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Impact of Mercury on the Liver and Ovary of Yellowfin Sea bream (*Acanthopagrus latus*) in the Persian Gulf

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ABSTRACT: This study reports the incidence of histological alterations in morphometric changes in the liver and ovary of *Acanthopagrus latus* collected from an urban stream impaired by anthropogenic activities and from a clean site (reference). Mercury concentration was determined using a standard cold vapor atomic absorption. The results showed that mercury concentration at the creek waters, and specially sediments along Mahshahr coast, was higher than in the other marine environment, and in Zangi was lower than in the other sites of sampling. Several liver lesions were identified, including enlarged and lateral nuclei, nuclear degeneration and vacuolation, oncotic, apoptic, focal, massive, centrilobular and periportal necrosis, atrophy, lipidosis, hydropic and cloudy swelling, oval cell proliferation, cirrhosis, hemorrhage, macrophage aggregates, bile stagnation, dilation of sinusoid, intracellular edema, and dark granules. Ovary histology revealed some changes in higher concentrations such as increase in the number of pre-vitellogenic oocytes, oocyte atresia, and adhesion in the ovaries of females that were sampled from the sites with high concentration of mercury. In this study, existence of mercury in Mahshahr coast is demonstrated and high incidence of histological alterations in the liver and ovary of *A. latus* is an evidence of the poor environmental quality of creek waters. Hence, histopathological changes were induced by the mercury adversely affected the proper functioning of these organs in these fish.

Keywords: *Histopathology, Liver, Mercury, Ovary, Yellowfin Sea bream*

1 INTRODUCTION

Fish can be used as a monitoring tool for the quality of the urban aquatic environment, and fish histopathology with a broad range of causes is increasingly being used as an indicator of environmental stress since it provides a definitive biological end-point of historical exposure (Khoshnood *et al.*, 2011; Xing *et al*., 2012 a,b). Fish tissues are sensitive indicators of marine pollutant, which can bio-accumulate pollution concentration as both organic and inorganic forms in the marine environment (Gochefeld, 2003). Correlation between exposure to environmental contaminants and fish hepatic lesions has been well established. For example, toxicopathic liver lesions in fish species are sensitive signs of hepatic injury, and have been used as the biomarkers of chemicals in environmental risk

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assessments (Stehr *et al.*, 2004).

Non-essential heavy metals such as mercury can be considered as serious pollutants of aquatic environments. Mercury is a toxic heavy metal, and is considered as ecosystem contaminant in marine food webs (Boening, 2000). Because of specific nature and abundance of mercury in some industrial effluents, it is known as one of the most disastrous heavy metals (Gochefeld, 2003 Cizdziel, 2003; Gilbertson and Carpenter, 2004). Therefore, mercury accumulation in the living organisms can lead to many ecophysiological abnormalities, which could be considered as biomarker of mercury toxicity (Martin-Diaz *et al.*, 2005; Hosseini *et al*., 2013).

Due to high value of natural resources and also oil and petroleum capacity, the Persian Gulf is surrounded by many industrial and sailing activities; these anthropogenic attempts can lead to bio-accumulation of hazardous chemicals by marine organisms. Continuous discharge of industrial effluents into the Persian Gulf is considered as a serious risk to the survival of aquatic animals. In the Persian Gulf ecosystem, costal and creek waters are susceptible areas for pollution accumulation (Sheppard, 1993; Agah *et al.*, 2009). Mahshahr creeks, located in the northwest of the Persian Gulf, are some of the most critical areas of the Persian Gulf that are influenced by the above mentioned conditions (Agah *et al*., 2009).

Although most studies focused on mercury distribution and accumulation, the literature on the effect of mercury on tropical fish and its toxic effects on their tissues and organs is rare (Oliveira Ribeiro *et al.*, 2002b). Exposure to marine pollutants can induce different lesions and injuries in different fish organs (Oliveira Ribeiro *et al.*, 2006).

Among the aquatic organisms, due to widespread distribution of fishes, they are attracted more concerns among the most scientists. Due to high market value, easy adaptation to captivity and availability of production technology in experimental conditions, Yellowfin Sea bream (*Acanthopagrus latus*) is considered the most commercially important marine fish in the Islamic Republic of Iran (Hesp *et al.*, 2004; Naderi *et al.*, 2012). However, because of its continuous exposure to toxic chemical-rich industrial effluents that are discharged into the Persian Gulf and their enclosed creeks, this fish has long been put under threat. Since *A. latus* is widely found in polluted creeks and also captured for human consumption, it can affect human health.

The importance of mercury for histopathological changes in various fish species has been confirmed by several studies (Oliveira Ribeiro *et al.*, 2002 a,b); however, damages caused through this heavy metal in the liver and ovary of Yellowfin Sea bream are largely unknown. Therefore, the aim of the present study was to determine the concentration of mercury in Mahshahr coast and also provide baseline knowledge on the prevalence of histopathological liver and ovary lesions in *A. latus* under environmental exposure to mercury.

2 MATERIALS AND METHODS

2.1 Environmental test

For environmental test, in the first step, 26 creeks in Mahshahr region (northwest of the Persian Gulf) (Figure 1) were analyzed to determine their water and sediment mercury levels. Then four highly polluted creeks (Jafari, Ghazaleh, Majidieh and Petroshimi) and one lowly polluted creek (Zangi) were chosen as sampling sites. At each creek, three sampling points were chosen. One water and sediment sample and three female fish (170 g, immature males) were collected from each sampling site.

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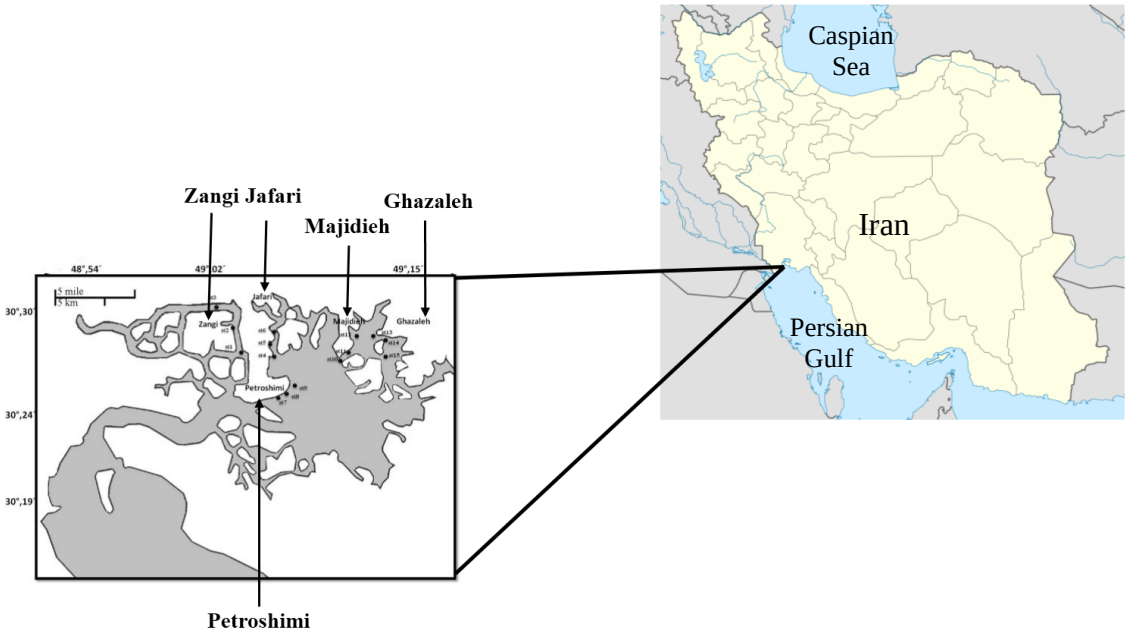

Figure 1: Map of the study area with reference to sampling areas. Sea breams were sampled from the Jafari, Ghazaleh, Majidieh, Petroshimi and Zangi sites in Mahshahr coast (Nabavi and Parsa, 2013).

2.2 Mercury analysis

The water samples were filtered with Millipore strainer (YM-10; Millipore Corporation, Billerica, MA, USA) (mesh size 0.45 µm). The filtrate was then acidified with 2 mg l^{-1} of 20% $K_2Cr_2O_7$ (w/v) prepared at nitric acid (Rand, 1976), and the resultant solution was stored at -4 ^oC until mercury analysis. For weight stabilizing, the sediments were freeze-dried (Shi *et al.*, 2005), sieved through 63 µm meshes, and allowed to settle; then the supernatant water was decanted and homogenized. Finally, the powdered sediment was dissolved in 60 mL container containing 4 mL of nitric acid and 2 mL of concentrated sulfuric acid. The mixture was digested at 90°C for l-2 h in a hot plate. Upon cooling, 1 mL $K_2Cr_2O_7$ or 0.5 mL BrCl was added. The solution was filtered using Whatman GFK No.1 filter paper, diluted to 50 mL with deionized water (Rand, 1976), and then preserved prior to Hg analysis.

Mercury concentrations were determined at the Department of Marine Chemistry Laboratory, Khorramshahr University of Marine Science and Technology, Iran using a standard cold vapor atomic absorption (CV-AAS) apparatus (Unicam 919, ATI Unicam, Cambridge, UK) equipped with Hg cold vapor generator (VGA 77; EPA, 1992) (Lewis and Chancy, 2008).

2.3 Histopathological analysis

A section of the liver and ovary right lob was dissected and examined macroscopically. The tissues samples were preserved in Bouin's solution over 24 h. The dissected tissues were washed in ice cooled 0.9% sodium chloride solution, and subsequently, fixed in 10% formalin solution for 48 h. Next, the tissues were transferred to 70% ethanol (Hosseini *et al.*, 2013). After incubation, dehydration and lastly xylene, paraffin mixture was put in tissue processor (Triangle Biomedical Sciences USA). Then the tissues were paraffinized and sectioned using an ultra microtome (Olympus CUT 4055E, USA) to obtain sections of 5 µm

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in thickness. The sectioned tissues were fixed on the microscope slides and air-dried for 24 h. The slides were later stained with hematoxylin and counter stained with eosin (Haschek *et al.*, 2010; Khoshnood et al., 2011). In this study, morphological abnormalities were identified through digital optical imaging technique (Nikon EC 600 Eclipse). The incidence of changes was reported by a qualitative evaluation, plus a semi-quantitative scale scored in four categories based on the intensity of alterations: none (1), mild (2), moderate (3) and severe (4) (Di Giulio and Hinton, 2008).

3 RESULTS AND DISCUSSION

3.1 Mercury

Significant differences were found among the sampling sites (creeks). Concentrations of mercury in the water and sediments were determined, and the highest concentration was related to the Mahshahr coast. The gradient of mercury concentrations in various sites was observed. Station of Zangi had the lowest mercury level in all measurements (3.91 ± 0.14 ug l^{-1} in water column; 458.29 ± 39.83 ppb in sediment), and other sites had higher levels of mercury concentrations. Stations of Majidieh and Petroshimi were noticeably close to an area with industrial activities (oil and petrochemistry, respectively), and the observed high mercury levels were predictable.

The analytical data were normalized to the distance from the creeks with water and sediment mercury (Figure 2).

The concentrations of both water and sediment mercury were strongly higher in the polluted creeks than in the clean ones; however, this increase in water mercury was realizable. In general, the highest concentrations of mercury for water and sediment had a similar pattern. These observations strongly suggest that anthropogenic activities can significantly increase mercury levels in the water and sediment, especially in closed creeks. These differences denote a contamination gradient according to the distance from the point source of mercury into the system as Petroshimi and Majidieh are the nearest and Zangi and Jafari the furthest creeks to the anthropogenic activities near the Mahshahr coast.

3.2 Histopathological analysis

The severity of hepatocyte abnormalities increased along with increase in mercury concentration. The tissues damages and injuries after mercury exposure are summarized in Tables 1-3 and Figures 4-6. Mercury induced various pathological alterations in the liver of Yellowfin Sea bream. The lesion index for liver showed difference between the Zangi site (lowest concentration) and other sampling sites. The most common pathological modifications observed in the samples were increase in lipid droplets (lipidosis) occurrence, nuclei change, necrosis, swelling, degeneration, hepatic cells' cytoplasmic vacuolization, hemorrhage in blood vessels, macrophage aggregates, cirrhosis, bile stagnation, dilation of sinusoid, atrophy, and pre-necrotic lesions within many hepatocytes compared to the Zangi samples. Lipid bodies were also sporadically visible in the cytoplasm of the hepatocytes. Furthermore, the presence of megalocytosis was higher in the mercury-exposed fish than the Zangi samples. Intoxicated hepatocytes were enlarged with clear staining vacuoles, which compressed the cytoplasm and nuclei to the cell margins. In some samples, the cord-like layout of the hepatocytes was conserved. However, in the severely intoxicated samples, there were severe disruptions in the normal Bilroth cord layout.

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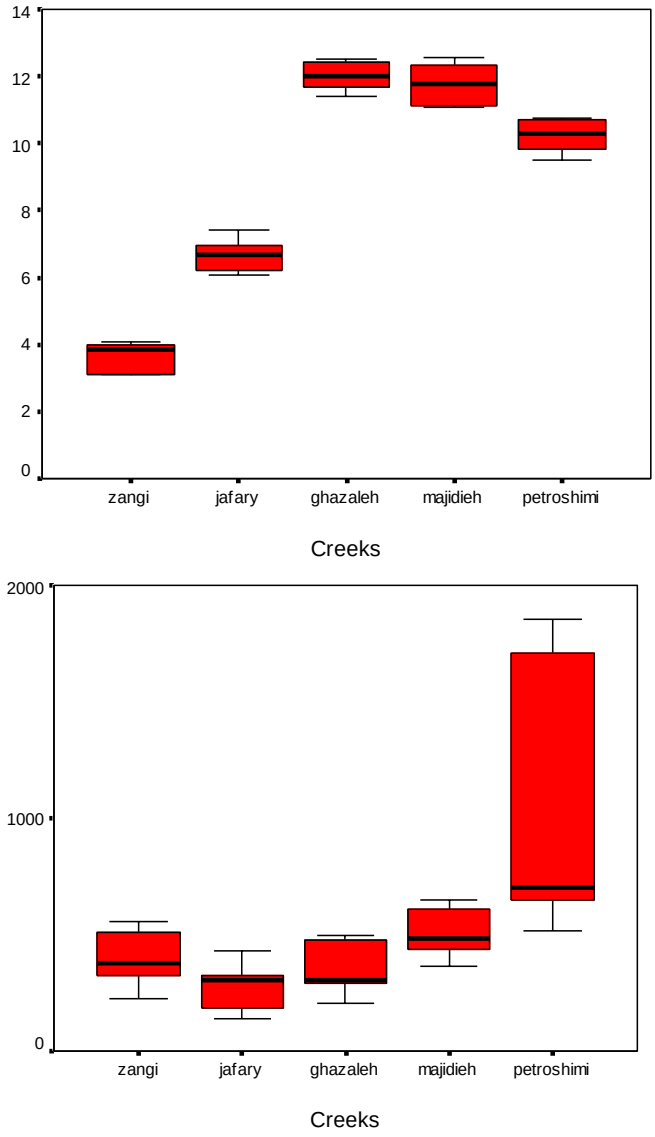

Figure 2: Environmental concentration of mercury chloride (µg l^{-1}) in the water and sediment of different creeks in Mahshahr coast with different sources of pollutant (box plots contain mean and standard deviation).

The Kupffer cells were absent in the samples. Cellular swelling was observed in some of the samples. Degenerated cell number was increased in the specimens originating from the sites with high concentrations of mercury as compared to the Zangi samples. These degenerated nucleuses were commonly observed in most of the intoxicated fish. The hepatic tissue of the Zangi fish showed the normal histological characteristics reported for teleosts (Cengiz *et al*., 2001). It was constituted by hepatocytes, which were large in size, polygonal in shape with centrally located nuclei, and polyhedral form with a spherical nucleus with one or more nucleolus. There were also the portal vein and the central vein, branching into the sinusoids. A large number of blood sinusoids (separating the hepatic cords one by one) were observed (Figure 3).

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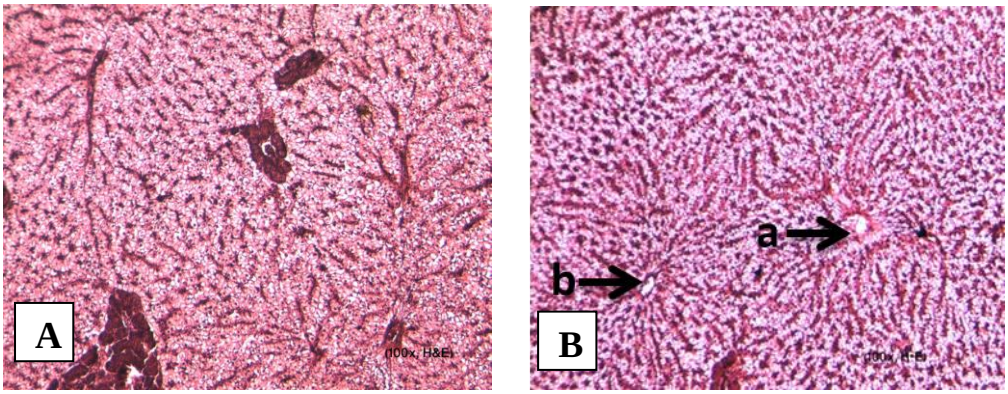

Figure 3: Light microscope features of hepatic parenchyma of Yellowfin seab ream in Zangi samples (A) hepatopancreas, (B) portal vein (a), and central vein (b) (100 x).

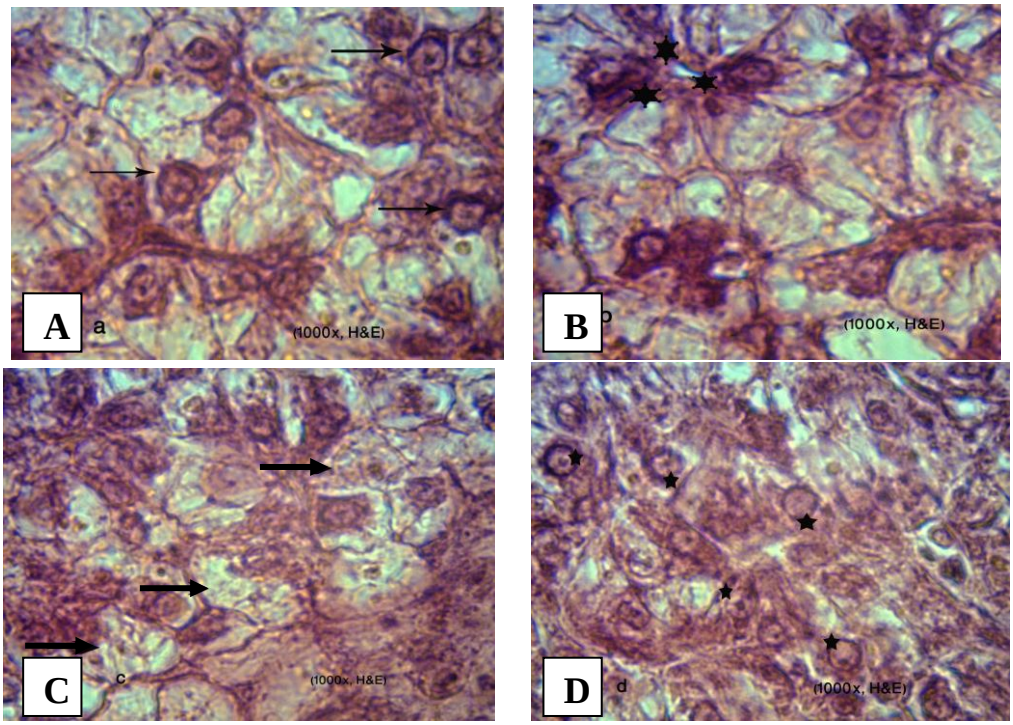

Figure 4: Light microscope features of hepatocyte nuclear lesions in the liver of Yellowfin Sea bream exposed to mercury (A) enlarged nuclei, (B) lateral nuclei, (C) nuclear degeneration, and (D) nuclear vacuolation (1000 x)

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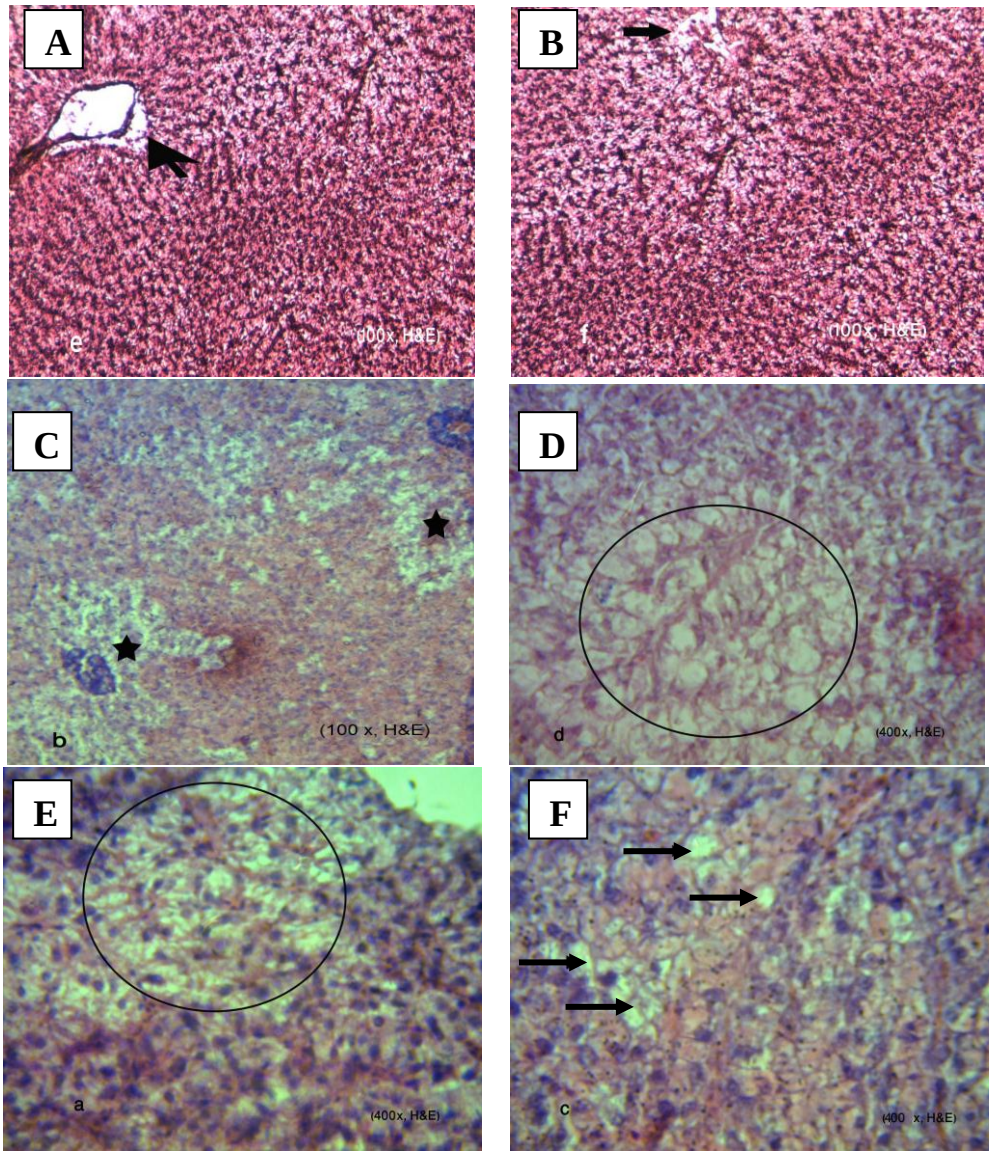

Figure 5: Light microscope features of necrosis lesions in the liver of Yellowfin Sea bream exposed to mercury (A) centrilobular necrosis, (B) periportal necrosis, (C) apoptotic necrosis (100 x); (D) massive necrosis, (E) oncotic necrosis, and (F) focal necrosis (400 x).

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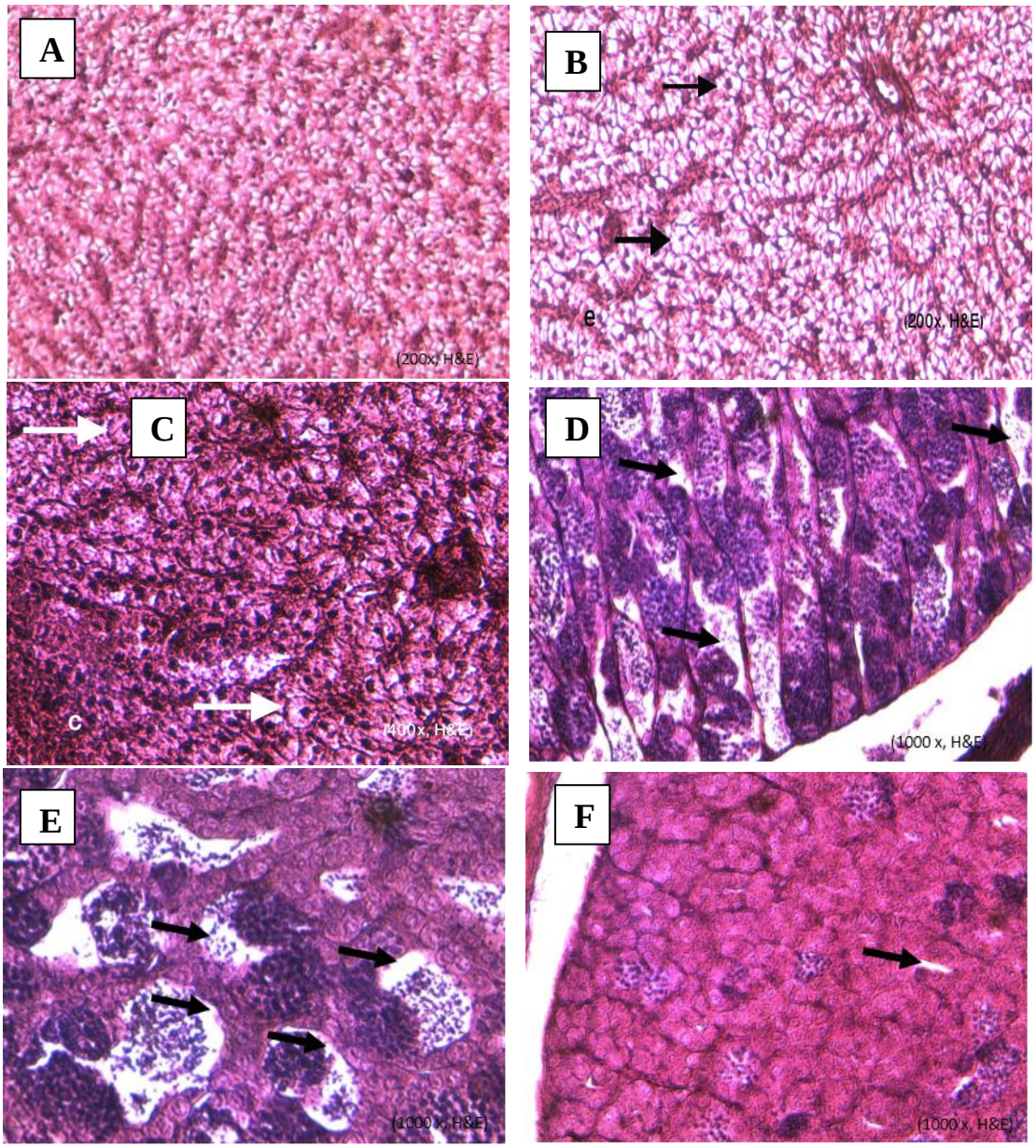

Figure 6: Light microscope features of other hepatocyte lesions in the liver of Yellowfin Sea bream exposed to mercury (A) oval cell proliferation, (B) cloudy swelling, (200 x); (C) megalocytosis (400 x); (D) atrophy, (E) lipidosis, and (F) hydropic swelling, (1000 x).

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Table 1: Summarized hepatocyte nuclear lesions in the liver of Yellowfin Sea bream during exposed to mercury

Lesion	***Environmental* exposure (creek)**				
	Zangi	Jafari	Ghazaleh	Majidieh	Pertoshimi
Enlarged nuclei	-	-	+	+	+
Lateral nuclei	+	-	+	+	-
Nuclear degeneration	-	+	++	+	++
Nuclear vacuolation	-	++	+	++	+

Score value: None (−), mild (+), moderate (++) and severe (+++)

Table 2: Summarized necrosis lesions in the liver of Yellowfin Sea bream exposed to mercury

Lesion	***Environmental* exposure (creek)**				
	Zangi	Jafari	Ghazaleh	Majidieh	Pertoshimi
Oncotic necrosis	-	++	+	+	+
apoptotic necrosis	-	-	++	+	++
Focal necrosis	+	++	++	+++	++
Massive necrosis	-	+	-	+	+
Centrilobular necrosis	-	-	+	++	+
Periportal necrosis	-	-	+	-	+

Score value: none (−), mild (+), moderate (++) and severe (+++).

Table 3: Summarized other hepatocyte lesions in the liver of Yellowfin Sea bream exposed to mercury

Lesion	***Environmental* exposure (creek)**				
	Zangi	Jafari	Ghazaleh	Majidieh	Pertoshimi
Atrophy	-	-	+	+	-
Lipidosis	+	+	++	++	++
Megalocytosis	-	-	-	-	-
Hydropic swelling	-	+	++	+	+
Cloudy swelling	+	++	+++	++	+++
Oval cell proliferation	-	-	+	++	++

Score value: none (−), mild (+), moderate (++) and severe (+++).

The bile ducts lined by cubic epithelial cells were distributed through the hepatic parenchyma and usually associated with the port vein. There were bile canaliculi, surrounded by the plasmatic membrane of the hepatocytes. In the Zangi samples, very homogeneous hepatic parenchymas were observed. The hepatocytes were arranged in cords, generally, two cells thick between two contiguous sinusoids. Microscopic analysis of the liver sections revealed a pattern of arrangement of hepatocytes different from that of mammals. A double row of hepatocytes was obvious. The bile preductular epithelial cells were found between the hepatocytes rows in the liver tubule; however, the nuclei of hepatocytes contrast with the flattened and elongated nuclei of different cell types. Also larger elements of the bile passage ways were present in the parenchyma of the examined fish liver.

With regard to the hepatic tubules, it can be said that tubules curve, forming a complex

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continuum of parenchyma, that tunneled by an extensive microcirculation. This large blood supply leads likely to intensive mercury exposure and accumulation in the hepatocytes and biliary epithelial cells. The results showed that mercury induced a wide range of histopathologiocal changes in the liver of Yellowfin Sea bream.

Histopathological changes were also noticed in the ovary tissue of mercury-exposed fish. The ovary of the exposed fish displayed increased numbers of pre-vitellogenic oocytes, oocyte atresia and adhesion (Table 4 and Figure 7).

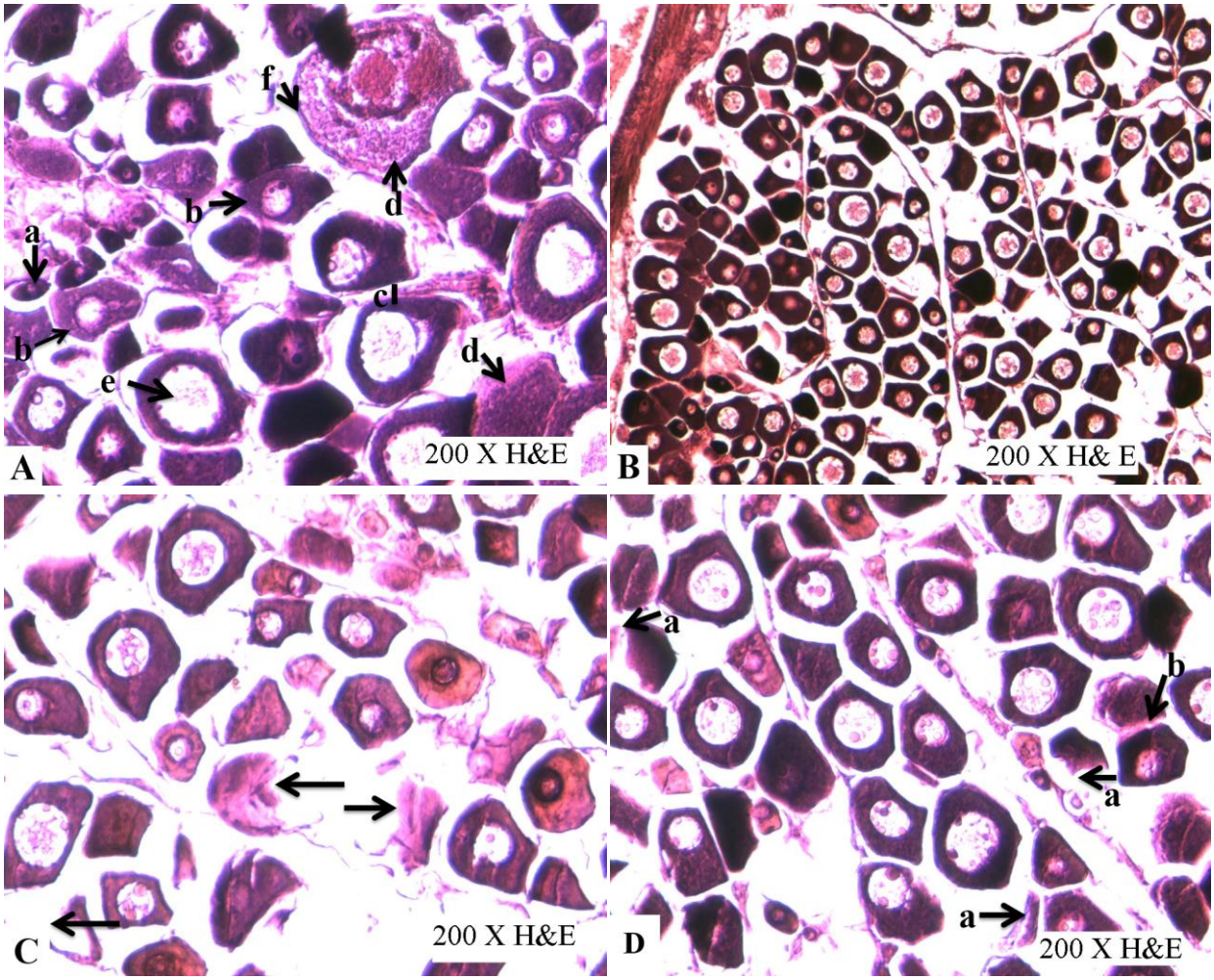

Figure 7: Light microscope features of the ovary of Yellowfin Sea bream in Zangi samples: (A) (a) stage II, (b) stage III, (c) stage IV, (d) stage V, (e) nucleus, and (f) follicular epithelium; (B) mercury-exposed fish: increase in the number of pre-vitellogenic oocytes; (C) mercury -exposed fish: oocyte atresia; and (D) mercury-exposed fish: (a) oocyte atresia, and (b) adhesion (200 x).

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Table 4: Summarized ovarian lesions of Yellowfin Sea bream exposed to mercury

Lesions	***Environmental* exposure (creek)**				
	Zangi	Jafari	Ghazaleh	Majidieh	Pertoshimi
Increased oocytes	-	+	+	++	++
Oocyte atresia	-	-	+	++	+++
Adhesion	-	-	+	+++	+++

Score value: none (−), mild (+), moderate (++) and severe (+++).

Since heavy metals are stable, and permanently exist in the environment, contamination by these metals is an important problem. Since the aquatic environments are final destination for many pollutants having by natural or synthetic origin, accumulation and persistence of heavy metals in these areas can threat biological life (Hosseini *et al*., 2013). The large vacuole in the cell may associate by nuclear atrophy. Vacuolation of hepatocytes is accompanied by the protein synthesis inhibition, energy decrease, disaggregation of microtubules, and shifts in substrate utilization (Oliveira Ribeiro *et al*., 2002a). Oncotic necrosis (karyolysis, swelling and loss of cell structure) can result in reduction in cell number (Haschek *et al.,* 2010). The resulted scarring will cause inflammation; however, the reduction in tissue or organ size is a response to toxicant exposure (Di Giulio and Hinton, 2008).

In chronic exposure, the affected area is depressed below the surface of the adjacent tissue (Stehr *et al.,* 2004). Focal necrosis may be observed apparently as small pale foci. The lesions are particularly evident when they include inflammatory cells (Haschek *et al.,* 2010). Apoptosis is defined as a controlled cell death program that serves as a regulation point for biologic processes, and may be considered as the counterpoint of cell division by mitosis (Timbrell, 2009). The swollen and necrotic hepatocytes appear to compress the vascular spaces. This can explain the reason of observed small blood content in the sinusoids in the necrotic central lobular areas (Di Giulio and Hinton, 2008). Periportal necrosis has been considered as peripheral lobular necrosis. Numerous oval cells may be found in the periportal area or when hepatocyte regeneration is completely, or at least partially, blocked (Haschek *et al.,* 2010). Massive necrosis is clearly evident on gross observation. In the sub-acute phase, the affected liver has abnormal coloration (pale), and looks like swollen slightly.

The swelling hepatocytes typically compressed or displaced the adjacent structures. Staining affinity was diminished, generally, giving the cells a pale or cloudy appearance. Hydropic change was observed in almost all the treated animal's cells by enlarged pale-staining cytoplasm. A cell with disrupted homeostasis can respond differently to maintain itself short of death; this is called *adaptation* (Haschek *et al.,* 2010). Atrophy is a simple adaptation way, at the cellular level; atrophy is often a response to decreased demand for the specialized functions of a particular cell. Atrophy may lead to cell death, either accompanied by apoptotic or oncotic necrosis (Hinton *et al.,* 2001). In contrast, hypertrophy is a response to increased metabolic demand for a specialized function provided by a special cell (Di Giulio and Hinton, 2008).

Lipidosis is often observable in the cells that should metabolize more lipids for energy (Haschek *et al.,* 2010). Cell swelling is an early change that occurs in most types of liver injury, which may be an introduction to more effective changes (Hinton *et al.,* 2001). Hydropic

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swelling is a reversible damage with accumulation of water within the cell cytosol or rough endoplasmic reticulum of hepatocytes. This type of swelling may be attributed to a failure to maintain intracellular sodium ion balance (Mommsen and Moon 2005). The swollen cells have a cloudy and granular cytoplasm. Hinton *et al.* (2001) reported that although swelling is an integral part of adaptation of injured cells, they are rarely as the major indication of toxic injury.

Hemorrhage of blood vessels occurred in some of the exposed fish, especially within the portal veins. Moreover, macrophage aggregates were considered as the stromal components of the liver. The intrahepatic blood vessels were dilated and congested with blood, and inflammatory leucocytic infiltrations were observed. Hepatopancreatic alveoli of the exocrine pancreas were observed in the parenchyma, and the melanomacrophage centers were positioned close to hepatopancreas in light-brown color.

The cirrhosis parts were nodular, and tan or yellow in color. Nodules of the regenerating hepatocytes were microscopically characterized by the cell plates greater than two cells thick, and were separated by connective tissue. Cirrhosis is defined as hepatic fibrosis with nodular regeneration. It is the final, irreversible symptom of a variety of chronic pollutant lesions (Hinton *et al.* 2001). It was compatible with our results with no effects in sub-acute and more lesions in chronic treatments; thus, it can be concluded that environmental fish was at "end-stage liver".

The toxicants targeting liver are typically characterized to be cytotoxic or cholestatic. Cytotoxic mechanisms affect hepatocytes, and make a variety of liver injury. Cholestatic mechanisms affect bile flow. Intrahepatic cholestasis takes place when bile flow is blocked within the liver as it flows through canaliculi, and bile ductules (Mommsen and Moon 2005).

The cholestatic mechanisms that lead to the blockage of bile are not understood very well. "Blockage" may be due to obstruction of transport mechanisms in the cell membrane of hepatocytes (Hodgson 2004).

The types of liver lesions depend on the type of toxicant, the severity of intoxication, and the type of exposure, whether acute or chronic (Hodgson 2004). Numerous studies have reported histopathological changes in the liver of fish exposed to variety of heavy metals in marine environments (Mommsen and Moon 2005; Rabitto *et al.*, 2005; Giari *et al.*, 2007).

Some of the symptoms observed in the hepatic cells in the present study (e.g. vacuolar degeneration and lipid droplet accumulation) are in line with those reported in the other species such as *D. labrax*, *Lates calcarifer* and *Carassius carassius* that exposed to other heavy metals (cadmium) (Giari *et al.*, 2007). In fact, the presence of necrosis is as common visible damages in many tissues affected by heavy metals (Rabitto *et al.*, 2005). In fish liver, the presence of necrotic area is also related to the concentration of pollutant(s) during the detoxifying process.

Liver lesions such as irregular shaped hepatocytes, vacuolation, and nucleus in a lateral position were also described in the Siluriformes *Corydoras paleatus* exposed to organophosphate pesticides (Fanta *et al.*, 2003). Pacheco and Santos (2002) described increased vacuolisation of the hepatocytes as a signal of degenerative process that suggests metabolic damage due to exposure to marine pollutants. Fish liver parenchyma exhibited the signs of degeneration (cytoplasmic and nuclear degeneration, and nuclear vacuolation), besides the focal necrosis, during the exposure to heavy metals such as mercury (Oliveira Ribeiro *et al.*, 2006). Exposure to other heavy metals also resulted in changes in the hepatocytes such as

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vacuolization, necrosis and nuclear condensation (Giari *et al.,* 2007; Figueiredo-Fernandes *et al.,* 2007).

Hypertrophy, vacuolization, nuclear and fatty degeneration of hepatocytes have also been observed in the fish exposed to organic pollutants (Gill and Epple, 1993). In contrast, Cengiz *et al.* (2001) showed that there were hepatic lesions including degeneration, hypertrophy, sinusoids enlargement, hemorrhage, reposition of nuclei, vacuolization, and infiltration of mononuclear lymphocyte as a result of heavy metal exposure in mosquito fish, *Gambusia affinis*.

The stagnant of bile indicates possible damage to the hepatic metabolism (Fanta *et al.,* 2003). Similar to the findings of Pacheco and Santos (2002), the results of this study showed an increase in the density of the macrophage aggregates. Since it is, generally, related to hepatic lesions such as degenerative and necrotic processes, macrophage aggregates' result is important (Van Dyk *et al.,* 2007). The present histological changes were not mercury-specific; rather they are associated with the response of hepatocytes to wide range of pollutants. The function of the melano-macrophages in fish liver has remained uncertain; however, some studies suggest that it could be related to destruction, detoxification or recycling of endogenous and exogenous compounds (Haaparanta *et al.,* 1996). Degeneration of the liver tissue and necrosis of central vein could be as a result of the accumulation of neutrophils and lymphocytes. Similar results have been reported in African catfish exposed to fuel oil for 14 days (Gabriel *et al.,* 2007).

We observed alterations in ovary structure in all of the contaminated groups, especially in Petroshimi and Majidieh creeks. Oocyte atresia could reflect a disruption in the normal processes of the final maturation of oocytes and spawning (McCormick *et al.,* 1989); subsequently, it could easily lead to the production of abnormal fry (Guillete *et al.,* 1994). A similar conclusion was drawn by Dutta and Maxwell (2003) when bluegill (*Lepomis macrochirus*) exposed to sub-lethal concentration of diazinon. These observations agree with previous reports of increase in oocyte atresia after exposure to mercury. Day and Bhattacharya (1988) and Victor *et al.* (1986) showed that the number of atresia increased in *Channa punctatus* and *Lepidocephalichthys* after exposer to 16.7 ppb and 0.2 ppm of mercury, respectively.

Mercury exposure leads to relative increase in oocyte number and a decrease of oocytes size; this can cause subsequent inhibition of ovarian growth or embryos with abnormal development (Norman Haldén *et al.* 2011). This is likely to be caused by degeneration of mature vitellogenic oocytes due to chemical exposure (Van den Belt *et al.,* 2002). Day and Bhattacharya (1988) reported a dose-dependent atrophy of oocytes in *Channa punctatus* treated with mercury.

We observed adhesion between the oocytes increase with a dose effect. Oocytes that adhered to one another are prevented from moving onto the next level of maturation.
Singh and Sahai, (1985) and Sahai, (1988) reported fusion of oocytes in the ovary of Rasbora danicornius and in Puntius ticto after exposure to malathion and endosulfan, respectively. The histological changes observed in the present study imply that the fish were responding to both the chronic and sub-acute concentrations of mercury.

4 CONCLUSION

In conclusion, mercury concentration was high in Mahshahr coast. This measurement was followed by investigating the effect of mercury on Yellowfin Sea bream. Due to extensive changes in the liver and ovary, it can be said that mercury concentration in this area can

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considered as a very toxic substance for Yellowfin Sea breams so that it can jeopardize their life, and ultimately, lead to reduce their populations in the Persian Gulf and their enclosed creeks, as reported previously in Atlantic salmon (*Salmo salar*) (Handeland *et al.*, 1996).

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اثر جیوه بر بافت کبد و تخمدان ماهی شانک (*Acanthopagrus latus*) در خلیج فارس

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چکیده

این مطالعه وقوع تغییرات بافت‌شناسی در کبد و تخمدان ماهی *Acanthopagrus latus* که از رودخانه‌های آلوده به مواد شیمیایی حاصل از فعالیت انسانی و منطقه پاک جمع آوری شده‌اند را گزارش می‌کند. غلظت جیوه با استفاده از جذب اتمی بخار سرد اندازه‌گیری شد. نتایج نشان داد غلظت جیوه در آب و به ویژه رسوبات منطقه ماهشهر بیشتر از سایر محیط های دریایی بود و منطقه زنگی کمتر از سایر مناطق نمونه برداری شده بود. چندین ناهنجاری در در کبد مشاهده شد از جمله ایجاد هسته‌های بزرگ جانبی تخریب و تورم هسته، واکوئله شدن، نکروز جانبی و مرکزی، تورم، تجمع چربی، افزایش تعداد تخمک، خونریزی، تجمع ماکروفاژ و هم‌چنین اتساع سینوسی، تورم داخل سلولی و تخریب گرانولوزا. هم‌چنین بافت‌شناسی تخمدان نیز نشان داد که تعداد تخمک‌های قبل از زرده‌سازی و تعداد تخمک‌های تخریب و چسبیده شده در ماهیانی که از مناطقی با غلظت‌های بالای جیوه نمونه‌برداری شده بودند، افزایش یافت. در این مطالعه، وجود جیوه در سواحل ماهشهر تایید شد و وجود تغییرات بافتی فراوان در بافت‌های کبد و تخمدان ماهی *A. latus* شاهدی بر کیفیت پایین آب در این منطقه می‌باشد. بنابراین تغییرات بافتی که از طریق جیوه به وجود آمده بود، مانع از انجام درست فعالیت این بافت‌ها در این ماهی می‌شود.

کلمات کلیدی: بافت شناسی، تخمدان، جیوه، شانک ماهی، کبد

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