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Genomic & biochemical changes in fishes due to pesticide pollution

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Abstract: *The objective of the present review paper is to study the various effects of pesticide pollution on freshwater fishes like Labeorohita and conclude about what to do as solution. Pesticides are used extensively in crop fields in present days. The focus of the following paper is the changes occurring at genomic and biochemical level in fishes. Usually, biochemical changes are observed in total protein content and/or total carbohydrate content.Lipids are also affected due to pesticide pollution. Genomic changes are nominal, although nucleic acids of two types, namely DNA and RNA, are affected in large scale.*

Keywords: *Pesticides, Chemicals, Fish, Protein, Carbohydrate, Nucleic acid, DNA, RNA*

I. Introduction

Chemical pollution in the environment by pesticides hasbeen increasing due to their extensive usage in agriculture.Alterations in the chemical composition of natural aquaticenvironments can affect the freshwater fauna, particularly fish. Many of these compounds or their metabolites haveshown toxic effects related to oxidative stress [Winston and et al., 1991].The aquatic environment is subject to an always growing range of man-made pollutants, reflecting the ever increasingrapid innovations of our technology to manufacture goods. These materials are used as tool to gratify a perceived increase in consumer demand on which our economy is based.The natural physiological functioning of an organism gets disturbed on exposure to toxicant stress. Toxicants affect first at cellular or at molecular level, but ultimately causes physiological, pathological and biochemical alterations.Pesticides are one of the most potentially harmful chemicals introduced into the environment. The contamination of surface waters by pesticides used in agriculture is a problem of worldwide importance.[Rathod et al., 2010].Most of the chemicals that are used as pesticides are generally toxic to many non-target species, including man, and other desirable forms of life that coinhabit the environment. [Jagadeesan et al., 2012].The production of pesticides started in India in 1952 with the establishment of a plant for the productionof BHC near Calcutta, and India is now the second largest manufacturer of pesticides in Asia after Chinaand ranks twelfth globally. [Mathur, 1999] The following is a list of 24 pesticides registered and used in India, classified as Potential Carcinogens by the US EPA:Acephate , Alachlor , Atrazine , Benomyl , Bifenthrin , Captan, Chlorothalonil, Cypermethrin, Dichlorvos, Diclofop-Methyl, Dicofol, Mancozeb, Methomyl, Metolachlor, Oxadiazon , Oxyflourfen , Permethrin , Phosphamidon , Propiconazole , Propoxur , Thiodicarb , Thiophanate Methyl , Triadimefon, Trifluralin.

Fish are particularly sensitive to pesticides and other toxic pollutants because they are able to uptake and retain the dissolved xenobiotic in water via active or passive processes[Bhuvaneshwari et al., 2013]. As a result, external changes, as well as internal changes occur, most of which lead to numerous deformities. Internal changes are mostly at biochemical and genomic level. Toxicological effects specifically target nucleic content and metabolic proteins.The magnitude of pesticide pollution was studied in the Indian fishes by various workers [Dalela*et al*., 1979; Dubale and Shah, 1984; Pandey and Shukla, 1980; Rashatwar and Ilyas, 1984; Sadhu and Mukhopadhyay, 1985; Shukla and Pandey, 1985, Ghosh and Chatterjee, 1989; Medda*et al.*, 1995, Bhattacharya *et al*., 1997; Munshi*et al.*, 1999, Rakesh*et al*., 2009, Naveed*et al.*,2010].Environmental contamination by pesticides may causephysiological and behavioral changes in fish and also affectfunctions such as reproduction and metabolism [Oruc- andUner, 1999; Bretaud et al., 2000].

II. Objectives

i) To find outthe changes that are observed at genomic level of fishes due to pesticide pollution
ii) To identify the biochemical properties of fishes that are affected by the pesticide pollution

III. Discussion

Genomic changes

The primary objective of the genetic characterization is to assessthe distribution and pattern of genetic variability at intra as well as inter-specific level populations,through the use of identified genetic markers.Nucleic acid content is considered as an index of capacity of an organism for protein synthesis. Nucleic acid content, i.e., DNA and RNA show a drastic change after being exposed to pesticide-dissolved water. Dichlorvos concentration of 0.01 ppm caused chromosomal aberrations in the form of centromericgaps, 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 [Rishi et al., 1995].Significant decrease in RNA and DNAcontent in *Clarias batrachus* exposed to endosulfan was recorded[Parveen et al., 1986].But, decrease in DNA content is rather obscure than that of RNA.The decrease may be attributed to the increased activity of DNAase[Tayyabaet al., 1981].Quinolphos induced significant decreases in RNA content of liver muscle and gill and DNA content of brain of fish*Oreochromismossambicus*was observed [Durairaj et al., 1992]. The toxicity of dichlorvos has also been related to alterations in DNA replication, which causes mutations [Gilot-Delhalle et al., 1983].The degradation of RNA level suggestsincreased proteolysis and possible utilization of the products of their degeneration for metabolic purposes. The significantdecrease in both protein and nucleic acids would suggest that pollutant harm the process of protein synthesis in the tissues of fishes exposed to pesticides. Exposure to genotoxic compounds could induce DNAdamage not only directly but also through othermechanisms, such as oxidative stress or inflammatory processes [Lebailly*et al.*, 1998].

Table 1. Fish germplasm resources in India

Ecosystem	**Total species**
Cold water	157
Warm water	608
Brackish water	113
Marine	1365
Total	2243

[Lakra et al., 2008]

Table 2. Characteristics of some commonly used insecticides along with their relative toxicity to fish.

Insecticide	Relative run-off potential	Relative leaching potential	Half life in days	Relative toxicity to fish[1]
Hydrdamethinon (Amdro®)	large	small	10	high
Diazinon	medium	large	30	high
Chlorpurifos (Durisban®)	large	small	30	very high
Malathion	small	small	1	very high
Acephate (Orthene®)	small	small	3	very low
Carbaryl (Sevin®)	medium	small	10	medium
Dimehoate (Cygon®)	small	medium	7	medium
Trichlorfon (Dylox®)	small	large	27	high
Dicofol (Kethane®)	large	small	60	high
Propargite (Omite®)	large	small	56	high

[1]Fish Toxicity based on catfish and bluegill. LC_{50} categories are rated as follows: very low = more than 100 mg/L, low = 10 to 100 mg/L, medium = 1 to 10 mg/L, high = 0.1 to 1 mg/L, very high = less than 0.1 mg/L. ®commercial name

[Agrawal et al., 2010]

The DNA content in control fish *Labeorohita*indifferent tissues are in the order of: Kidney > Liver > Brain > Muscle Under exposure to sublethal and lethalconcentrations of quinalphos technical grade and 25% EC the DNA content in liver and kidney increased but was found todecrease in brain and muscle. The decreasing order of DNA content in different tissues is in the order of: Technicalsublethal: Kidney > Liver > Brain > Muscle, Technical lethal: Kidney > Liver > Brain > Muscle, 25% EC sublethal:Kidney > Liver > Brain > Muscle, 25% EC lethal: Kidney > Liver > Brain > Muscle.

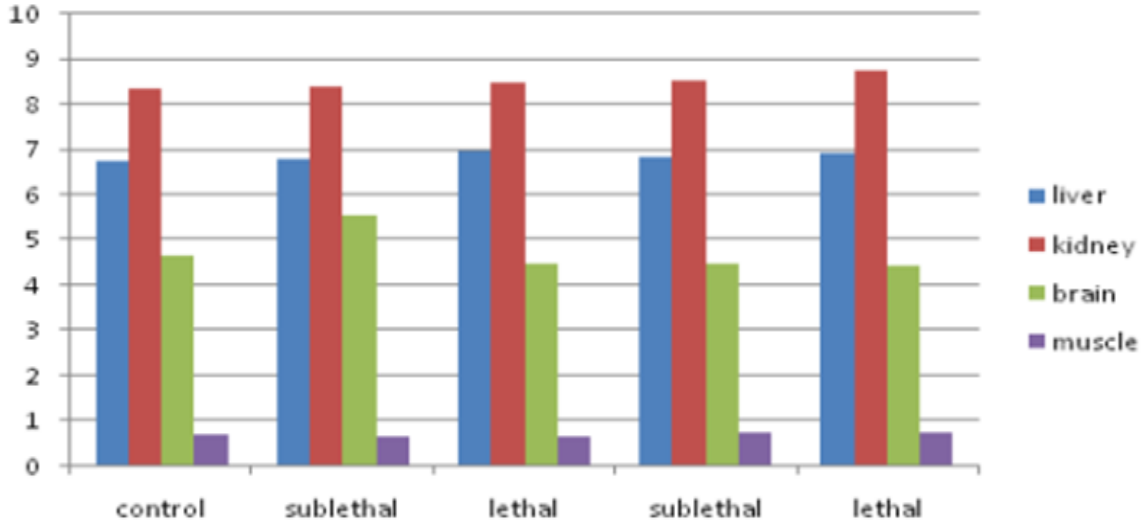

Figure 1: Change in the amount of DNA (mg/gr Body Wet Weight of the Tissue) and % Change over the Control inDifferent Tissues of Fish *Labeorohita*Exposed to Sublethal and Lethal Concentrations of Quinalphos [Rathnamma et al., 2013]

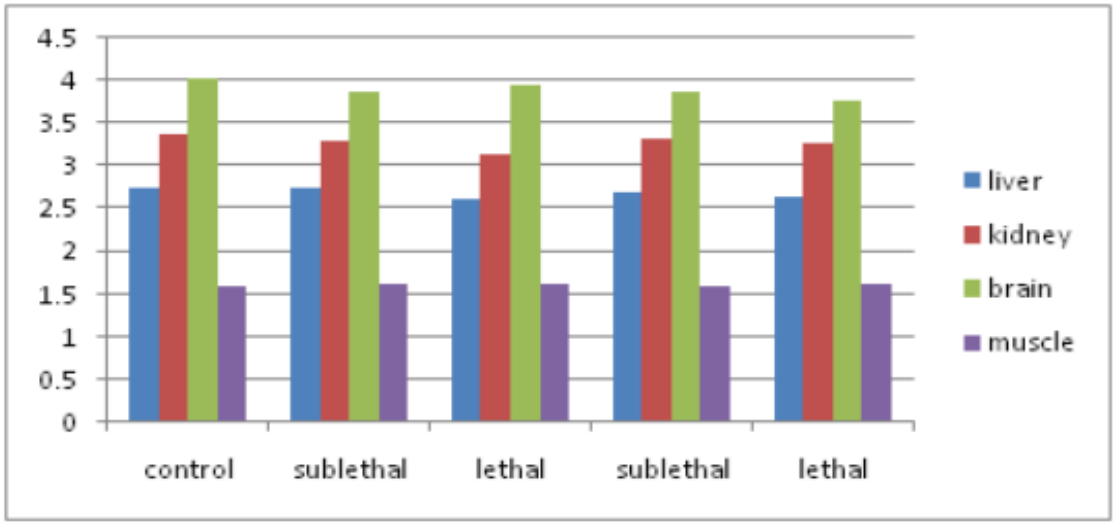

Figure 2: Change in the Amount of RNA (mg/gr Body Wet Weight of the Tissue) and % Change over the Control inDifferent Tissues of Fish *Labeorohita*Exposed to Sublethal and Lethal Concentrations of Quinalphos[Rathnamma et al., 2013]

The RNA content in control fish *Labeorohita*in different tissues are in the order of: Kidney > Liver > Brain >Muscle. Under exposure to sublethal and lethal concentrations of quinalphos technical grade and 25% EC it was found thatThe RNA content in control fish *Labeorohita*in different tissues are in the order of: Kidney > Liver > Brain >the liver, kidney and muscle RNA content was decreased but the brain RNA content was found to increase. The decreasingorder of RNA content in different tissues is in the order of: Technical sublethal: Kidney > Liver >Brain > Muscle,Technical lethal: Kidney > Liver > Brain > Muscle, 25%EC sublethal: Kidney > Liver > Gill > Brain > Muscle, 25% EClethal**:** Kidney > Liver >Brain > Muscle, The results indicate heterogeneous levels of DNA and RNA in the tissues ofbrain, liver, muscle, and kidney. Zeljezic et al., (2001) studied that the mixtureof pesticides such as atrazine, alachlor,cyanazine, 2,4-dichlorophenoxyacetic acid and malathioncausedincrease in the level of DNA damage. Freeradicals produced by oxidative processes can attackDNA at bases or sugars, causing primarily single strandbreaks, as well as, secondary double strand breaks [Ferri*et al.*, 1994; Sarker*et al.*, 1995; Spencer *et al.*, 1996].

Table 3. LC50 values of fishes after exposure to pesticides

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]

[Murthy et al., 2013]

Biochemical changes

Biochemical parameters show a significant decrease after exposure to pesticides and effluents.

Appreciable decrease in protein level of liver, muscle, intestine, gill and blood of *Heteropneustesfossilis*was noticed after the exposure of fish to nickel for 30, 60 and 90 days [Nanda et al., 2000].

In *Catlacatla*, total protein concentration experiences a decrease after treatment with insecticide profenofos [Jagdeesan et al.,2012]. In addition, Holbrook (1980) stated that the toxicant can directly stop protein synthesis.

Decrease in the liver and muscle protein level has been reported in *Cyprinuscarpio* exposed to endosulfan [Jenkins et al., 2003].

Total carbohydrate content is reduced as toxic water as pesticides like 2,4-Dichlorophenoxyacetic acid or 2,4-D may induce oxidative stress leading to generation of free radicals and cause lipid peroxidation as molecular mechanisms involved in pesticide-induced toxicity in the fish *Channastriatus* [Anushiya et al., 2013].Decreased levels of total free sugar were reported in all theregions of brain except cerebral hemisphere of *Labeorohita*and in the medulla oblongata and in the spinal cord of *Labeo* and *Cyprinus*community when exposed to concentration of phosalone [Ravi, 1984].

Oxidative stress is defined as a situation when steady-state reactive oxygen species (ROS) concentration is transiently or chronically enhanced, disturbing cellular metabolism and its regulation and damaging cellular constituents [Lushchak, 2011].Pesticides may induce oxidative stress leading to thegeneration of free radicals and cause lipid peroxidation as molecular mechanisms involved in pesticide-inducedtoxicity[Agrawal et al., 1991; Khrer, 1993].A sudden increase in lipid peroxidation and in turn oxidative stress results in degradation of enzymatic antioxidants. Enzymes like glutathione peroxidase, catalase, glutathione transferase, etc, have significant antioxidant properties. They show a level down after treatment with pesticide-dissolved water.

Maruthanayagam et al. (2004) studied the effect of monocrotophos on *Cyprinuscarpio*to understand the toxic effects of toxicant on the nucleic acids. The conclusion was that, the pesticide lead to several changes in the biochemical markers like DNA and RNA which may be due to the enhanced activity of the enzyme DNAase and the inhibition of RNA polymerase function. But during recovery period, the DNA andRNA levels increased progressively indicating a probable form of the disruption of internal organs.

Lakshmanan et al. (2013) designed an experiment to assess the impact of Dichlorvos on tissue glycogen, totalprotein and albumen content in the selected tissues of *Oreochromismossambicus*. In their study, when*O. Mossambicus*is treated with sub lethal doses of Dichlorvos for all the exposure periods, it shows

asignificant decrease in the liver, kidney and muscle protein content and it is suggested that depletion oftissue total proteins after 7 days exposure period may be due to increased proteolysis thereby contributingto the availability of free amino acids that may be fed to the tricarboxylic acid (TCA) cycle and furtherpossible utilization of its products for metabolic process. Several workers have observed the decrease inprotein content in liver when organisms were subjected to pesticide treatments. But their investigationprovides the first report on the effect of Dichlorvos in fish fingerlings of *O. mossambicus.*

IV. 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 takethe 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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