Aquatic ecosystems are used as storage houses for many chemicals, discharged by industries, domestic, sewage and agriculture wastes. The continuous release of these chemicals disturbs the water quality and aquatic organisms because of their persistence, bioaccumulation, toxicity and biomagnifications in the food chain. Aquatic organisms including fishes accumulate toxicants directly from contaminated water or indirectly through the food chain (Tilak *et al*., 2004). All the animals have some capacity to tolerate the toxic concentrations because, when the pollutant enters the body it get metabolized and excreted outside. However, when animals cross the tolerance limits, they show changes in the behaviour, morphology of cells and tissues.

India is a developing country and major pollution in the aquatic environment has been done by the industrial activities. Therefore, industrial wastes due to their persistence, toxicity and high concentration in the water are considered to be the interest of study. These wastes contain heavy metals, polycyclic aromatic hydrocarbons, polychlorinated biphenyls and other toxic compounds, which directly or indirectly enter in the body of aquatic organisms and affect their survival. Majority of these toxicants, when crossed their permissible limits become potentially genotoxic and carcinogenic, which are responsible for DNA damage in aquatic organisms.

Fishes are relatively sensitive to changes in their surrounding environment and their health may reflect the status of any aquatic ecosystem (Gupta *et al*., 2009; Mokhtar *et al.*, 2009). The toxic discharges adversely affect the water quality, feeding and swimming behaviour of fishes (Kothari *et al.*, 1990; Atif *et al.*, 2005; Laovitthyanggoon, 2006; Kumar *et al.*, 2007). Environmental stress causes a variety of detectable and recognizable behavioural changes in fishes. The behavioural changes constitute an index to measure the toxicity of any toxicant. Thus, behaviour is considered as a promising tool in ecotoxicology. In fishes, behavioural changes are used as a diagnostic end point for screening and differentiating chemicals on basis of their mode of action, which also provide early information about the presence

of pollutants in the aquatic system (Drummond *et al.,* 1986). Important contributions about the behavioural change in fishes under experimental conditions have been earlier reported by many workers (Hoar, 1952; Baerends *et al.,* 1955; Smith and Hoar, 1967; Sage, 1968; Baskaran *et al*., 1989).

Toxic impact of pollutants may also responsible for the physiological, biochemical or pathological alterations in the organisms (Shivakumar *et al.*, 2005). Genotoxicity tests have been used to detect the toxicity in fishes. These are chromosomal aberration test, micronucleus test and erythrocyte aberration test. Chromosomal aberration test (CAT) is a sensitive biomarker in monitoring the toxicity. Moreover, micronucleus and erythrocyte aberration tests are also important because fish erythrocytes possess nucleus, so, any change in structure and shape of nucleus in the erythrocytes act as diagnostic tool to judge the genotoxicity in the fishes.

Histopathology is used for the histo-morphological evaluation of microscopic alterations in the affected organs and tissues (Campbell, 1997). Histopathological biomarkers reflect the overall health (at sublethal and chronic levels) of the entire population in the ecosystem. These are widely used in the laboratory as well as in the field studies to check the safe concentrations of toxicants in the environment. Detection of toxicants in the tissues in relation to other biomarkers reflects the overall health of the ecosystem. All these changes activate the metabolic pathways, which ultimately damage the cells, tissues and organs of affected organisms. So, detailed study about genotoxic and histopathological alterations is required to understand the toxic effects of industrial wastes on fishes.

Presently, *Labeo rohita* has been selected to evaluate the genotoxicity and histopathological changes caused by the tannery and paint industrial effluents. 96h LC_{50} value of both the effluents are 15.48% (tannery) and 31.62% (paint), respectively. Fishes have been subjected to two sublethal concentrations (1/2 and 1/8) on the basis of 96h LC_{50} of both the effluents. These concentrations are 7.74% and 1.93% for tannery effluent, while 15.81% and 3.95% for paint effluent.

Genotoxic and hisopathological effects of both the effluents have been determined on basis of:

1. Behavioural responses and morphological changes
2. Chromosomal analysis
3. Micronucleus and erythrocytes Aberrations
4. Histopathology

Behavioural responses and morphological changes:

Control fishes maintained in the well aerated water are active against slight disturbances, show well synchronized movements, usually settled at the base and rarely came on the surface. No mortality has been observed in control.

Fishes treated with two sublethal concentrations of tannery and paint industrial effluents have showed various behavioural responses and morphological changes from 24h to 120h. Behavioural responses include erratic and rapid swimming, gulping of air, opercular movements, loss of equilibrium, hitting against the wall, restlessness, sluggishness and deaths of fishes. Morphological changes reveal loosening of scales, redness in eyes, sinking of eyeball, profuse mucous secretions, bleeding from gills and hemorrhages. Behavioural and morphological changes are showing the concentration and time dependent response for both the effluents. However, these changes are more pronounced in tannery effluent as compared to paint effluent.

Similar types of behavioural and morphological changes due to the effect of heavy metals, pesticides, herbicides and industrial effluents have also been reported in many fishes by the various workers. Earlier, behavioural and morphological changes due to heavy metals and industrial wastes have also been studied in *Labeo rohita*. These are loss of equilibrium, jerky movements, hitting against wall, death of fishes, convulsion, gulping of air, restlessness, erratic swimming, fast opercular movements, profuse mucous secretions, hyperactivity, loss of scales, haemorrhages, impaired schooling behaviour, lack of body coloration, sluggishness/lethargy, surfacing of fishes, fishes jumping out of water or avoidance due to heavy metals like cadmium chloride

(Wankhede and Dhande, 1999); cadmium (Maruthanayagam *et al.,* 2002); potassium dichromate (Vutukuru, 2005); sodium cyanide (Dube and Hosetti, 2010; aluminium chloride (Selvam *et al*., 2014) and lead nitrate (Brraich and Kaur, 2015). Moreover, similar changes have also been observed due to the impact of paper/pulp industry (Srivastava *et al.,* 2007); municipal wastes (Kaur and Dua, 2012) and tannery industry effluent (Walia *et al.,* 2013b, Walia and Handa, 2016).

Few reports are also available on the effect of other chemicals like organochlorine, organophosphate and carbamate pesticides in *Sarotherodon mossambicus* (Khalaf, 1990); carbofuran in goldfish (Saglio *et al.,* 1996); diazinon 60EC in *Anabas testudineus, Channa punctatus, Barbodes gonionotus* (Rahman *et al.,* 2002); lindane and diazinon in *Clarias garipinus* (Adedeji *et al.,* 2008); cypermethrin anf k-cyclothrin in *Channa punctatus* (Kumar *et al*., 2007); dimethote in *Heteropneustes fossilis* (Pandey *et al.,* 2009, Srivastava *et al.,* 2010); chloropyrifos in *Cyprinus carpio* (Halappa and David, 2009); cypermethrin in *Labeo rohita* (Marigoudar *et al*., 2009), *Oreochromis niloticus* (Yaji *et al*., 2011); carbaryl and malothion in *Clarias batrachus* (Wasu *et al.*, 2009); fenvalerate in *Labeo rohita, Catla catla, Cirrhinus mrigala* (Susan and Sobha, 2010) and endosulphan in *Channa striatus* (Ganeshwade *et al.,* 2012).

Behavioural responses and morphological changes in fishes are mainly due to the inactivation of acetylcholine as suggested by various workers (Fulton and Key, 2001; Rao *et al*., 2005; Mushigeri and David, 2005; Agrahari *et al*., 2006; Raju, 2000; Prashanth *et al.,* 2005; Prashanth and Patil 2006). According to them, restlessness, erratic and jerky swimming, loss of equilibrium, hitting against the wall and hyperactivity in fishes might occur due to the inactivation of acetylcholinesterase (AChE), which lead to accumulation of acetylcholine at synaptic junctions. Surfacing or gulping of air might be due to the demand of high oxygen level after exposure (Katja *et al*., 2005). Bisht and Agarwal (2007) suggested that profuse mucous secretion is considered to be a defense mechanism to neutralize the effect of toxicants by coagulation and also prevent the entry in the body. These secretions also cover the gills and

inhibit the diffusion of oxygen (Muniyan and Veeraragghavan, 1999; David *et al.*, 2002). Santhakumar *et al.* (2000) and Prashanth and Patil (2006) also concluded that increase in opercular movements in fishes is related to adaptation for the respiration and avoid contact with toxicants. Banerjee (1997) suggested that haemorrhages/lesions or degenerative changes in the skin and muscles of fishes may be due to replacement of loose connective tissue.

In present study, fishes have also showed various behavioural and morphological changes because both the effluents contain heavy metal, which are responsible for inactivation of acetylcholine as suggested by earlier workers. High concentrations of TDS, TSS, BOD and COD also cause hypoxic conditions, acidic/alkaline pH resulting in avoiding, jumping and haemorrhages in the fishes.

Chromosomal aberrations:

In *Labeo rohita*, diploid chromosome number is 2n = 50. The chromosomes are comparatively small and condensed, but easily recognizable. They are differentiated on basis of centromere as 5 pairs of metacentrics, 9 pairs of submetacentrics and 11 pairs of telocentrics chromosomes. So, the chromosomal formula is 10m + 18 sm + 22t and fundamental arm number is 78. However, sex chromosomes are not distinguishable. Zhang and Reddy (1991) were the first to describe the chromosome formula for *Labeo rohita* and present observations are also in accordance to their observations.

Fishes have been treated with two sublethal concentrations of tannery and paint industrial effluent for 24h, 48h, 72h, 96h and 120h time durations. Somatic metaphases have been obtained from the kidney tissue. Eleven types of aberrations like chromosome fragments, ring chromosomes, terminal chromatid deletions, minutes, terminal association of chromosomes, centromeric gaps, stickiness, clumping, pyknosis, stretching and pulverizations have been observed. All these aberrations show time and concentration dependent response with respect to their controls.

In tannery effluent, at low concentration (1.93%), there is decrease in all types of chromosomal aberrations with increase in exposure periods, particularly

after 96h and 120h. Overall, the frequency of Cf is highest followed by Tcd, Cg, whereas the frequency of Stk and stch are lowest. Mitotic indices also show increasing trend upto 48h then there is prominent increase from 72h to120h. In the high concentration (7.74%), there is increase in chromosomal aberrations after 24h to 120h. Overall, the frequency of Rc is highest followed by Cf and Cg, while frequencies of C, Stk and Stch are lowest. Mitotic indices indicate the decreasing trend, while after 48h, there is drastic decrease upto 120h.

In paint effluent, at low concentration (3.95%), the frequency of chromosomal aberrations decreases from 24h to 120h. Overall, the frequencies of M, Tcd, Cf are highest, whereas Stch and P are lowest. Stk, Stch and P are not observed at 120h. Mitotic indices show decreasing trend from 24h to120h. In high concentration (15.81%), the frequency of chromosomal aberrations increases from 24h to 120h. Overall, frequencies of Tcd, M, Cf are highest, while Tac and Stch are lowest. Stch is absent at 120h. Mitotic indices also show increasing trend from 24h to120h.

Earlier, various types of chromosomal aberrations resulted due to the effect of various toxicants have also been reported. Chromosomal aberrations by heavy metals like chromium in *Boleophthalmus dussumieri* (Krishnaja and Rege, 1982); methyl mercury chloride in *Fundulus heteroclitus* (Perry *et al.*,1988); mercuric chloride in *Oreochromis mossambicus* (Manna and Mukherjee, 1989), *Clarias gariepinus* (Mahboob *et al*., 2013); fluoride in *Clarias batrachus* (Tripathi *et al.*, 2009); copper in *Cyprinus carpio* (Stouthart *et al.*, 1996); lead in *Channa punctatus* (Mathew and Jahageerdar, 1999), *Hoplias malabaricus* (Cestari *et al.*, 2004, Ferraro *et al.*, 2004), *Hoplias malabaricus* (Ramsdorf *et al*., 2009); cadmium chloride in *Oreochromis mossambicus* (Chandra and Khuda-Bukhsh, 2001), *Channa punctatus* (Parveen and Shadab, 2012); copper sulphate in *Labeo rohita* (Krishna and Gupta, 2002); chromium (VI), mercuric chloride, arsenic trioxide and copper sulphate pentahydrate in *Channa punctatus* (Yadav and Trivedi, 2006, 2009); copper sulphate and [$(CHCOO)_3$ Pb] in *Oreochromis niloticus* (Mohamed *et al.*, 2008)

and sodium arsenate in *Clarias lazera* (Radwan and Ghaly, 2008) have been studied.

Chromosomal aberrations in fishes due to the pollutants present in the polluted water have been observed. Effect of polluted river in *Mytilus galloprovincialis* (Al-Sabti and Kurelec, 1985); paper and pulp mill effluents in *Micropterus salmoides* (Denslow *et al*., 2004), *Channa punctatus* (Kumari and Ramkumaran, 2006), *Mugil cephalus* (Hafez, 2009), *Clarias gariepinus* (Obiakor *et al.*, 2010), *Hypophthalmicthys molitrix* (Rose *et al.*, 2010); drainage canal receiving sewage and other discharges in *Oreochromis niloticus* and *Tilapia zillii* (Mahmoud *et al.*, 2010); chromium effluent in *Cirrhinus mrigala* (Saravanan *et al.*, 2012); tannery industry effluent in *Labeo rohita* (Walia *et al.*, 2013c, Walia and Handa, 2016) and dyeing industry effluent in *Cirrhinus mrigala* (Kaur *et al.*, 2013a, Walia and Kalotra, 2016) have been reported.

Chromosomal aberrations resulted due to other chemicals like triphenyltin hydroxide in catfish (Visoottiviseth *et al.* 1998); dichlorvos in *Channa punctatus* (Rishi and Grewal, 1995a); lambda-cyhalothrin in *Mystus gulio* (Velmurugan *et al.*, 2006), butataf herbicide in nile tilapia (Ramadan, 2007); antineoplastics- bleomycin, mitomycin-C and doxorubicin in *Boleophthalmus dussumieri* (Gadhia *et al.*, 2008) and cypermethrin in *Channa punctatus* (Ansari *et al.*, 2011) have also been studied.

Majority of the workers suggested that chromosomal aberrations in fishes are resulted due to the error in DNA duplication. These toxic chemicals disrupt DNA replication during S phase, interfere with nucleotide synthesis (Mattar *et al.*, 1992) leading to malformation of DNA molecules (Landolt and Kocan, 1983) and these toxins may have strong oxidative effect on membrane phospholipid proteins and nucleic acids (Chorvatovicova *et al*., 1992). Later, Evans (1977) concluded that it may be a consequence of miss-replication of damaged DNA. Further, Natarajan and Obe (1978) enumerated that DNA break is ultimate reason for chromosomal aberrations and OH and O_2 radicals are most biologically relevant oxygen species (ROS), which react with DNA and

result in chromosomal aberrations. Tsalev and Zaprianov (1983) also explained that heavy metal like chromium reduces to chromium (IV) in the body, which generates reactive free radicals.

During the present study, genotoxic effect of both the effluents resulted in chromosome aberrations might also be due to the error in the DNA replication because presence of heavy metals. Both the effluents also contain chromium which result in formation of free radicals resulted in chromosomal aberrations as suggested by earlier workers.

Micronucleus test and Erythrocytes aberration test:

Generally, erythrocytes in fishes are fairly large in size and having well defined boundary, centrally placed elliptical/rounded nucleus in the cytoplasm. Dot like micronucleus is present in the erythrocytes, while it lies close to the main nucleus or at the peripheral position. Besides one micronucleus, two and three micronuclei have also been recorded in the erythrocytes. In both concentrations of effluents, occurrence of one micronucleus is predominant as compared to two and three micronuclei. Overall, in higher concentrations of both the effluents, the frequencies of all types of micronuclei are more. However, fishes treated with 7.74% concentration of tannery effluent show steady increase in the frequency of micronuclei from 24h to 120h, whereas in 1.93% concentration, the number of cells decreases from 24h to 48h then sharp decrease upto 120h. On the other hand, fishes treated with 15.81% concentration of paint effluent possess steady increase from 24h to 96h then sharp increase at 120h, while in 3.95% concentration, steady decrease in the number of cells with micronuclei from 24h to 120h.

Eleven types of erythrocytes Aberrations have been recorded, which further divided into nuclear and cellular Aberrations. In both the effluents, fishes in lower concentrations show decrease in frequency of erythrocytes Aberrations from 24h to 96h then sharp decrease at 120h, while in higher concentrations there is slow increase from 24h to 48h then sharp increase from 72h to 120h. Micronuclei and erythrocyte Aberrations show concentration and time dependent response for both the effluents. However, intensity of micronuclei and

erythrocytes Aberrations is more in tannery effluent as compared to paint effluent.

Earlier, micronuclei and erythrocytes Aberrations have been recorded due to the effect of heavy metals like cadmium in *Channa punctatus* (Gupta and Rajbanshi, 1988); rainbow trout (Castano *et al*., 1998); chromium in *Carassus auratus* and *Carassus gibelio* (Al-Sabti and Metcalfe, 1995); potassium dichromate in *Dreissena polymorpha* (Mersch *et al*., 1996), *Oreochromis niloticus* (Da-Rocha *et al*., 2011), *Labeo rohita* (Praveena *et al*., 2014); mercuric chloride in *Cyprinus carpio* (Nepomuceno *et al*., 1997), *Therapon jarbua* (Nagarajan *et al*., 2009); mercuric nitrate and cadmium chloride in *Salmo trutta* and *Phoxinus phoxinus* (Sanchez-Galan *et al*., 1999); mercuric nitrate in *Paecilia latipinna* (Ayllon and Vazquez, 2000); mercury in *Channa punctatus* (Gupta and Dua, 2002); cadmium chloride in *Oreochromis mossambicus* (Chandra and Khuda Bukhsh, 2004), *Oreochromis niloticus* (Ozkan *et al*., 2011), *Channa punctatus* (Parveen and Shadab, 2011); cadmium, chromium and copper in *Cyprinus carpio* (Zhu *et al*., 2004); cadmium chloride and copper sulphate in *Cyprinus carpio, Carassius gibelio, Corydoras paleatus* (Cavas *et al*., 2005), *Gambusia affinis* (Muranli and Guner 2011); copper and zinc in *Oncorhynchus mykiss* (Bagdonas and Vosyliene, 2006); *Puntius altus* (Jiraungkoorshul *et al*., 2007); copper sulphate, arsenic trioxide, mercuric chloride in *Channa punctaus* (Yadav and Trivedi, 2009); chromium trioxide in *Channa punctatus* (Choudhary *et al*., 2012); hexavalent chromium in fishes (Kumar *et al*., 2012); zinc acetate in *Heteropneustes fossilis* (Talapatra *et al*., 2014); phenol in *Clarias gariepinus* (Aita, 2014) and cobalt chloride in *Oreochromis mossambicus* (Suganthi *et al*., 2015), *Labeo rohita* (Nagpure *et al*., 2015).

Micronuclei and erythrocytes Aberrations due to industrial effluents and polluted water discharges have been studied. Impact of papermill in *Heteropneustes fossilis* (Das and Nanda, 1986); in perch (Al-Sabti and Hardig, 1990); *Chela atapar*, *Mystus vittatus* (Tripathy and Das, 1995); *Dicentrachus labrax* (Gravato and Santos, 2003); industrial effluents in *Clarias lazera*

(Odeigah and Osanyinpeju,1995); old military factory in *Salmo trutta* (Ayllon *et al*., 2000); textile effluent in *Oreochromis niloticus* (Cavas and Ergene-Gozukara, 2003); petrochemicals, fertilizer industries, few tanneries and sewage discharges in *Lates calcalifer, Liza parsia, Liza tade, Mugil cephalus, Rhinomugil corsula, Terapon jarbua* and *Scatophagos argus* (Mallick and Khuda-Bukhsh, 2003); petroleum refinery and chromium processing plant effluents in *Oreochromis niloticus* (Cavas and Ergene-Gozukara, 2005a); tannery effluent in *Oreochromis niloticus* (Matsumoto *et al*., 2006), *Channa punctatus* (Nagpure *et al*., 2015); refinery effluent in nile tilapia (Souz and Fontanetti, 2006); polluted water in *Clarias gariepinus, Mugil cephalus, Alburnus orontis* (Ergene-Gozukara *et al.,* 2007); El-Tabia pumping station in *Mugil cephalus* (Hafez, 2009); textile industry effluent in *Oreochromis niloticus* (Perera and Pathiratne, 2010), *Clarias gariepinus* (Ayoola *et al*., 2012, Oriaku *et al*., 2012); petroleum refinery effluents in *Oreochromis niloticus* (Hoshina and Morales, 2008); crude oil in *Mytilus edulis* (Barsiene *et al*., 2010); sewage polluted water in five fishes (Poongothai *et al.,* 1996); polluted estuary in *Centropomus parallelus* (Kirschbaum *et al*., 2009); polluted water lake in *Cyprinus carpio* (Saleh, 2010), polluted river Gomti in *Channa punctatus* and *Mystus vittatus* (Kushwaha e*t al.,* 2012); dyeing industry effluent in *Cirrhinus mrigala* (Kaur *et al.,* 2013c) and tannery industry effluent in *Labeo rohita* (Walia *et al.,* 2015) have been reported.

Micronuclei and erythrocyte Aberrations due to the effect of other chemicals have also been reported by the several workers. Effect of 2,4-dichlorophenoxyacetic acid and butachlor in *Clarias batrachus* (Ateeq *et al.*, 2002); ethyl methane sulphonate in *Oreochromis mossambicus* (Guha and Khuda-Bukhsh, 2003); carbaryl in *Anabas testudineus* (Bhunya and Sahoo 2004); sterigmatocystin in *Oreochromis niloticus* (Abdel-Wahhab *et al.,* 2005); glyphosate in *Carassius auratus* (Cavas and Konen, 2007); sodium arsenate in *Clarias lazera* (Radwan and Ghaly, 2008); malathion in *Channa punctatus* (Parveen and Shadab, 2011) and synthetic sindoor in *Heteropneustes fossilis* (Malla *et al*., 2011) have been studied. There are few reports about the

micronuclei and erythrocytes aberrations due to on tannery effluent in fish, *Oreochromis niloticus* (Matsumoto *et al*., 2006) and *Channa punctatus* (Nagpure *et al*., 2015), whereas, there is no report on paint effluent.

The mechanism responsible for micronuclei and erythrocytes Aberrations in fishes have not been fully explained, it has been suggested that nuclear budding in interphase cause blebbed and lobed nuclei, which result in the formation of micronuclei. The entire process represents the mechanism of elimination of amplified genes from the nuclei (Shimizu *et al*., 1998 and Crott *et al*., 2001). Further, Von Sonntag (1987) and Steenken (1989) hypothesized that these Aberrations arise due to damage caused to the genetic material by free radical produced under oxidative stress due to the toxicants. Aneuploidy is another abnormality that resulted due to tubulin failure and mitotic fuses caused by aneugenic actions of toxicants and resulted in formation of binucleated cells and notched nuclei (Ventura *et al*., 2008 and Fernandes *et al*., 2007). Ateeq *et al.* (2002) elaborated the sequence of cellular degradation under the impact of toxicants and also suggested that toxicants cause hypoxic conditions, which result in depression of ATP that lead to abnormal shape of erythrocytes. Further, toxicants also interrupted the lipid solubility of membranes of erythrocytes result in vacuolated cells and echinocytes cells and ultimately, lead to apoptosis.

In present study, fishes exposed to both the effluents possess micronuclei and erythrocytes Aberrations. These might be due to the presence of heavy metals in both the effluents, which results in hypoxic conditions and formation of free radicals as suggested by earlier workers.

Histopathology:

In fishes, majority of the histopathological studies have been carried out on gills, kidney and liver because they are major target organs responsible for vital functions like respiration, excretion, accumulation and biotransformation of xenobiotics, respectively (Gernhofer *et al.,* 2001). Gills are most vulnerable to environmental toxicants because they are externally located, close contact with water and are principal site for the uptake of toxicants through membrane.

Kidney is the second effector organ in ionic regulation and plays an important role in excretion of many bio-transformed derivatives of toxicants. Liver is the third important organ to maintain the external homeostasis, metabolism of toxicants and accumulation of foreign compounds. However, few reports are also available on histopathological changes in skin, muscle, spleen and intestine of fishes, which are providing defense, bone protection, haematopoiesis and absorption/excretion, respectively (Hossain *et al*., 2002; Athikesavan *et al.*, 2006; Bhatnagar *et al*., 2007; Altinok and Capkin, 2007; Dumitrescu *et al*., 2010; Patel and Bahadur, 2011; Dhevakrishnan and Zaman, 2012; Navraj and Yasmin, 2012; Sounderraj *et al*., 2012; Al- Balawi *et al.*, 2013; Al-bairuty *et al*., 2013). Majority of the work provides qualitative details of the organs, although Bernet *et al*. (1999) gave formulae to detect the semi-quantitative estimation of histopathological changes.

During the present study, fishes exposed to two sublethal concentrations of tannery (7.74% and 1.93%) and paint (15.81% and 3.95%) industrial effluents after 120h have been selected for histopathological analyses of gills, kidney and liver. Histopathological alterations in all the organs have been recorded, classified and their semi-quantitative estimation has been done according to the formulae given by Bernet *et al.* (1999). Histopathological changes are divided into four reaction patterns *viz.,* circulatory disturbances, regressive changes, progressive changes and inflammation for all the three target organs.

Gills:

In present study, at low concentrations (1.93% tannery and 3.95% paint) main alterations like haemorrhages, intracellular oedema, lamellar telangiectasia, hyperplasia, damaged lamellae, loss of lamellae and lymphatic infiltrations have been observed, while at high concentrations (7.74% tannery and 15.81% paint) significant changes like haemorrhages, intracellular oedema, broken cartilage, damaged lamellae, necrosis, loss of lamellae, hyperplasia, hypertrophy, lamellar telangiectasia, aneurysm, lymphatic infiltrations of cells

have been recorded. However, intensity of these alterations is more in tannery as compared to paint effluent.

The first report on histopathological alteration in the gills of *Barbus stigma* exposed to distillery effluent has been given by Haniffa and Sundaravadhanan (1984). They observed partial destruction in gill epithelium, pillar cells, blood capillaries, cartilage cells, separation of epithelial layer of secondary lamellae from basement membrane and gill filaments with mucous. Later, effects of effluents, heavy metals, pesticides, herbicides on gills of various fishes have been done by many workers and they have recorded different types of histopathological alterations.

Bulge on tip of lamellae, necrosis, hypertrophy, aneurysm, haemorrhages, oedema hyperplasia, fusing/curling of lamellae, epithelium lifting leading to contraction, sloughing of respiratory epithelium, disintegration/destruction of primary and secondary gills epithelium, capillaries and filaments have been recorded due to the effect of heavy metals like mercury in *Puntius sophore* (Khangarot and Somani, 1980), *Sarotherodon mossambicus* (Naidu and Ramamurthi, 1983), *Rasbora danicoinus* (Gupta and Rajbanshi, 1995), *Trichomycterus brasiliensis* (Oliveira *et al.,* 1996), *Trichomycterus brasiliensis* (Ribeiro *et al.,* 1996); cadmium chloride in *Puntilus conchonius* (Gill *et al*., 1989); *Cyprinus carpio* (Suresh *et al*., 1993), *Channa punctatus* (Sastry and Shukla, 1994), *Lates calcarifer* (Thophon *et al.*, 2003); nickel chloride in *Colisa fasciatus* (Nath and Kumar, 1989), *Hypophthalmichthys molitrix* (Athikesavan *et al*., 2006); mercuric chloride in *Heteropneustes fossilis* (Prasad, 1994); *Ctenopharyngodon idella* (Khan *et al.*, 2004); *Oncorhynchus mykiss* (Van Heerden *et al*., 2004); zinc chloride in *Heteropneustes fossilis* (Hemalatha and Banerjee, 1997); copper in *Solea senegalensis* (Arellano *et al.,*1999), *Cyprinus carpio* (De Boeck *et al.,* 2001), mercuric sulphate in *Cirrhinus mrigala* (Gupta and Kumar, 2006); cadmium and zinc in *Salmo trutta* (Besirovic *et al*., 2010); cadmium sulphate in *Tilapia mossambica* (Jalaludeen *et al*., 2012); lead acetate in *Clarias gariepinus* (Al-

Balawi *et al.*, 2013); copper sulphate and copper nano particles in *Oncorhynchus mykiss* (Al-Bairuty *et al*., 2013).

Similar changes have also been noticed due to the exposure of effluents like industrial effluent in *Heteropneustes fossilis* (Anitha and Sree, 1995); sago effluents in *Clarias batrachus* (Ramesh and Nagarajan, 2007); textile wastewaters in *Gumbusia affinis* (Sharma *et al.*, 2007); waterborne copper in *Oreochromis niloticus* (Figueiredo-Fernandes *et al*., 2007); dyestuff and chemical wastewater in *Clarias lazera* (Abdel-Moneim *et al*., 2008); papermill effluent in *Rasbora daniconius* (Pathan *et al*., 2010); malachite green dye in Nile tilapia (EL-Neweshy and Abou Srag, 2011); textile mill effluent in *Labeo rohita* (Nikalje *et al*., 2012); papermill and other industrial wastes in *Channa punctatus* (Kaur and Dua, 2015). Moreover, reduced secondary lamellae, swelling in pillar cells, ruptured secondary lamellae have also been reported in *Rasbora daniconius* exposed to papermill effluent (Pathan *et al*., 2010).

Histopathological alterations like intense vasodilation of lamellar vascular axis and filament epithelium proliferation have been noticed only due to the effect heavy metals like copper in *Solea senegalensis* (Arellano *et al*.,, 1999), *Cyprinus carpio* (De Boeck *et al.*, 2001), *Oncorhynchus mykiss* (Van Heerden *et al*., 2004); waterborne copper in *Oreochromis niloticus* (Figueiredo-Fernandes *et al*., 2007); cadmium and zinc in *Salmo trutta* (Besirovic *et al*., 2010); copper sulphate and copper nano particles in *Oncorhynchus mykiss* (Al-Bairuty *et al*., 2013).

Thickening of secondary lamellae have been observed only due to impact of effluent like textile wastewaters in *Gumbusia affinis* (Sharma *et al.*, 2007), *Labeo rohita* (Nikalje *et al.*, 2012); papermill and other industrial wastes in *Channa punctatus* (Kaur and Dua, 2015). Inflammatory cells and mild telangesitica have been recorded in Nile Tilapia exposed to malachite green dye (EL-Neweshy and Abou Srag, 2011) and collapsed pillar cells in *Clarias lazera* due to dyestuff and chemical wastewater (Abdel-Moneim *et al*., 2008).

Earlier, effect of tannery effluent on gills in *Labeo rohita* (Dhanapalkiam *et al*., 2004); *Oreochromis mossambicus* (Navraj and Yasmin,

2012), *Tilapia mossambica* (Sounderraj *et al*., 2012) and *Danio rerio* (Sivakumar *et al*., 2015) have also been reported. They observed fusion/clumping of primary lamellae, swelling in primary and secondary lamellae, filament cell proliferation, cellular infiltration, haemorrhages and epithelial lifting, hyperplasia, oedema, fusion of secondary lamellae, necrosis, hypertrophy, vacuolization, erosion and clubbing of lamellae. During the present study, similar histopathological alterations in *Labeo rohita* exposed to tannery effluent have been noticed and results are in accordance to the earlier reports. Moreover, aneurysm, broken cartilage, damaged lamellae, loss of lamellae, lamellar telangiectasia, curling of lamellae are recorded for the first time due to the effect of tannery effluent. Histopathological effect of paint industrial effluents on gills of *Labeo rohita* has also been done for the first time.

Gills are most affected organ because they play major role in osmotic regulation, ion regulation, respiration, uptake of metals, storage and transfer to internal components *via* blood transport. Mucus secretion is often the first line of defense to metal/ toxicant exposure in the gills and can temporarily protect the underlying epithelium from injury by fusion of lamellae (Handy and Maunder, 2009). Heavy metals in the aquatic ecosystem can enter the gills through calcium ions *via* chloride cells and interact with enzymes and metallothionein (MT), which are low-molecular-mass cysteine-rich metal-binding proteins having a high affinity for heavy metallic ions (Reid and McDonald, 1991; Alazemi *et al*., 1996; Oronsaye and Brafield, 1984; Gill *et al*., 1988; Fu *et al*., 1990). After entry into the tissues, swelling of the secondary lamellae occurs, which cause telangiectasia (an increase in the diffusion distance across the epithelium for gas exchange). Later, Garcia-Santos *et al.* (2006) concluded that telangiectasia causes lesion in pillar cells and responsible for the emergence of lamellar aneurysms.

Lifting of epithelium, lamellar fusion, gill damage, cellular damage in gills like hemorrhages, hyperplasia and hypertrophy are caused due to hypoxic conditions caused by pollutants (Skidmore, 1970; Skidmore and Tovell, 1972,

Burton *et al.,* 1972; Gardner and Yevich 1970; Bilinski and Jonas, 1973; Gardner and La-Rocha, 1973). These changes might act as defense mechanisms that reduce the branchial superficial area in contact and increases the diffusion barrier to the pollutants with the external milieu (Lauren and McDonald 1985; Van Heerden *et al.* 2004). The alterations like necrosis, damaged lamellae, loss of lamellae are due to the above effect. Oedema appears to be a common feature of the gill pathology when any types of toxicants come in contact with gills. Toxicants disturb the branchial Na+, K+-ATPase pump, which lead to solute accumulation in the epithelial cells and disturb the osmotic influx of water. This exchange protects the epithelial cells of the lamellae and prevents the entry of waterborne pollutants in to the bloodstream (Arellano *et al.*, 1999).

In present study, fishes exposed to both the effluents resulted in various histopathological alterations in gills of *Labeo rohita* might be due to the presence of heavy metals in both effluents, which interact with metallothionenin *via* chloride cells in the gills as suggested by earlier workers. Further, increased hypoxic condition disturbs Na+, K+ ATPase pump, which also damage the gill tissue.

Kidney:

In present study, at low concentrations (1.93% tannery and 3.95% paint), main alterations like hypertrophy, hyperplasia, lymphatic infiltration, dilation of Bowman's capsule and widen lumen of tubules have been observed. Moreover, at high concentrations (7.74% tannery and 15.81% paint), significant changes like intracellular oedema, haemorrhages, lymphatic infiltrations, hypertrophy, occlusion of tubules, necrosis, increased Bowman's space, narrowing of tubular lumen, shrunken glomerulus, dilation of Bowman's capsule, decrease in hematopoietic tissue, widen lumen and hyperplasia have been recorded. However, intensity of these alterations is more in tannery as compared to paint effluent.

Earlier, effect of heavy metals effluents, pesticides and herbicides on the kidney of fishes have been done by many workers. They have noticed various types of structural alterations like degeneration in glomerulus/interstitium

tissue, Shrunken glomeruli/ dilation of Bowman's capsule; increase in Bowman's space, shrinkage of proximal tubule cells with pyknotic nuclei, increase in tubular lumen, increased in intra-tubular hematopoietic tissue, glomerular expansion, destroyed proximal tubular lining with disintegrated cytoplasmic material due to the impact of elson, mercuric chloride and aqueous ammonia in *Channa punctatus* (Banerjee and Bhattacharya, 1994); mercuric chloride in *Cirrhinus mrigala* (Sastry and aggarwal, 1997), *Ctenopharyngodon idella* (Khan *et al.*, 2004); nitrate in *Cyprinus carpio* (Iqbal *et al*., (2004)*;* mercuric sulphate in *Cirrhinus mrigala* (Gupta and Kumar, 2006); sodium dichromate in *Carassius auratus* (Velma and Tchounwou, 2010)*;* zinc sulphate in *Clarias batrachus* (Joshi 2011); cadmium sulphate in *Tilapia mossambica* (Jalaludeen *et al*., 2012); lead acetate in *Clarias gariepinus* (Al-Balawi *et al.*, 2013); potassium dichromate in *Labeo rohita* (Praveena and Rao, 2013); sodium cyanide in *Cyprinus carpio* (David and Kartheek, 2014) and dyestuff and chemical wastewater in *Clarias lazera* (Abdel-Moneim *et al*., 2008).

Vacuolization, necrosis, hyperplasia, accumulation of proteinaceous content in Bowman's capsule, hydropic swelling of tubular cell and numerous dark granule accumulations in many tubules have been recorded due to heavy metals in wild fishes (Kumanda *et al*., 1973; Al- Mohanna, 1994; Dallinger *et al.*, 1987); copper in *Heteropneustes fossilis, Channa punctatus* (Gupta and Rajbanshi, 1979, 1986); cadmium in *Cyprinus carpio* (Thiruvalluvan *et al*., 1997); *Leiostomus xanthorus* (Hawkins *et al*., 1980), *Salmo gairdneri* (Forlin *et al*., 1986), *Lates calcarifer* (Thophon *et al.,* 2003), elson, mercuric chloride and aqueous ammonia in *Channa punctatus* (Banerjee and Bhattacharya, 1994); zinc in *Cirrhinus mrigala* (Gupta and Sharma, 1994), *Channa punctatus* (Gupta and Shrivastva, 2006); mercuric chloride in *Cirrhinus mrigala* (Sastry and agarwal, 1997), *Ctenopharyngodon idella* (Khan *et al.,* 2004); nickel chloride in *Coregonus clupeaformis* (Ptashynski and klaverkamp, 2002) and *Hypophthalmichthys molitrix* (Athikesavan *et al*., 2006); mercuric sulphate in *Cirrhinus mrigala* (Gupta and Kumar, 2006); chromium in *Oncorhynchus tshawytscha* (Farag *et al*., 2006); cadmium and zinc in *Salmo trutta* (Besirovic

et al., 2010); sodium dichromate in *Carassius auratus* (Velma and Tchounwou, 2010)*;* cadmium sulphate in *Tilapia mossambica* (Jalaludeen *et al*., 2012); potassium dichromate in *Labeo rohita* (Praveena and Rao, 2013); copper sulphate and copper nano particles in *Oncorhynchus mykiss* (Al-Bairuty *et al*., 2013); sodium cyanide in *Cyprinus carpio* (David and Kartheek, 2014) However, vacuolization has also been seen due to the effect dyestuff and chemical wastewater in *Clarias lazera* (Abdel-Moneim *et al*., 2008) and malachite green dye in nile tilapia (EL-Neweshy and Abou Srag, 2011).

Destroyed/degenerated tubules, desquamation of tubular cells, karyolysis, rupture of glomerular wall, haemorrhages, disintegration /disorganization of cells of both renal /hematopoietic systems, thickening of basal lumen of nuclei, enlargement of renal tubules, disorganized blood capillaries, desquamation of epithelial lining, oedema, dilation of renal tubules, pyknosis and dilatation of capillaries have been observed due to the exposure of heavy metals in wild fishes (Kumanda *et al*., 1973; Dallinger *et al.*, 1987, Al- Mohanna, 1994); cadmium and methyl parathion in *Cyprinus carpio* (Thiruvalluvan *et al*., 1997); cadmium chloride in *Leiostomus xanthorus* (Hawkins *et al*., 1980), *Salmo gairdneri* (Forlin *et al*., 1986), *Coregonus clupeaformis* (Ptashynski and klaverkamp, 2002), *Lates calcarifer* (Thophon *et al.*, 2003); elson, mercuric chloride and aqueous ammonia in *Channa punctatus* (Banerjee and Bhattacharya, 1994); zinc in *Cirrhinus mrigal*a (Gupta and Sharma, 1994), *Channa punctatus* (Gupta and Shrivastva, 2006); mercuric chloride in *Cirrhinus mrigala* (Sastry and agarwal, 1997), *Ctenopharyngodon idella* (Khan *et al.*, 2004); nickel chloride in *Hypophthalmichthys molitrix* (Athikesavan *et al*., 2006); mercuric sulphate in *Cirrhinus mrigala* (Gupta and Kumar, 2006); chromium in *Oncorhynchus tshawytscha* (Farag *et al*., 2006); cadmium and zinc in *Salmo trutta* (Besirovic *et al*., 2010); sodium dichromate in *Carassius auratus* (Velma and Tchounwou, 2010)*;* cadmium sulphate in *Tilapia mossambica* (Jalaludeen *et al*., 2012; Sounderraj *et al*., 2012); sodium cyanide in *Cyprinus carpio* (David and Kartheek, 2014); dyestuff and chemical

wastewater in *Clarias lazera* (Abdel-Moneim *et al*., 2008) and malachite green dye in Nile Tilapia (EL-Neweshy and Abou Srag, 2011).

Damage to the epithelium of some renal tubules, increased Bowman's space, expansion of renal tubules, tubular dilation and shrinkage in lumen of collecting tubule have been observed due to the effect of copper in *Heteropneustes fossilis* and *Channa punctatus* (Gupta and Rajbanshi, 1979, 1986); chromium in *Oncorhynchus tshawytscha* (Farag *et al*., 2006); sodium dichromate in *Carassius auratus* (Velma and Tchounwou, 2010); copper sulphate and copper nano particles in *Oncorhynchus mykiss* (Al-Bairuty *et al*., 2013) and sodium cyanide in *Cyprinus carpio* (David and Kartheek, 2014).

Effect of tannery effluent on the kidney of fishes have also been reported in *Oreochromis mossambicus* (Navraj and Yasmin, 2012), *Tilapia mossambica* (Sounderraj *et al*., 2012). They observed very few changes like exfoliation and swollen cells with pyknotic nuclei and destroyed/ degenerated tubules. However, detailed study of histopathological alterations in the kidney of *Labeo rohita* has been done during the present investigation. However, there is no report on the histopathological changes in kidney due to the effect of paint industrial effluent.

In fish kidney, metals/pollutants enter kidney *via* gills and effect the kidney through metallothionein (MT), which inhibit Na+/H+ exchange and also disturb the osmoregulation process. These changes lead to various structural damages in the tissue like hydropic swelling, hyaline droplets, hypertrophy, pyknosis, accumulation of dark granules (Vilella *et al*., 1991; George and Olsson, 1994, Olsson *et al*., 1996; Chan and Rennert, 1981; Hamer, 1986; Dunn *et al*., 1987; Kagi and Schaffer, 1988; Roesijadi, 1992; Venkataramana and Radhakrishnaiah, 1987; Singhal and Jain, 1997). Heavy metals are also responsible for internal exhaustion (hypoxia) which changes the metabolic activity and forms diavalent ions of metals, causing kidney lesions (Rasquin and Rosenbloom, 1954).

In present study, fishes exposed to both the effluents resulted in various histopathological alterations in the kidney of *Labeo rohita* might be due to

presence of divalent ions of heavy metals, which interact with metallothionenin *via* gills, inhibit the Na+, K+ ATPase pump result in disturbed osmoregulation and metabolic pathways as suggested by earlier worker.

Liver:

In fish, liver plays an important role in metabolic processes and detoxification of many xenobiotics. Toxicants cause severe alterations in liver. In the present study, at low concentrations of both effluents (1.93% tannery and 3.95% paint), main alterations like fatty acid degradation, dilation of sinusoids, presence of melano-macrophage centers, hypertrophy of cell, disintegration of central vein have been observed, while in high concentrations (7.74% tannery and 15.81% paint), significant changes like hemorrhages, disintegration of central vein, necrosis, dilation of sinusoids, melano-macrophage centres, hyperplasia, fatty acid degradation, intracellular odema and aneurysm have been recorded. However, intensity of these alterations is more in tannery as compared to paint effluent.

Earlier, effect of heavy metals effluents, pesticides and herbicides on the liver of fishes have been done by many workers. Vacuolization, pyknotic nuclei, haemorrhages, hypertrophy, hyperplasia, blood vessel congestion, loss of nuclear membrane of hepatocytes, cirrhosis, distended parenchyma, distended sinusoids, degeneration of nuclei, rupture of blood sinusoids, loss of shape of hepatocytes, cloudy swellings, atrophy, loss of polygonal shape of liver cells, formation of spaces in the tissues, splitting of the cells, karyomegaly, intense hemolysis, necrosis, clumping of chromatin and its aggregation at the centre in liver hepatocytes, basophilic cell foci and presence of dark cells have been observed due to the effect of heavy metals like zinc in fishes (Wong *et al*., 1977); lead in *Channa punctatus* (Sastry and Gupta, 1978 b); copper and cadmium in *Cyprinus carpio* (Dalela and Sharma, 1984); copper in *Ictaburus nebulous* (Benedetti *et al.,* 1981), *Channa punctatus* (Khangarot, 1992); zinc chloride in *Channa punctatus* (Singh and Bhati, 1994); waterborne copper in *Oreochromis niloticus* (Figueiredo-Fernandes *et al.,* 2007; Antonio *et al*., 2007); copper and zinc in *Puntius conchonius* (Kumar and Pant, 1981);

copper sulphate in *Colisa fasciatus* (Singh, 1983), *Tinca tinca* (Roncero *et al.*, 1992), *Brachyodanio rerio* (Paris-palacios *et al.*, 2000), *Catla catla* (Patel and Bahadur, 2011); DDT, malathion and mercury in *Sartherodon mossambicus* (Ramlingam, 1988); copper sulphate and copper nano particles in *Oncorhynchus mykiss* (Al-Bairuty *et al*., 2013); mercury chloride *Gambusia affinis* (Bakre, 1985); mercuric fungicide in *Channa punctatus* (Ram and Sathyanesan, 1987); nickel in *Anabas testudineus* (Jha and Jha, 1994); nickel chloride in *Hypophthalmichthys molitrix* (Athikesavan *et al*., 2006); mercury in *Channa punctatus* (Patil, 1995); zinc sulphate in *Clarias batrachus* (Bhoraskar and Kothari, 1997; Joshi, 2011); chronic exposure of heavy metals in *Cyprinus carpio* (Pourahamad and Brien, 2000; Varanka *et al*., 2001); cadmium chloride in *Lates calcarifer* (Thophon *et al.*, 2003); sodium perchlorate in *Poecilia sphenops* (Burcu *et al*., 2009); polluted water containing heavy metal salt in *Oreochromis niloticus* (Osman *et al*., 2009); sodium dichromate in *Carassius auratus* (Velma and Tchounwou, 2010); chlorides of chromium, nickel and zinc in *Labeo rohita* (Bhatkar, 2011); cadmium sulphate in *Tilapia mossambica* (Jalaludeen *et al*., 2012); chlorides of mercury and copper in *Channa gachua* (Deore and Wagh, 2012), *Tilapia mossambica* (Sounderraj *et al*., 2012)*;* lead acetate in *Clarias gariepinus* (Al-Balawi *et al.*, 2013). However, industrial effluents like textile effluent in *Oncorhynchus mykiss* (Marlasca and Sampera, 1998); paper/pulp and kraft paper industries wastewater in *Microgadus tomcod* (Couillard *et al.*, 1999), *Carassius auratus*; *Dicentrarchus labrax* (Mario *et al.*, 2011); cassava mill effluent in *Clarias gariepinus* (Adeyemo, 2005); sago effluent *Oreochromis niloticus* (Wade *et al.*, 2002); dyestuff and chemical wastewater in *Clarias lazera* (Abdel-Moneim *et al*., 2008) have also been responsible for similar alterations in the liver.

Pigmented macrophage aggregates, oedema, degeneration of hepatocytes leading to tumor and sycytium formation, proliferation of hepatocytes, dilation of sinusoid, bile canliculi, bile pigments, cellular damage and increase in blood sinuses, disarray of hepatic cords, fatty acid degradation, infiltration of blood filled spaces due to inflammatory cells, red blood cell

occlusion in portal vessels, vacuolar degeneration of hepatocytes, engorged blood vessel congestion have also been noticed due to the effect of mercuric chloride in *Gambusia affinis* (Bakre, 1985); mercuric fungicide in *Channa punctatus* (Ram and Sathyanesan, 1987); DDT, malathion and mercury in *Sartherodon mossambicus* (Ramlingam, 1988); mercury in *Channa punctatus* (Patil, 1995); copper sulphate in *Brachyodanio rerio* (Paris-Palacios *et al.*, 2000), *Catla catla* (Patel and Bahadur, 2011); cadmium chloride in *Lates calcarifer* (Thophon *et al.*, 2003); sodium perchlorate in *Poecilia sphenops* (Burcu *et al*., 2009); cadmium sulphate in *Tilapia mossambica* (Jalaludeen *et al.*, 2012); *Heteropneustes fossilis* (Sastry and Subhadra, 1982, 1985) and paper/pulp and kraft paper industries wastewater in *Microgadus tomcod* (Couillard *et al.*, 1999), *Carassius auratus* and *Dicentrarchus labrax* (Mario *et al.*, 2011).

Blood congestion in sinusoids, increased liver glycogen and hydropic swelling of hepatocytes, shrinkage in the liver cells, degenerated nuclei, splitting of connecting tissues and hyperchromatic nuclei have been observed due to zinc in fish (Wong *et al*., 1977; Kumar and Pant, 1981) and cadmium chloride in *Heteropneustes fossilis* (Sastry and Subhadra, 1982, 1985); *Lates calcarifer* (Thophon *et al.*, 2003). However, enlargement of hepatocytes has been observed due to impact of nickel in *Anabas testudineus* (Jha and Jha 1994) and nickel chloride in *Hypophthalmichthys molitrix* (Athikesavan *et al*., 2006).

Vacuolization, necrosis and hyperplasia have been reported due to the effect of tannery effluent *Heteropneustes fossilis* (Sastry and Subhadra, 1982, 1985), *Oreochromis mossambicus* (Navraj and Yasmin, 2012); *Tilapia mossambica* (Sounderraj *et al*., 2012) and *Danio rerio* (Sivakumar *et al*., 2015). However, detailed study of histopathological alterations in the liver of *Labeo rohita* has been done during the present investigation. However, there is no report on the histopathological changes in liver due to the effect of paint industrial effluent.

Histopathological alterations in the liver like blood congestion leading to aneursym, accumulation of dark granules, vacuolization or intracellular oedema and hydrophic swelling of hepatocytes are due to the presence of Metallothionein (MT), which uptake by gills and circulates to the liver through gills and responsible to the tissue damage (Brown *et al.*, 1984; Olsson *et al.*, 1989; Olsson and Hogstrand, 1987; Wicklund-Glynn and Olsson, 1991; Glynn *et al*., 1992). More numbers of melano-macrophage in the effected liver also involves in recycling of endogenous materials from damaged cells (Haaparanta *et al*., 1996).

In present study, fishes exposed to both the effluents resulted in various histopathological alterations in liver of *Labeo rohita* might be due to the presence of heavy metals, which interact with metallothionenin *via* gills circulate to the liver and responsible for tissue damage. Further, the increased numbers of melano-macrophage also indicate the recycling of endogenous materials from damaged cells as suggested by earlier workers.

Inflammatory cells or lymphatic infiltrations in gills, kidney and liver are formed due to necrosis and degeneration of tubular epithelium by toxicants (Kurtovic *et al*., 2008). Generation of reactive oxygen species by heavy metals leads to per-oxidation of unsaturated fatty acids in cell membranes also leading to necrosis (Lloyd *et al*., 1998 and Susa *et al*., 1996). During the present study, lymphatic infiltrations in gills, kidney and liver of *Labeo rohita* exposed to both the effluents might be due to the presence of heavy metals as suggested by earlier workers.

Semi-Quantitative Histological Assessment:

Earlier, the organ indices have been calculated in *Salmo trutta* from four Swiss rivers (Zimmerli *et al.*, 2007); *Astyanax altiparanae* from Cambe stream, (Silva and Martinez, 2007); *Clarias gariepinus, Clarias ngamensis, Oreochromis andersonii, Serranochromis angusticeps* from Okavango delta, (Van Dyk *et al*., 2009); *Hydrocynus vittatus* from Pongolapoort Dam, (McHugh *et al*., 2011); *Siganus canaliculatus* exposed to crude oil and dispersed oil (Agamy, 2012). During present study, all types of histological

alterations in the selected target organs have been analyzed by means of the semi-quantitative histological assessment protocol (Bernet *et al*., 1999). The aim of the semi-quantitative assessment is the proper interpretation of the histopathological evaluations because control of both the effluent is also showing minor structural disorders (Bernet *et al*., 2004).

The mean index values of gill, kidney and liver range in class II ((index <10-25), which represent the moderate alterations at low concentrations of both the effluents, while these values are placed in class III (index <26-35), which show pronounced alterations at high concentrations of both effluents as compared to control. I_{rp} index (Reactive index) values clearly depict that there are more regressive changes in tannery effluent as compared to paint effluent. Total reaction index values ($TotI_{rp}$) and Total organ indices ($TotI_{org}$) indicate that tannery effluent is more toxic and causes more damage in the organs of fishes as compared to paint effluent. Organ indices (I_{org}), gills are highly affected than kidney and liver in both the effluents because gills are directly in contact with toxicants present in water.

Labeo rohita, a popular edible carp exposed to tannery and paint industrial effluents has showed genotoxic and histopathological damage. The results depicts that tannery effluent is more toxic than paint effluent. Low concentrations of both the effluents are also showing aberrations in chromatin as well as in tissue structure. It clearly indicates that even low concentrations are also harmful to aquatic fauna. The cumulative effect of both the effluents on fishes has been done for the first time, while few reports are available on tannery effluent but effect of paint effluent has been done for the first time.

The fishes proved to be very sensitive to the effluents. The industries discharge their effluent directly into the river ecosystem without proper treatment. It is highly recommended for both small scale as well as large scale industries to treat their waste water in treatment plant before dumping. Legal action should be taken to stop dumping of untreated wastes to rivers. Healthy aquatic environment should be maintained to save fish fauna as well as human health.