyprinus carpio* accumulate higher pesticide levels, increasing oxidative stress [68]. Acute exposure to high doses of insecticides disrupts liver enzymes and promotes oxidative stress. Meanwhile, chronic exposure to low doses of insecticides results in glycogen depletion and metabolic imbalances.

3.1.4. Effect of Insecticide Exposure on Hematological Abnormalities

Within various aquatic species, insecticide exposure has been documented to elicit significant hematological aberrations. Notably, a dose of 2.4 μg/L of endosulfan decreased hemoglobin, increased the white blood cell count, and increased the plasma glucose in *Prochilodus lineatus* [98]. Similarly, a dose of 2.6 μg/L of endosulfan caused a decrease in hematological parameters such as erythrocyte volume and hemoglobin in *Cichlasoma dimers* [96]. Hematological manifestations, denoted by decreased erythrocyte and leukocyte counts, an increase in the size of erythrocyte cells, and decreased HB and hematocrit values, were observed at a low dose of endosulfan (0.42 μg/L) in *Monopterus albus* [95]. In addition, the exposure of *Tilapia guineensis* to 0.002mg/L of chlorpyrifos resulted in a reduced number of leucocytes and erythrocytes [72]. Collectively, insecticides mediate hematological imbalances by affecting various parameters, including cell count, Hb levels, and hematocrit (Table 1). Furthermore, acute exposure to high-dose insecticides, including chlorpyrifos, rapidly reduces RBC counts and leukocytosis. Meanwhile, chronic exposure to lower doses of insecticides results in anemia and metabolic dysfunction [72].

3.1.5. Effect of Insecticide Exposure on the Immune Response

Limited data are available regarding insecticides' impact on the immune system. For example, at a concentration of 7.83 ppm, diazinon reduces the mitochondrial membrane potential in spleen mononuclear cells. These cells play a crucial role in intracellular signaling in immune cells, and the insecticide also reduces the number of phagocytic active cells and diminishes splenocyte proliferation and phagocytic activity [73–75,103]. Additionally, *Cyprinus carpio*, when exposed to a 428 μg/L dose of atrazine, showed the increased expression of interleukin-1β (IL-1β) and interleukin receptor I (IL-1RI) in the spleen and head kidney [77]. A similar effect was also observed when chlorpyrifos (116 μg/L) was exposed to the same species [77,78].

Insecticides substantially affect fish's immune responses by mediating immune suppression, inflammation, and oxidative stress-related immune dysfunction. Various molecular mechanisms mediate immune dysfunction, such as disparities in immune cell populations, altered cytokine expression, impaired phagocytic activity, and increased levels of oxidative stress markers (Table 1). The disruption of immune homeostasis can be species-specific, particularly for active predator species that rely on their immune resilience, making them more susceptible to pesticide-induced suppression. In contrast, bottom-dwelling species endure more tremendous immune stress due to constant exposure to insecticide-contaminated sediments.

3.1.6. Effect of Insecticide Exposure on Reproduction and Growth

The impact of insecticide exposure on the adverse health outcomes, related to reproduction and growth, of various aquatic species has been examined by several studies. Notably, the administration of endosulfan at a dose of 2.6 μg/L induces testicular damage in *Cichlasoma dimers* [96]. Likewise, exposure to endosulfan at a dose of 1.2 μg/L in *Lepomis macrochirus* results in substantial damage to seminiferous tubules in the testis [99,100]. The exposure of *Jordanella floridae* to an endosulfan dose of 10.8 μg/L (LOEC) exhibits various adverse effects on growth, reproduction, and survivability [102]. In addition, three different doses of diazinon in *Channa striata*, 0.016 and 0.079 mg/L or 0.35 mg/L, respectively, caused growth inhibition [79]. Insecticides affect fish reproduction and growth by disrupting endocrine regulation and gametogenesis, which subsequently results in reproductive failure, delayed development, and growth retardation. Key molecular mechanisms associated with insecticides include gonadal atrophy, which significantly reduces reproductive success

that arises from hormonal imbalances that disrupt sexual differentiation. In addition, delayed hatching and larval deformities lead to poor offspring viability and stunted growth (Table 1).

3.2. Herbicides and Their Effect on Fish Health

Herbicides are pesticides that target plants to kill unwanted weeds, shrubs, and trees and they are commonly used in agriculture [107]. In general, most herbicides do not exhibit acute toxicity to fish. However, certain herbicides are uncouplers of oxidative phosphorylation, while others disrupt cell division. Sublethal effects of herbicides may manifest in reproduction, stress, olfaction, and fish behavior [108]. Moreover, it substantially affects fish's health by interfering with several neurological, metabolic, oxidative stress, and hematological processes, eventually resulting in neurotoxicity, genotoxicity, and hepatotoxicity. Table 2 summarizes the impact of various herbicides on fish health, species-specific toxicological response, and toxic effects.

Table 2. Impact of herbicides on fish health.

Fish Species	Herbicide	Toxic Dose	Effect on Fish	Reference
Cnesterodon decemmaculatus	DIC and 2,4-dichlorophenoxyacetic acid (2,4-D)	410 mg/L	↑Oxidative damage, ↑catalase, ↑GST, and ↑AChE activity.	[109]
Anguilla anguilla	molinate	11.15 mg/L (one-third of the 96 h LC(50)	↓ChE activity, ↓blood proteins, ↓hematocrit, ↓HG, ↓erythrocytes, and ↓leukocytes	[110]
Leporinus obtusidens	clomazone	0.5 mg/L	↓AChE brain activity	[111]
	quinclorac	0.375 mg/L	↓AChE brain activity and ↑AChE tissue activity	[111]
	propanil	3.6 mg/L	↑AChE tissue activity	[111]
	metsulfuron methyl	0.002 mg/L	↑AChE tissue activity	[111]
Astyanax sp.	diuron	30 mg/L	↑GST activity and ↓catalase activity	[112]
	glyphosate	0.006 mL/L	↑membrane damage	[112]
Pimephales promelas	bromacil	185 mg/L	↓growth	[113]
	diuron	23.3 mg/L	abnormal or dead fry and ↓survival	[113]
Cnesterodon decemmaculatus	Panzer	15.68–16.70 mg/L	↑micronuclei frequency (genotoxic effect)	[114]
	Credit	91.73–98.50 mg/L	↑micronuclei frequency,	[114]
Leporinus obtusidens	clomazone	376 µg/L	↓AChE activity in the brain and muscles, ↓TBARS in brain, muscle, and liver tissues, and ↓catalase in the liver	[115]
	propanil	1644 µg/L	↓AChE activity in the brain and muscles, ↓ TBARS in brain, muscle, and liver tissues, and ↓catalase in the liver	[115]

Table 2. *Cont.*

Fish Species	Herbicide	Toxic Dose	Effect on Fish	Reference
Oreochromis niloticus	acetochlor	2.625 mg/L	Alteration in erythrocytes, ↓RBC count, ↑cholesterol, ↑total plasma protein, ↑urea, ↑creatinine, ↑albumin, ↑globulin, ↑albumin/globulin ratio, ↑ALP activity, ↑ALT activity, ↑and AST activity.	[116]
	bispyribac-sodium	0.800 mg/L	Alteration in erythrocytes, ↓RBC count, ↑cholesterol, ↑total plasma protein, ↑urea, ↑creatinine, ↑albumin, ↑globulin, ↑albumin/globulin ratio, ↑ALP activity, ↑ALT activity, ↑and AST activity.	[116]
	bentazon	36.00 mg/L	Alteration in erythrocytes, ↓RBC count, ↑cholesterol, ↑total plasma protein, ↑urea, ↑creatinine, ↑albumin, ↑globulin, ↑albumin/globulin ratio, ↑ALP activity, ↑ALT activity, ↑and AST activity.	[116]
	bensulfuron-methyl	2.50 mg/L	Alteration in erythrocytes, ↓RBC count, ↑cholesterol, ↑total plasma protein, ↑urea, ↑creatinine, ↑albumin, ↑globulin, ↑albumin/globulin ratio, ↑ALP activity, ↑ALT activity, ↑and AST activity.	[116]
	halosulfuron-methyl	1.275 mg/L	Alteration in erythrocytes, ↓RBC count, ↑cholesterol, ↑total plasma protein, ↑urea, ↑creatinine, ↑albumin, ↑globulin, ↑albumin/globulin ratio, ↑ALP activity, ↑ALT activity, and ↑AST activity.	[116]
	quinclorac	11.250 mg/L	Alteration in erythrocytes, ↓RBC count, ↑cholesterol, ↑total plasma protein, ↑urea, ↑creatinine, ↑albumin, ↑globulin, ↑albumin/globulin ratio, ↑ALP activity, ↑ALT activity, ↑and AST activity.	[116]
Leporinus obtusidens	glyphosate	3 mg/L	↓ AChE brain activity, ↑hepatic glycogen and glucose, ↓glycogen and glucose in the liver, and ↑ammonia in liver and muscle tissue.	[117]

3.2.1. Impact of Herbicide Exposure on Hepatic Dysfunctions, Metabolism Disorders, and Oxidative Stress Responses

The investigation into the consequences of insecticide exposure on hepatic dysfunctions, metabolism disorders, and oxidative stress responses has underscored significant implications for various aquatic species. Research focusing on the impacts of the herbicides dicamba (DIC) and 2,4-dichlorophenoxyacetic acid (2,4-D) at a concentration of 410 mg/L has demonstrated the induction of oxidative damage alongside an elevation in catalase and glutathione S-transferase (GST) activities in the species *Cnesterodon decemmaculatus* [109]. The species undergoing treatment displayed increased micronuclei frequency, a genotoxic effect, due to the exposure to different doses of glyphosate-based formulations (Panzer): 15.68–16.70 mg/L and 91.73–98.50 mg/L [114].

Further examination by Moraes et al. into the effects of different herbicides on *Leporinus outsiders* emphasized that a dose of 0.375 mg/L of clomazone exhibits more pronounced adverse effects, including a decrease in liver catalase [111,115]. Lower doses of propanil, at 1644 µg/L, also cause a decrease in liver catalase [115]. Another study reported the effect of 3 mg/L glyphosate (Roundup) on the same species and demonstrated increased hepatic glycogen and glucose, decreased lactate and protein in the liver, and increased ammonia levels in the liver and muscle tissue. [117]. A dose of 0.006 mL/L of glyphosate caused membrane damage in *Astyanax* sp., and a dose of 30 mg/L diuron (Hexaron) increased glutathione S-transferase activity and lowered the catalase activity [112]. Fathy et al. revealed that acetochlor (2.625 mg/L) caused an alteration in the shape of erythrocytes, a decrease in RBC count, and an increase in levels of cholesterol, albumin, globulin, albumin/globulin ratio, total plasma protein, urea, and creatinine, altering the activity of ALP, ALT, and AST [116]. Likewise, a study conducted by Fathy and colleagues emphasized that the exposure of *Oreochromis niloticus* to bispyribac-sodium (0.800 mg/L), bentazon (36.00 mg/L), bensulfuron-methyl (2.50 mg/L), halosulfuron-methyl (1.275 mg/L), and quinclorac (11.25 mg/L) resulted in an altered erythrocyte shape, a decreased RBC count, increased levels of cholesterol, urea, creatinine, albumin, and globulin, an increased albumin/globulin ratio, and an increase in the activity of ALP, ALT, and AST [116]. Herbicides mainly mediate hepatic dysfunction by disturbing liver enzyme activity, dysregulating glycogen metabolism, and inducing oxidative stress responses. The most commonly reported molecular process is glycogen depletion, which decreases hepatic glycogen and eventually disrupts energy metabolism. Lipid peroxidation demonstrated an increase in the MDA and a decrease in the catalase and GSH levels, collectively resulting in oxidative stress damage. Erythrocyte alterations also decrease the RBC count, hematocrit values, and blood protein content, hindering oxygen transport and disturbing metabolic homeostasis. Additionally, the increase in plasma glucose is concomitant with an increase in cholesterol level and total plasma protein, significantly contributing to metabolic stress (Table 2). Species characteristics can affect the extent to which the toxicity of herbicides causes hepatic dysfunction. For example, predatory species such as *Oreochromis niloticus* and *Anguilla anguilla* are more susceptible to higher herbicide levels, resulting in severe hepatic and oxidative stress-related damage [110,116]. Furthermore, small-bodied species such as *Pimephales promelas* and *Astyanax* sp. experienced significant enzyme fluctuations, contributing to metabolic shifts [112,113]. Moreover, exposure duration and herbicide classification can shape the severity of hepatic dysfunction. Acute exposure to high doses of herbicides such as acetochlor causes rapid metabolic disruption, erythrocyte depletion, and liver enzyme inhibition. Chronic exposure to low-dose herbicides such as diuron induces progressive enzyme imbalances and oxidative stress [112]. Collectively, the data highlight the need for increased awareness and the regulatory examination of herbicide use and its potential impacts on aquatic ecosystems and organisms.

3.2.2. Effect of Herbicide Exposure on Neuronal Abnormalities

A study on the exposure of the *Anguilla anguilla* to a dose of 11.15 mg/L (one-third of the 96 h LC(50) of molinate resulted in decreased ChE activity in tissues [110]. Moraes and colleagues examined the effects of different herbicides on *Leporinus outsiders*. They revealed that AChE activity decreased in the brain at a dose of 0.5 mg/L of clomazone and 0.375 mg/L of quinclorac. A dose of 0.375 mg/L of clomazone exhibits more pronounced adverse effects, including decreased AChE activity in the brain and muscle and a decreased presence of thiobarbituric acid-reactive substances (TBARSs) in the brain, muscle, and liver tissues [111,115]. While higher doses of 3.6 mg/L of propanil and 0.002 mg/L of metsulfuron-methyl increased AChE activity in the tissue, lower doses of propanil 1644 µg/L induced a decrease in AChE activity in the brain and muscle along with a reduction in TBARS in the brain, muscle, and liver tissues [115]. Another study reported the effect of 3 mg/L glyphosate (Roundup, Bayer, Leverkusen, Germany) on the same species and demonstrated a decrease in brain AChE activity [117]. The species *Pimephales promelas* suffered reduced growth when exposed to 185 mg/L of bromacil, as well as abnormal or dead fry, and displayed decreased survival when exposed to 23.3 mg/L of diuron [113].

Herbicides exert their neurotoxic effect on fish by interfering with AChE activity, neurotransmitter signaling, and oxidative stress balance, giving rise to phenotypic traits such as neuromuscular dysfunction, cognitive impairment, and metabolic stress. Several molecular mechanisms are involved in the herbicide-mediated neurotoxic effect in fish, including AChE inhibition, which distorts synaptic transmission and reflexes in the brain and muscle tissues. Herbicides can also mediate neuroinflammation by inducing oxidative stress, increasing lipid peroxidation, and mitigating antioxidant defenses, ultimately resulting in neuronal cell damage and the development of inflammation. The disruption of ion channels mainly decreases Na^+/K^+-ATPase activity, significantly hindering neuronal excitability and impairing sensory responses. This induces metabolic stress in neurons by inducing elevated glucose and lactate levels. This causes substantial impairment to energy regulation, leading to neurodegeneration (Table 2). The severity of herbicide-mediated neurotoxic effects can significantly affect certain species. Predatory fish such as *Leporinus obtusidens* experienced greater neurotoxicity due to higher neuronal metabolic rates [111,115,117]. Additionally, body size can exaggerate the herbicide effect in small fish such as *Astyanax* sp., which is more vulnerable to oxidative stress-related neurodegeneration [112]. Furthermore, dose, exposure duration, and herbicide class can affect the extent of neuronal abnormalities. Acute exposure to high doses of herbicides such as glyphosate leads to progressive AChE depletion and neuronal stress [117]. Chronic exposure to low-dose herbicides, such as propanil and clomazone, causes neurotransmitter disruption and neuromuscular impairment [111,115]. Collectively, these findings highlight the necessity for further research into the toxicity limits of herbicides and regulatory actions to safeguard aquatic environments.

3.3. Fungicides and Their Effect on Fish Health

Fungicides are a category of pesticide specifically designed to control fungi that contribute to plant diseases and threaten crop yields. The use of fungicides has become widespread globally. Fungicides affect fish toxicity through several mechanisms, mainly oxidative stress, reducing reproductive capabilities, and inhibiting AChE activity in the brain. They also inhibit metabolism. These effects lead to impaired enzymatic functions, reproductive issues, metabolic stress, and neurotoxicity, as depicted in Table 3.

Table 3. Impact of fungicides on fish health.

Fish Species	Fungicide	Toxic Dose	Effect on Fish	Reference
Danio rerio	kresoxim-methyl	195 µg/L	↑catalase, ↑peroxidase, ↑carboxylesterase activities, ↑malondialdehyde content in larvae, and ↑oxidative stress in adults	[118]
	pyraclostrobin	81.3 µg/L	↑catalase, ↑peroxidase, ↑carboxylesterase activities, ↑malondialdehyde content in larvae, and ↑oxidative stress in adults	[118]
Oryzias latipes	triadimefon	2.0 and 3.5 µM	↑CYP3A, ↑CYP1A activity, ↑ CYP3A38 and ↑CYP3A40, ↑CYP26B, ↑pregnant x receptor, ↑retinoid acid receptor γ1, and ↑p53 gene expression	[119]
	myclobutanil	2.0 and 3.5 µM	↑CYP3A activity, ↑CYP3A38, ↑CYP3A40, ↑CYP1A, ↑pregnant x receptor, ↑p53 and ↑catalase expression	[119]
Pimephales promelas	prochloraz	0.1 mg/L	↓CYP19 aromatase activity in brain and ovarian homogenates, ↓productiveness of fish, and ↓steroidogenesis	[120]
	fenarimol	1.0 mg/L	↓CYP19 aromatase activity in brain and ovarian homogenates, ↓productiveness of fish, and ↓steroidogenesis	[120]
Carassius auratus	Tattoo (mancozeb)	10 mg/L (Tattoo) Corresponding to 3 mg/L (mancozeb)	↑SOD, ↑catalase, ↑glutathione peroxidase, ↑protein carbonyls in the liver and kidney, ↓GAPDH activity in the kidney, ↑lipid peroxide level in the brain, ↓glutathione reductase activity in the brain	[121]
Oncorhynchus kisutch	mancozeb	2.05 mg/L (EC 50)	↑AChE brain activity	[89]
Carassius auratus	Tattoo (mancozeb)	10 mg/L	Moderate lymphopenia, ↑protein carbonyl groups in the blood, ↓thiols levels, ↑lipid peroxide, ↑SOD, ↑catalase, and ↑GAPDH activity	[122]
Clarius batrachus	mancozeb	22.87 mg/L	↓protein, ↓amino acids, ↓glycogen, ↓nucleic acids, and ↓enzyme succinic dehydrogenase in the liver and muscles, ↑lactic dehydrogenase, ↑protease, ↑GOT, and ↑DPT in the liver and muscles	[123]

3.3.1. Impact of Fungicide Exposure on Hepatic Dysfunctions, Metabolism Disorders, and Oxidative Stress Responses

Mao et al. examined the effect of kresoxim-methyl (195 µg/L) and pyraclostrobin (81.3 µg/L) on *Danio rerio*. They highlighted the increased catalase, peroxidase, and carboxylesterase activities and malondialdehyde content in larvae, as well as increased oxidative stress in adults [118]. Another study on the effects of 2.0 and 3.5 µM of both triadimefon and myclobutanil, respectively, on *Oryzias latipes* emphasized that the fungicides induced hepatic cytochrome P450 (CYP) 3A4, CYP1A, CYP3A38, CYP3A40, and CYP26B activity, along with an increase the gene expression of pregnant x receptor, retinoid acid receptor γ1, and p53 [119]. In the *Carassius auratus*, a dose of 10 mg/L (Tattoo) corresponding to 3 mg/L (mancozeb) increased the activity of SOD, catalase, and glutathione peroxidase, enhanced the content of protein carbonyls in the liver and kidney, reduced glucose-6-phosphate dehydrogenase (GAPDH) activity in the kidney, increased lipid peroxide levels in brain, and reduced glutathione reductase activity in brain [121,122]. Likewise, the exposure of *Clarius batrachus* to 22.87 mg/L of mancozeb resulted in decreased protein, amino acids, glycogen, nucleic acids, and enzyme succinic dehydrogenase in the liver and muscles, while increasing lactic dehydrogenase, protease, and liver and muscle enzymes, such as glutamic-oxaloacetic transaminase (GOT) and dihydropyrimidine dehydrogenase (DPT) [123].

Moreover, fungicides can impact fish health by dysregulating hepatic enzyme activity, metabolic balance, and oxidative stress, collectively resulting in hepatic dysfunction due to impaired detoxification and metabolic imbalances. The molecular characteristics of fungicides' hepatic dysfunctions involve increased oxidative stress due to the induction of lipid peroxidation, catalase, and glutathione peroxidase, which contributes to disorderly cellular homeostasis. The dysregulation of metabolic enzymes also plays a role, affecting steroidogenesis and energy metabolism. Additionally, reducing glycogen and increasing lactic dehydrogenase and protein oxidation activate stress responses, resulting in liver cell damage and impaired hepatic function. Fungicide toxicity responses differ depending on fish species, dose, exposure duration, and the specific fungicide class. *Danio rerio* and *Oryzias latipes* are predatory fish that accumulate higher fungicide residues, leading to severe oxidative stress-related hepatic damage. *Pimephales promelas* and *Carassius auratus* have high metabolic rates, resulting in significant enzyme suppression and subsequently affecting detoxification processes. In addition, dose, exposure duration, and the specific fungicide class also contribute to the fungicidal mediation of hepatic dysfunction, resulting in variation in fish response. Acute exposure to high doses of prochloraz and mancozeb inhibits liver function, leading to metabolic and detoxification failure. Chronic exposure to low doses of triadimefon and kresoxim-methyl progressively depletes metabolic enzymes and induces oxidative stress.

3.3.2. Effect of Fungicide Exposure on Reproductive Abnormalities and Neuronal Abnormalities

The consequences of fungicide exposure extend to reproductive and neurological functions. A study on the effects of 2.0 and 3.5 µM of triadimefon and myclobutanil on *Oryzias latipes* found that the fungicides induced hepatic CYP3A activity and enhanced gene expression [119]. The inhibition of the activity of CYP19 aromatase in brain and ovarian homogenates reduces the fecundity of fish. For instance, exposing *Pimephales promelas* prochloraz at 0.1 mg/L and fenarimol at a dose of 1.0 mg/L inhibits steroidogenesis [120]. The use of mancozeb at a dose of 2.05 mg/L (EC 50) increased AChE activity in the brain in the *Oncorhynchus kisutch* species [89]. This discussion highlights the diverse effects of fungicide exposure on different biological systems in aquatic organisms, emphasizing the need to reevaluate their use and application in agriculture and aquaculture to minimize

environmental and ecological consequences. Fungicides can disrupt hormone balance and reproductive and neurological function, leading to fertility issues, impaired spawning, and neurotoxic effects. Fungicides can induce endocrine disruption by affecting the function of CYP450, causing hormonal blockade, and impairing gamete development, resulting in reduced reproductive success via a marked decrease in fertility. This consequently leads to lower reproductive output and a population decline.

Furthermore, like other classes, fungicides mediate neurotoxic effects by modulating neurotransmitters, including primarily inhibiting AChE and inducing oxidative stress, which causes neuromuscular impairment and cognitive dysfunction (Table 3). Variability is evident across species; for instance, egg-producing species such as *Pimephales promelas* are more prone to reproductive failure due to aromatase inhibition [120]. Predatory fish such as *Oncorhynchus kisutch* experience more significant neuronal damage due to neurotransmitter imbalance [89]. In addition, dose, exposure duration, and the specific fungicide class also contribute to fungicidal mediation of hepatic dysfunction, resulting in variation in fish response. Acute exposure to high doses of prochloraz causes immediate reproductive failure [120]. The variation in dose-dependent and species-specific effects remains unclear; further ecotoxicological studies and regulatory measures are warranted to mitigate the impact of these factors on aquatic ecosystems.

4. Pesticides and the Nutritional Value of Fish

Pesticide exposure significantly affects the nutritional value of fish by influencing protein levels, lipid metabolism, and carbohydrate regulation, which leads to reduced growth, health, and overall nutritional value. Several pesticides sustainably decrease total protein, mainly in muscle, liver, gills, and kidney tissues, indicating impaired protein synthesis and metabolism. In addition, pesticides affect lipid metabolism, causing liver lipid accumulation in specific tissues, decreasing PUFA content, and distorting membrane integrity, immune function, and nutritional value. Additionally, pesticides can increase carbohydrate levels in the gill, muscle, and kidney while decreasing glycogen storage in the liver, suggesting metabolic disruption, as depicted in Table 4. The subsequent section will shed light on the effect of various pesticides in protein, lipid, carbohydrate, and PUFA content in fish and demonstrate how this affects fish health and compromises their nutritional values.

Table 4. Impact of pesticides on the nutritional value of fish.

Fish Species	Pesticide	Toxic Dose	Effect on Fish	Reference
Oreochromis mossambicus	cypermethrin	10% EC	↓total protein	[17]
	lambda-cyhalothrin	2.5% EC	↓total protein	[17]
	malathion	57% EC	↓total protein	[17]
Channa punctatus	malathion	0.l mg/L	↑ free amino acid in muscle, kidney, heart, and stomach, ↓protein content in muscle, gill, and liver	[104]
Anguilla anguilla	chlorpyrifos	0.365 ppm	↑cholesterol and ↓protein in the liver	[62]
	fenitrothion	Sublethal 0.02 ppm	↓protein	[71]
Cnesterodon decemmaculatus	DIC and 2,4-dichlorophenoxyacetic acid (2,4-D)	410 mg/L	↓total protein	[109]

Table 4. *Cont.*

Fish Species	Pesticide	Toxic Dose	Effect on Fish	Reference
Oreochromis niloticus	acetochlor	2.625 mg/L	↑total plasma protein	[116]
Tilapia mossambica	monocrotophos	52.36 mg/L	↓protein, ↑carbohydrate levels in the gill, muscle, and kidney, and ↓cholesterol in the gill, muscle, and kidney	[124]
Clarius batrachus	mancozeb	22.87 mg/L	↓protein, ↓amino acids, ↓glycogen, ↓and nucleic acids in the liver and muscles	[123]
Cirrhinus mrigala	Cartap hydrochloride	0.339–0.436 mg/L	↓glycogen, ↓total protein, ↓nucleic acids in gill, liver, brain, kidney and muscle tissues	[93]
Channa punctata	endosulfan	Endosulfan A (0.05 and 0.45 ppb) and Endosulfan B (3 and 11 ppb)	↓protein, ↓glycogen, ↓lipid in the liver, ↓glycogen in muscle, ↑protein and ↑glycogen in the kidney, and ↑protein in the brain	[125]
	malathion	0.4 mg/L; 1/20th of 96 h LC50 value	↓serum glucose and ↑protein content	[105]
Gadus morhua	chlorpyrifos	0.5 mg/kg	↑omega-6 and ↓omega-3	[126]
Clarias batrachus	γ-BHC	2 and 8 ppm	Altered lipid metabolism	[127]
	malathion	1 and 4 ppm	↑liver lipids and Altered lipid metabolism	
Rhamdia quelen	trichlorfon	11 mg/L	↓total PUFAs	[63]

4.1. Protein

Protein metabolism and amino acid content may adversely affect various fish organs. Proteins are crucial to cells' structures and functions and essential for their sustenance [104]. Total protein might provide a practical diagnostic approach to examining the biological functionality of the cell to maintain homeostasis [17]. Evidence suggests that total protein content can be adversely affected when fish or other marine organisms are exposed to pesticides.

Numerous studies assert that the total protein content of fish may be altered following pesticide exposure (Table 4). Fluctuations in the level of inhibition or toxicity differ depending on the particular pesticide, the dose of the pesticide, and the length of exposure. Biochemical changes in fish's free amino acid content may occur in response to exposure to various pesticides. Increased exposure or contact with pesticides can lead to protein breakdown in fish (Table 4). Clinical studies have also shown that pesticides can alter fish's serum amino acid levels [104,128]. Amino acids are the fundamental components of protein, and changes in serum amino acid levels may directly result from toxic stress following pesticide exposure [104]. Results from Table 4 suggest that pesticides can promote alterations in the free amino acid content of plasma and various organs, including muscle, liver, kidney, and heart. Referring to the impact of pesticides on the nutritional value of fish species, Table 4 provides an elaborate account of the toxic doses used per species. A decrease in the total protein content of the *Oreochromis mossambicus* was observed due to

the doses of 10% EC of cypermethrin, 2.5% EC of lambda-cyhalothrin, and 57% EC of malathion [17].

Different malathion doses in *Channa punctatus* caused different effects. The 0.l mg/L dose increased the free amino acid content in the muscle, kidney, heart, and stomach and decreased the protein content in the muscle, gill, and liver [104]. Meanwhile, the 0.4 mg/L dose of 1/20th of the 96 h LC50 value of malathion reduced serum glucose and increased protein content [105]. The same species showed increased cholesterol in the liver and reduced protein when exposed to 0.365 ppm of chlorpyrifos [62]. Endosulfan A at concentrations of 0.05 and 0.45 parts per billion (ppb) and endosulfan B at concentrations of 3 and 11 ppb resulted in a drop in protein, glycogen, and lipid levels in the liver of *Channa punctata*. Additionally, it caused a reduction in muscle glycogen levels and increased protein and glycogen levels in the kidney [125]. A decrease in protein resulted from exposure to sublethal 0.02 ppm fenitrothion in the *Anguilla anguilla* [71]. Similarly, total protein content was reduced in *Cnesterodon decemmaculatus* with DIC and a 2,4-dichlorophenoxyacetic acid (2,4-D) dose of 410 mg/L [109]. Acetochlor, administered at 2.625 mg/L, increased the total plasma protein of *Oreochromis niloticus* [116]. A monocrotophos dose of 52.36 mg/L affected the *Tilapia mossambica* by causing a decrease in protein content, an increase in carbohydrate levels in the gill, muscle, and kidney, and a decrease in cholesterol in gill, muscle, and kidney [124]. Likewise, a reduction in protein, amino acids, glycogen, and nucleic acids in the liver and muscles was caused by a dose of 22.87 mg/L of mancozeb in the *Clarius batrachus* [123].

4.2. Fat

Fat is an essential macronutrient that is commonly found in fish. Pesticide pollution affects fat or lipids [129]. Fatty acids are the fundamental building blocks of all lipid molecules, playing a crucial role as one of the primary components of cellular membranes. Fatty acids are utilized as a source of energy and can be transported and processed for numerous purposes. They exhibit variations in length, degree of unsaturation, and substituent groups. Polyunsaturated fatty acids (PUFAs) are forms of essential fatty acid that can be found in several aquatic species. They have a crucial role in preserving the properties of cellular membranes, regulating hormone levels, and preventing or delaying the onset of diseases [12]. Fish muscles are renowned for their high content of PUFAs. They are primarily called omega-3 and omega-6 fatty acids, as these are the two fundamental categories of PUFAs. Omega-3 fatty acids are considered beneficial fats due to their proven ability to reduce the likelihood of specific human illnesses, such as cardiovascular disease. Omega-3 can be classified into two primary categories: eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) [4]. The omega-3 to omega-6 ratio differs among several fish species. Various factors may contribute to the observed variations in the lipid profile within each species. Farmed fish, such as trout, salmon, and sea bass, have higher proportions of omega-3 to omega-6 fatty acids than their wild counterparts. Pesticide exposure can cause alterations in the lipid composition of fish.

Indeed, OCPs have been observed to build up in the fatty tissues of unintended species and are linked to many ailments, such as cancer [29]. Chlorpyrifos-methyl has been found to induce alterations to inflammatory markers and lipid mediators in fish. Lipid mediators derived from omega-3 and omega-6 fatty acids, such as linolenic acid, arachidonic acid (ARA), EPA, and DHA, promote the synthesis of inflammatory mediators. Omega-6-derived mediators induce inflammation, whereas omega-3 mediators alleviate inflammation [126,130]. Table 4 illustrates the impacts of pesticides on the levels of fatty acids and the composition of lipids in fish. Olsvik et al. investigated the ability of PUFA supplementation to reduce the harmful effects of chlorpyrifos. They administered EPA and

ARA supplements to Atlantic salmon. They found that PUFA supplementation changed the lipid composition of cells, reduced the absorption of chlorpyrifos, and caused an increase in the quantity and size of lipid droplets in the cells. This study suggests that including lipids offers a satisfactory level of protection against the harmful effects of complement component properdin [126]. A dose of 05 mg/kg of chlorpyrifos in *Gadus morhua* resulted in an increase in omega-6 linoleate fatty acids and a decrease in omega-3 fatty acids [130]. A study observed altered lipid metabolism and elevated liver lipids when *Clarias batrachus* was exposed to gamma-BHC (2 and 8 ppm) and malathion (1 and 4 ppm), respectively [127]. Pesticides reduce the nutritional quality of fish by interfering with protein synthesis, lipid metabolism, and carbohydrate storage. The molecular effects include protein depletion, caused by decreases in total protein and amino acids. This leads to impaired muscle growth and tissue repair. Lipid metabolism disruption, caused by increasing liver lipids and omega-6, decreasing omega-3, and decreasing PUFAs, collectively alters the fatty acid composition, affecting energy reserves (Table 4). Variability in the response to the toxic effects of pesticides is evident across different species, pesticide doses, exposure durations, and pesticide classes. Fish with carnivorous feeding habits, such as *Gadus morhua* and *Channa punctatus*, are more susceptible to higher levels of pesticides, resulting in more significant depletion of macronutrients.

In contrast, omnivorous species like *Oreochromis niloticus* and *Clarias batrachus* experience more severe disruptions in lipid metabolism, which can negatively impact their energy balance and growth. Acute exposure to high chlorpyrifos doses alters omega-3 and omega-6 fatty acids ratios, affecting human consumption's nutritional value [130]. In contrast, chronic exposure to low doses of fenitrothion depletes protein levels and fatty acids [71].

5. The Impact of Pesticides on Fish and Human Health

Harmful substances can enter the human body via the ingestion of contaminated sources such as fish and other food items within the food chain [17]. Pesticide residues in food can produce undesirable effects in terms of human hormone regulation and enzymes. These effects can also lead to unwanted adverse health outcomes, chronic diseases, or cancer [2]. Indeed, OCPs impair the processes of fat and glucose metabolism, leading to a range of adverse consequences such as cancer, reproductive abnormalities, neurobehavioral abnormalities, and toxicity to the endocrine and immune systems [131]. The mechanisms for these adverse health outcomes include endocrine disruption, oxidation stress, and epigenetic changes. Various OCPs, including DDT, dieldrin, and toxaphene, are the most concerning human health chemicals [29]. Particular POPs, such as DDT, aldrin, dibenzodioxin, mirex, toxaphene, heptachlor, dieldrin, and polychlorinated biphenyls (PCBs), have been implicated in impacting the human reproductive and endocrine systems [132]. PCBs have been linked to reduced birth weight and cognitive and behavioral impairments in the offspring of women who consume high amounts of contaminated fish or have had occupational exposure [133].

High levels of exposure to DDT and DDE pesticides result in elevated serum levels of certain OCPs that are associated with diabetes in some populations [134]. Male subfertility has also been correlated with the presence of various OCPs and elevated levels of mercury in the diet [135]. Moreover, even at low concentrations, POP exposure leads to side effects related to the endocrine, reproductive, immune, and nervous systems [132]. POPs have been shown to accumulate in humans via the consumption of contaminated aquatic organisms [136]. Exposure to POPs has been linked to significant health consequences, including neurobehavioral disorders that can lead to learning difficulties, attention problems, cognitive disabilities, and reduced performance.

Changes in the immune system have also been suggested, resulting in defects in reproductive behavior, reduced lactation periods for mothers, cancer, and diabetes [132,137]. OPs function as ChE inhibitors, leading to permanent ACh neurotransmitter overlay across a synapse. The failure of a nerve impulse to cross the synapse results in muscle twitching, followed by paralysis and death. The nature of these pesticides makes them more toxic to vertebrates and invertebrates than other pests [18]. OCPs, carbamates, and OPs have been found to promote oxidative stress due to altered mitochondrial function. They can also lead to disruptions in neuronal and hormonal status in the body. OPs and carbamates act as AChE inhibitors and can affect the nervous system, muscles, liver, pancreas, and brain. OPs and carbamates impair enzymatic pathways by metabolizing all three macronutrients, including protein, carbohydrates, and fat [128]. These chemicals can affect cell growth, metabolism, protein synthesis, reproduction, and electrolyte balance in the human body [132]. Notably, the effects of the detrimental pollutant surpassed the dietary benefits [9]. The genotoxic and mutagenic effects of pesticides negatively impact both fish and humans. The consumption of fish from pesticide-contaminated regions leads to bioaccumulation and biomagnification, causing mutagenic and potential carcinogenesis effects [138]. For instance, several studies on fish from pesticide-contaminated areas emphasized substantial DNA damage and oxidative stress, insinuating that pesticides can endanger fish health and potentially harm human consumers through fish consumption [139,140]. Even though the level of pesticides might be undetectable in humans, chronic exposure might induce deleterious effects such as genotoxic, mutagenic, and carcinogenic effects [141]. Collectively, these studies shed light on pesticides' harmful effects on both fish and human health and indicate that a considerable effort must be made to address the risks associated with the impact of pesticides on aquaculture, marine species, and human health, such as establishing large-scale environmental monitoring programs and assessing the bioaccumulation levels of pesticides in aquatic species via large-scale biomonitoring studies. In addition, there is a pressing need to employ risk assessment models that evaluate how pesticides might affect marine ecosystems and human health. Finally, we advocate for tighter safety standards and more stringent maximum pesticide residue limits in aquaculture products.

6. Conclusions

Pesticides can have a beneficial impact on specific aspects of food security and human health. Pesticides lead to increased food production in agriculture and aid in relieving many vector-borne illnesses like malaria and typhus. Despite the noted benefits, improper disposal, usage, and handling can lead to high amounts of pesticides in the ecosystem. Pesticides can promote various adverse effects in fish, including oxidative stress, changes in fish composition, alterations in hematologic parameters, and altered metabolism. These changes can leave fish susceptible to disease and negatively impact the quality of fish. In many countries, fish and other marine species are vital sources of protein, omega-3s, and micronutrients. Pesticides can influence the fatty acids profile in fish, threatening individuals who ingest these fish. Although fish are noted to have significant health benefits related to cardiovascular disease, the harmful effects related to contamination might overshadow those benefits. Pesticide exposure, whether through diet or other means, is associated with severe adverse health effects in humans, including non-communicable diseases, reproductive impairments, cognitive impairments, and cancer. Particularly, populations at risk, such as pregnant women and children, face a higher risk of experiencing harmful health consequences due to consuming contaminated fish. Undoubtedly, a considerable effort must be made to quantify the impact of pesticides on aquaculture, marine species, food quality, and human health.

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Abbreviations

The following abbreviations are used in this manuscript:

AChR	acetylcholine receptor
ALT	alanine transaminase
ALP	alkaline phosphatase
ARA	arachidonic acid
EPA	eicosapentaenoic acid
DHA	docosahexaenoic acid
AST	aspartate aminotransferase
AzMe	azinphos methyl
BChE	butyrylcholinesterase
CFP	complement factor properdin
ChE	cholinesterase
DDE	dichlorodiphenyldichloroethylene
DDT	dichlorodiphenyltrichloroethane
DPT	dihydropyrimidine dehydrogenase
ERK1/2	extracellular signal-regulated kinase 1/2
G6PDH	glucose-6-phosphate dehydrogenase
GOT	glutamic-oxaloacetic transaminase
GSH	glutathione
GSSG	glutathione disulfide
GST	glutathione S-transferase isoenzymes
IL-1RI	interleukin receptor I
IL-1Î²	interleukin-1Î²
LDH	lactate dehydrogenase
MDH	malate dehydrogenase
OPs	organophosphates
PCBs	polychlorinated biphenyl
OCPs	organochlorines
PDH	pyruvate dehydrogenase
pERK1/2	phospho extracellular signal-regulated kinase 1/2
PGF2Î±	prostaglandin f2alpha
POPs	persistent organic pollutants
PUFAs	polyunsaturated fatty acids
ROS	reactive oxygen species
SOD	superoxide dismutase
TBARS	thiobarbituric acid-reactive substances
TEPP	tetraethyl pyrophosphate

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