A Review of Dichlorvos Toxicity in Fish

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DOI : http://dx.doi.org/10.12944/CWE.8.1.08

(Received: February 08, 2013; Accepted: February 23, 2013)

ABSTRACT

Among the wide majority of pesticides, dichlorvos (2, 2-dichlorovinyl dimethyl phosphate), an organophosphate compound, is commonly used as agricultural insecticide. It is extremely toxic to non-target organisms like fish and hampers fish health through impairment of metabolism, sometimes leading to death. As one of the few organophosphates still registered for use, dichlorvos has elicited worldwide concern for many reasons. This study is a review of potential adverse effects of dichlorvos in fish.

Key words: Dichlorvos, Toxic, Acute, Chronic, Fish.

INTRODUCTION

Use of pesticide has become a necessary evil for developing countries like India where it is estimated that approximately 30% of its crop yield valued at Rs.60,000 crores are lost due to pest attack each year1. Amongst others, organophosphorus pesticides (OPs) are the most commonly used pesticides in the world due to their quick degradation2. Unfortunately, OPs lack target specificity and can cause severe, long lasting population effects on terrestrial and aquatic non-target species, particularly vertebrates3. Quick degradation is probably the reason why, irrespective of reports of health hazardous, developing countries especially, in the Asia-Pacific region, use these chemicals for agricultural and public health purposes 4-5.

Dichlorvos (2, 2-dichlorovinyl dimethyl phosphate) was first introduced in 19616. It has a molecular formula C_4H_7Cl_2O_4P and molecular weight to be 220.98 (Fig 1). It is also known by its trade name DDVP, Dedevap, Nogos, Nuvan, phosvit or Vapona7. It is one of the most commonly used organophosphate pesticides in developing countries8. It is classified by the WHO as a Class IB, ‘highly hazardous’chemical9. Dichlorvos is usually used as an agricultural insecticide on crops and stored products but is also used as an anti-helminthic (worming agent) for dogs, swine, and horses, as a botacide; agent that kills fly larvae10. It is poisonous if swallowed, inhaled, or absorbed through the skin11. It is extremely toxic pesticides to aquatic organisms and hampers fish health through impairment of metabolism sometimes leading to death. As one of the few organophosphates still registered for use, dichlorvos has elicited worldwide concern for many reasons. Although dichlorvos serves as a contact and stomach insecticide for food and non-food crop pests, it is also toxic to fish and other aquatic organisms12-14. Dichlorvos is also commonly used in fish farming to eradicate crustacean ectoparasites. It is specially used in the treatment of sea lice (Lepophtheirus salmonis and Caligus elogatus) on commercial salmon farms. But this pesticide often ends up producing both lethal and sub-lethal effects on the fish15 and even zooplanktons16. At only 1 ppm, dichlorvos, showed both acute and chronic toxicity in fish17. Some other workers have also noted adverse effects of dichlorvos in fish18-20. The present study is an attempt to review the potential adverse effects of dichlorvos in fish.
Acute toxicity of dichlorvos on fish

The acute toxicity of dichlorvos to fish has been previously determined by a number of researchers. Its toxicity for freshwater and estuarine fish is moderate to high, and it does not bioaccumulate in fish\(^{21}\). For freshwater and estuarine fish, 96-h\( \text{LC}_{50} \) values range from 0.2 to 12 mg/L\(^{22}\). For marine fish, the toxicity was estimated to be more than 4 mg/L for adults and pre-adults of Atlantic salmon (Salmo salar)\(^{23}\). The 96-h\( \text{LC}_{50} \) value of dichlorvos obtained for fingerlings of European sea bass (Dicentrarchus labrax) was 3.5 mg/L\(^{15}\). A comparison of the 96-h\( \text{LC}_{50} \) values published for several teleost fish species\(^{22}\) indicates that fingerlings of the European sea bass are more resistant to dichlorvos exposure than the most part of the other species of estuarine and freshwater fish studied. However, the comparison with fathead minnow (Pimephales promelas) or with mosquito fish (Gambusia affinis) of similar size indicates that sea bass fingerlings are more sensitive to dichlorvos, since 96-h\( \text{LC}_{50} \) values of 12 and 5.3 mg/L have been reported\(^{22}\) for both species, respectively. In a study, it was found that 100% of 100 g salmon (Salmon salar) survived after 24 h of exposure to 1, 3, and 5 mg/L of dichlorvos\(^{44}\). The 96-h\( \text{LC}_{50} \) value of dichlorvos obtained for Labeo rohita was 16.71 ppm. The fish in the same study exhibited erratic swimming, copious mucus secretion, loss of equilibrium and hitting to the walls of test tank prior to mortality in acute toxicity tests\(^{25}\). The 96-h \( \text{LC}_{50} \) values of Dichlorvos has been reported in Cirrhinus mrigala to be 9.1 ppm\(^{26}\), in Zebra fish, the 24-post fertilization \( \text{LC}_{50} \) value of dichlorvos in the semi static test was 39.75 mg/L\(^{27}\) and 48-h \( \text{LC}_{50} \) values to be 0.5-10 mg/L of dichlorvos formulations in carp\(^{28}\). A study report indicated that 96-h \( \text{LC}_{50} \) value in rainbow trout was 0.93 mg/L\(^{29}\) and on golden orfe was 0.45 mg/L\(^{30}\). In another study, 100% lethality at 10 mg/L in fry of rainbow trout was found\(^{41}\). In Tilapia mossambica with three size groups, 96-h \( \text{LC}_{50} \) values were found to be 1.4-1.9 mg/L, the smaller sizes being more sensitive\(^{32}\). 24, 48 and 96-h \( \text{LC}_{50} \) values of dichlorvos in common carp to be 3.8, 2.7, 2.3 mg/L respectively and 4.1, 4.0 and 3.7 mg/L in Java carp respectively\(^{33}\). In harlequin fish found 24-h \( \text{LC}_{50} \) value to be 12 mg/L and 48-h \( \text{LC}_{50} \) value to be 7.8 mg/L\(^{24}\) while 24 and 48-h \( \text{LC}_{50} \) in bluegill sunfish to be 1 and 0.7 mg/L respectively\(^{26}\). Again, 96-h \( \text{LC}_{50} \) in bluegill and spots were reported to be 0.48 and 0.55 mg/L respectively\(^{36}\).

Chronic toxicity of dichlorvos on fish

Effects on Choline esterase activity

Dichlorvos is an organophosphorus insecticide reported to be neurotoxic due to its irreversible inhibitory effect on AChE\(^{37}\). The enzyme AChE degrades the neurotransmitter acetylcholine in cholinergic synapses. The inhibition provokes an accumulation of acetylcholine in synapses with disruption of the nerve function that can end in the death of the organism. Several authors have been reporting significant inhibition of ChE activity in fish at sub-lethal concentrations of dichlorvos\(^{38-41}\). In sea bass, dichlorvos significantly inhibited the activity of ChE in the selected tissues, both in vitro and in vivo conditions. Differences in ChE sensitivity were found in relation to the age of the fish and the tissue analysed. Sea bass fingerlings are able to tolerate high levels of head and muscle ChE inhibition before death\(^{15}\). Similar results were obtained for pinfish (Lagodon rhomboides)\(^{42}\) and European eel (Anguilla anguilla)\(^{43}\). Brain tissue showed higher ChE activity than muscle, whole blood or plasma tissues. On the contrary, in fingerlings, the highest ChE activity was obtained in muscle. This is in agreement with the results obtained by other researchers\(^{44-46}\). Skeletal muscle presented higher ChE activity than brain tissue in juveniles of goldfish (Carassius auratus) of size ~5 g exposed to three different pesticides\(^{47}\). Chronic dichlorvos exposure impaired mitochondrial energy metabolism and neuronal apoptotic cell death in brain\(^{48}\). AChE activity of Tilapia mossambica in relation to the interacting effects of aging and sub-lethal
concentrations of dichlorvos was studied\(^4\). The enzyme activity of brain and liver decreased with increasing size (and age) and dichlorvos exposed fish showed considerable inhibition of brain and liver AChE. There was a positive correlation between dichlorvos concentration and the time of exposure when the degree of enzyme inhibition was considered. Brain exhibited a higher degree of enzyme inhibition in all age groups of fish as compared to liver. Small fish were more susceptible to the insecticide with respect to AChE activity. When transferred to clean water, most of the exposed fishes recovered their AChE activity and the recovery was greater in liver than in brain\(^4\).

**Effects on Antioxidants**

It has now been established that OP pesticides induced oxidative stress\(^5\). An antioxidant defence system (ADS) is needed to protect biomolecules from the harmful effects of ROS. Fish are endowed with defensive mechanisms to neutralize the impact of reactive oxygen species (ROS) resulting from the metabolism of various chemicals. These include various antioxidant defence enzymes such as superoxide dismutase (SOD), catalase (CAT), glutathione peroxidase (GPOx), glutathione S-transferase (GST), and glutathione reductase (GR). Low molecular weight antioxidants such as glutathione (GSH), ascorbate (vitamin C), vitamin A and E are also reported to contribute in the quenching of oxy radicals\(^5\). ROS which is not neutralized by this antioxidant defense system damages all biomolecules. One of the most important targets of ROS is the membrane lipids which undergo peroxidation (LPO). Thus, LPO estimation has also been successfully employed to signify oxidative stress induced in aquatic animals by such chemicals\(^6\). Decreased GSH levels and also decreased MnSOD activity were observed, in the brain mitochondria isolated from low-level chronic dichlorvos treated rat\(^3\). Also, in fish exposed to dichlorvos for 24 h, at concentrations of 1 or 5 mg/L, a dose-dependent increase was noted in the activities of SOD and CAT in the liver and brain. A rise was also observed in the level of GSH and changes were noted in MDA level in these organs. The increase in GSH was noted mainly in the brain and was accompanied by a decrease in MDA level. The decrease was greater at the exposure to the higher dose of the compound\(^3\).

**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\(^3\). 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\(^5\) and cellular hyperproliferation as a result of local irritation\(^5\).

Dichlorvos has carcinogenic potential, which has been reviewed on several earlier occasions by several workers\(^5\).

**Immune response**

Dichlorvos has the potential to induced altered immune response in fish and it was reviewed by Dunier *et al.* 1991\(^6\).

**Developmental effects**

Dichlorvos exposure during early development in Zebra fish caused clear behavioural impairments detectable during the post hatching period. It also showed mortality and developmental abnormalities\(^7\).

**Histopathology**

Histopathology is an important tool in assessing pesticide toxicity\(^6\). The histopathological effects of liver tissues in *Cirrhinus mrigala* chronically exposed to dichlorvos showed hepatic lesions in the liver tissues were observed which were characterized by cloudy swelling of hepatocytes, congestion, vacuolar degeneration, karyolysis, karyohexis, dilation of sinusoids and nuclear hypertrophy. In the same study, changes in gills such as hyperplasia, desquamation, and necrosis of epithelial, epithelial lifting, oedema, lamellar fusion, collapsed secondary lamellae, curling of secondary lamellae and aneurism in the secondary lamellae were observed after exposure to dichlorvos\(^8\). The effects of sub-lethal doses of dichlorvos on lipid composition and metabolism
of rainbow trout skin cells in primary culture were investigated and it was suggested that dichlorvos may have direct effects on fish skin that could have important consequences for fish health in general. In another study in air-breathing catfish *Clarias batrachus* exposed to lethal and sublethal concentrations of the dichlorvos, significant cytoarchitectural changes in the oocytes, including pronounced vacuolation, degeneration and deformation were observed. Clumping of the cytoplasm and karyohypertrophy were also evident and rupture of the cell wall, with extrusion of the cytoplasm and the nuclei, was observed in the same study. Pesticides are also reported to cause changes in structure and functions of fish gonads. Although the studies with dichlorvos are scarce, the effects of sublethal concentrations of dichlorvos (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 Gonadosomatic index decreased with the increase in concentration, whereas it increased with increase in exposure at all concentrations.

**CONCLUSION**

Dichlorvos 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 dichlorvos 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 dichlorvos 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 insecticide 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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