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A review on toxicity of pesticides in Fish

K. Shankar Murthy [1*], B.R.Kiran [2] and M.Venkateshwarlu [3]

1. Research & Teaching Assistant in Botany and Biotechnology, Directorate of Distance Education, Kuvempu University, Shankaraghatta- 577 451, Karnataka, India.

2. Research & Teaching Assistant in Environmental science, Directorate of Distance Education, Kuvempu University, Shankaraghatta- 577 451, Karnataka, India.

3. Director, Directorate of Distance Education and Professor, Department of Applied Zoology, Kuvempu University, Shankaraghatta- 577 451, Karnataka, India

Abstract

Pesticides usage in agricultural fields to control pests is extremely toxic to non target organisms like fish and affect fish health through impairment of metabolism, sometimes leading to mortality. Present study is a review of potential adverse effects of pesticides and pollutants in fish. Furthermore, the data generated could be useful in the environmental risk assessment of freshwater and marine organisms.

Keywords

Acute, chronic, pesticides, toxicity, fish.

***Corresponding Author:** K. Shankar Murthy [1], Email: skdde.ku11@gmail.com

I. INTRODUCTION

In recent years, the high rate of increase in human population and rapid pace of industrialization have created problem of disposal of waste waters. The domestic wastes and untreated or partially treated industrial effluents, supplemented with pollutants like heavy metals, pesticides and many organic compounds, have greatly contributed to massive fish death of aquatic ecosystems. These toxic chemicals and metals have changed the quality of water that affect the fish and other aquatic organisms [1-2].

II. PRODUCTION, USAGE OF PESTICIDES IN INDIA AND THEIR BENEFITS

The production of pesticides started in India in 1952 with the establishment of a plant for the production of BHC near Calcutta, and India is now the second largest manufacturer of pesticides in Asia after China and ranks twelfth globally [3]. There has been a steady growth in the production of technical grade

pesticides in India, from 5,000 metric tons in 1958 to 102,240 metric tons in 1998. In 1996–97 the demand for pesticides in terms of value was estimated to be around Rs. 22 billion (USD 0.5 billion), which is about 2% of the total world market [4].

The pattern of pesticide usage in India is different from that for the world in general. In India 76% of the pesticide used is insecticide, as against 44% globally [3]. The use of herbicides and fungicides is correspondingly less heavy. The main use of pesticides in India is for cotton crops (45%), followed by paddy and wheat.

The primary benefits are the consequences of the pesticides' effects – the direct gains expected from their use. For example the effect of killing caterpillars feeding on the crop brings the primary benefit of higher yields and better quality of cabbage. The three main effects result in 26 primary benefits ranging from protection of recreational turf to saved human lives. The secondary benefits are the less immediate or less obvious benefits that result from the primary benefits. They may be subtle, less intuitively obvious, or of longer term. It follows that for secondary benefits it is therefore more difficult to establish cause and effect, but nevertheless they can be powerful justifications for pesticide use. For example the higher cabbage yield might bring additional revenue that could be put towards children's education or medical care, leading to a healthier, better educated population. There are various secondary benefits identified, ranging from fitter people to conserved biodiversity [4].

III. PESTICIDES: TOXICITY TO AND OCCURRENCE IN AQUATIC SYSTEMS

To identify the major routes of pesticide exposure to aquatic systems and biota, the following parts of a global biocycle should be considered:

1. The water column, which usually first comes in contact with pesticides;
2. Organic substrates (algae, mosses, vascular hydrophytes, leaf litter, and branches);
3. Inorganic substrates, which include sedimentary material ranging from microscopic silts to coarse sand particles.

Naturally, pesticide content of interstitial water and sediments is usually much lower than that in the water column; and lithic biotopes are mostly less contaminated than standing waters. For instance, permethrin residues after application to a 640 ha forested block in Ontario attained peak concentrations of 147 µg/l in ponds, but only 2.5 µg/l in streams; while accumulation and persistence of pesticides in bottom sediment was negligible [5].

The determination of pesticide content in water, organic substrates, sediments (and animal tissues as well) is solely chemical. The solid materials or animal/plant tissues are usually homogenized, and 2−3 or less are extracted (e.g., with acetone/hexane), evaporated to a small volume, cleaned and dried, and analysed by a gas chromatographic procedure.

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The effects, including acute toxicity, of pesticide-contaminated water on non-target organisms (NTOs) should be determined using biological methods. The concept of *in situ* bioassays is based on exposure of test animals at field sites without disturbing contaminated sediment, and the determination of percentage survival. The process of exposing fish to test the toxicity of water is relatively simple: cages containing fish are hung in the water column or anchored at the bottom, and mortality is measured after exposure for 96 h or longer.

Histopathological effects of pesticides in fishes have been studied intensively. Pathological changes occur mainly in the liver, blood vessels, kidneys, and gills. Liver cells exhibit cytoplasmic granularity, partial loss of liver plate radial orientation, and shrinkage of some liver cell mass. Glomeruli in the posterior kidney show pycnotic changes of cell nuclei, vocalization of cytoplasm, and atrophy of some cells. Gill filaments and lamellae show the precipitated masses that have plugged the central capillaries. The pathological changes of large blood vessels caused by methoxychlor are described in bluegill by Kennedy *et* al.[6]. Also changes in haematocrit levels and in morphology and quantity of blood cells have been found in various fish species. Boyd [7] found that sublethal amounts of several chlorine insecticides induced abortions. The highest levels of DDT (2.0 mg/kg per week for 156 days) produced more mature ova than the untreated fish; also mortality among sac-fry was higher when one of the gametes came from a treated parent than when both gametes came from untreated fish. The reproduction of some other species (e.g, cutthroat goldfish) seems to be unaffected by DDT treatment [8]. Table 1 depicted acute toxicity of pesticides to various fish species.

Figure 1. Fish bioassay for toxicity studies (www.popstoolkit.com)

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Table 1: Acute toxicity (LC_{50}) of pesticides to fish species

Sl. No.	Name of the pesticides	Test organism	Duration of exposure	LC50 value	Reference
1	Cypermethrin	*Labeo rohita*	96 hrs	4.0µ/L	Marigoudar et al., 2009 [9]
2	Methyl parathin	*Catla catla*	96 hrs	4.8ppm	Illyazhanan et al., 2010[10]
3	Malathion	*Heteropneustes fossilis*	96 hrs	**0.98ppm**	Sanjoy Deka & Rita Mahanta, 2012 [11]
4	Pyrethroid Lambela Cyhalothrin	*Danio rerio*	96 hrs	0.119µ/L	Badre Alam Ansari & Kafeel Ahmed, 2010 [12]
5	Cypermethrin	*Colisa fasciatus*	96 hrs	0.02mg/L	Shailendra Kumar Singh et al., 2010 [13]
6	Rogor	*Puntius stigma*	96 hrs 72 hrs	7.1ppm 7.8ppm	Bhandare et al., 2011 [14]
7	Malathion	*Labeo rohita*	96 hrs	15mg/L	Thenmozhi et al., 2011[15]
8	Dimethoate	*Heteropneustes fossilis*	96 hrs	2.98mg/L	Rakesh K. Pandey et al., 2009 [16]
9	Elsan	*Channa punctatus*	48 hrs	0.43ppm	Sambasiva Rao ety al, 2009[17]
10	Endosulfan	*Channa striatus*	96 hrs	0.0035ppm	Ganeshwade et al., 2012[18]
11	Metasystox	*Nemacheilus botia*	96 hrs	7.018 ppm	Nikam et al., 2011 [19]
12	Acephate	Fathead minnow	96 hrs	>1000 mg/L	Waynon Johnson & Mack Finley ,1980 [20]
13	Alaclor	Rainbow trout	96 hrs	2.4(1.8-3.1) mg/L	Waynon Johnson & Mack Finley ,1980 [20]
14	Akton	Channel catfish	96 hrs	400(295-542) µg/L	Waynon Johnson & Mack Finley ,1980 [20]
15	BHC	Gold fish	96 hrs	348(261-466) µg/L	Waynon Johnson & Mack Finley ,1980 [20]
16	Carbaryl	Lake trout	96 hrs	690(520-910) µg/L	Waynon Johnson & Mack Finley ,1980 [20]
17	Carbofuran	Yellow perch	96 hrs	147 (115-188) µg/L	Waynon Johnson & Mack Finley ,1980 [20]
18	DDT	Rainbow trout	96 hrs	8.7 (6.8-11.4) µg/L	Waynon Johnson & Mack Finley ,1980 [20]
19	Endosulfan	Channel catfish	96 hrs	1.5 (1.3-1.7) µg/L	Waynon Johnson & Mack Finley ,1980 [20]

IV. EFFECT ON ACETYLCHOLINESTERASE

AchE activity is more sensitive for organo phosphates and carbamate pesticides than other contaminants, but the inhibition of this enzyme have been also used to indicate the exposure and effects of other contaminants in fishes. It has been shown that the addition of crude oil to brain homogenate in amounts equivalent to sediment concentration inhibited AChE activity in fishes [21].Minier *et al*.[22] reported that muscle AChE of flounder from polluted sites with high level of PAH was inhibited by 40%. Also, a reduction of 40% of brain AChE was observed in Mullus barbatus from three pollutes sites of Salento Apulia (Italy), related with presence of great variety of compounds (PAH, heavy metals and pesticides) present in the sediment [23]. The reduction in swimming performance in fish after exposure to organo-phosphates could be attributed to the inhibition of AChE [24-25]. The present results illustrate that after prolonged exposure to PF induced tissue-specific peroxidative damage in brain, gill, viscera and muscle tissues of O mossambicus and the most affected tissue was gill. Earlier studies show that LPO may be induced in various tissues by a variety of environmental pollutants [26]. Significant depression of cholinesterase activities in brain and liver tissues of *Oreochromis niloticus* following single and multiple exposure of chlorpyrifos (an organophosphate insecticide) and carbosulfan (a carbamate insecticide) in the laboratory was reported by Chandrasekara and Pathiratne [27] and Baby Joseph and Justin Raj [28].

V. CHROMOSOMAL ABERRATIONS AND CARCINOGENIC EFFECTS

Dichlorvos concentration of 0.01 ppm caused chromosomal aberrations in the form of centromeric gaps, chromatid gaps, chromatid breaks, sub-chromatid breaks, attenuation, extra fragments, pycnosis, stubbed arms etc in kidney cells of *Channa punctatus* after exposure periods of 24, 48, 72 and 96 h [29]. Interestingly, there was an inverse relationship between duration of exposure and aberration frequency. Longer exposures to dichlorvos were associated with lower frequencies of aberrations. The toxicity of dichlorvos has also been related to alterations in DNA replication, which causes mutations [30] and cellular hyperproliferation as a result of local irritation [31-33].

VI. EFFECT ON PROTEIN CONTENT

Appreciable decrease in protein level of liver, muscle, intestine, gill and blood of *Heteropneustes fossilis* was noticed after the exposure of fish to nickel for 30, 60 and 90 days [34]. Jha & Jha [35] have observed protein depletion in liver, muscle and gonads of *Anabus testudineus* under the stress of nickel chloride. Decrease in the liver and muscle protein level has been reported in the *Channa punctatus* exposed to phenyl mercuric acetate [36] and also *Channa punctatus* exposed to oleandrin [37]. A similar

decreased liver protein level has also been found in *Mystus vittatus* exposed to nuwan [38], *lepidocephalicthus thermails* exposed to copper [39], *Channa punctatus* exposed to arsenic [40], *Channa straiatus* exposed to mercury, cadmium and lead [41], *Cirrhina mrigala* exposed to lead acetate [42], *Cyprinus carpio* exposed to endosulfan [43], *Labeo rohita* exposed to cypermethrin [44] and *Heteropneustes fossilis* exposed to petroleum oil [45].

VII. EFFECT OF PESTICIDES TO SALMONID FISH

Pesticides are capable of killing salmon and other aquatic life directly and within a short period of time. For example, in 1996 the herbicide acrolein was responsible for the death of approximately 92,000 steel-head, 114 juvenile coho salmon, 19 resident rainbow trout, and thousands of non-game fish in Bear Creek, a tributary of the Rogue River. Deaths of threatened and endangered species from accidental contamination of waterways are of grave concern. The loss of each individual in a sensitive population makes recovery efforts that much more difficult. Fortunately, these deaths are relatively infrequent [46].

In contrast to dramatic fish kills, the effects of sublethal concentrations of pesticides are more subtle and go largely unseen and unregulated. Sublethal concentrations of pesticides do not cause immediate death, but can interfere with the biology of the organism in other ways and can ultimately impact the survival of the species. Laboratory studies show that sublethal concentrations of pesticides can affect many aspects of salmon biology, including a number of behavioral effects:

- Long-term exposure to certain pesticides can increase stress in juvenile salmonids and thereby render them more susceptible to predation.

- Certain pesticides can alter swimming ability, which in turn can reduce the ability to feed, to avoid predators, to defend territories, and to maintain position in the river system.

- Many pesticides interrupt schooling behavior, a critical tactic for avoiding predation, during salmon migration. Disruption of schooling behavior is thought by some researchers to be a classic method for examining sublethal effects of pesticides because the effect is so common.

- Several pesticides (and other pollutants) have been shown to cause fish to seek sub-optimal water temperatures, thus subjecting them to increased dangers of disease and predation.

- Some herbicides have been shown to inhibit normal migration to the sea, resulting in severe disruption of the life cycle. There is a dearth of research looking at this effect for common insecticides.

• Several studies suggest that certain pesticides can impair salmonid's ability to transition from freshwater to seawater. There is a need for further research in this area, placing particular emphasis on the critical period of transition that takes place in the estuary.

• Adult salmon adjust their migration patterns to avoid polluted areas, resulting in delayed spawning.

Effect on Immune Systems & Endocrine Disruptors- Indirect effects

In addition to changes in behavior, exposure to relatively low concentrations of pesticides can disrupt the immune system of salmon. Evidence for these effects in salmonids is not as extensive as for disruption of behavior, but the data available suggest that pesticides can have serious negative impacts on the immune system. Such disruption results in the onset of disease and even death. Fish and other organisms are especially vulnerable to endocrine-disrupting effects during the early stages of development. Pesticides at low concentrations may act as mimics or blockers of sex hormones, causing abnormal sexual development, feminization of males, abnormal sex ratios, and unusual mating behavior. The unique plasticity of sex differentiation in fish suggests that these animals may be very susceptible to disruption of sexual characteristics by pollutants. Pesticides can also interfere with other hormonal processes, such as thyroid functioning and bone development.

Pesticides can indirectly affect fish by interfering with their food supply or altering the aquatic habitat, even when the concentrations are too low to affect the fish directly. Such indirect effects greatly reduce the abundance of food organisms which in turn reduces the growth and probability of survival of the fish. In addition, removal of aquatic vegetation can decrease habitat suitability and increase the salmon's susceptibility to predation. These indirect effects are subtle, but evidence suggests that in complex ecosystems indirect effects can be even more important than direct effects.

VIII. TOXICITY TO CYPRINID AND CAT FISH

Mastan and Shaffi [47] have made an attempt to investigate the sub-lethal effect of Organophosphates on the various enzymes such as phosphate activated glutaminase and L-Keto acid activated glutaminase in the different regions of brain of *Labeo rohita.* They were then exposed to sub-lethal concentrations of dichlorvas, monocrotophos and phosphamidon for acute and chronic studies. In exposed fishes, phosphate glutaminase and X-ketoacid glutaminase registered significant changes in different brain regions under both acute and chronic studies. Differential response of glutaminases to monocrotophos, dichlorvas and phosphamidon in different brain regions in *L. rohita* might be concerned with their involvement in metabolism and degree of response to the above pesticides and vice-versa.

The effects of pesticides on blood characteristics and histological changes in erythrocytes of the fish species *Cyprinus carpio* and *Puntius ticto* were studied by Satyanarayana et al. [48]. The fishes were exposed to sub lethal concentrations of different chlorinated pesticides namely aldrin, dieldrin, DDT, BHC and chlordane for 10, 20 and 30 d in continuous flow-through test. Results showed an increase in haemoglobin content of both *Cyprinus carpio* and *Puntius ticto* in case of aldrin and dieldrin. Haemoglobin content reduced from an initial 13 g/100 ml to 8.07 and 10.15 g/100 ml in case of Cyprinus at the end of ten days exposure to aldrin and dieldrin respectively, and gradually increased to 8.7 g/100 ml and 10.15 g/100 ml after 20 d of exposure. The haemoglobin content after 30 d exposure to aldrin and dieldrin was 10.15 g/100 ml and 11.6 g/100 ml respectively. In case of *Puntius ticto*, the haemoglobin content in control fishes recorded was 12.8 g/100 ml while in case of fish exposed to aldrin, the haemoglobin content reduced initially on ten days exposure to 10.15 g/100 ml and increased to 11.6 g/100 ml and 13.0 g/100 ml during twenty days and thirty days exposure respectively. This trend was also observed with dieldrin in both the fishes studied. Red blood cells were also counted in case of all the pesticides and exposure periods with respect to *Cyprinus carpio* and *Puntius ticto*. Irrespective of the species and pesticide, the RBC counts uniformly showed decreasing trend with the increase in exposure period, while packed cell volume, PCV(%) showed increasing trend with respect to increase in exposure period in case of aldrin and dieldrin in both the fishes. But DDT, BHC and chlordane showed decreasing trend in PCV(%) values with increasing periods of exposure [48].

Geyer et al.[49] provides an explanation for a 40-fold difference in the acute toxicity (LC50) of gamma-hexachlorocyclohexane (gamma-HCH, Lindane) in 14 different fish species, based on well recognized principles of toxicokinetics and toxicodynamics in combination with a compilation of data from the literature and some original data. The 48-h median lethal concentration (48-h LC50) of gamma-HCH in 14 fish species, belonging to 6 families, range from 22 to 900 micrograms/l. A significant positive linear relationship was found between lipid content (% of wet weight) and the 48-h LC50 of gamma-HCH in these fish species, revealing that the toxicity of gamma-HCH in various fish species is decreasing with increasing total lipid content. If median lethal concentrations are normalized for 1% lipid content, then the range of 48-h LC50s is reduced to between 18 and 32 micrograms/l. It is concluded that lipids of aquatic organisms can serve (among other functions) as a protective storage site against the toxic effects of gamma-HCH and, possibly, of other lipophilic, persistent organic chemicals which are bioconcentrated in body lipids. Therefore, in organisms with higher lipid content, a smaller fraction of a lipophilic chemical will reach target organs (liver, lung, central and peripheral nerves, etc.) to cause adverse effects. Results suggest that this correlation can be used to extrapolate the acute toxicity (48-h LC50) of gamma-HCH to other fish species if their lipid content is known.

Toxicity of Fenvalerate a synthetic pyrethroid to some fresh water fish species was evaluated by Satyavardhan [50]. The toxicity is determined by using both static and continuous flow through systems and also noticed some specific behavioral characteristics during the experimentation periods in different time intervals. The LC_{50} values were calculated for 24h, 48h, 72h and 96h respectively. When the fish were exposed to sub lethal and lethal concentration of Fenvalerate , technical grade and 20% EC., they were migrated to the bottom of the test chamber immediately. This is because of the toxic stress. Their Schooling behavior was totally disturbed and they are swimming independently and this was followed by irregular, erratic and dangling movements with the imbalanced swimming activity. The swimming behavior was in a cork screw pattern and rotating along horizontal axis and followed by "S" jerks, sudden, rapid and non-directed sport of forward movement likely to be busted swimming. The fish were exhibited peculiar behavior that is the fish were trying to leap out from the test chamber which can be viewed as escape phenomenon. Respiratory disruption was observed due to cough and yawning this is because of toxic stress. They often barrel rolled or spiraled at regular intervals and engulfed the air through mouth before respiration ceased. A change in color of the gill lamellae from reddish to light brown with coagulation of excess mucous on the gill lamellae was observed. The symptoms of Fenvalerate poisoning in the fish include loss of schooling behavior, swimming near the upper surface, hyper activity, zigzag movement, loss of buoyancy, elevated cough , increased gill mucous secretion, flaring of the gill arches, head shaking and restlessness before death.

According to Ramachandra Mohan [51], lower dosage of Malathion brings about a reduction in the ovarian weight and retard the growth of the pre-vitellogenic oocytes. A higher dosage of Malathion on the other hand results in the degeneration of the immature oocytes and rupture of follicular epithelium. The findings suggest that the histopathological changes in the ovary might be a reflection of the disturbance in the endocrine/hormonal imbalance.

During an investigation by Chatterjee *et al.* [52], where *Heteropneustes fossilis* were exposed to sub-lethal doses (0.5, 1, and 2 mg/L) of carbofuran (CF) for 30 days at 25 ± 1°C, it was observed that CF altered both the area and the percentage occurrence of the various types of primary oocytes in the ovary compared to that of the control fish. The degeneration of follicular walls, connective tissues and vacuolization in the ooplasm of the stage II and III oocytes were observed in CF-treated fish (0.5–2 mg/ L). It appears that CF at sub- lethal concentrations inhibits oocyte maturational processes in catfish.

Dutta *et al.* [53], observed effects of a sub-lethal (1.2 mg 1^{-1}) organophosphate, Malathion, in the ovary of breathing catfish, *Heteropneustes fossilis*. He noticed microscopic changes that occured on ovigerous lamellae. He observed Clumping of cytoplasm, Degeneration in the follicular cells, shrinkage of nuclear material, increased atretic oocytes, along with ruptured follicular epithelium.

Dutta *et al*. [54], studied the effects of the insecticide, diazinon (an organophosphorous compound), on the ovaries of bluegill (*Lepomis macrohirus*). He noticed adhesion of primary follicles, cytoplasmic retraction in oocyte II, cytoplasmic degeneration, increased atretic oocytes, damages to the oocyte IV, Partial destruction of the ovigerous lamellae and vitellogenic membrane destruction of follicles, Severe damage of the ovigerous lamellae, increased intrafollicular spaces, vacuolated cytoplasm, extrusion of karyoplasm and necrosis in the cytoplasm.

Sanjoy Deka and Rita Mahanta [11] carryout an empirical study to investigate the effect of sub-lethal Malathion on liver, kidney and ovary of the freshwater catfish " *Heteropneustes fossilis.".* LC_{50} value of Malathion was calculated by probit analysis [55] and LC_{50} for 96 hours is found to be 0.98 ppm. Sub-lethal concentration of 0.2 ppm is prepared by using standard technique [56]. For this study, Control Group was being freed from the treatment of Malathion where as experimental group was treated with sub-lethal Malathion concentration of 0.2 ppm. After 10days of exposure to Malathion, primary follicles began to show adhesion and as well as cytoplasmic retraction in oocyte occured. Cytoplasmic degeneration and the number of atretic oocytes increased. Damages to the oocyte started to occur. Cytoplasmic retraction and clumping was more visible in oocyte. Partial destruction of the ovigerous lamellae and vitellogenic membrane occurred. So In 10 days-treated ovary,overall deformed tissue was observed in the Section of the ovary of *Heteropneustes fossilis.*

The toxicity bioassay test of commercial grade endosulfan (35% EC) was conducted by Tripathi and Verma [57]. They reported sublethal concentration of endosulfan decreased the activity of citrate synthase (CS) and glucose 6-phosphate dehydrogenase (G6-PDH) in the brain, liver and skeletal muscle of the freshwater catfish, C. batrachus. The brain lactate dehydrogenase (LDH) activity was also reduced in response to endosulfan toxicity. The maximum reduction in activities of these enzyme was 34%-43%. Withdrawal of endosulfan restored the enzyme activity to control level in all the three tissues. The recovery in enzyme activity appears to be due to dissociation of endosulfan or its metabolite(s) from the enzyme molecules and/or fresh synthesis of enzymes. The treatment of actinomycin D or cycloheximide partially inhibited the withdrawal-dependent increase in enzyme activity. This substantiates de novo synthesis of enzyme during recovery period. Since the reduction in enzyme activity was more pronounced in response to actinomycin D, endosulfan might be inhibiting the transcription process. But endosulfan did not produce any significant effect on DNA content and RNA/DNA. However, the RNA and protein contents of brain, liver and skeletal muscle decreased significantly in tissues. The maximum decrease in RNA and protein was approximately 30%-37%. Withdrawal of endosulfan from the medium for 21 days restored the RNA, and protein contents nearly to their control levels. The treatment of actinomycin D or cycloheximide partially inhibited the withdrawal-dependent increase in these macromolecular contents.

This effect was more pronounced in case of actinomycin D which again supports the possibility of endosulfan-induced inhibition at transcription level. Their study suggested endosulfan-induced impairment of metabolism in fish, which appeared to be due to inhibition of transcription at some unknown points.

Ilavazhahan et al. [10] was taken experiment in test dose of 1ppm to 10ppm to study the effect of methyl parathion toxicity in the fingerlings of *Catla catla.* The fish exposed to this toxicant were severely affected. Increase in opercula movement, loss of equilibrium, frequent surfacing, changes in body colour, increased secretion of mucus, irregular swimming activity, rapid jerk movement were observed. No mortality was recorded at 1ppm concentration of the pesticide. Fish survived through 48hr up to toxic concentration of 3ppm and 50% mortality was recorded at 4.8ppm of concentration. The mortality recorded was 80% and 100% at 8 and 10ppm concentration respectively. 96hr LC_{50} of pesticide methyl parathion was noted as 4.8 ppm.

The effects of sublethal concentrations of the organophosphate pesticide Dichlorvos (Neon) (0.65 mg/l, 0.90 mg/l and 1.17 mg/l) on the gonadosomatic index of the fish, *Cyprinus carpio communis* was studied. The Ganadosomatic index (GSI) decreased with the increase in concentration, whereas it increased with increase in exposure at all concentrations. It may also be noted that the reduction in GSI values was maximum at highest concentrations of the pesticide in series. Ovaries of the Dichlorvos treated fish showed histomorphological disorders. Furthermore, the reduced GSI was found directly proportional to the pesticide concentration and duration of exposure [58].

Christopher Didigwu Nwani et al.[59] was undertaken study to evaluate the lethal toxicity and stress of commercial formulations of carbosulfan (Aatank) insecticide, glyphosate (Roundup) and atrazine (Rasayanzine) herbicides toward freshwater air-breathing fish *Channa punctatus* (Bloch). The 96 h LC_{50} values, determined in a semi-static system by probit analysis as 0.268, 32.540 and 42.380 mg/l for carbosulfan, glyphosate and atrazine, respectively, indicated that the fish were more sensitive to carbosulfan than the other two herbicides. There were large variations in the safe levels estimated by different methods for the pesticides. In addition to dose and dose-time dependent increase in mortality rate, stress signs in the form of behavioral changes were observed in response to the test pesticides.

Marigoudar et al.[9] conducted bioassay test to determine the acute toxicity (LC_{50}) of technical grade pyrethroid insecticide, cypermethrin (92.25%) in freshwater indigenous carp, *Labeo rohita.* Carp fingerlings were exposed to different concentrations (2.0 to 6.0 µg/L) of cypermethrin for 96 h. The acute toxicity value was found to be 4.0 µg/L and one fifth of LC_{50} (0.57 µg/L) was selected for sub acute studies. Behavioural patterns and oxygen consumption were studied in lethal (1, 2, 3 and 4 d) and sub lethal concentrations (1, 5, 10 and 15 d). Carp in toxic media exhibited irregular, erratic and darting

swimming movements, hyper excitability, loss of equilibrium and sinking to the bottom, which might be due to inactivation of (AChE) acetyl cholinesterase activity which results in excess accumulation of acetylcholine in the cholinergic synapses leading to hyperstimulation. Variation in oxygen consumption (1.289 to 17.409 %; 20.580 to 109.77 %) was observed in both lethal and sub lethal concentrations of cypermethrin respectively. Alterations in oxygen consumption may be due to respiratory distress as a consequence of impairment in oxidative metabolism. Fish in the sublethal concentration were found under stress, but that was not fatal.

Vishal Rajput et al.[60] reported that, Imidacloprid and sodium fluoride were highly toxic but at lethal dose because remarkable protein loss was reported at lethal concentration but at sub-lethal level their toxicity was moderate. But Butachlor caused remarkable protein loss at lethal as well as sub-lethal concentration.

Thenmozhi et al.[15] used malathion for *in vivo* study on fresh water fish *Labeo rohita* to know its toxicity. The acute toxicity tests were conducted during certain intervals in various concentrations (5, 10, 15, 20, 25 and 30 mg/L) of malathion. While treating with malathion, the percentage of fish mortality was assessed during 24, 48, 72 and 96 hours. The lethal and sub-lethal concentration of malathion were found to be LC_{100} (25 mg/L) and LC_0 (5 mg/L), respectively. The antioxidant enzyme activity (Catalase 43.1±2.3, 16.5±0.57, 23.9±0.17 µ moles of phenol liberated/min/100mg protein and Glutathion-S-transferase (GST) 270.5±0.16, 143.2±1.03, 215.5±0.72 µ moles of phenol liberated/min/100mg protein), in the liver, muscle and gill, respectively increased during the accumulation of malathion, whereas it decreased (Catalase 17±1.44, 7.9±0.23, 10.7±0.69 µ moles of phenol liberated/min/100mg protein and GST 219.5±1.12, 108.1±0.34, 160.2±0.46 µ moles of phenol liberated/min/100mg protein) in the liver, muscle and gill respectively during depuration period. The effects of malathion resulted in the gradual decrease of nucleic acids, protein, free amino acids (FAA) and glycogen. During recovery period, the levels of biochemical components progressively increased indicating a probable recovery from the disruption of internal organ. Hence, the pesticide intoxication has made defective consequences in the normal metabolic pathways which led increasing the rate of morality in fish population.

Ganeshwade [61] exposed freshwater fish *Puntius ticto* to lethal (5.012 ppm) and sublethal (2.50 ppm and 1.253 ppm) concentration of Dimethoate for 96 hrs and 60 days durations. The protein level decreased in lethal and sublethal exposures. Significant decrease in glycogen, slight decrease in protein where as increased cholesterol and ascorbic acid content has been noted to both concentrations exposure.

IX. EFFECT ON OTHER FISHES

The toxicity of the carbamate insecticide carbaryl and its metabolite, 1-naphthol, to four species of fish was studied by Tilak et al. [62]. The calculated 96 h LC 50 values of carbaryl for *Catla catla* (Ham.), *Anabas testudineus* (Bloch), *Mystus cavasius* (Ham.) and *Mystus vittatus* (Bloch) are 6.4, 5.5, 4.6 and 2.4 ppm respectively and that of 1-naphthol are 4.3,3, 0.33 and 1.1 ppm respectively. The degradation product of the insecticide was found to be more toxic than the parent compound, to all the four fish species.

Pesticides that leach into aquatic habitats may influence various physiological processes that may impact upon the larvivorous potential of fishes. John Ravindran et al. [63] observed on the effect of 0.001 and 0.01 mg/l of the insecticide, Hostathion (Triazophos) and 0.5 and 1.0 mg/l of the fungicide, Kitazin (Iprobenfos) on the larvivorous potential of *Oryzias carnaticus*, an indigenous larvivorous fish in India. The mean consumption by fishes were 3.1 and 4.1 times lower than control and was lowest on Day 2 and Day 1 in Hostathion treated solution. It was 4.6 and 4.8 times lower than control and consumption was lowest on Day 1 at both concentrations in Kitazin treated solutions. Mean consumption did not reach the level of control during the four day exposure period. Their results indicated larvivorous potential to be affected when fishes were exposed to sub-lethal concentrations of both studied pesticides.

Organophosphorous pesticides are frequently used against pest because of their high insecticidal property low mammalian toxicity, less persistence and rapid biodegradability [14]. These also affect non-target organisms either directly or indirectly. In rice land agroecosystem, all organisms including larvivorous fishes can be affected [64]. At lower concentrations, physiological functions including the larvivorous potential are affected and results of the present study indicate the same. Indiscriminate and prolonged use may lead to mortality and depletion of the fish population [63]. Re-establishment of fish population takes a longer duration when compared to the mosquito population. Since vector mosquito control using larvivorous fishes is considered to be an environmental friendly and safe alternative to insecticides [65], judicious use of pesticides preserving the natural habitat of these fishes is important. Integrated pest management, biological and genetical control for agricultural pests will help in the preservation of the biotic communities in the rice land ecosystem. Further, there is a need to monitor ecotoxicity whenever larvivorous fishes are used as a biocontrol agent in vector mosquito control programmes.

Shailendra Kumar Singh et al [13] has reported toxicological and biochemical alterations of Cypermethrin (Synthetic Pyrethroids) against freshwater Teleost fish *Colisa fasciatus* at different Season. Their result has shows strong piscicidal activity in freshwater teleost fish *Colisa fasciatus* for all the exposure periods (24h or 96h) in time as well as dose dependent manner. The LC_{50} values decreases

from 0.009 (24h) to 0.006 (96h) in winter season (water temp. 16°C) and 0.06 (24h) to 0.02 (96h) in summer season (water temp. 28°C). Sub-lethal doses (40 and 60% of LC_{50}) of cypermethrin after 96h was also significantly alter the levels of total protein, total free amino acid, in muscle and liver tissues, nucleic acids (DNA and RNA) in gonadal tissues and the activity of enzyme acetylcholinesterase (AChE), lactic dehydrogenase (LDH) and succinic dehydrogenase (SDH) in nervous tissue of the freshwater teleost fish *C. fasciatus* in time and dose dependent manner.

Impact of sub lethal concentrations dimethoate on oxygen consumption of *Tilapia mossambica* was reported Shereena et al.[66]. The rate of oxygen consumption of the fish was studied under the concentrations of 0.15ppm, 0.2ppm, 0.3ppm and 0.6ppm under 1/20, 1/15, 1/10, and 1/5 of sub lethal concentration respectively during the 24, 48,72 and 96 hours of exposure. The initial elevation in the oxygen consumption could be explained in terms of acceleration of oxidative metabolism during the initial hours of exposure, as a result of sudden response to the toxic stimulus of pesticide. Their study reveals that the dimethoate is toxic to the fish, *Tilapia mossambica* and even low concentration of dimethoate stress create respiratory disturbance which ultimately leads to the deterioration of general health of the fish.

Sucharita Bose et al.[67] described the effect of Thiamethoxam on the growth and liver total protein of this exotic fish *Oreochromis niloticus*. Their results revealed that various sublethal doses of Thiamethoxam had significant impact on growth and liver total protein of this fish. The weight, length and breadth showed a decreasing trend in all doses of pesticide though there was a variation in protein level at different dosed fish. That was probably to cope up with the increasing doses so that the fish could overcome the toxic effect of Thiamethoxam to make a balance between growth and metabolism. This was in conformity with the effects of thiodon pesticides on *Clarias gariepinus* [68].

Lakshmanan et al. [69] was designed to assess the impact of Dichlorvos on tissue glycogen, total protein and albumen content in the selected tissues of *Oreochromis mossambicus*. In their study, when *O. mossambicus* is treated with sub lethal doses of Dichlorvos for all the exposure periods, it shows a significant decrease in the liver, kidney and muscle protein content and it is suggested that depletion of tissue total proteins after 7 days exposure period may be due to increased proteolysis thereby contributing to the availability of free amino acids that may be fed to the tricarboxylic acid (TCA) cycle and further possible utilization of its products for metabolic process. Several workers have observed the decrease in protein content in liver when organisms were subjected to pesticide treatments. But their investigation provides the first report on the effect of Dichlorvos in fish fingerlings of *O. mossambicus.*

X. IMPACT OF WATER POLLUTION ON HEALTH OF FISH & SHELLFISH

As there is little evidence of pollution affecting the health of fish and shellfish on a global scale. Although there is no dispute that pollution can affect the aquatic organisms under laboratory conditions and may be responsible for the decline of populations of such animals in some inland waters and some estuaries, most of the evidence for pollution causing or increasing disease in fish in open waters is circumstantial. Historical data proves that almost all fish and shellfish diseases known today have been described since the end of the last century. However, it is also known that water pollution, especially in inland waters, has for the past 400-500 years been the result of urbanization and industrialization. This has resulted in some major rivers becoming devoid of or deficient in fish stocks. The concern that pollution may influence the health status of fish and shellfish stocks has increased over the past 20 years. Initial attention was paid to epidermal diseases, including fin-rot in demersal fish, and protozoan diseases in molluscs in the heavily polluted bays and estuaries in North America. As the interest in this subject spread, it became political, and often controversial, especially amongst the North Sea countries. The disagreements have largely been settled amongst scientists because international bodies, such as the International Council for Exploration of the Sea (ICES), established workshops to investigate sampling methods and disease-reporting techniques. As there are variable, interacting biological and physical influences in the aquatic environment, it is difficult to establish the background prevalence's of diseases in populations of fish and shellfish. Examples of the influences of climatic changes are presented, and these show that short-term catastrophes can be directly related. However, a more long-term problem is water acidification resulting largely from anthropogenic activities. In parts of Scandinavia this has, and is, leading to decimation of fish stocks in inland waters. In general, diseases in fish and shellfish are very localized, but there is concern amongst scientists that certain cancers, especially liver tumours, occurring in demersal fish inhabiting polluted estuarine and coastal waters, are related to the release of chemicals, e.g. hydrocarbons, pesticides and heavy metals [70].

Potentially harmful substances-e.g. pesticides, heavy metals and hydrocarbons-are often released into the aquatic environment. When large quantities of pollutants are released there may be an immediate impact as measured by large-scale sudden mortalities of aquatic organisms, e.g. fish kills resulting from contamination of waterways with agricultural pesticides. Lower levels of discharge may result in an accumulation of the pollutants in aquatic organisms. The end results, which may occur long after the pollutants have passed through the environment, include immunosuppression, reduced metabolism, and damage to gills and epithelia. However, the link between adverse water quality and fish diseases is not

proven. Many surveys have indicated a greater proportion of diseased fish in polluted compared to non-polluted sites. Yet, the value of such surveys may be questioned [71].

XI. CONCLUSION

Long term exposure of organisms to pesticides means a continuous health hazard for the population. So, human population is at high risk by consuming these toxicated fishes. This implies that one should take the necessary precaution in the application of pesticides to protect the life of fish and other aquatic fauna. It is likely that approaches using molecular biology techniques will revolutionize toxicological applications that are cheaper and do not require the use of animals to detect environmental stressors. Pesticide toxicity in fish has been studied by several workers who have shown that at chronic level, it causes diverse effects including oxidative damage, inhibition of AchE activity, histopathological changes as well as developmental changes, mutagenesis and carcinogenicity. With reports of toxicants usage and its adverse effects on non-target organisms like fish, it has become essential to formulate stringent rules against indiscriminate use of this pesticide. Since pesticide is present in the environment with other similar organophosphate compounds, additive responses to organophosphate compounds may induce lethal or sublethal effects in fish. It is, therefore, a matter of great public health significance to regularly monitor the pesticide residues in foods and humans in order to assess the population exposure to this pesticide. Besides, for a safe use of this pesticides more experimental work should be performed to determine the concentration and time of exposure that do not induce significant sub-lethal effects on fish.

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