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A Review on Toxicity of Pesticides in Catfishes: Reproductive, Haematological and Biochemical Aspects

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Author's contribution

The sole author designed, analyzed, interpreted and prepared the manuscript.

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Review Article

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ABSTRACT

This review article focuses on the effect of pesticides on reproductive functions, haematological and biochemical changes in catfishes. Pesticides are chemical substances that are released into the environment to control the populations of harmful pests. Pesticide exposure leads to toxicity in aquatic organisms, including fishes which are particularly sensitive to pesticides. The acute and sublethal concentrations of pesticides in the aquatic environment result in different lethal alterations, including changes in reproductive functions, histology, haematology, proteins, glucose, lipids, enzymes, etc. Pesticides act as endocrine disruptor compounds and have the potential to impair reproductive function in catfishes. Alterations in haematological and biochemical parameters are used as efficient biomarkers in assessing the toxicity of pesticides in fishes.

Keywords: Pesticides; reproduction; haematological; biochemical; catfish; toxicity.

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1. INTRODUCTION

Pesticide pollution in the aquatic environment is increasing due to their extensive usage in agriculture and fish farming. Variations in the chemical composition of aquatic environments can affect the survival of aquatic organisms including fishes. In the present era, the use of pesticides in agriculture are inevitable, but their effects on non-target organisms outweigh their impact on the target pests. Different types of pesticides are used in agriculture, but their toxicity to fishes varies with each pesticide type and insecticides are typically the most toxic [1,2]. The major insecticides that are usually used are organophosphate, organochlorine, carbamates, pyrethroids and necotenoides [1,3]. Pesticides enter the aquatic environment by surface run-off, leaching, drift, etc. and exposure to pesticides induces behavioural dysfunctions, physiological disturbances, histopathological damages, haematological alterations, biochemical changes, immune-suppression, hormone disruption, etc. in fishes [2,4,5,6,7,8,9,10]. Also, long- term exposure to pesticides induces mortality, physical, and morphological changes in fish [11]. The effect of the toxicant in fish is concentration and time-dependent [12]. A high level of pesticides in the blood of vertebrates has been reported to trigger reproductive dysfunction in vertebrates, including fish [13]. Pesticide exposure usually caused external and internal changes, most of which led to numerous deformities. Internal changes induced by pesticides are mostly at haematological, biochemical, and genomic levels [1,4,9]. Hematological and biochemical parameters are consistently used as indicators of the physiological or sublethal stress response in fish [14].

There are available reports on the influence of pesticides in inducing reproductive dysfunction in fishes [15,16]. Pesticides have the potential to disrupt the development and reproduction of fish and act as the endocrine-disrupting agents [17,18] causing reproductive endocrine disruptions [19,20,21]. Pesticides cause reproductive failure primarily by disrupting secretory activities of the hypothalamic-hypophyseal-gonadal axis [22]. Pesticides have been reported to trigger damage to fish gonads such as inhibits oocyte maturational processes, ovarian disruption including atretic follicles, intersex and disorganization of ovarian structure, spawning, hatching, and slow progression of spermatogenesis [15,23,24,25,26,27]. Sex steroids play an important role in the sex differentiation of lower vertebrates, including fishes and a small change in their levels caused by endocrine disruptors significantly affects the gonadal development of aquatic vertebrates [18]. It has been reported that the gonadal-somatic index, testosterone (T), and estradiol- 17beta (E) declined in the catfish captured from the polluted river when compared with the catfish captured from the reference site [13]. Thus, this article attempts to review the impact of some pesticides on the reproductive functions, haematology, and biochemical parameters of catfishes.

2. EFFECT OF SOME PESTICIDES ON REPRODUCTIVE FUNCTIONS

2.1 Effects of Organophosphate Pesticides

The toxicity of organophosphorus pesticides in inducing reproductive dysfunction in fish has been reported. The effect of organophosphate pesticide Malathion on reproductive organs of *Heteropneustes fossilis* has been investigated by Deka and Mahanta [24]. They exposed *H. fossilis* to Malathion at a concentration of 0.2 ppm for 10, 20 and 30 days and observed severe histological changes in the ovary of the fish. It showed adhesion of primary follicles, retraction and damage of oocyte, degeneration, and clumping of cytoplasm, the number of atretic oocytes increased, destruction of the ovigerous lamellae, and vitellogenic membrane, intrafollicular spaces increased, vacuolated cytoplasm, extrusion of karyoplasm and necrosis in the cytoplasm. Similarly, Dutta et al. [28] has reported that long-term exposure to the sub-lethal doses of malathion (1.2 mg 1^{-1}) caused severe damage to the ovary of *H. fossilis*. Clumping of cytoplasm appears after 24 hr of exposure to Malathion, and after 48 hr clumpings intensified with the degeneration of follicular cells. After 72 hr of exposure, nucleoli increased in number, shrinkage of nuclear materials and oocytes became adhered. With 96 hr of exposure, it was observed that nuclear materials of all the oocytes shrunk to a smaller clump, oocytes fused together along with ruptured of the follicular epithelium.

Malathion exposure of juvenile catfish *Clarias batrachus* even at low doses (1 and 10 µg/L) for 21 days revealed slow progression of spermatogenesis in the testis, while in the ovary, the oil droplet oocytes are higher after 10 µg/L malathion treatment [26]. They further noticed that exposure to low doses of Malathion hinder

and modulate early gonadal development by targeting gene expression pattern of transcription factors, activin A, sex steroid or orphan nuclear receptors, and steroidogenic enzymes in 50 days post-hatch catfish fingerlings. The impact of Malathion on endocrinology of catfish has also been observed. Exposure of *C. batrachus* for 30 days to Malathion reduced the levels of testosterone (T) and estradiol-17β (E2) in a dose-dependent manner during all the phases of reproductive cycle studied [20]. Reduction in the estrogen level in the blood serum of *H. fossilis* was also observed following 72 hours of exposure to a sub-lethal dose (1.2 mg L^{1}) of Malathion [28]. It appears that the pesticide Malathion, even at sublethal concentrations adversely affects gonadal activity in both male and female catfish.

According to Singh and Singh [29], exposure of *H. fossillis* to Cythion at sub-lethal dose for four weeks during the spawning phase of the annual reproductive cycle effectively reduced the testicular 32P uptake and suppressed gonadotrophin secretion from the pituitary gland. Reduced ovarian 32P uptake and a significantly decreased level of total gonadotropin in the pituitary gland and serum have also been reported when *H. fossilis* were exposed for 96 hours to Malathion [30]. Also, the pesticides Cythion and Paramar M50 were observed to effectively decrease the gonadotrophic potency, with a resulting reduction in ovarian 32P uptake in sham-hypophysectomised fish. In hypophysectomised fish, Cythion and Paramar M50 do not affect ovarian 32P incorporation. However, these pesticides significantly decreased the level of the GnRH-like factor in the hypothalamus, suggesting that Cythion and Paramar M50 adversely affect ovarian activity through the hypothalamic- pituitary-ovarian axis [22].

2.2 Effects of Organochlorine Pesticides

Organochlorine pesticides are synthetic pesticides used in the chemical industry and agriculture. Organochlorine is known for its high toxicity, bioaccumulation, and slow degradation. These pesticides have been developed to disturb the physiological activities of the target organism; however, often non-target species are also severely affected by their application [31]. Endosulfan produced a significant increase in plasma estradiol-17β (E2) level and decreased plasma testosterone (T) level but not 11 ketotestosterone (11-KT) in catfish *Clarias gariepinus* [32]. The effects of a low dose of Endosulfan (2.5 µg/L) and Flutamide (33 µg/L), alone and in combination, in male juvenile *C. batrachus* were investigated by Rajakumar et al. [12]. Exposure of the fish for 100 days post-hatch (dph) decreased the expression of testis-related transcription factors (dmrt1, sox9a, and wt1), steroidogenic enzymes (11β-hsd2, 17β-hsd12 and P450c17), steroidogenic acute regulatory protein, and orphan nuclear receptors (nr2c1 and Ad4BP/SF-1). A combination of Endosulfan and Flutamide treatments also elevated the levels of testosterone (T) and 11-ketotestosterone (11-KT). Levels of testosterone (T) and 11-ketotestosterone (11-KT) were also increased after Flutamide treatment. They further demonstrated that a low dose of Endosulfan modulated testis growth by decreasing the progression of spermatogonia's differentiation to spermatocytes, and this result suggests that even at a low dose, the two pesticides impair testicular development in the fish either directly or indirectly at the level of the brain.

In female Juvenile *C. batrachus* of 50 days post-hatch (dph), treatments with Endosulfan (2.5 ppb) and Flutamide (33 ppb) for 100 dph, enhanced expression of ovary-specific transcription factors, steroidogenic acute regulatory protein, and *aromatases*, while transcripts of tryptophan *hydroxylase2* (tph2) and gonadotropin-releasing hormone declined in the brain of treated groups with maximum reduction in the Endosulfan group when compared with the control group. There is also an increase in the number of previtellogenic but a smaller number of immature oocytes in the treated groups which indicates accelerated ovarian growth [33]. They also reported elevated *ovarian aromatase* activity and plasma estradiol-17β levels in the treated groups with the maximum being in the Endosulfan group. It seems that Endosulfan alone or in combination causes synchronous precocious ovarian development better than Flutamide. This result suggests that both Endosulfan and Flutamide alter ovarian growth by triggering precocious development in catfish. Treatment of male and female freshwater catfish, *H. fossilis*, with safe (0.1 and 1.0 mg/L) and sublethal concentrations (10 mg/l) of gamma-hexachlorocyclohexane revealed that GSI and plasma gonadotropin (GtH) were significantly decreased in both the sexes. In male fish, the plasma testosterone and 11-ketotestosterone, and in female fish the plasma testosterone and 17beta- estradiol significantly declined in response to gamma-hexachlorocyclohexane exposure [19].

During the investigation of Singh and Singh [30], Endrin (Hexadrin) has been observed to retard gonadotrophin secretion, reducing ovarian 32P uptake in female freshwater catfish, *H. fossilis*. In males, exposure at sub-lethal dose to Hexadrin for four weeks during the spawning phase effectively suppresses gonadotrophin secretion and reduced the testicular 32P uptake [29]. Also, it has been reported that the pesticides Aldrin and Hexadrin were effective in reducing the gonadotrophic secretion, with a reduction in ovarian 32P uptake in sham-hypophysectomised fish, whereas, in the hypophysectomised fish, these pesticides remarkably reduce the ovarian 32P uptake and significantly reduced the level of the GnRH-like factor in the hypothalamus [22].

2.3 Effects of Carbamate pesticides

Carbamate interferes with reproductive function in catfish. When *H. fossilis* were exposed to sublethal doses (0.5, 1, and 2 mg/L) of Carbofuran for 30 days, the gonado-somatic index of the fish treated with 1 or 2 mg/L was found to decrease significantly compared to that of the controls, but the dose of 0.5 mg/L was found to be ineffective [23]. Also, they have investigated the impact of Carbofuran on the egg maturational processes of *H. fossilis*. Histomorphological observations of the ovary of the fish after Carbofuran treatment (0.5, 1, and 2 mg/L) for 30 days at 25 ± 1°C, revealed the alteration in the area and the percentage of occurrence of the different types of primary oocytes when compared to that of the control fish. It was noticed that the stage I primary oocytes were predominantly higher in Carbofuran-treated fish than in stage II and stage III. The follicular walls degeneration and connective tissues, and vacuolization in the ooplasm of stage II and III oocytes were observed in Carbofuran-treated fish. Their findings indicate that Carbofuran at sublethal concentrations inhibits oocyte maturational processes in the catfish. The serum and ovarian content of 17beta-Estradiol in pre-spawning and spawning of *H. fossilis* was found to be reduced under Carbofuran treatment with sublethal doses (0.5 - 2 mg/ml) for 30 days. The serum and ovarian vitellogenin levels of fish were also reduced but only during the Pre-spawning stage. It was also noticed that the pituitary gonadotrophs of the pre-spawning fish failed to release gonadotropin hormone following Carbofuran treatment [17]. Based on this finding, they suggest that Carbofuran acts as an antiestrogenic, endocrine-disrupting agent, possibly targeting the pituitary-gonad axis of the fish.

2.4 Effect of Pyrethroid

Exposure of *Clarias gariepinus* to cypermethrin at 0 (ethanol solvent control), 0.07, 0.014, 0.028, 0.056 and deltamethrin at 0.22, 0.44, 0.88 and 1.76 µg/L, for 7, 14, 21, and 28 days, reveals the presence of ovotestis (intersex), oocytes atresia, cytoplasmic degeneration and clumping of vitellogenic oocytes in females, while male fish displayed enlargement and degeneration of testicular seminiferous tubules after 28 days exposure to cypermethrin and deltamethrin [21]. Eni et al. [21] observed respective and apparent concentration- and time-dependent increase and decrease of plasma estradiol-17β (E2) and testosterone (T) levels, compared to control. The significant increase in estradiol-17β (E2) levels paralleled gonadal ovotestis (intersex) condition in exposed fish, indicating endocrine disruptive effects of cypermethrin and deltamethrin that favour the estrogenic pathway.

The effects of cypermethrin (0, 2, 4, 8, 16, and 32 µg L-1) on the embryo and the larvae of *Gangetic mystus* (*Mystus cavasius*) was elucidated by Ali et al. [34]. According to this study, increasing cypermethrin concentrations decreased the hatching success and significantly increased the mortality of embryo and larvae. Their result revealed several malformations in embryos and larvae when exposed to the two highest concentrations of cypermethrin. Their findings suggest that 2 µg L-1 cypermethrin concentration in the aquatic environment may have deleterious effects on the development and reproduction of *Gangetic mystus*.

2.5 Effects of Rotenone

There is a scarcity of information on the impact of Retenone on Catfish reproduction. The effect of Rotenone in altering neurobehavioral and reproductive functions of freshwater catfish, *Mystus cavasius*, has been reported by Badruzzaman et al. [35]. According to Badruzzaman et al., at the neuroendocrine level, dopamine (DA) regulates fish's behavioral and reproductive functions. Treatment of *Mystus cavasius* with rotenone at 0, 2.5, 25, and 250 µg/L for 2 days significantly reduced dopamine (DA) and 3,4-dihydroxyphenylacetic acid (DOPAC) and after fish were treated with 250 µg/L concentration of rotenone for 2 days, there was a significant reduction of DA, DOPAC, and

DOPAC/DA in diencephalon, DA and DOPAC in pituitary, and in the telencephalon only DA, compared with control fish. In this study, Rotenone treatment was observed to significantly increase gonadosomatic index with many mature vitellogenic oocytes in ovaries and lowered dopaminergic activity in the fish. These findings seem to suggest that Retenone affect reproductive functions and behaviour via dopaminergic neuronal cell loss in the brain of *Mystus cavasius*.

2.6 Effect of Tihan

Agbohessi et al. [25], studied the acute toxicities of Tihan 175 O-TEQ, as well as it active ingredients Flubendiamide and Spirotetramat for embryo-larval of the African catfish *C. gariepinus*. For embryo-larval stages, Tihan is toxic but Spirotetramat and Flubendiamide produced more toxicity, their LC50 48hr constituted 20, 8.44 and 2.0 ppm, respectively and all of these compounds decreased hatching rates. Tihan and Spirotetramat affect the larval swimming coordination, Flubendiamide induced tail cleavage. According to Agbohessi et al. [32] chronic exposures to Tihan (LC50 96 hr = 440 ppb & 880 ppb) reduced fertilization rate, hatching rate, ova, and larval weight, delayed hatching, and increased abnormalities in the F1 generation. In male gonads, foam cells in lobular lumen, fibrosis, necrosis, and immature cells released in the lobular lumen and female gonads melano-macrophage centers (MMCs), necrosis, fibrosis, and vacuolation were observed. Tihan doses also significantly decreased plasma estradiol-17β (E2) and 11 ketotestosterone (11-KT), but not testosterone (T). The findings also revealed altered gonad histology and induced high proportions (18–30% of males) of ovotestis in males and follicular atretic oocytes in females but only one case of ovotestis was observed at the highest dose of Tihan treatment.

2.7 Effects of Herbicides

Herbicides are widely used to control the harmful effects of pests and weeds on agricultural production and fish farms. The herbicide after being used, ultimately find their way into different aquatic ecosystems and be highly toxic to non-target organisms including fishes. Herbicides may have some effects on the development, growth, reproduction, and behavior of fish [36]. Findings of Opute et al. [37] revealed a significant decrease in the level of follicle stimulating hormone (FSH), leutenizing hormone (LH), prolactin (PRL) and testosterone (T), and increasing the concentration of progesterone after 28 days of exposure of juveniles *Clarias gariepinus* to a concentration of 2.5, 25, 250, and 500 µg L^{-1} Atrazine. Histologically, treatment of ovaries with the lowest concentration (2.5 µg L^{-1}) revealed that atretic oocytes with broken membranes invaded many of the dead ova and empty spaces. In other treatments (25, 250, and 500 µg L^{-1}), interfollicular spaces, vacuolation in oocyte formation, and dissolution of oocyte walls were observed, but at the highest atrazine concentration (500 µg L^{-1}), disruption of the yolk vesicle and clumping of the cytoplasm in maturing oocytes was reported.

Soni and Verma [38] studied the effects of pretilachlor SL-I (1/20th LC50), SLII (1/15th LC50), and SL-III (1/10th LC50) for 30, 45, and 60 days on the plasma sex steroid profile, plasma vitellogenin concentration, and gonadal *aromatase* activity in *Clarias batrachus*. Depending on concentration and duration of exposure, this herbicide decreased the plasma testosterone levels in both male and female fish. Significant increases in the plasma 17β-estradiol, plasma vitellogenin concentration, and gonadal *aromatase* activity were observed only in the male fish, but no significant changes were observed in female fish. This finding seems to suggest that herbicide pretilachlor acts as an endocrine disrupting agent but act differentially in male and female fishes.

Treatment of *Clarias batrachus* with a sublethal concentration of Pendimethalin for 30, 45, and 60 days result in a remarkable increase in plasma 17β-estradiol (E2) in males but the plasma 17β-estradiol in females was not affected. Plasma concentrations of testosterone were significantly decreased in both sexes. It was noticed that plasma vitellogenin (VTG) and gonadal *aromatase* activity increased in male fish irrespective of concentration and duration of exposure. However, in female fish there was concentration and time-dependent reduction in plasma vitellogenin levels but unchanged gonadal *aromatase* activity. These findings revealed that the ability of Pendimethalin to induce reproductive toxicity in male and female fish differs considerably [39].

Thus, it is seen that pesticides have the potential to alter reproductive processes in different species of catfishes. Pesticides seem to induce reproductive failure by affecting gonadal steroid production and disrupting the secretory activities of the hypoyhalamic-pituitary-gonadal axis of the

fish. It was noticed that the gonads get affected differently by the pesticides during different stages of their development, disrupting, and influencing the reproductive performance of catfishes through decreased gonadal steroids and reproductive axis activity. Both testis and ovaries show a wide range of abnormalities when catfish are exposed to certain pesticides. Disruption of spermatogenesis and oogenesis, ovarian development and growth are explicitly evident of the toxic effect of pesticide in catfishes. Therefore, the reproductive health of catfish can be used as bioindicators of reproductive toxicants in aquatic environments.

3. EFFECT OF SOME PESTICIDES ON HAEMATOLOGICAL PARAMETERS

Haematological parameters, such as erythrocyte and leucocyte count, throbocyte, etc. have been considered as bioindicators of toxicity in fish following exposure to organochlorine, organophosphate, Carbamate, and pyrethroid insecticides [40]. The impact of pesticides on haematological parameters of catfish has been reported by several authors. Joshi et al*. [41*] reported that Lindane and Malathion exposure of *C. batrachus* for a period of 30 days causes a decrease in red blood cells (RBCs) count and haemoglobin (Hb) content. They also found that the erythrocyte sedimentation rate (ESR) remained unchanged in Malathion treated group, whereas Lindane treated group showed increased values for ESR. Also, pesticides such as Diazinon, Malathion, Carbofuran, Cypermethrin, Dichlorvos, Acetelic, Chlorpyrifos, etc. induced a significant reduction in RBCs, Hb content and haematocrit values in *C. gariepinus* [42,43,44,45,46], *Clarias albopunctatus* [47] and *H. fossilis* [48]. Significantly lower RBC and haematocrit values have also been observed under Termifos exposure in *C. gariepinus* [6]. Herbicide Ronstar significantly reduced the erythrocyte count and Hb concentration in *C. gariepinus* juvenile [49]. On the other hand, organophosphate pesticide, Phostoxin and DD-Force induced significant ($P < 0.05$) elevation in RBC and platelets values in the exposed fish *C. gariepinus* [8]. These alterations in the RBCs, haemoglobin and haematocrit value might be due to direct or feedback responses of structural damage to RBC membranes resulting in haemolysis and impairment in haemoglobin synthesis [50]. Thrombocyte count significantly increases ($p > 0.05$) in *C. gariepinus* following exposure to Diazinon pesticide [42]. In fish, the pronephros, or head kidney, is a basic organ forming the blood elements, and is also a reservoir of blood cells [51,52,53,54,55]. Therefore, the decrease in the haematological parameters may be correlated to the dysfunction of haematopoietic system of the fish. The decrease in haemoglobin content in the pesticide treated fish may also be due to increased rate of destruction of haemoglobin or due to decreased rate of synthesis of haemoglobin or due to dysfunction/suppression of haemopoietic organ, and the decrease in thrombocytes is related to decreased thrombocyte production or increased destruction of thrombocytes [48].

Alteration in the haematological indices like the pack cell volume (PCV), mean corpuscular hemoglobin (MCH), mean corpuscular hemoglobin concentration (MCHC), mean corpuscular volume (MCV) have also been observed in the catfish following exposure to different pesticides [42,44,45,46]. Termifos treatment induced a significant elevation in the MCH, MCV and mean MCHC values in the fish *C. gariepinus* [6]. In *C. batrachus*, PCV decreased under Lindane and Malathion exposure [41]. However, significant elevation in PCV has been observed in *C. gariepinus* following exposure to organophosphate pesticide Phostoxin and DD Force [8]. It has also been reported that Ronstar (Herbicide) treatment induced significant reduction in PCV, MCH and MCV in juvenile *C. gariepinus* [49].

Various pesticides including Malathion, Lindane, Diazinon, Carbofuran, Acetelic, Chlorpyrifos, Dichlorvos, etc. drastically induced changes in the white blood cell (WBCs) count in *C. gariepinus, C. batrachus, C. albopunctus, H. fossilis* [41,42,43,44,46,48,56]. Malathion and Diazinon exposure decreased WBC count, the relative and absolute lymphocyte counts, but there was a significant increase in both the relative and absolute count of developmental forms of neutrophile granulocytes, myelocytes ($p < 0.05$) and metamyelocytes ($p < 0.05$) in *C. gariepinus* [42,43]. Also, WBC count has been reported to decrease under Termifos (chlorpyrifos-based pesticide) exposure in *C. gariepinus* [6].

On the other hand, in the study conducted by Joshi et al. [41] Malathion and Lindane exposure for 30 days has been reported to increase the WBC count in *C. batrachus*. An increase in WBCs count under Carbofuran, Clorpyrifos and Dichlorvos exposure has also been observed in catfish (43, 44, 48). Phostoxin and DD Force (Organophosphate pesticides) also induced

significant (P < 0.05) elevation in WBC values in the exposed fish *C. gariepinus* [8]. Carbofuran exposure caused a significant increase in WBCs count, neutrophils, eosinophils, basophils, and monocytes, while the lymphocytes were decreased [44]. Significant leucocytosis was reported in *C. albopunctatus* fingerlings following exposure to sublethal concentrations of insecticide Actellic [56]. Oluah et al. [49] reported significant leucocytosis, lymphocytosis, neutropenia and monocytopenia in the *C. gariepinus* juvenile fish exposed to Ronstar herbicide. According to Prakash and Verma [48], Chlorpyrifos exposure significantly decreased monocytes, eosinophil and basophil in the treated group. However, depending on concentration and duration of exposure, it produced a significant increase in WBCs count, lymphocytes and neutrophils causing lymphocytosis in Chlorpyrifos exposed *H. fossilis*. Such Lymphocytosis effect might be due to an immunological reaction to produce more antibodies to cope with the stress induced by the toxicant. Significant leucocytosis and macrocytic anaemia have been observed in *C. albopunctatus* under Acetelic exposure [56]. The increase in WBC count may be correlated with an increase in antibody production, which helps the exposed fish to cope with the pesticide stress [41]. The changes in values of both the erythrocyte and leucocyte profile after exposure to pesticides may also be referred to the disruption of haematopoiesis and a decrease in non-specific immunity of the fish [42].

4. EFFECT OF SOME PESTICIDES ON BIOCHEMICAL PARAMETERS

The pesticides toxicity on biochemical substances like proteins, lipids, glycogen, etc. has been investigated in various organs of fishes [2,7,43,57]. Once toxic substances such as pesticides enter the body of the fish, they damage and weaken the mechanism concerned, leading to physiological, pathological and biochemical disorders or metabolic dysfunction [8,58,59,60]. Nuvan, a highly hepatotoxic organophosphate that predominantly damages the liver, exhibited dose- and duration-dependent alterations in many biochemical components such as serum protein, serum albumin, serum bilurubin, serum creatinine and urea in *H. fossilis* [59]. In *C. gariepinus,* organophosphate pesticide Malathion produced a remarkable decrease in plasma protein levels and increase in plasma glucose levels after exposure to sublethal concentration (0.5, and 2.0 mg/L) for 4 weeks [43]. A declining trend in blood protein concentration has also been observed under Termifos treatment, whereas blood glucose concentration showed an ascending trend and a positive correlation with Termifos concentration [6]. The total protein, albumin and globulin levels were significantly elevated, and the plasma glucose, total lipid, urea and creatinine levels were significantly increased under Carbofuran exposure [44]. A marked decrease in total protein and serum albumin, and a significant increase in urea and creatinine have been reported under Delamethrin (pyrethroid pesticide) treatment [61]. Depletion in total protein by organophosphate pesticides may be due to the inhibition of RNA synthesis disturbing the protein metabolism [59]. In the study conducted by Ahmad and Gautam [59] on *H. fossilis*, they found that hypoproteinemia and hypoalbuminemia after 60 days of exposure to Nuvan (organophosphate pesticide) which may be due to the liver damage where most plasma protein synthesis usually occurs. Also, Creatinine showed a significant increase after Nuvan exposure which might be due to the harmful effects on kidney tissues and inability of the damaged kidney to filter urea up to normal levels. Tyagi [57] reported that Methyl Parathion exposure alters the lipid profiles such as level of triglyceride, total cholesterol, high density lipoprotein (HDL), low density lipoprotein (LDL) and very low-density lipoprotein (VLDL) in *C. batrachus*. Tyagi [57] further observed that Methyl Parathion pesticide also alter the functioning of the kidney of fish by changing the level of urea, uric acid and creatinine up to an alarming level as these changes significantly produce a harmful effect on fish. Ahmad and Gautam [59] found a significant increase in total billirubin (hyperbilirubenaemia) following exposure to Nuvan which may be attributed to the great damage of hepatocytes, obstruction of the bile duct or a haemolysis, and the higher levels of serum billirubin caused liver damage in the exposed fish.

Glycogen in liver and muscle decreased in the fish exposed to 0.5, 1.0 and 2.0 mg/L of Malathion [43]. Ibrahim and Harabawy [7] observed a highly significant reduction in Glycogen levels in the liver and gills ($p< 0.001$), a significant decrease ($p< 0.05$) in kidney and muscles, and insignificant changes ($p>0.05$) in gonads of *C. gariepinus* exposed for 35 days to Carbamate pesticide. The reduction in glycogen content in the body tissue of the fish implies its rapid utilization for energy generation, to cope with the toxic stress induced by the pesticide [2].

Changes in plasma enzyme activity are used as indicators of tissue injury, environmental stress, or a diseased condition. Variation or fluctuation in total protein and albumin, serum glutamate oxaloacetate *transaminase* (SGOT) and glutamate pyruvate *transaminase* (SGPT) indicate the continuous decay of different tissues and muscle where the pesticide is accumulated [58]. Exposure of *C. gariepinus* to sub-lethal concentrations (0.5, 1.0 and 2.0 mg/L) of Malathion, the organophosphate pesticide for 4 weeks increased activities of Alanine *aminotransferase* (ALT), glutamate oxaloacetate *transaminase* (GOT) and glutamate pyruvic *transaminase* (GPT). Magnesium and calcium ions were also affected, but the effects were insignificant [43]. Under Carbofuran (0.16 and 0.49 mg/L) exposure for 35 days, there caused a significant ($p<0.05$) increase in aspartic *aminotransferase* (AST) and alanine *aminotransferase* (ALT) and a significant ($p<0.05$) decrease in alkaline *phosphatase* (ALP) activity in *C. gariepinus* [44]. The two metabolic enzymes, *glucose-6-phosphate dehydrogenase* (G6PDH) and *lactate dehydrogenase (LDH)* exhibited significant decreases ($p<0.05$) in the liver, kidney, gills, gonads and muscles of Carbofuran exposed fish [7]. Deltamethrin (Pyrethroid pesticide) induced a significant increase in serum Alanine *transaminase* (ALT), and Aspartate *transaminase* (AST) activity in fish [61].

The oxidative stress markers such as *superoxide dismutase* (SOD), glutathione (GSH) and *glutathione S-transferase* (GST) of liver, kidney, gills, gonads and muscles were also affected by Carbofuran treatment [7]. Lipid peroxidation significantly increased in the liver, kidney, and gill of *C. gariepinus* under Deltamethrin treatment [61]. Likewise, Carbofuran exposure for 35 days significantly increased ($p<0.05$) lipid peroxidation (LPO) levels in the liver, kidney, gills, and muscles except for gonads after 5 weeks of exposure [7]. *Catalase* (CAT) activity was significantly decreased in the liver, kidney and gills of catfish *C. gariepinus,* following 48 hr exposure to 0.75 µg/L pyrethroid pesticide Deltamethrin [61]. Carbofuran exposure for 35 days also showed a significant decline ($p<0.05$) in *catalase* activity in the liver, kidney, gills and muscle of *C. gariepinus* [7]. This significant reduction in *catalase* activity might be due to the influx of super oxide radicals, which have been reported to decrease *catalase* activity [61].

Inhibition of *Acetylcholinesterase* (AChE) is a specific biomarker for exposure to organophosphate and carbamate insecticides [62]. *Acetylcholinesterase* (AChE) activity is more sensitive for organophosphates and carbamate pesticides than other contaminants, however the inhibition of this enzyme has also been used to indicate the exposure and effects of other contaminants in fishes [1]. Reduction in the *acetylcholineterase* level under sublethal concentrations of the organophosphate pesticide, Phostoxin and DD Force has been observed in the catfish *C. gariepinus* [8]. Herbicide Clomazone significantly reduced the *acetylcholinesterase* activity in brain and muscle tissues, and in contrast, Quinclorac and Metsulfuron methyl caused an increase in *acetylcholinesterase* activity in the brain and inhibit the enzyme activity in muscle tissue [63]. Somnuek et al. [62] conducted the Real-time PCR examination of AChE gene expression in brain tissue of hybrid catfish, exposed to sublethal concentrations of Chlorpyrifos (0.43, 4.3 and 43 µM) and Carbaryl (1.19, 11.9 and 119 µM) for 24 hr and noticed that AChE gene expression was significantly elevated (12.4 times) in catfish exposed to 43 µM chlorpyrifos in comparison to the control group ($p<0.05$), but Carbaryl did not produce any significant change. Mdegela et al. [47] investigated the AChE activities in plasma, eye and brain homogenates of *C. gariepinus* using Ellman's method and 5,5'-dithiobis-2-nitrobenzoic acid (DTNB) chromophore. They found that Chlorfenvinphos significantly inhibits AChE activities in plasma (84%) and eye homogenate (50%) whereas the AChE activities in brain homogenate were comparable between Chlorfenvinphos exposed fish and controls. Mdegela et al. [47] and Doherty et al. [8] reported that *acetylcholinesterase* is a useful biomarker in assessing aquatic environments contaminated by pesticides or *anticholinesterases*.

5. CONCLUSION

From the present review, it is obvious that different pesticides present in the aquatic environment can affect the reproductive functions, haematology, and biochemical parameters of catfishes in different ways. The acute toxicity effects and alterations in reproductive functions, blood characteristics and biochemical parameters observed for all pesticides reviewed imply that commonly used pesticides that find their way directly or indirectly into the aquatic ecosystem remain a serious

health concern to the fish. Thus, based on the observations on the histological and hormonal alteration of gonadal tissue, changes in RBCs, WBCs, proteins, lipids, glucose, antioxidant defense system following exposure to different pesticides, it can be concluded that pesticides even at sublethal concentrations, are highly toxic to fish and impose life-threatening effects on fish. They can decrease the fish population by inducing reproductive dysfunction and by weakening the immune defense mechanisms of the fish.

COMPETING INTERESTS

Author has declared that no competing interests exist.

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