

A Histological Study on Tilapia (*Oreochromis Mossambicus*) for Assessment of Bioaccumulation of Pollutants and its Impact at Koyali and Dumad Pond of Vadodara District, Gujarat

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ABSTRACT

The results of the diverse toxicant exposure at small levels are presented below. The relationship between toxic expressions in experimental and in situ exposure leads to the conclusion that the pathological changes brought about in the organs of fish from Koyali pond are similar to or only slightly more severe than those observed for low dose exposure for 30 days in experimental groups. While in-situ exposure involves a diversified and vast variety of environmental circumstances, the research setup's environmental parameters are significantly less varied because they are tightly controlled. After 30 days liver, muscles and gills were severely affected and different cells have shown necrosis, inflammation, oedema and vacuolar degeneration. These effects were not seen clearly in 15 days treatment. Results suggests that timely bioremediation and pollution control at study sites is required as soon as possible.

INTRODUCTION –

In the last 50 years, Vadodara's industrial sector has expanded significantly. Industrial buildings have largely replaced agricultural fields as the predominant form of land use. The region's inconsistent solid waste disposal practices have not actually reduced pollution. In the end, this would result in the widespread distribution of dangerous substances in both surface water and ground water. As a result, the industrial area surrounding Vadodara has been polluting its subsurface area for more than three decades. Pollution of a range of resources has led to the biomagnification of toxicants in diverse trophic statuses of different ecosystems. In present study histological study will be carried out on *Tilapia* (*Oreochromis mossambicus*) to examine the bioaccumulation of harmful pollutants and its effects on organs of *Tilapia*. *Tilapia* is classified under Phylum - Chordata, Class – Actinopterygii, Order – Perciformes. The creation of biological monitoring methods based upon fish provides the opportunity to monitor water pollution and respond quickly to small concentrations of directly hazardous substances (Ali *et. al.*, 2007).

MATERIALS AND METHOD –

The metropolitan area of Vadodara is surrounded by latitudes of 22°18' N and longitudes of 73°16' E. The Vadodara urban cluster spans over 140 km². The main chemical heavy industrial zone is in the area towards the north-west of Vadodara city. The Koyali Pond is 9.57.08 hectares in size, while the Dumad Pond is 8.80.20 hectares in size. Both are located at latitudes of 22o34.34" N and longitude of 73o11.21" E.

Samples were collected from the study ponds and without any further exposure or experimentation tissue samples were processed for various analyses. Fishing was done during early morning hours.

Four to six adult *Tilapia* (*Oreochromis mossambicus*) measuring 15-18 cm (weight 26-30 g) were collected each time, seasonally from Koyali and Dumad ponds and brought live to the laboratory. The liver, gills and muscles soft tissues were dissected out and stored in -80°C. Small pieces of tissues were preserved in 10 percent formalin solution for 24 hours, rinsed under running tap water for 3-4 hours, dehydrated in ethyl alcohol grade, cleared in xylene and clove oil and the paraffin blocks were made. 5µm sections were stained with hematoxylin and eosin and observed under microscope for structural details. Microphotographs were taken at 20X, 40X or 100X for documentation purpose.

RESULT AND DISCUSSION –

The bile ducts are suitably arranged, and the central vein, sinusoid, and portal sections of the fish liver are parenchymatous in usual fashion, predominantly composed of polyhedral hepatocytes with big central nuclei and conspicuously coloured chromatin. Endothelial cells surround the blood sinus holes. Between the hepatocytes and the sinusoids, at the edge of the sinusoids, are indeed the reticuloendothelial cells. A few lymphocytes are also visible in the portal area. Seven days after the toxicant exposure at a 10% dose level, there had been no discernible changes. A few pericentral hepatocytes showed some degree of enlargement. By day 15, there were noticeable cytoplasmic alterations in the higher dose group. Cytoplasmic disintegration was evident, although nuclear alterations were barely perceptible

(Vachhrajani and Verma, 2014). The major vein and the sinusoids' endothelium lining both suffered severe damage. By day 30, there had been significant change to the normal parenchymatous look, and the cord-like patterns of the hepatocytes had almost disappeared. The nuclei had severely broken down. The degradation of tiny vacuoles and cytoplasm were observed. Extensive eosinophilic staining revealed serious cytoplasmic abnormalities in the liver of one of the fish. Many of these histological anomalies could be seen in the fish that were taken from Koyali. Large vacuoles were seen, and cell injury was evident. In the periphery area, there were multiple locations where lymphocyte infiltration was visible (Vachhrajani and Verma, 2014).

In the buccal cavity of tilapia, four gill arches extend on either side. Gill arches on the anterior borders guard the delicate gill filament. The striated abductor and adductor muscles that are connected to the bone and cartilage that supports the arches help to facilitate gill movement. The core cartilaginous support, afferent and efferent arterioles, and thin epithelial coating of the gill filaments are all present. Secondary lamellae begin on the superior and inferior surfaces of primary lamellae. The secondary lamella's thin epithelial layer is supported by pillar cells and sits on basement membrane. Melanocytes, lymphocytes, macrophages, mucous, and chloride cells are additional cell types that can be discovered in primary and secondary lamellae (Vachhrajani and Verma, 2014).

The chloride cells, the mucous cells, and the gill filaments are all situated at the base of the secondary lamellae. After being exposed to the heterogeneous effluent, cells showed obvious signs of hypertrophy and hyperplasia. On day 15, secondary lamellae fused as well. The mucosal epithelium and submucosa of the gill racker showed substantial damage after 30 days. Day 30 in the high dose group showed increased damage and a significant amount of clubbing at the tips in the secondary lamellae. The major lamellae thickened erratically. The circumstances were slightly worse in the tissues taken from Koyali fish, with gill filaments damaged (Vachhrajani and Verma, 2014).

Due to damage to epithelial covering cells and supportive pillar cells the architecture of secondary lamellae collapsed. The secondary lamellae were oedematous and infiltration of erythrocytes was also seen. The architecture of secondary lamellae collapsed as a result of damage to epithelial covering cells and supporting pillar cells. Erythrocyte infiltration and oedematous secondary lamellae were both seen (Vachhrajani and Verma, 2014).

The muscles were arranged as normal myofibril bundles encircled by perimysium, or loose connective tissues. Epimysium, a dense connective tissue, was packed around the bundle of muscle fibres. At the outermost part of each muscle fibre, there were many nuclei. The muscle's histoarchitecture was unaffected after 7 days of toxicant exposure. On day 15, the muscular organisation was substantially harmed in the high

dose group. Histoarchitecture is actively influenced by connective tissue injury (Vachhrajani and Verma, 2014).

The fish gathered from Koyali displayed a significant loss of connective tissues as well as some muscle degeneration. The loose organisation of the muscle bundles is a sign of perimysium rupture and disintegration. Focused necrosis, inflammatory cell aggregates, vacuolar disintegration, muscle cell shrinkage, and oedema were the main pathological alterations at high dose exposure for 30 days, as well as those in the tissues of Koyali pond fish (Vachhrajani and Verma, 2014).

CONCLUSION –

Due to their lipophilic nature, heavy metals have a very lengthy biological half-life and more potential to be easily absorbed. The most research is done on their accumulation in animal tissues (Rao, 2006; Vachhrajani and Verma, 2014; Metwally and Fouad, 2008). The primary determinant in metal bioaccumulation is the discrepancy between an organism's rate of intake and rate of excretion, which results in the net accumulation of heavy metals (Krishnamurthy, 1999; El-sheibly, 2009). Oxidative stress is brought on by heavy metal exposure, and biomarkers are altered (Lenartova, 1997; Rao *et al.*, 2006; Farombi *et al.*, 2007; Tejada-Vera, 2007; Firat *et al.*, 2009). Oxidative stress is brought on by heavy metal exposure, and biomarkers are altered. The condition of oxidative stress throughout fishes in connection to aquatic pollution had also been reviewed, in which it was suggested that while many researchers have studied pollutant-induced ROS increase as well as glutathione depletion, the results in several cases do not establish direct quantitative or dose response relationships (Handy, 1996; Livingston, 2001 and 2003; Hamed, 2004; Vachhrajani and Verma, 2014; Humtsoe, 2007; Valkova, 2007; Shen *et al.*, 2007; Laura, 2008; Hossam, 2009).

The liver is an chief organ associated with biotransformation of toxicants. The important enzymes of toxicant biotransformation are concentration in the pericentral region (Rani and Ramamurthi, 1986 and 1989; Madrasmi, 2007). The hepatocytes of this region were severely damaged in the high dose group in the fish from Koyali pond of vadodara. Considering biomagnifications through the food chain, ingestion is the most probable mode of exposure for the fishes under study. The entero-hepatic circulation exposes the liver tissue in particular to greater toxicant concentrations, increasing the likelihood of poisoning (Staicu *et al.*; 2005; Vachhrajani and Verma, 2014). This also gives an opportunity to liver to accumulate the toxicants. Present findings very clearly demonstrate that in Koyali pond water the metal toxicants are present which get accumulated in the liver tissues of the fishes and induce severe structural alterations. Significant cellular alterations were brought on by 21 days of experimental exposure to water-borne copper in the liver and gills (Dhanpakkium and Ramaswamy, 2001; Henson *et al.*, 2006; Figueiredo-Fernandes *et al.*; 2007). Exposure to aluminum also caused damage to fish tissues (Hadi *et al.*;

2009). Fish exposed in experiments to a wide range of toxicants at various concentrations for short-term to long-term periods of time showed very comparable types of lesions (James and Sanpath, 1999; Jaroli and Sharma, 2005; Madrasmi, 2007). The gills are shown to be partially dose responsive in present findings. Studies by Mohamed (2009) on tilapia from the pollution receiving site lake Qarun, Egypt,

demonstrated findings comparable to those observed in fishes from Koyali pond. The results of the two investigations directly link environmental contamination to hazardous expressions in aquatic systems. Peebua (2005) exposed tilapia for different durations to variety of toxicants including pesticides and metals.

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