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Histopathological Effects of Triphenyltin Hydroxide on Liver, Kidney and Gill of Nile Tilapia (*Oreochromis nilotica*)

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The histopathological effects of triphenyltin hydroxide (TPTH) on the liver kidney and gill of Nile tilapia (*Oreochromis nilotica*) one month old was studied by light microscopy. Two concentrations of TPTH were used: 1 mg l^{-1} and 3 mg l^{-1}. The fish were sacrificed at the end of one, two, three and four months. The results showed that the hepatocytes underwent a variety of changes from congestion and dilatation of sinusoidal space, pallor of cytoplasm, vacuolation and accumulation of hyaline droplets. Subcapsular and scattered focal necrosis was also observed. In the kidney, hydropic degeneration and accumulation of hyaline droplets in the tubular epithelial cells were noted. In addition, a congestion of peritubular capillaries and detachment of tubular epithelial cells were observed. In more severe case there was a collapse of glomerular capillary tuft with a widening of the Bowman's capsule. There were some changes in the gill filaments and lamellae, namely hyperplasia of the covering gill epithelium, congestion of gill capillaries and vessels, and aneurysmal formation of gill lamellar capillaries. These alterations were time-and dose-dependent. Copyright © 1999 John Wiley & Sons, Ltd.

Keywords: triphenyltin hydroxide; *Oreochromis nilotica*; liver; kidney; gill; histopathology

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INTRODUCTION

In general, the histology of teleost fish liver differs from that of mammals in that the parenchyma is an indistinguishable lobule, comprising branched, one- to two-cell thick laminae of hepatocytes, separated by a sinusoid.[1] In aquatic ecosystems with abundant accumulation of pesticides and related chemicals, the liver is usually the organ with the highest pesticide accumulation and greatest damage or impairment.[2]

Several studies had shown a variety of changes of fish liver resulting from exposure to pesticides and other toxic chemicals. Acute and extensive necrosis of liver cells may occur in toxic conditions but focal necrosis is more common.[2,3] Couch[2] found inflammation and loss of glycogen and lipid in the livers of estuarine fish after exposure to endrin, a lindane and dichlobenil. General swelling of parenchymal cells was observed occasionally in rainbow trout (*Salmo gairdneri*) exposed to parathion and in redear sunfish exposed to hydrothol.[2]

General changes observed in the hepatocytes exposed to toxic chemicals are an abundance of fat vacuoles, lipid deposition and pyknosis. Couch[2] found pyknotic hepatocytes in bluegills exposed to dichlobenil. Abundance of fat vacuoles was found in several species of fish exposed to many types of pesticides, particularly organochlorine and organophosphate.[4,5]

In the tilapia, *Oreochromis mosambica*, exposed to Aquatin (an organotin molluscicide) and Brestan (triphenyltin acetate), Cruz[6] observed extensive necrotic hepatocytes and fibrosis and sinusoid congestion in the liver. In addition, he also found several pathological changes in kidney: necrosis of renal tubule and depletion of hematopoietic tissue resulting from the exposure to Aquatin.[6] The

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exposure to the Brestan showed necrotic renal tubules, eosinophilic casts in the tubule lumen, depletion of hematopoietic tissues and lymphocytic infiltration.[6]

The teleost gills consist of a gill arch, gill filaments and gill lamellae. The epithelium covering the gills is the squamous epithelium, usually at least two layers thick. The layers of epithelium are occasionally separated by intercellular spaces that contain macrophages. Since gills are the organs responsible for gas exchange, osmoregulation and excretion of nitrogeneous wastes, changes in the gill epithelium are a good warning of environmental impacts and infectious disease processes.[7]

A wide variety of pathological changes in the gills of trout (*Salmo gairdneri* and *S. clarki*), bluegill (*Lepomis macrochirus*) and goldfish (*Carassius auratus*) were induced after exposure to acute or chronic levels of pesticides.[8] Gill damage has also been described in bluegills, goldfish or trout after exposure to heavy metals, organic toxicants, organic solvents, gases and wood-fiber waste.[8] Proliferation of gill epithelium and lamellar hemorrhage in bluegills were found after four days' exposure to dichlobenil. The lesion was sharply defined by blood vessel engorged filaments. Fused lamellae were found after 63 days of exposure. Adjacent lamellae fused together in fish exposed to diuron for 21 days.[8] Gill lesion, hyperplasia of gill epithelium, accumulation of hemorrhagic exudate or clavate globate lamellae congestion were found in blood of goldfish treated with 1.0 mg l^{-1} mirex for 56 days.[8] Edema of lamellae and vasodilatation of arterioles and capillaries of gill filaments were induced after exposure to 6 mg l^{-1} of the sodium salt of 3-trifluoromethyl-4-nitrophenol (TFM) in both larval lamprey and rainbow trout.[9]

Cruz *et al.*[6] reported gill damage for *Oreochromis mosambica* by the molluscicides Aquatin (AQTN) and Brestan (BTN). They observed hyperplasia and fusion of gill lamellae as the result of AQTN. For BTN, there was a slight epithelial lifting, separation of capillaries from the epithelium and hyperplasia of gill lamellae.[6] Similarly, Chliamovitch[10] studied the effects of tributyltri oxide (TBTO) on the gills of rainbow trout and showed separation of the gill epithelium from the basement membrane and pillar cells; there was also a swelling of the secondary lamellae and dilatation of blood vessels.

Since the Nile tilapia (*Oreochromis nilotica*) is one of the important economic fish of Thailand, it is a major concern to investigate histopathological changes of the liver, kidney and gill as a result of pesticides, in this case triphenyltin hydroxide (TPTH).

MATERIALS AND METHODS

Nile tilapia (*O. nilotica*) one month old were obtained from the National Inland Fisheries Institute, Department of Fisheries, Ministry of Agriculture and Cooperatives, Bangkok, Thailand. They were acclimated and raised in 16 cylindrical fiberglass aquaria 130 cm in diameter and 80 cm in height

A total of 640 fish were divided equally into four groups as follows:

Group I	Reared in tanks with water
Group II	Reared in tanks with 0.0015% dimethyl sulfoxide (DMSO)
Group III	Reared in tanks with 1 mg l^{-1} triphenyltin hydroxide (TPTH) in 0.0015% DMSO
Group IV	Reared in tanks with 3 mg l^{-1} TPTH in 0.0015% DMSO

Group I and II were the control groups while groups III and IV were the experimental groups. The experiment was carried out for four months. During each month, ten fish were sampled from each tank. They were dissected and the livers, kidneys and gills were removed and fixed in 10% buffered formalin, pH 7.2 for 24–72 h at ambient temperature. The tissues were dehydrated through a graded series of ethanol infiltrated and embedded in paraplast. Sections 4 μm thick were cut on a rotary microtome, stained with hematoxylin and eosin, and viewed under an Olympus BH-2 light microscope.

RESULTS

Liver

Control groups

The histology of livers of the Nile tilapia treated with 0.0015% DMSO and those without treatment was rather similar. The liver was made up of hepatocytes that were not oriented into distinct lobules but were arranged in branched laminae two cells thick, separated by sinusoids (Figs 1A, 1B). Hepatocytes were polygonal cells with a central spherical nucleus and a densely stained nucleolus.

The hepatocyte sinusoid came from branches of hepatic portal vein and hepatic artery. The blood flowed from the sinusoid through the central vein to the hepatic vein, which was surrounded by the exocrine pancreas within the liver (Fig. 2A). Livers of two- to five-month-old fish of control groups showed normal hepatocytes and hepatic capsule (Figs 1A; 1B, 2A, 2B; 3A, 3B). However, in some specimens in the 0.0015% DMSO-treated group, there were mild congestion of sinusoidal spaces and slight widening of subcapsular space (Fig. 3B).

Experimental groups

Treated with 1 mg l^{-1} TPTH. At one month's exposure to TPTH, mild sinusoid dilatation was observed and a small number of lipid droplets were found in the hepatocyte cytoplasm of Nile tilapia liver (Fig. 1C).

At two months' exposure, homogeneous eosinophilic droplets or hyaline droplets were first seen in the hepatocyte cytoplasm. A few lipid droplets were also observed in the cytoplasm at this interval (Fig. 1D). No necrosis of the liver cells was observed.

At three months' exposure, most specimens showed abundant hyaline droplets in the cytoplasm (Fig. 2C). Some specimens still revealed lipid droplets and mild sinusoid congestion.

At the end of four months, more specimens showed a great number of hyaline droplets and lipid accumulation in the hepatocytes (Fig. 3C). Sinusoids were slightly dilated and congested. Scattered necrotic cell areas around the vascular region were seen in some specimens.

Treated with 3 mg l^{-1} TPTH. At this dosage, changes in the liver were similar to those observed in the group treated with 1 mg l^{-1} TPTH but they occurred at an earlier time. At the end of one and two months' exposure, congestion and dilatation of sinusoidal spaces and lipid accumulation in the hepatocytes were seen (Fig. 1E). Hyaline droplets appeared at the end of two months' exposure. However, in some specimens the capsules seemed to be normal in appearance.

From three months until the end of the experiment, most specimens showed a greater degree of sinusoidal congestion, and numerous hyaline droplets in the cytoplasm (Fig. 2D). Scattered foci of necrosis and subcapsular necrosis were seen only in a few specimens. Lipid vacuolation in the hepatocyte cytoplasm was found in almost every specimen and appeared massive, particularly at four months' exposure (Fig. 3D). Lipid accumulation was more severe than that in the group treated with 1 mg l^{-1} TPTH after the same period.

Kidney

Control groups

The histology of the kidneys of Nile tilapia from the control groups (with or without 0.0015% DMSO) showed similar results (Figs 4A, 4B; 5A, 5B; 6A). At one month's exposure, the kidneys showed a normal appearance of nephrons and hemopoietic tissues. After two to four months, most specimens showed normal features, but some showed the presence of hyaline droplets on the epithelium and more distortion and congestion of glomeruli (Figs 5A, 5B, 6A).

Experimental groups

Treated with 1 mg l^{-1} TPTH. At one and two months, a few specimens showed some eosinophilic droplets or hyaline droplets in the cytoplasm of the first proximal tubular cells (Fig. 4C). Glomeruli were distorted in some specimens at the end of two months. From three to four months, most specimens showed marked accumulation of hyaline droplets in the first proximal tubular epithelial cells and also a glomerular distortion (Figs 5C, 6B).

Treated with 3 mg l^{-1} TPTH. During the first two months, some specimens exhibited small hyaline droplets in the first proximal tubule epithelium. The glomerulus showed a mild dilatation; congestion and distortion of capillaries were also observed (Fig. 4D). After three to four months, similar results to those of two months' exposure were observed, but more severe. Hyaline droplets were larger, and increased in number. Glomeruli became more distorted and capillaries were markedly congested, especially at the end of the experiment (Figs 5D, 6C).

Gills

Control groups

The most sensitive parts of Nile tilapia gills are the gill filaments or primary lamellae which are attached to a cartilagenous gill arch. Another part is a leaf-like structure the gill lamellae or secondary lamellae, which has a single layer of thin epithelial cells surrounding the capillary. Blood enters lamellae from the afferent arteriole of filaments and exits through the efferent arteriole.

The histology of gills of the Nile tilpia of the control groups (reared in water or 0.0015% DMSO)

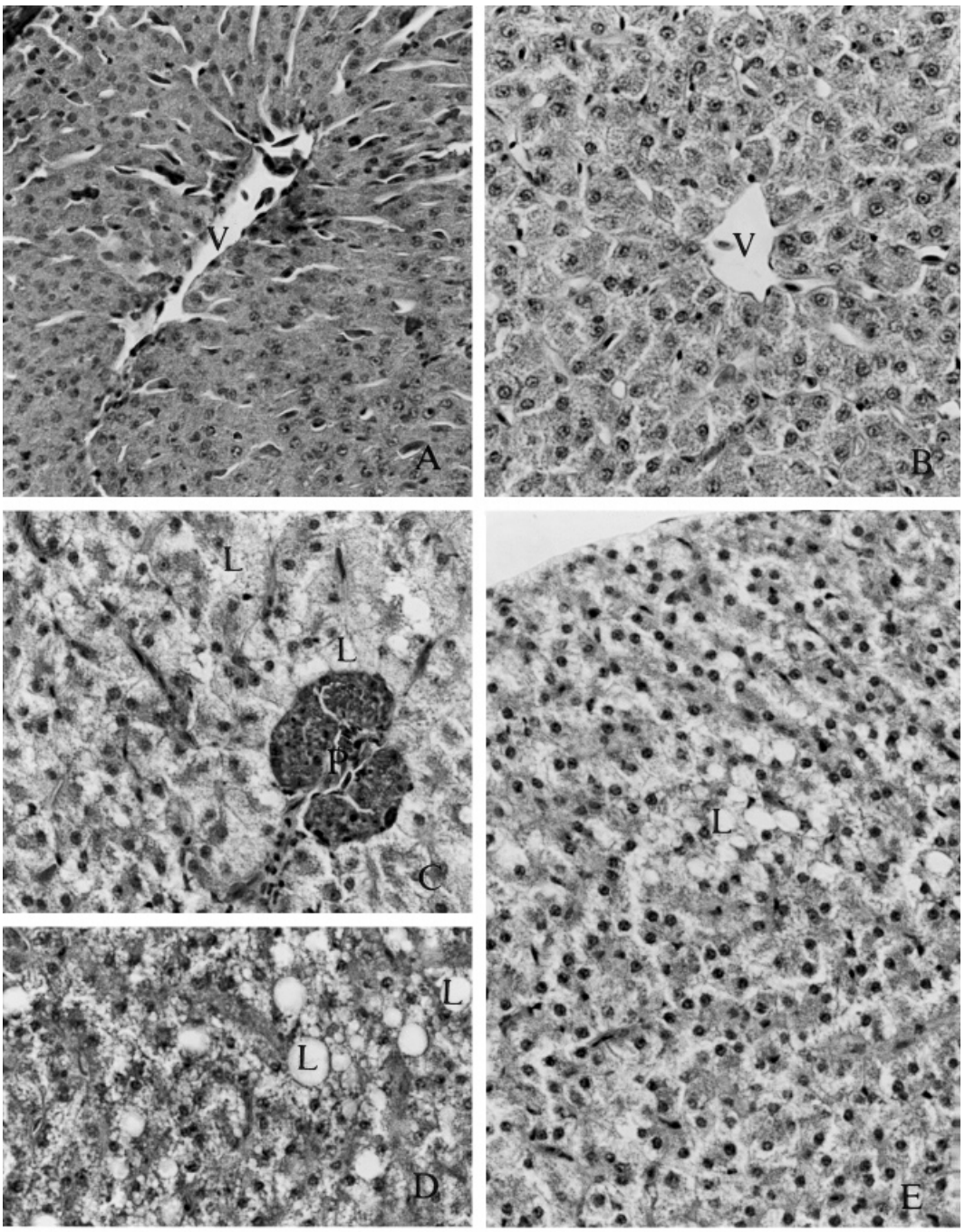

Figure 1 Nile tilapia livers of control and TPTH-treated groups after one to two months of exposure (×400). (A) Control (water). Liver is composed of laminae of hepatocytes arranged around a central vein (V) into which blood flows from sinusoids. (B) Control (0.0015% DMSO), showing normal features of laminae of hepatocytes and sinusoids. (C) Treated with 1 mg l^{-1} TPTH for one month showing normal features of liver. Note a few small lipid vacuoles (L) around exocrine pancreas (P). (D) Treated with 1 mg l^{-1} TPTH for two months, showing numerous vacuoles resulting from removal of lipid and glycogen (L). Hepatocytes remain normal. (E) Treated with 3 mg l^{-1} TPTH, showing normal features of liver with a mild degree of lipid vacuolation.

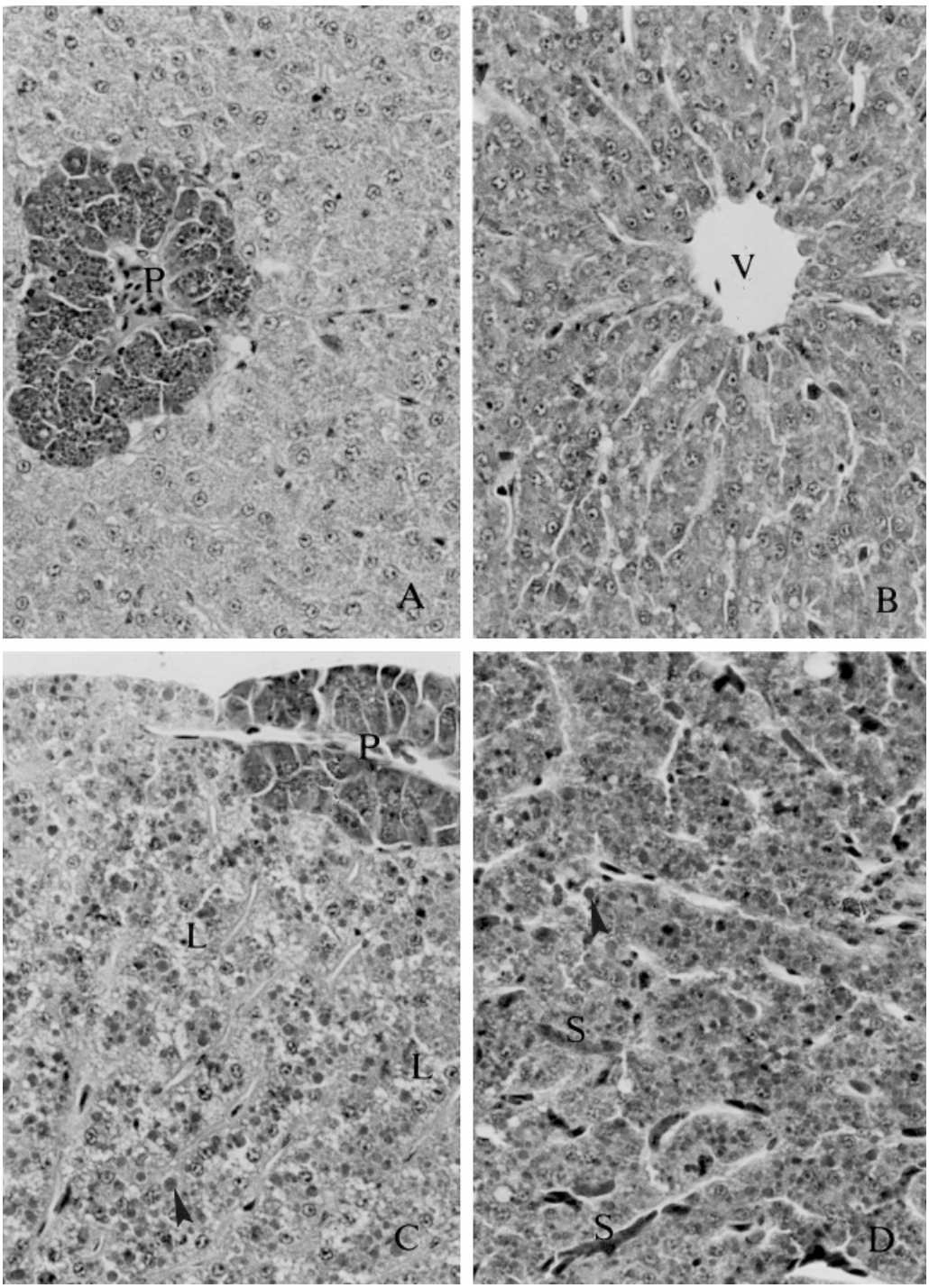

Figure 2 Nile tilapia livers of control and TPTH-treated groups after three months of exposure (×400). (A) Control (water), showing normal profile of sinusoids and hepatocytes. (B) Control (0.0015% DMSO), showing normal features of liver. (C) Treated with 1 mg l^{-1} TPTH, showing hepatocytes with hyaline droplets (arrowheads) and a mild degree of lipid vacuolation. P, exocrine pancreas. (D) Treated with 3 mg l^{-1} TPTH, showing numerous hyaline droplets in hepatocytes (arrowhead), dilatation and congestion of sinusoids (S).

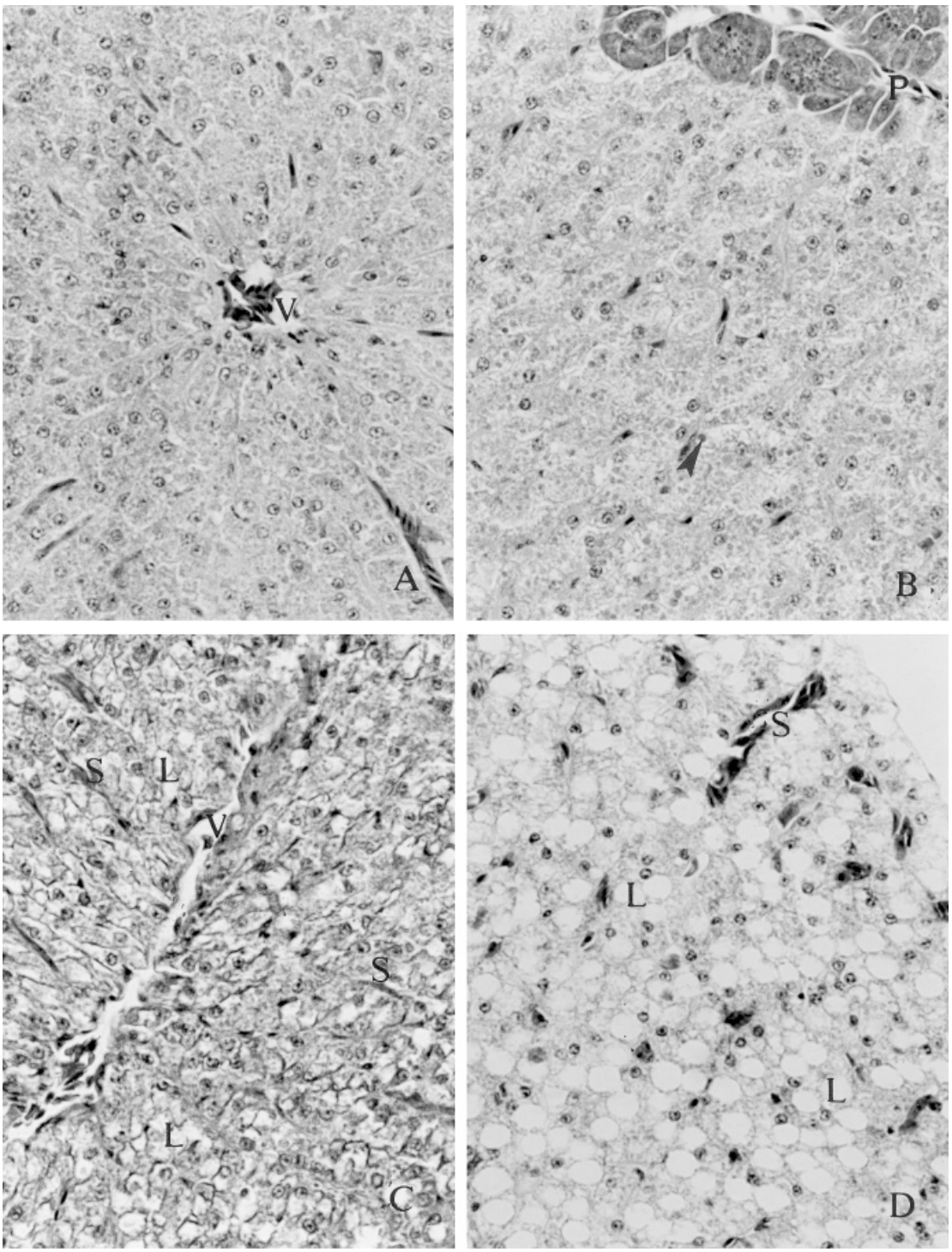

Figure 3 Nile tilapia livers of control and TPTH-treated groups after four months of exposure (×400). (A) Control (water), showing normal hepatocytes and sinusoids. (B) Control (0.0015% DMSO), showing small hyaline droplets in hepatocytes. P, exocrine pancreas. (C) Treated with 1 mg l^{-1} TPTH, showing congestion of sinusoids (S) and central vein. Note a marked inerease in lipid vacuolation (L). (D) Treated with 3 mg l^{-1} TPTH, showing more severe sinusoidal congestion (S) and lipid vacuolation (L).

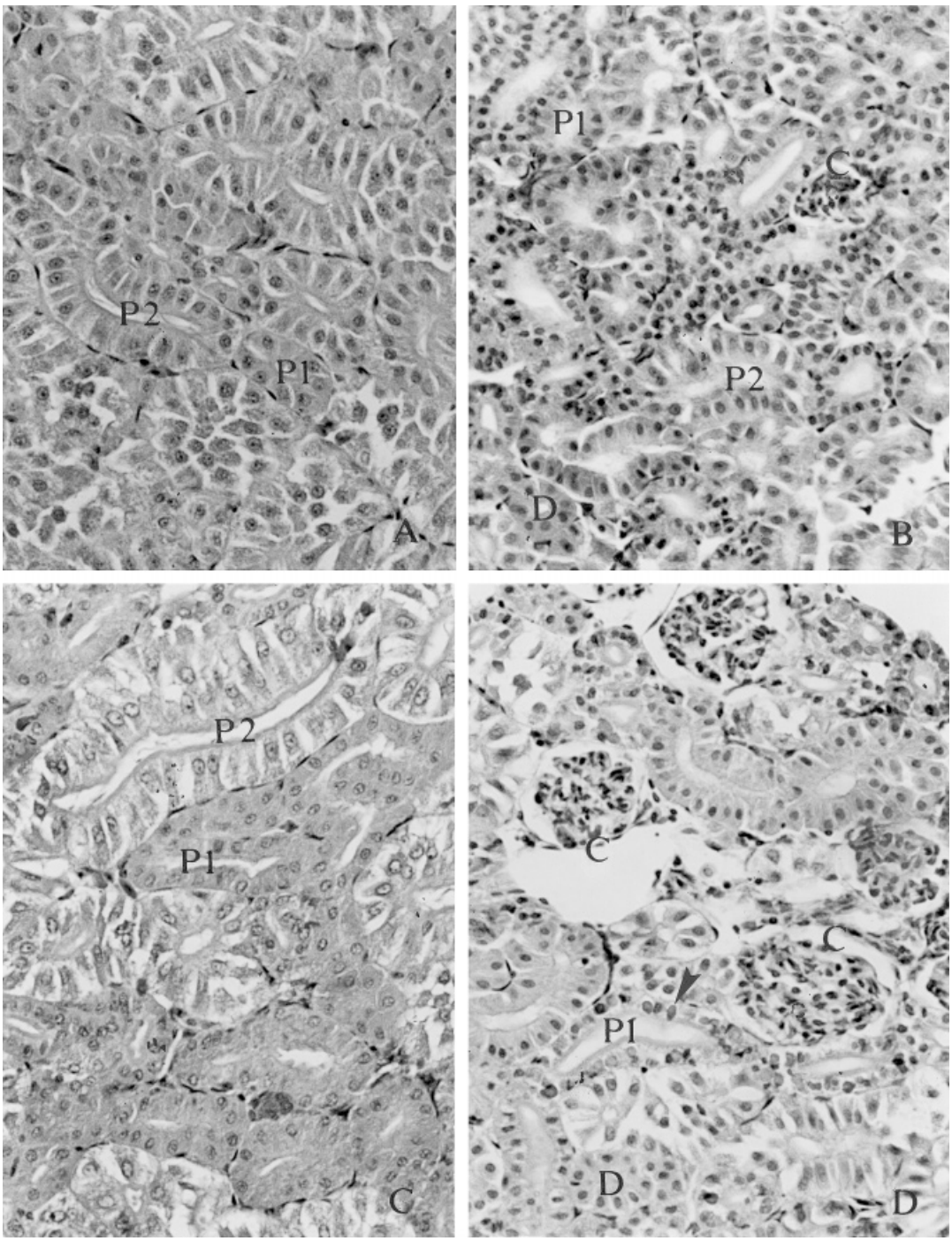

Figure 4 Nile tilapia kidneys of control and TPTH-treated groups after one to two months of exposure (×400). (A) Control (water), showing normal profile of epithelial of first proximal tubule (P1) and second proximal tubule (P2). (B) Control (0.0015% DMSO), showing normal appearance of renal corpuscle (C), epithelial cells of first (P1) and second (P2), proximal tubules, and distal tubule (D) (C). Treated with 1 mg l^{-1} TPTH. There are no significant changes in proximal (P1, P2) and distal tubules. (D) Treated with 3 mg l^{-1} TPTH, showing eosinophilic hyaline droplets (arrowhead) in proximal tubule epithelial cells. Renal corpuscles (C) and distal tubule are not altered.

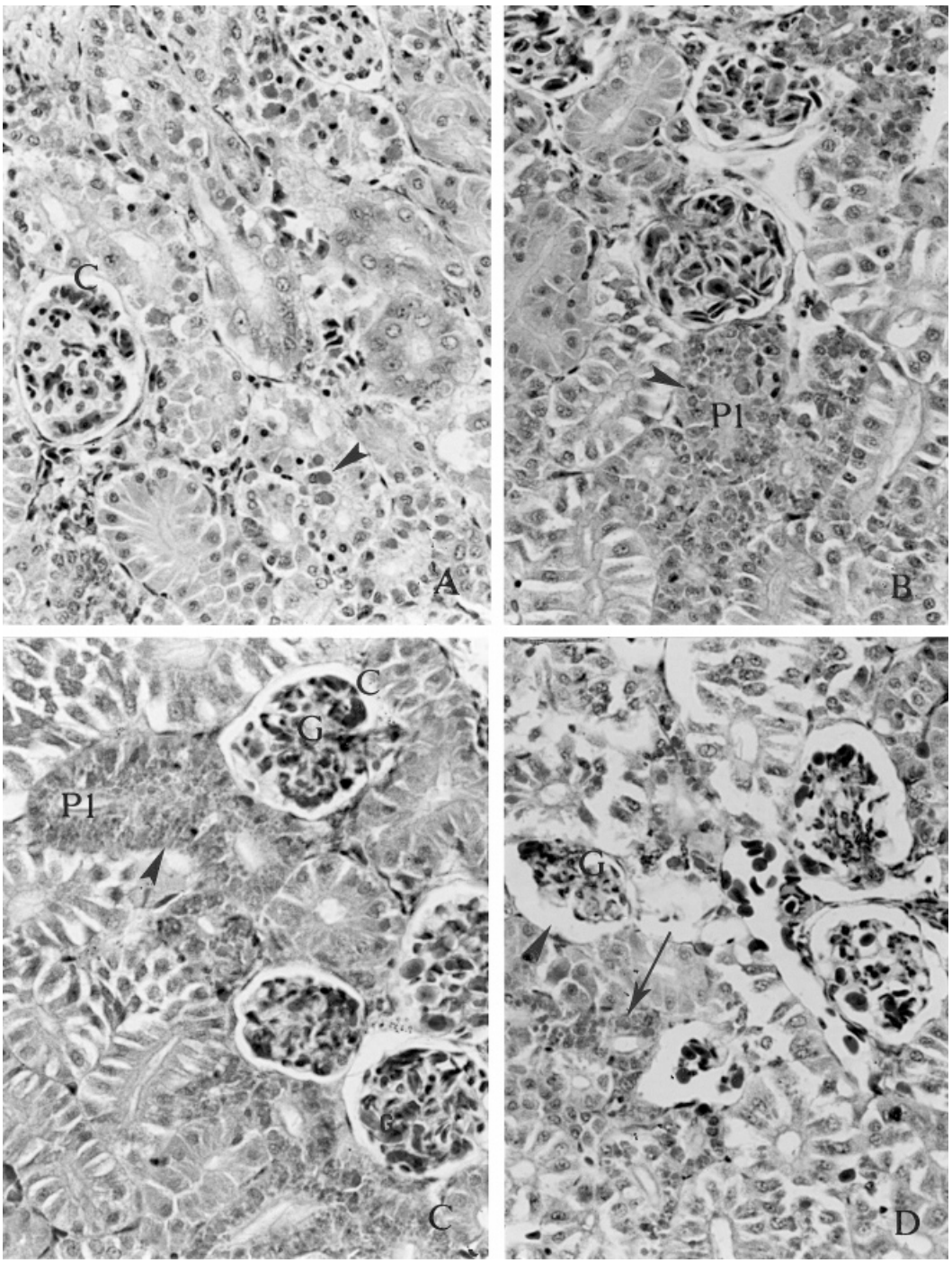

Figure 5 Nile tilapia kidneys of control and TPTH-treated groups after three months of exposure (×400). (A) Control (water), showing normal renal corpuscle (C). Note the presence of hyaline droplets in epithelial cells (arrowhead). (B) Control (0.0015% DMSO), showing similar features to (A). Note clusters of hyaline droplets in first proximal epithelial cells (arrowhead). (C) Treated with 1 mg l^{-1} TPTH, showing mildly dilated capillaries in glomerulus (G) and numerous hyaline droplets in P1 epithelial cells. (D) Treated with 3 mg l^{-1} TPTH showing glomerulus distortion and broader Bowman's capsule (arrowhead). The arrow points to hyaline droplets.

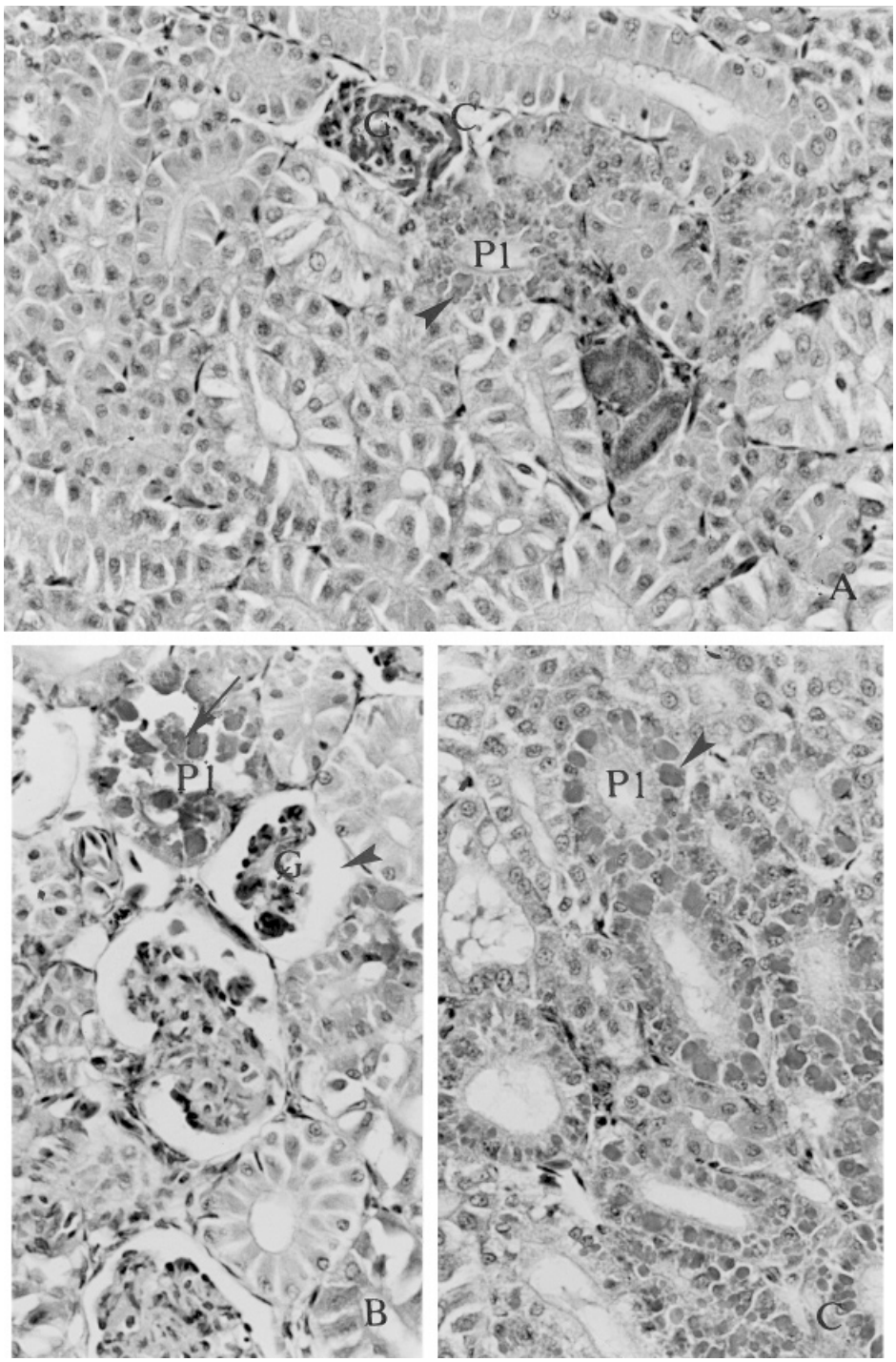

Figure 6 Nile tilapia kidneys of control and TPTH-treated groups after four months of exposure (×400). (A) Control (water), showing normal appearance of glomerulus (G) and renal corpuscle (C). Hyaline droplets (arrowhead) in the first proximal epithelial cells (P1) are observed. (B) Treated with 1 mg l^{-1} TPTH, showing more severe glomerulus contraction (G) and widening of Bowman's capsules (arrowhead). First proximal epithelial cells (P1) are filled with large hyaline droplets (arrow). (C) Treated with 3 mg l^{-1} TPTH, showing numerous large hyaline droplets (arrowhead) in epithelial cells of first proximal tubules.

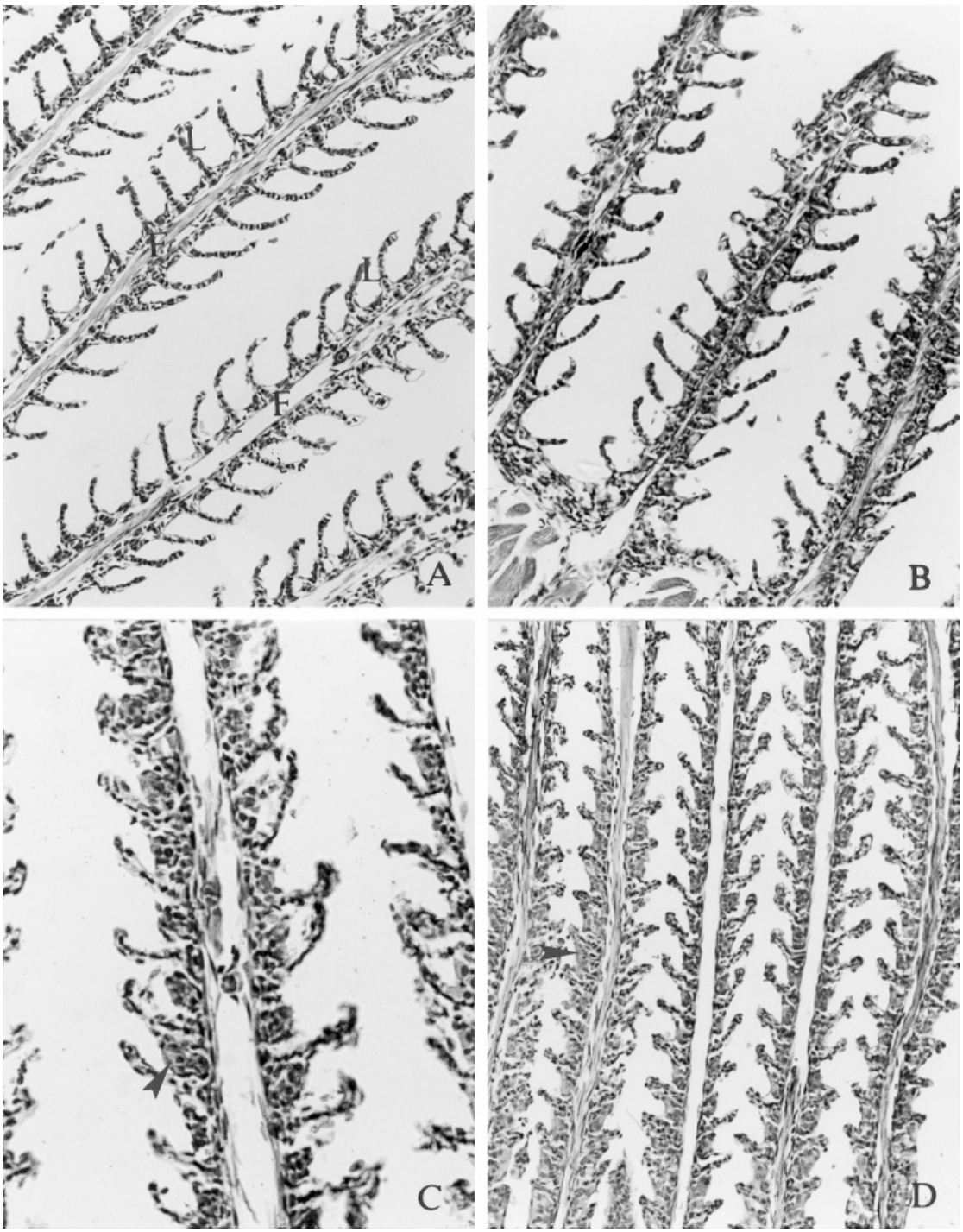

Figure 7 Nile tilapia gills of control and TPTH-treated groups after one month of exposure. (A) Control (water), showing normal appearance of gill filaments (F) and lamellae (L). Note stratified squamous epithelium covering gill filaments and a thin single-layer epithelium of gill lamellae (×200). (B) Control (0.0015% DMSO), showing normal gills (×200). (C) Treated with 1 mg l^{-1} TPTH, showing slightly thickened epithelium of filament (arrowhead) (×400). (D). Treated with 3 mg l^{-1} TPTH, showing slightly thickened epithelium of filament (arrowhead) (×400).

showed similar results. At one and two months, most gills showed the normal epithelial lining of gill filaments and lamellae (Figs 7A, 7B, 8A, 8B). The gill filaments were covered with a stratified squamous epithelium. The gill lamellae had a single layer of thin epithelial cells surrounding the capillary. After three and four months, most gills showed the normal appearance of filaments and lamellae (Figs 9A, 9B). Only some specimens showed a mild degree of change to lining epithelium. Gills were edematous and the capillaries were dilated and congested. Only a few specimens showed a slight thickening of epithelium of gill lamellae.

Experimental groups

Treated with 1 mg l^{-1} TPTH. At one month's exposure, the Nile tilapia gills showed a slight thickening of filament epithelium (Fig. 7C). This change was considered non-specific and was observed in some specimens. At two month's exposure, edema and congestion of lamellae were observed (Fig. 8C).

The Nile tilapia exposed to 1 mg l^{-1} TPTH for three to four months showed similar histopathology of the gills (Figs 9C, 9D). The gill lamellae showed edema, as revealed by the separation of epithelium from the capillary (Fig. 9D). There were vasodilatation and congestion of arterioles and capillaries of lamellae in most specimens. Several large balls of erythrocytes or aneurysms at lamellae were observed (Fig. 9C).

Treated with 3 mg l^{-1} TPTH. At one month's exposure, the Nile tilapia gills showed some edema of gill lamellae, and a mild dilatation and congestion of arterioles and capillaries (Fig. 7D). At two months' exposure, most gills exhibited edema of gill lamellae. A few aneurysms were also observed at gill lamellae (Fig. 8D).

The Nile tilapia exposed to 3 mg l^{-1} TPTH for three to four months showed similar changes in gill lamellae (Figs 9E, 9F). These changes were relatively similar to those of Nile tilapia treated with 1 mg l^{-1} TPTH for three to four months, but they were more severe. Marked swelling or edematous change of the lamellae was evident by the separation of the epithelium from the capillary (Fig. 9F). Large aneurysms at gill lamellae were apparent (Fig. 9F). There was also a slight thickening of the gill filament epithelium (Fig. 9F).

DISCUSSION

In the present study, it is interesting to note that histopathological changes in the liver exposed to 1 and 3 mg l^{-1} TPTH showed concentration- and time-related induced changes, namely congestions of blood vessels and sinusoids, lightening of cytoplasm of hepatocytes (which may be due to the removal of glycogen and accumulation of hyaline droplets), and presence of vacuoles with sharp demarcation from fat accumulation, focal necrosis and lamellar or subcapsular necrosis. All the histopathological changes are reversible except for the last two types of necrosis which are irreversible.

The vacuolation of cytoplasm by removal of lipid, and accumulation of glycogen in hepatocyte cytoplasm were the predominant findings. Fat vacuolation is a non-specific change. Almost any toxic agent would produce this. However, in this series of experiments the degree of fat accumulation was time- and dose-dependent. Similarly, glycogen accumulation induced by TPTH was also time- and dose-dependent. Glycogen accumulation seems to be more prominent in the earlier period of the experiment whereas lipid accumulation predominated in longer-duration exposures. Hepatocellular vacuolation had been reported by many authors. Wester and Canton[11,12] reported that bis(tri-*n*-butyltin) oxide (TBTO) and di-*n*-butyltin dichloride (DBTC) had similar effects causing glycogen and fat accumulation in the hepatocyte cytoplasm of medaka (*Oryzias latipes*) and guppy (*Poecilia reticulata*). Bruno and Ellis[13] also reported similar result of vacuolation in the hepatocytes of Atlantic salmon (*Salmo salar*) after exposure to tributyltin (TBT). The higher degree of vacuolation in longer fish exposures is still unclear.

Fat accumulation is also commonly found in pesticide-treated livers, particularly pesticides in the organochlorine and organophosphate groups. King[4] and Mount[5] found lipid droplets in the hepatocytes of brown trout and bluntnose minnows after exposure to DDT and endrin, respectively. Furthermore, Couch[2] noted the marked lipid accumulation in sheephead minnow hepatocytes after treatment with Dursban insecticide. Increase of fat vacuolation was common in fatty-liver degeneration which affected microsomal and mitochondrial functions and thus precluded the synthesis of lipoprotein.[11] However, it was found that massive fat accumulation in the liver did not affect its function.[11]

The impaired glycogen breakdown that caused

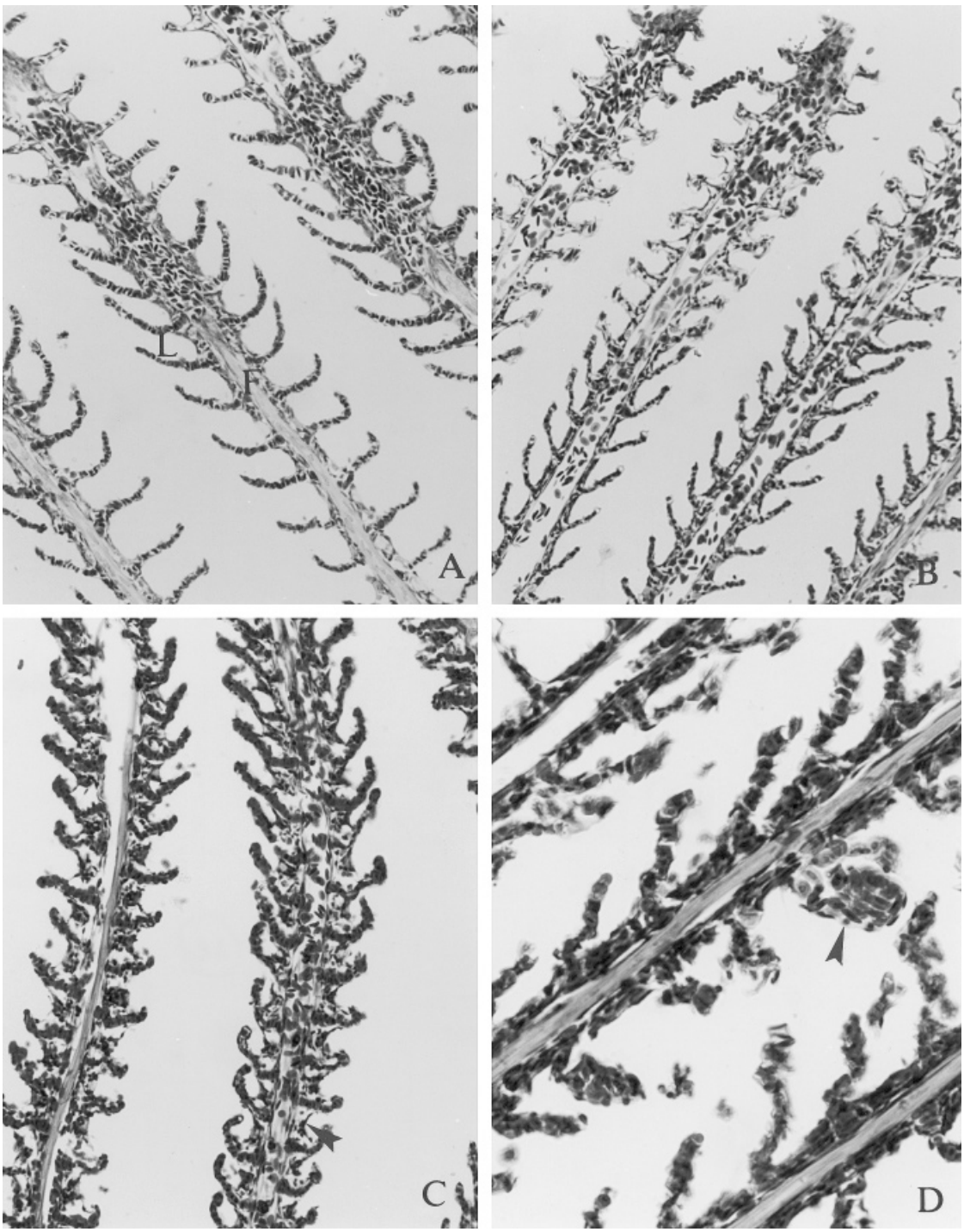

Figure 8 Nile tilapia gills of control and TPTH-treated groups after two months of exposure. (A) Control (water), showing normal appearance of gill filaments (F) and lamellae (L) (×200). (B) Control (0.0015% DMSO); most gill filaments and lamellae show normal appearance (× 200). (C) Treated with 1 mg l^{-1} TPTH, showing edema (arrowhead) and congestion of lamellae (×200). (D) Treated with 3 mg l^{-1} TPTH, showing gill lamellae packed with erythrocytes (arrowhead) (×400).

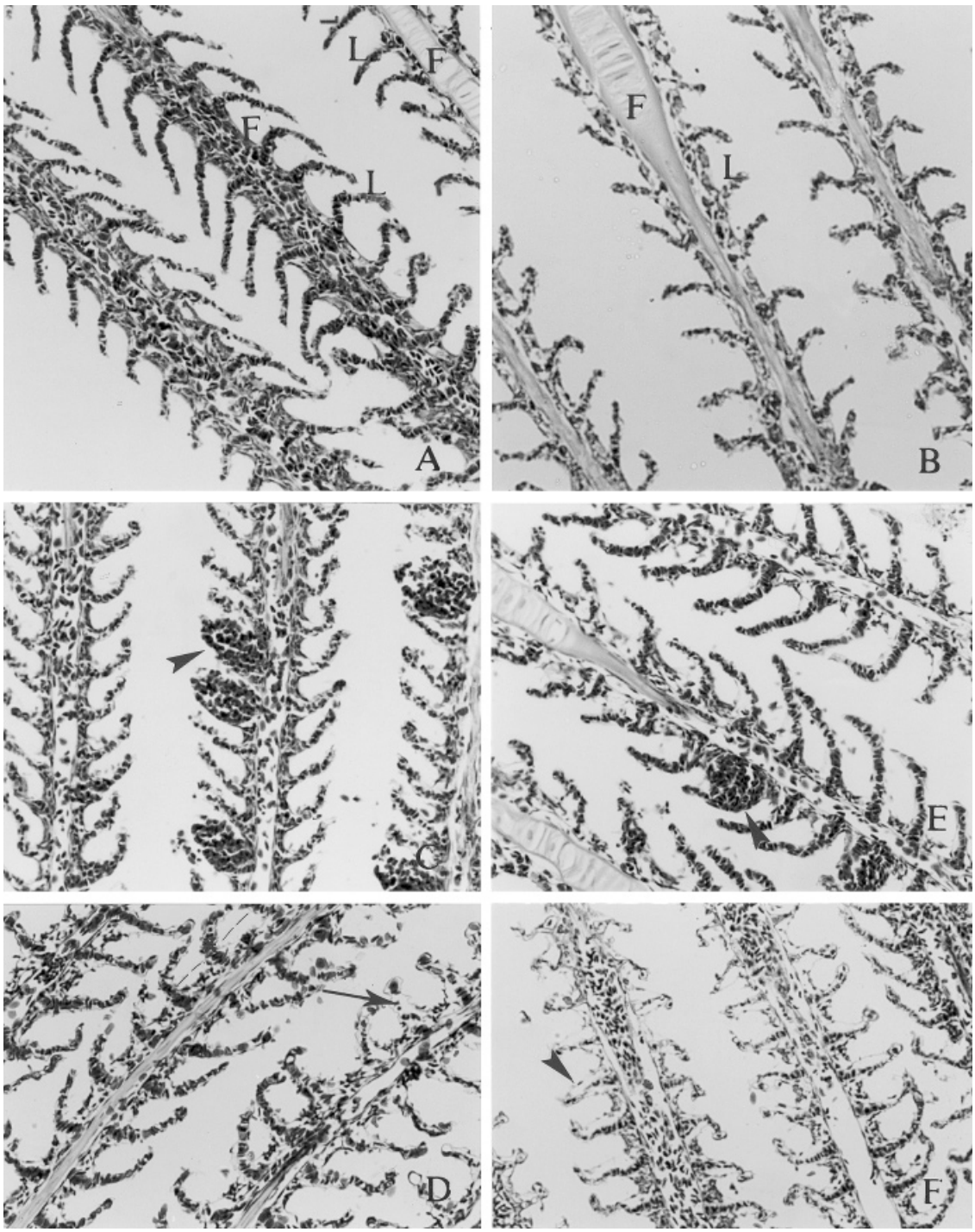

Figure 9 Nile tilapia gills of control and TPTH-treated groups after three to four months of exposure (magnification ×200). (A, B) Controls (water and 0.0015% DMSO), showing normal appearance of gill filaments (F) and lamellae (L). (C, D) Treated with 1 mg l^{-1} TPTH, showing edematous lamellae (arrowhead) (C) and aneurysm (arrow) (D). (E). Treated with 3 mg l^{-1} TPTH, showing marked swelling or edematous changes of gill lamellae (arrowhead). (F). Treated with 3 mg l^{-1} TPTH, showing a marked aneurysm at most lamellae (arrowhead).

glycogen accumulation could be a result of interference of TBT with the key enzymes directly (e.g. phosphorylase, phosphokinase) or indirectly via inhibition of induction of cAMP production. Glycogen depletion was also found in rainbow trout fry exposed to TBTC. Wester and Canton[11] described hyaline droplets, and homogeneous eosinophilic granules, in the hepatocytes of guppy after exposure to β-hexachlorocyclohexane. They suggested that these phosphoprotein droplets were caused by a proliferation of the rough endoplasmic reticulum so as to increase protein synthesis. Moreover, the necrosis of rat liver exposed to dibutyltin chloride was shown.[15] Studies employing lead nitrate [$Pb(NO_3)_2$] and mercuric chloride ($HgCl_2$) revealed disorganization of hepatic plates and focal hepatocyte necrosis with portal and peritubular infiltration of inflammatory cells.[16]

The first proximal tubules of the Nile tilapia kidney were most affected, with hyaline droplet accumulation. Some glomeruli were collapsed or distorted with dilated and congested capillaries. These lesions were dose- and time-dependent. Similar findings were confirmed by Hickman and Trump,[17] who noted that renal changes in visceral glomerular and tubular epithelial cells (particularly the proximal tubule) in fish were the result of filtration and subsequent resorption and lysosomal degradation of macromolecules. Furthermore, Wester *et al.*[14] reported that the glomerulus and proximal tubules of guppy exposed to β-hexachlorocyclohexane showed an accumulation of hyaline droplets, confirmed by slab-gel electrophoresis. These hyaline droplets were found not only in the renal epithelium but also in the glomerular Bowman's capsule, hepatocytes and heart endothelial cells.

Pathological changes resulting from exposure to triorganotins have been reported in medaka exposed to (TBTO) and DBTC.[12] Predominant changes in renal tubules such as dilatation, epithelial atrophy, degeneration, regeneration and proteinaceous casts, including cellular debris (tubulonephrosis) were observed. Glomerular lesions included dilatation of glomerular loops and Bowman's capsule and glomerulopathy.[12] However, it was concluded that the lack of knowledge on the toxicological pathology of this organ in fish and the lack of specific antibodies to investigate further possible immune-mediated glomerulopathies precluded further interpretation of this change.

The Nile tilapia gills exposed to 1 and 3 mg l^{-1} TPTH exhibited very prominent lesions. These included separation or edema of gill filaments and lamellae, congestion of lamellae (that often progressed to become a large ball of packed erythrocytes i.e. an aneurysm.). These histopathological changes are dose- and time-dependent, i.e. the higher the concentration of TPTH, the more severe and rapid was the incidence of lesions.

There are several studies that report on gill lesions after exposure to pesticides. Christie and Battle[3] found that 3-trifluoromethyl-4-nitrophenol (TFM) induced edema of lamellae and vasodilatation of arterioles and capillaries of the gill filaments in both larval lamprey, *Entosphenus lamottei* and rainbow trout. In addition, Gilderhus[18] reported that the gill lamellae of bluegills exposed to the herbicide sodium arsenite became swollen and edematous or packed with erythrocytes. Eller[8] noted that the gills of goldfish treated with mirex showed epithelial hyperplasia along the filament and aneurysm.

Histopathological changes resulting from organotin exposure have been reported in *S. gairdneri* treated with TBTO.[10] The lesions were detachments of the gill epithelium from the basement membrane, swelling of secondary lamellae, dilatation of blood vessels, thickening of epithelium at the bases of secondary lamellae, and vacuolization of secondary lamellae.[10] Chliamovitch noted that the toxicity of TBTO in gill lesion would provoke rapid suffocation by destruction of the gill epithelium and inhibition of the main metabolic pathways. Trialkyltin causes swelling not only of mitochondria but also of erythrocytes. Nevertheless, the swelling of red blood cells might be a consequence of a lower oxygen pressure.

The Atlantic salmon, *Salmo salar*, exposed to TBT showed lamellar separation and multiple points of epithelial hyperplasia at the tip of lamellae.[13] These features were explained in that tributyltin might interfere with the normal growth process of fish gills. Furthermore, the treatment of *O. mosambica* with Aquatin (20% organotins compound) and Brestan (60% triphenyltin acetate) showed similar alterations of the gills, such as epithelial hyperplasia and separation of the epithelial layer from supportive tissue.[4] These alterations were usually directly related to gill function disorders, which might affect the physiology or cause fish death.[8,10] Smith and Piper[20] and Mitchell *et al.*[21] reported that hyperplasia and separation of the epithelium associated with asphyxiation, partial or complete loss of secretory or excretory function, impairment of oxygen–carbon dioxide exchange and loss of plasma electrolytes or protein.

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REFERENCES

1. J. M. Grizzle and W. A. Rogers, *Anatomy and Histology of the Channel Catfish*, Department of Fisheries and Allied Aquaculture, Auburn, Ala.: Auburn University, Agricultural Exptl. Station, United States, 1976.
2. J. A. Couch, Histopathological effects of pesticides and related chemicals on the livers of fishes. In: *The Pathology of Fishes*, William, R. E. (ed.) University of Wisconsin Press, Wisconsin, 1975, pp. 559–584.
3. R. F. Roberts, The pathology and systematic pathology of teleost. In: *Fish Pathology*, Ronalde, R. J. (ed.) Bailliere Tindall, London, 1978, pp. 55–91.
4. S. F. King, *U.S. Fish Wildlife Serv. Spec. Sci. Rep. Fish.* **58**, 20 (1962).
5. D. E. Mount, *U.S. Fish Wildlife Serv., Res. Rep. Fish* **58**, 38 (1962).
6. E. R. Cruz, M. C. Dela Cruz and N. A. Sunaz, *Second International Symposium on Tilapia in Aquaculture, Bangkok, Thailand*, 1987, pp. 99–110.
7. M. K. Stoskope, Fish histology. In: *Fish Medicine*, W. B. Saunders, Mexico, 1993, pp. 35–41.
8. L. L. Eller, Gill lesion in freshwater teleostes In: *The Pathology of Fishes*, Ribellin, W. E. (ed.), University of Wisconsin Press, Wisconsin, 1975, pp. 305–325.
9. R. M. Christie and H. I. Battle, *Can. J. Zool.* **41**, 51 (1963).
10. Y. P. Chliamovitch, *J. Fish. Biol.* **10**, 575 (1977).
11. P. W. Wester and J. H. Canton, *Aquat. Toxicol* **10**, 143 (1985).
12. P. W. Wester, J. H. Canton, A. A. J. Van Iersel, E. I. Krajne and H. A. M. G. Vaessen, *Aquat. Toxicol.* **16**, 53 (1990).
13. D. W. Bruno and A. E. Ellis, *Aquaculture* **72**, 15 (1988).
14. P. W. Wester, J. H. Canton and A. Bisschop, *Aquat. Toxicol.* **16**, 271 (1990).
15. J. M. Barnes and P. N. Magee, *J. Path. Bact.* **75**, 267 (1958).
16. X. Sastry and X. Gupta, *Lead nitrate and mercuric chloride* (1978).
17. C. P. Hickman and B. P. Trump, The kidney. In: *Fish Physiology*, Hoar, W. S. and Randall, D. J. (eds), New York Academic Press, New York, 1969, pp. 91–239.
18. P. A. Gilderhus, *Trans. Am. Fish Soc.* **95** 289–296 (1966).
19. G. R. Gardner, Chemically induced lesions in estuarine or marine teleost. In: *The Pathology of Fishes*, Ribellin, W. E. (ed.), University of Wisconsin Press, Wisconsin, 1975, pp. 657–693.
20. C. F. Smith and E. G. Piper, *J. Fish Res. Board. Can.* **29**, 328 (1986).
21. A. J. Mitchell, J. M. Grizzle and J. A. Plumb, *J. Fish Dis.* **1**, 115 (1978).

