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EFFECT OF POLLUTANTS IN COASTAL WATER OF JEDDAH ON THE 1-HISTOLOGICAL STRUCTURE OF GILLS AND INTESTINE OF THE FISH *SIGANUS RIVULATUS* (FORSKAL). SAUDI ARABIA

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Key words: Histopathology, pollution, gills, intestine, siganus rivulatus.

ABSTRACT

The rapid development of industry as well as the anthropogenic activities has created serious problems of uncontrolled discharge in natural water sources mainly in areas close to sources of emission. These discharges create unhealthy environment and subsequent enjurious effects on aquatic life. The present study was carried out to study gills and intestine histopathology in the fish *Siganus rivulatus*. This will by waste discharges reveal any toxic effects of unfavorable water quality in the Red sea coastal water of Jeddah. The chemical analysis water and sediments from the study area and biochemical analysis of some body organs (gills and intestine) in *Siganus rivulatus* revealed contamination with heavy metals mainly Fe, Pb & Cu in water and sediment and Fe, Zn & Cu in fish organs. Several histopathological changes were observed in fish organs would serve useful purpose in evalulting the toxic effects of various pollutants. In gills impairment of gaseous exchange and osmoregulatory ability of fish were detected. These were swelling and fusion of adjacent gill lamellae; edematous separation, sloughing and necrosis of the respiratory epithelium; aneurysm, clubbing, atrophy and lysis of the lamellar blood sinuses and hyperplasia, spreading and damage of chloride cells. In addition to mucous cells proliferation; parasitic infection and inflammatory infiltration. Histology of intestine showed mainly vacuolar degeneration, cloudy swelling, necrosis and massive desquamation of mucosal epithelium; parasitic infection and cellular infiltration. Histopathological lesions score in examined tissues were evaluated.

1. INTRODUCTION

The area under investigtion is the Red sea coastal zone in front of the middle part of Jeddah city, This is the biggest and oldest commercial center in Saudi Arabia. Municipal wasts (mixed sewage and industerial wastes) and port activities are the most important discharges to the coastal area and contribute both organic and metallic contaminants (Behairy and Saad, 1984; El-Rayis, 1989; Basaham, *et al.* 1999).

Number of laboratory experiments with different toxic compounds abundantly occurring in the aquatic ecosystem have been performed with fish. They were shown to induce severe pathological changes (Abdel-Rahman, 1997; El-Feki, 1998 using heavy metals; Lemaire. *Et al.* 1992 using aromatic hydrocarbons; El-Elaimy *et al.* 1990; Hilmy, 1996 using pesticides; Sederake, 1992; Zaki. *Et al.* 1999 a, b using dentifyin effluents and Ackermann, *et al.* 2002 using nonylphenol).

Also some field studies have attempted to show that fish being exposed to local sources of pollutants in coastal or inland waters exhibited histopathological and macroscopic pathological changes. As a result, these changes have been shown to be reliable biomarkers of the effects of exposure to pollutants in the ecosystem (Khadre, 1991; 1992; Saleh and Hamza, 1986; Abdel – Aziz, 1994; Kamel and Fathalla, 1995).

Since gills and gastrointestinal tract in fishes considered the main passage for entrance of pollutants to the internal body organs like liver and kidney through the blood (Takashima and Hibiya, 1995). The present study was undertaken to characterized the histopathologicial alterations in gill and intestine of rabbit fish, *Siganus rivulatus* which is a widely distributed and economically important fish in Saudi Arabia.

2. MATERIALS AND METHODS

The area of Jeddah coastal water under investigation (21º 29` - 21º30 N and 39°10` - 39º11`E) located in the middle part of Jeddah city and extends between the Islamic Harbour south of Jeddah and the desalination plant north of the city (Fig. 1). Some patches of submerged coral reefs barrier exist in the study area (Behairy and Saad, 1984).

Some morphometric measurements were cavried out for experimental fish as total weight, total length and condition factor.

Meteorological parameters were recorded through these months (sampling monthes Autumn and winter seasons). These parameters dentify air and water temperature. Relative humidity and pericipition rate (Table 1). By using methods of APHA (1995) Physico – chemical parameters (Table 3) and concentration of some haevey metals were recorded in five sites of the study area (Tables, 4 & 5), as well as estimation of concentration and bioconcentration factor of heavy metals in gills and intestine of the selected fish .

The average weight (150 ± 20 g) were caught by trammel and impounding nets from five sites along the study area at arate of twice monthly and for three months. The fish were dissected and the gills and intestine were removed. Gill samples were fixed in EDTA (ethylene diamine tetra acetic acid) about 3 days for decaleification and intestine was fixed in 10% buffered formaline. Tissue samples were dehydrated, waxed blocked, sectioned at 3 µm and stained with haematoxylin and eosin (H. & E.) and Periodic Acid Schiff`s (PAS) (Bancroft and Steven, 1977).

To dentifying different types of cells in the gill tissue, slices of gills were fixed in 2.5% glutaraldehyde followed by 0.1 M osmium tetroxide, dehydrated and embedded in Epon. An LKB ultramicrotome was used to obtain semithin sections (0.1 – 1.0 µ m) stained with toluidine blue (T. B.) and ultrathin sections (60 – 70 µ m) stained with uranyl acetate and lead citrate (Reynold, 1963) and examined in joel 100 CX electron microscope.

For histopathological examinations, section of gills and intestine for each fish were scanned at 40 x or 100 x magnification. Lesions in tissue sections were semiquantitatively recorded using a four point scale (Kocan ., 1996); non (0); scarce (1); moderate (2), dominant or severe (3).

The examined fish were arbitrary divided into three groups according to gradual intensity of the histopathological lesions in studied organs. In each fish groups for each lesion, mean scorces (± SE) were calculated. Normal structure and histopathological lesions in gills and intestine were identified following Roberts (1978) and Takashima & Hibiya (1995).

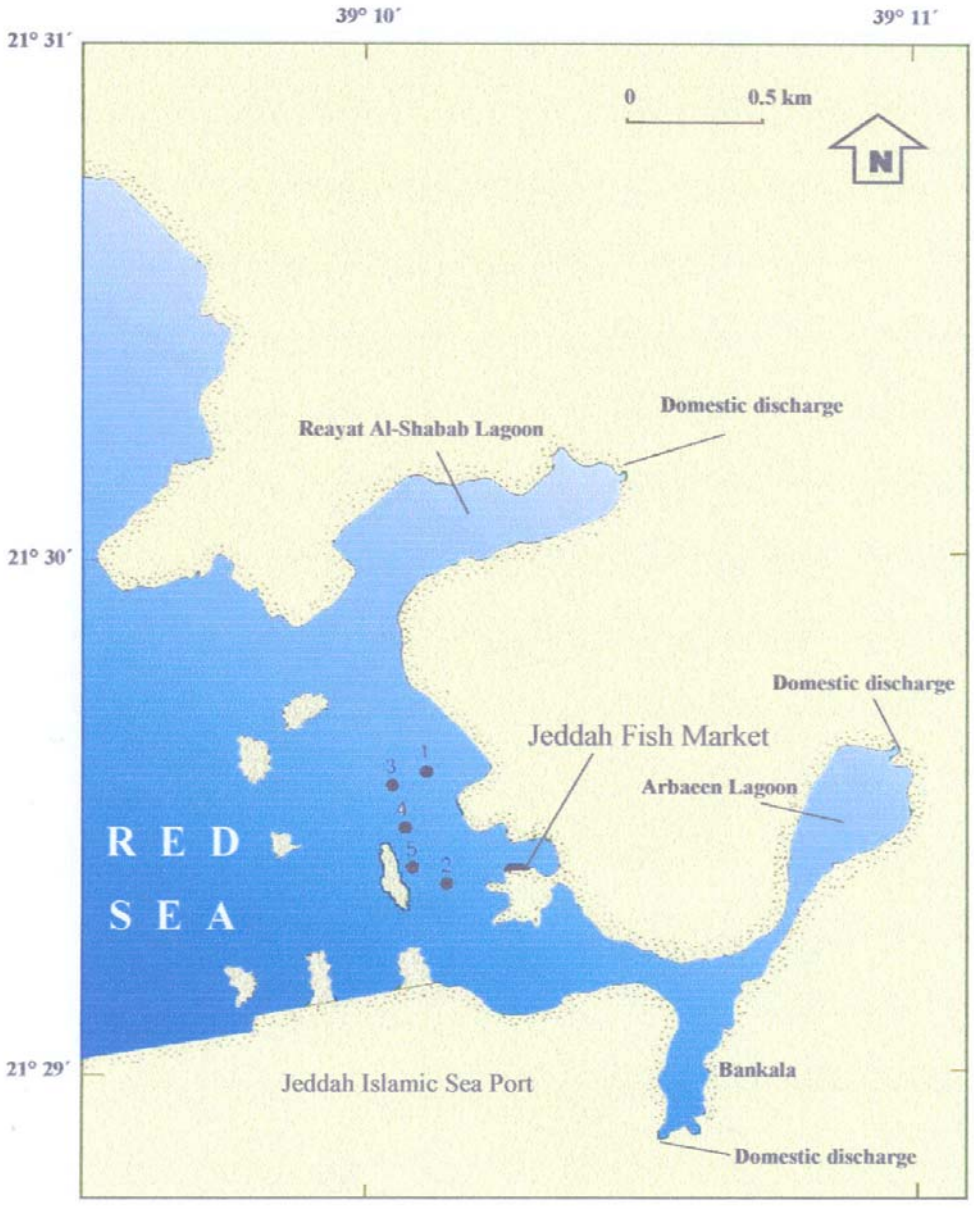

Fig. (1): Map showing the five sampling stations of Jeddah fish market

3. RESULTS AND DISCUSSION

The ecological studies in Table 1 showed astrong positive correlation between water and ainbient air temperature and a slight differences in mean values of temperature were observed between Autumn and winter seasons increase of concentration of biological and chemical oxygen demand in table 2 indicate to presence of organic pollutants in water of the studied area.

The concentration of heavy metals in sediments is higher than in water, for example Fe (36.22 ± 16.07 and 963.62 ± 219 µg/g for water and sediments respectively) followed by Pb, Ni, Zn, Cu, Mn, Cd and cr being last. (Tables 3 & 4) Gills and intestine of fish are the main passage for the entrance of pollutants to the in ternal organs through the blood, and the presence of high concentration of some heavy metals as Fe in non eaten organs as gills 389.41 ± 52.83 µg/g, liver 1147.69 ± 35764 attributed to the ability of these organs to accumulate these metals and to their blooded nature and high haemoglobin content, which Fe considered as its main constituent.

3.1. Gills

Some examined fish (16.67%) were characterized by maintaining the normal histological and cellular architecture in gills in comparison with that present in normal marine fish (Roberts, 1978; Takashima & Hibiya, 1995). Where primary and secondary lamellae of the gill filaments could be easily detected (Fig. 2). The secondary lamellae on both sides of the primary lamellae were erect or slightly curved. They composed of central lamellar blood sinuses constricted by contractile pillar cells and a layer of the flattened respiratory epithelial cells, which cover the surface of the lamellae in a wavey line separated from the lamellar blood sinuses by a basement membrane (Figs. 2 & 3). At intervals along the respiratory epithelia as well as crypts between lamellae, there were ovoid mucous cells which characterized by their basal nuclei, numerous secretary vesicles and a pical openings (Figs. 2 & 4). Also, in the basilamellar regions situated on the primary lamellae, were few chlorid cells which were described before in various marine fishes (Kessel and Beams, 1962 & Virtanen, 1986). They are spheroid and characterized by their fairly large number of mitochondria and smooth endoplasmic reticulum and are responsible for ionic regulation (Figs. 2 & 5).

Histopathological alteration were detected in the gill filaments of most captured fish (83.33%) with great individuals variability and characterized primarily by dilation of the lamellar blood sinuses and cruling of the secondary lamellae with separation of the respiratory epithelia and proliferation of chlorid cells in basilamellar regions (Fig. 7). there was epithelial layer separation from the remaining part of the lamellae with chloride cells proliferation. This lead to lamellar swelling fusion with swollen respiratory epithelial cells. (Fig. 8)

In some instances, pillar cells damage lead to loss of supportive properties with subsequent blood stasis in lamellar capillaries with separation of respiratory epithelium (Fig. 10).

The edematous separation of the respiratory epithelium and lamellar clubbing may be due to water born toxins and this departure from normal structure affect the functional efficiency of the gills for gas transport and ionic regulation (Kantham and Richard, 1995). Also, the pathological changes in the chloride cells may indicate osmoregulatory dysfunction, which is the main function of the chloride cells (Virtanen, 1986). Chloride cells proliferation due to an added function of oxygen transport due to injury to gill tissue proper. In certain abnormal conditions chloride cells may to be an oxygen transport functions (Tamse, ***et al.*** 1995).

Fusion of adjacent secondary lamellae on one or both sides of the primary lamellae due to an obliteration of inter lamellar space and

necrosis of the respiratory epithelia followed by atrophy and degeneration of central blood sinuses were clearly discernible in gill filaments examined . (Figs. 9 & 11) and a part from RBCs and leukocyte infilteration only damaged and intact chloride cells were still recognizable (Figs. 11 & 12) .Fusion of lamellae decreases gill surface area . This may allow slower up take of toxins but create an anoxic condition that will ultimately harm the fish (Takashima & Hibiya, 1995).

Also previous studies reported swelling and fusion of the gill lamellae in fish exposed to sublethal concentration of heavy metals where metals bind strongly to the plasma membrane of the lamellar epithelial cells increaing their permeability to water and ions (Mallat, *et al.* 1995; Abdel - Rahman, 1997; El-Feki, 1998; Bin Dohaish, 2001). Also, heavy metals might inhibit ion carries in chlorid cells and thus, an increase in their number would be compensatory (El-Feki, 1998).

On the other hand, mucous cells proliferated and hypertrophoid on the fused surface of the secondary lamellae (Figs. 9, 11 & 12). This may be considered as a protective response to bindes toxins transport. The set of protective reactions culuminats in the formation of mucous layer that could coagulate and up in the cessation of gaseous exchange and death (Tams, *et al.* 1995) .

Leukocytes infilteration caused their accumulation in the subepithelial spaces of secondary lamellae and necrotic gill tissues (Figs. 12 & 14). This may be an inflammatory reaction response to different chemical toxic substances (EL-Feki, 1998) or to phagocyte the toxicant particles and tissue debrises (Muhvich, *et al.* 1995).

Some form of parasites are usually present on gill of all fish in this study, parasitized gills showed haemorrage congestion, cellular infilteration and necrosis (Figs. 13, 14 & 15). Eosinophilic infilterations were clearly detected in parasitic infected gill filaments (Fig. 14) Eosinophilic granules contain a protein that is toxic to parasites but also causes lysis of infected tissues (Robbins, 1995).

3.2. Intestine

Histological examination revealed great variability in the intestinal lesions severity existed among most fish caught including focal deformation with caseous necrosis of mucosal epithelial layer of some villi, enlargment of the intestinal villi due to vacuolar degeneration (Fig. 16) or cloudy swelling of the mucosal epithelial cells (Fig. 17). Lymphocytic infilteration, dissociation and reduction of muscular bundles and serosal lysis were also detected (Fig. 16). In some instance, the columnar epithelial layer inbetween the intestine villi carry long hair like extensions and lymphotic sinuses and heavily cellular infilteration were detected in the intestinal tissue underlying (Fig. 18). This may be represent important link in the intestinal immune system which catch antigen and pass it into macrophage and lymphocyte underlying it to activate immune responses against antigen (Junqueira, *et a*l 1998). Minor lesions were observed in the intestine and they were largely due to presence of parasites. Parasitic infection accompanied by sloughing and damage of the intestinal villi, haemorrhage and leukocyte infilteration were observed in the intestinal lumen of many fish examined (Figs. 19 & 21). Eosinophilic infilteration was clearly accumulated in parasitic infected intestines (Fig. 20) where they stimulated by parasitism and increase in number in inflammatory sites (Patt and Patt, 1969 & Robbins, 1995). Necrotic lesions were also obsesred in some specimens (Fig. 22).

3.3. Evaluation of histopathological lesions

The examined fish (150 samples) were divided arbitrary into three groups according to gradual intensity of histopathological lesions in gills and intestine (Table 5). First group fish represents 16.67% (25 samples) of examined fish and characterized by normal histological and cellular architecture in gills and intestines with low histopathological lesions scores (L.S. ≤1). The mojor lesions

included separation of respiratory lamellae and mucous cell proliferation in gills.

The second group represent 60% (90 samples) of examined fish and characterized by wide variaty of pathological lesions in the gills and the intestines with some dominant lesions (L. S = 3) characterized in gills by lamellar swelling and fusion, chloride cells proliferation and spreading followed by mucous cell proliferation and lymphocytic infilteration (3 > L. S. > 2.5). In the intestine the dominant pathological lesions were associated with the presence of parasites and included sloughing celluler infiltration and necrosis (L. S. ≥ 2.5).

Severe histopathological abnormalities were recorded in gills and intestine of 35 samples (23.33%) of fish examined and represented by sloughing and lysis of the gill lamellae and parasitic infection in gill tissue (L.S. ≥ 2.5). In intestine, were represented by necrosis and sloughing of the mucosal epithelium, parasitic infection, extension of the intestinal lumen and loosening and reduction of the muscular bundles (L. S. ≥ 2.5).

In accordance with the presents study (Khadre, 1991) recorded massive desquamation of mucosal epithelium and lymphocytic infiltration in the intestine of *Tilapia zilli* caught from a contaminated lake in Egypt. Kohler and Halzel (1980) observed similar results, in addition to parasitic infection in flounder caught from Elbe estuary, England. Wood – word *et al.* (1995) found that brown trout that were fed on benthic invertebrate contaminated with two heavy metals (Cd, Pb) showed degenerative changes in the intestinal mucosal cells. Similar pathological changes were also mentioned in *Mugil cephalus* caught from polluted water by crude oil (Mazhar, *et al.* 1987).

It is worth mentining that chemical analysis of water and sediments from the studying area and biochemical analysis of some body organs of *Siganus rivulatus* caught from the same area revealed contamination with heavy metals mainly "Fe, Pb & Cu" in water and sediments and fish organs (Bin Dohaish, 2001). Thus, it can be inferred that histopathological changes in fish organs would serve useful purpose in evaluating the toxic effects of various pollutants present in the Red sea coastal water area of Jeddah.

Table (1): Meteorological records in the study area through Autumn and Winter seasons.

Season	Months	I	ll	lll	IV
Autumn	September	32.4	32.1	79.0	0.0
	October	30.0	29.0	80.0	0.0
	November	27.6	27.2	77.0	0.0
	Mean	30.0	29.4	78.7	0.0
	SD	2.4	2.5	1.5	0.0
Winter	December	27.4	24.7	78.0	10.0
	January	25.9	21.0	76.0	11.0
	February	25.6	22.0	70.0	11.0
	Mean	26.3	22.6	74.7	10.7
	SD	1.0	1.9	4.2	0.6

I **Air temperature (°C)**
II **water temperature (°C)**
III **Relative humidity (%)**
IV **Precipitation (mm/month)**

Table (2): Mean (and range) values of the physico-chemical parameters measured in surface waters of the Five sites of the study area.

Parameters		**PH**	**Hardness (mg /L)**	**DO (mg O2 /L)**	**BOD ($mg\ O_2$ /L)**
sites	1	7.80	6,850	4.95	3.81
	2	8.14	9,450	3.93	5.02
	3	8.16	7,750	5.35	2.68
	4	8.14	10,750	6.29	6.13
	5	8.17	15,250	7.17	7.93
Range		7.80 – 8.17	6,850 – 15,250	3.93 – 7.17	2.68 – 7.93
X ± SD		8.08 ± 0.16	10,010 ± 3,294	5.54 ± 1.25	5.11 ± 2.04
Parameters		**COD**	**NO_2 -N**	**NH_4 -N**	**PO_4 -P**
		(mg O2 /L)	**(µg at /L)**	**(µg at /L)**	**(µg at /L)**
sites	1	20.00	0.01	0.13	0.08
	2	20.00	0.15	0.11	0.78
	3	20.00	0.39	0.18	0.11
	4	40.00	0.17	0.13	0.12
	5	40.00	0.17	0.13	0.07
Range		20.00 – 40.00	0.01 – 0.39	0.11 – 0.18	0.07 – 0.78
X ± SD		28.00 ± 10.95	0.18 ± 0.13	0.14 ± 0.03	0.23 ± 0.31

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Table 3: Concentration of the heavy metals (µg /L) wet weight in the surface water of the studied sites.

Sample	Fe	Ni	Mn	Cr	Zn	Cd	Pb	Cu
1	19.64	5.36	1	0.52	1.44	0.84	3.2	0.52
2	53.68	4.36	1.88	0.72	1.4	1.4	10	2.24
3	19.44	1.6	1.84	1	1.44	1.92	5.2	2.4
4	48.96	3.28	0.88	0.92	2	0.8	12.8	1.32
5	39.36	9	0.52	1.24	1.68	1.04	4.8	1.44
Range	19.44 – 53.68	1.60 – 9.00	0.52 – 1.88	0.52 – 1.24	1.40 – 2.00	0.80 – 1.92	3.20 – 12.80	0.52 – 2.40
Mean ± SD	36.22 ± 16.07	4.72 ± 2.77	1.22 ± 0.61	0.88 ± 0.27	1.59 ± 0.25	1.20 ± 0.47	7.20 ± 4.03	1.58 ± 0.76

Table 4: Concentration of the heavy metals (µg /g) in the surface sediments of the studied sites

Sample	Fe	Ni	Mn	Cr	Zn	Cd	Pb	Cu
1	1050	19.112	17.612	13.85	8.437	4.75	50.875	7.587
2	1040	22.762	17.412	13.275	5.25	5.162	53.5	6.837
3	705	20.637	15.425	12.75	4.387	5.45	46.625	6.75
4	779.352	25	18.088	16.469	9.398	5.812	64.054	8.964
5	1243.75	21.7	19.112	12.562	4.55	5.225	48.625	39.712
Range	705.00 – 1,243.75	19.11 – 25.00	15.43 – 19.11	12.56 – 16.47	4.39 – 9.40	4.75 – 5.81	46.63 – 64.05	6.75 – 39.71
X ± SD	963.62 ± 219.44	21.84 ± 2.22	17.53 ± 1.35	13.78 ± 1.58	6.40 ± 2.34	5.28 ± 0.39	52.74 ± 6.83	13.97 ± 14.42

Table 5 : Histopathological Lesions scores in gills and intestines of *Siganus rivulatus* captured .

Lesions Score	Group I 25 Samples 16.67%	Group II 90 Samples 60.0%	Group III 35 Samples 23.33%
	Mean ± SD	Mean ± SD	Mean ± SD
GILL LESIONS			
Stasis and Separation of respiratory epithelia	1.160 ± 0.624	2.375 ± 0.490	2.000 ± 0.490
Proliferation & spreading of chlorid cells	0.360 ± 0.638	3.000 ± 0.000	1.400 ± 0.516
Sloughing of the respiratory epithelia	0.460 ± 0.374	1.525 ± 0.640	3.000 ± 0.000
Lamellar swelling	0.000 ± 0.000	3.000 ± 0.000	2.000 ± 0.000
Lamellar fusion	0.000 ± 0.000	3.000 ± 0.000	2.000 ± 0.471
Lymphocytic infilteration	0.000 ± 0.000	2.750 ± 0.439	1.800 ± 0.667
Mucous coagulation	0.000 ± 0.000	2.900 ± 0.304	2.300 ± 0.789
Disintegration of Lamellar tissue	0.000 ± 0.000	0.750 ± 0.840	2.800 ± 0.000
Parasitic infection	0.480 ± 0.653	1.050 ± 1.124	2.500 ± 1.434
INTESTINAL LESIONS			
Lymphocytic infilteration	0.000 ± 0.000	2.800 ± 0.405	0.700 ± 0.823
Necrosis and Sloughing of Mucosal epithelia	0.000 ± 0.000	2.125 ± 0.607	3.000 ± 0.000
Parasitic infection	0.200 ± 0.408	0.625 ± 1.125	2.800 ± 1.549

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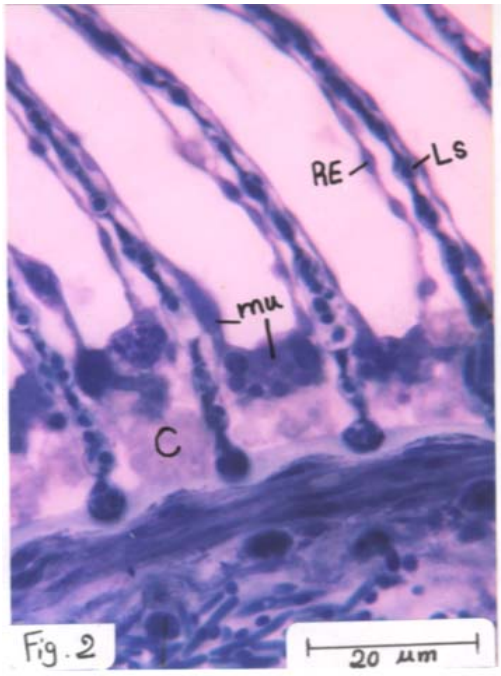
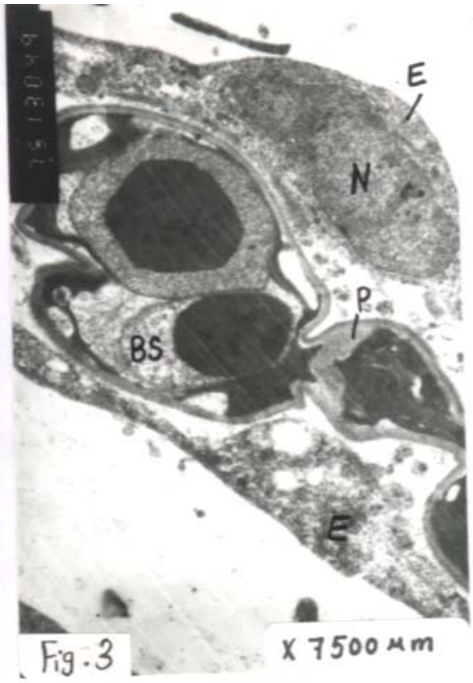


Fig. 2



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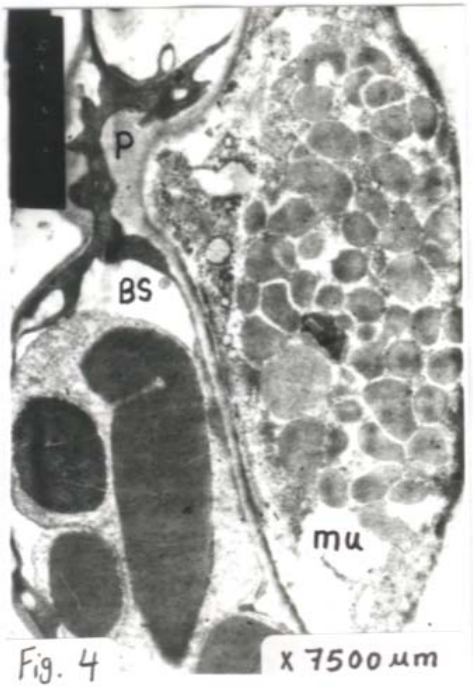

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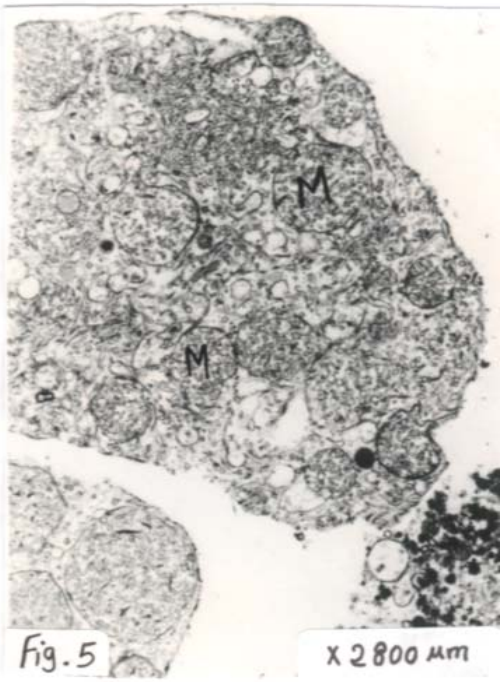

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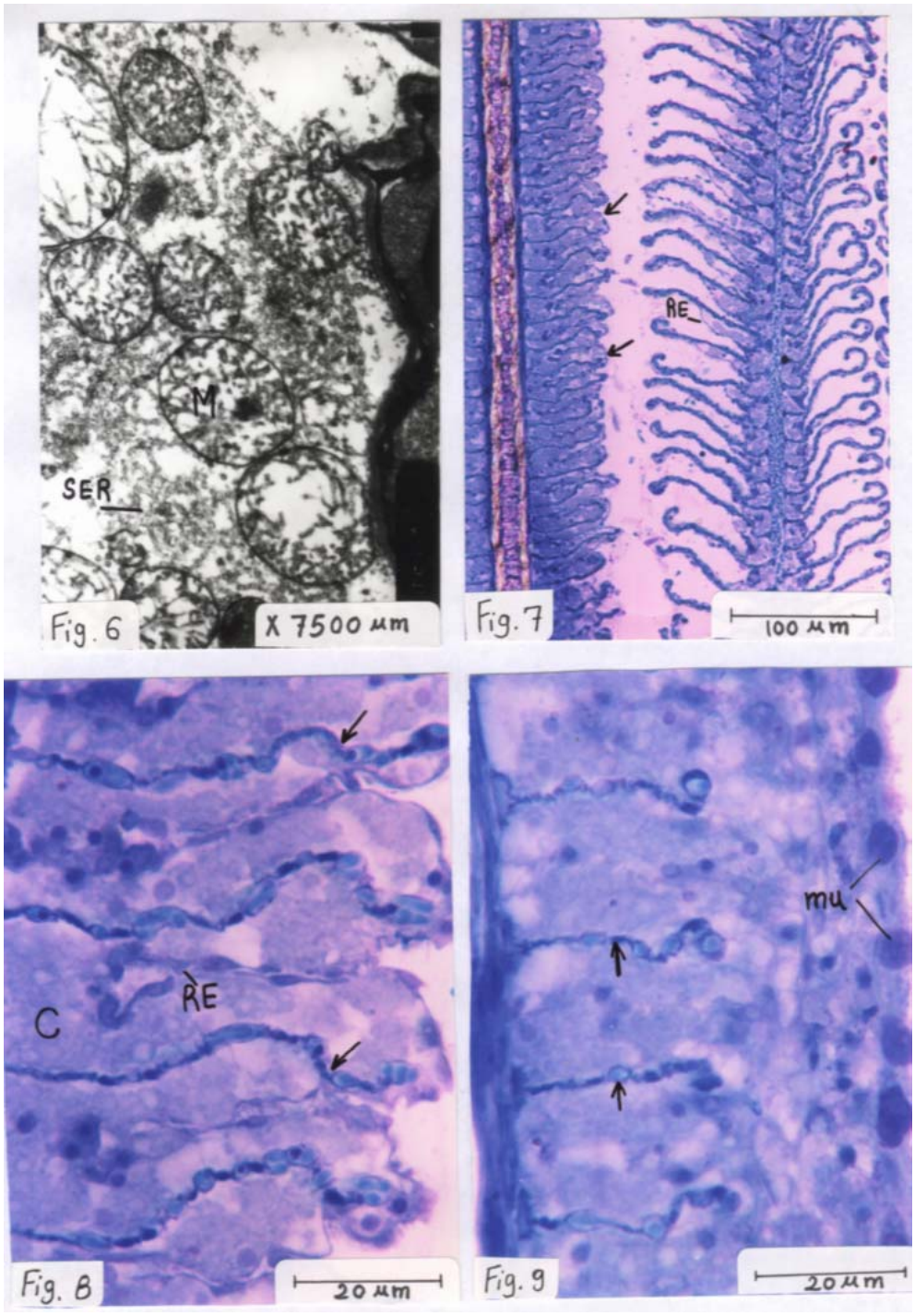

Fig. 6

Fig. 7

Fig. 8

Fig. 9

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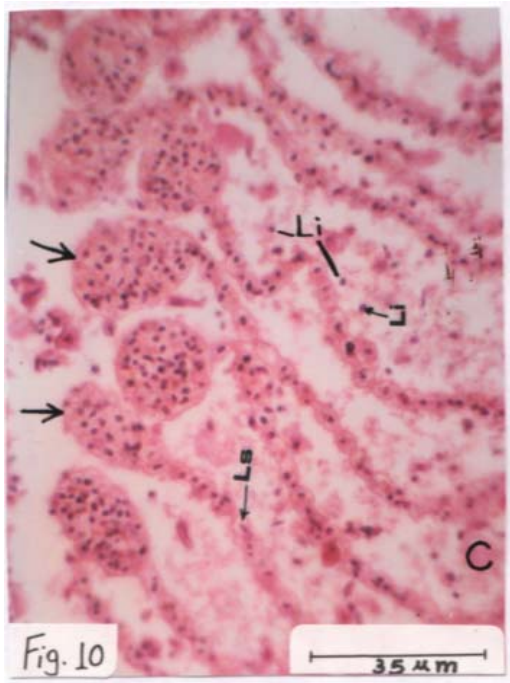


Fig. 10 35 µm

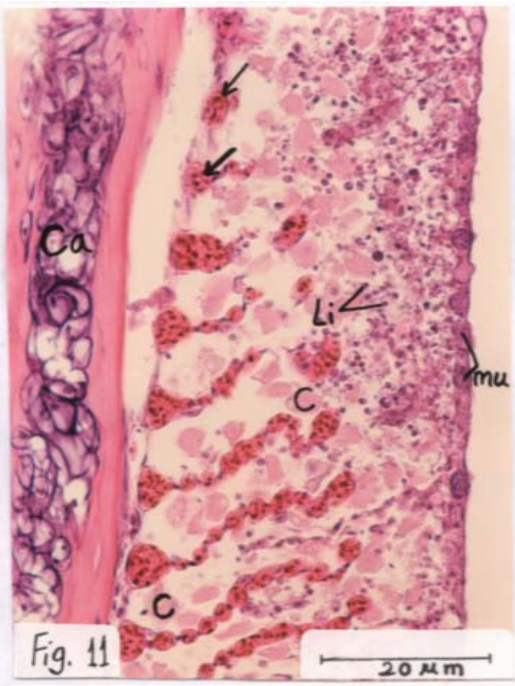


Fig. 11 20 µm

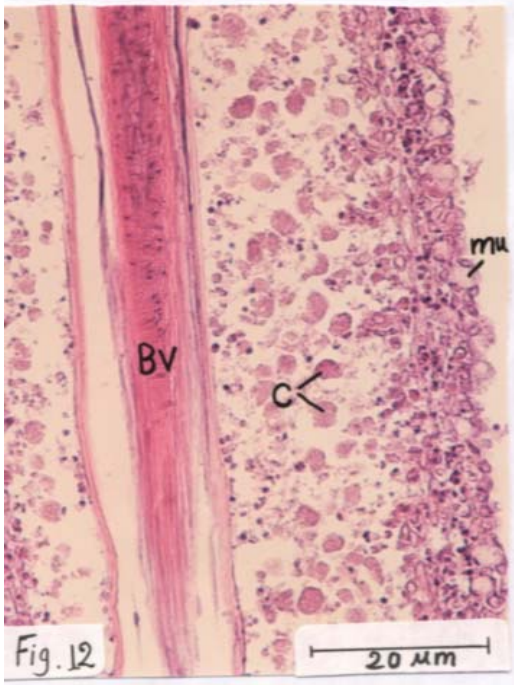


Fig. 12 20 µm

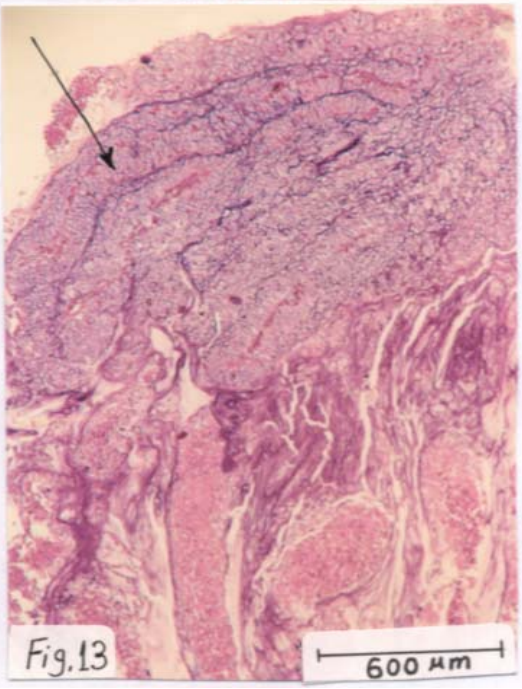
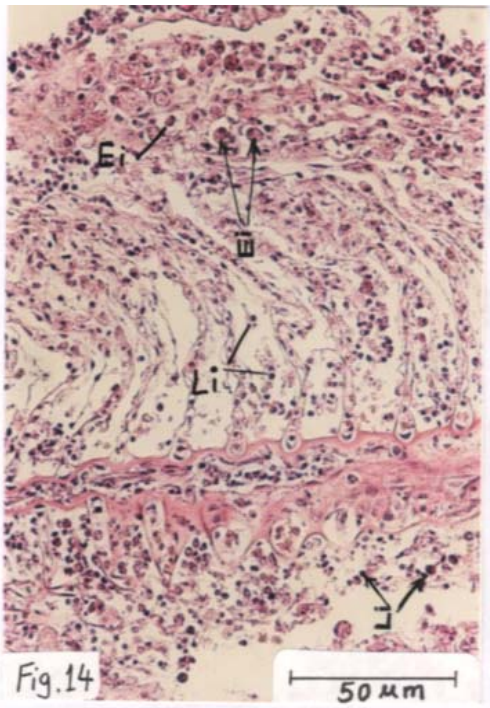


Fig. 13 600 µm


Ei
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Li
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Fig.14
50 µm

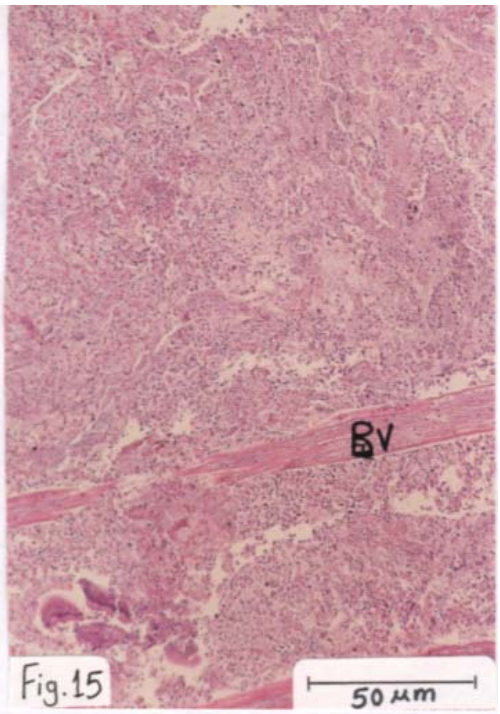
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Fig.15
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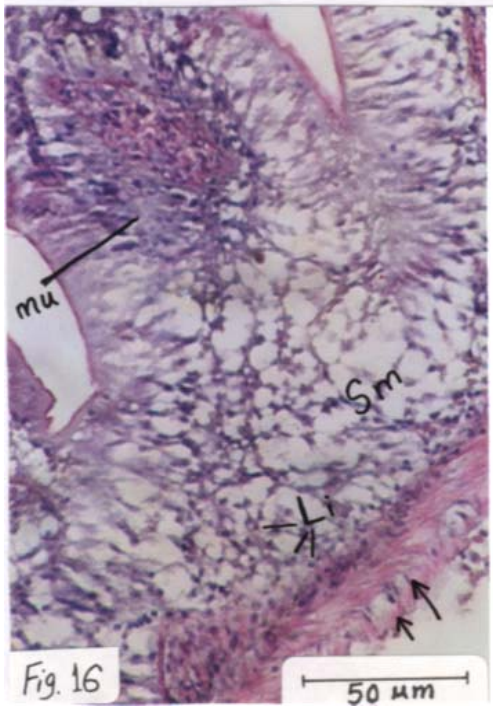
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Fig. 16
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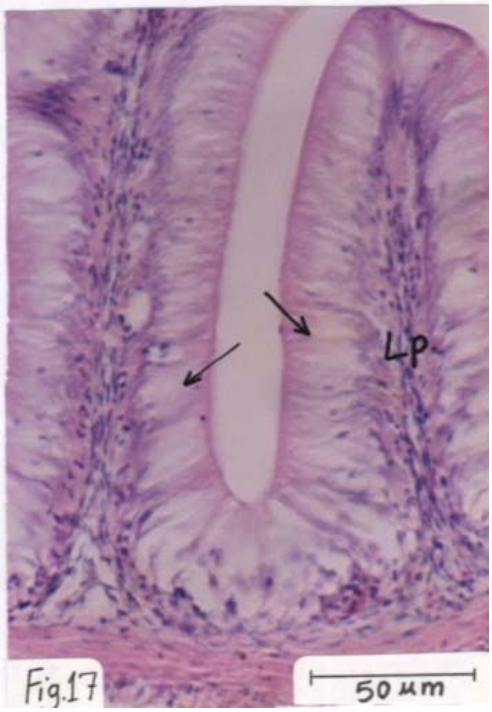
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Fig.17
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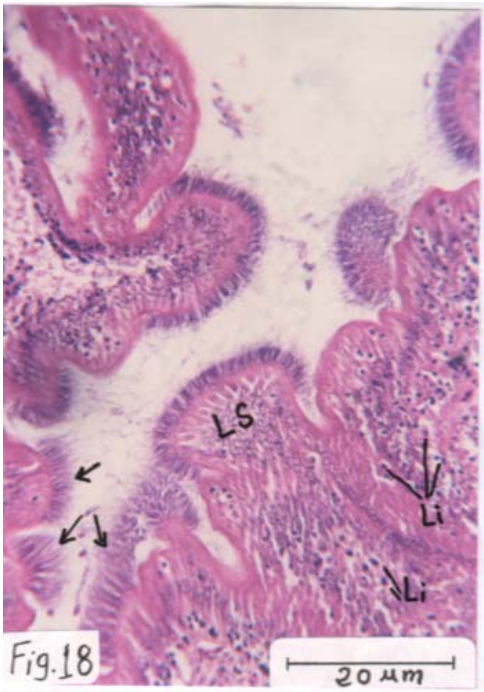


Fig. 18

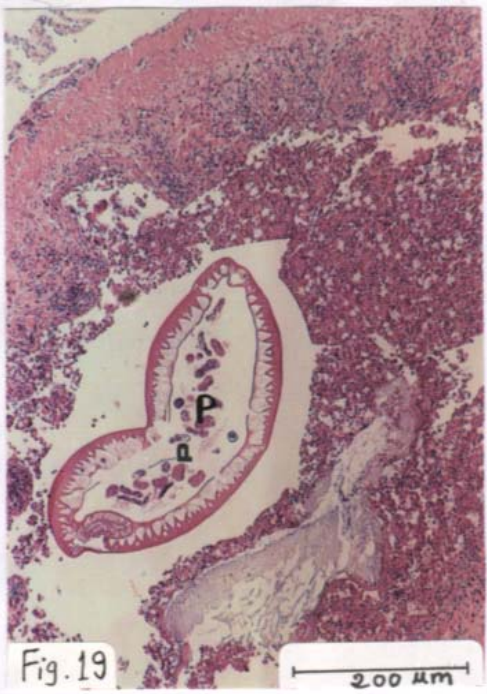


Fig. 19

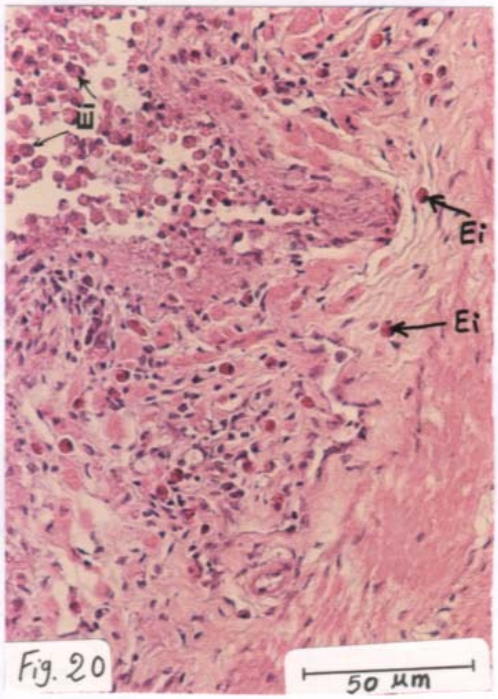


Fig. 20

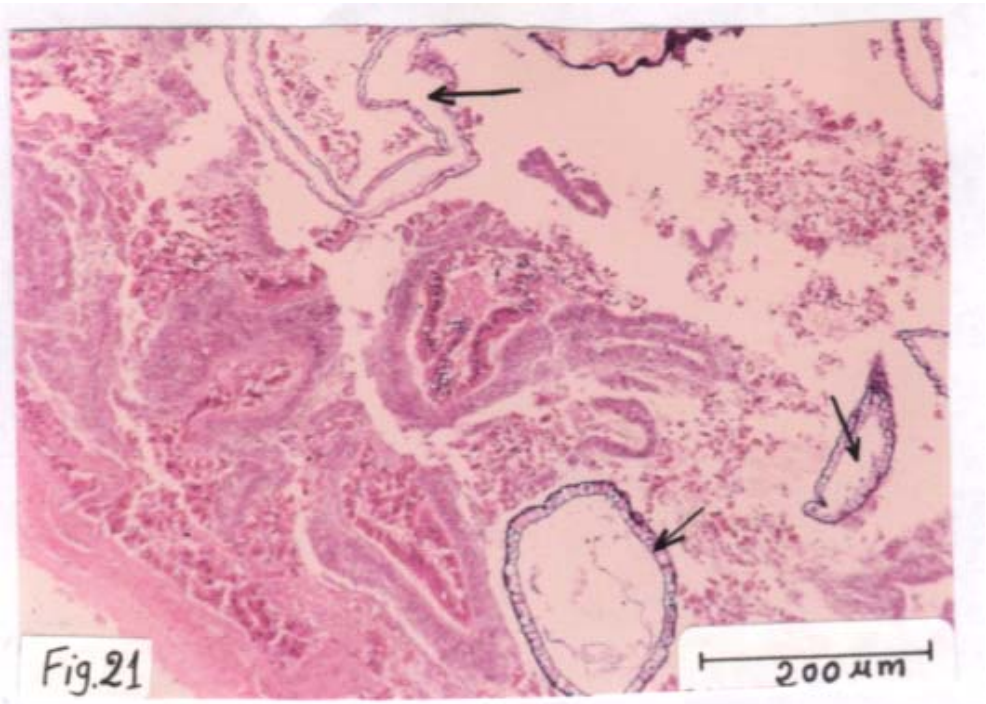
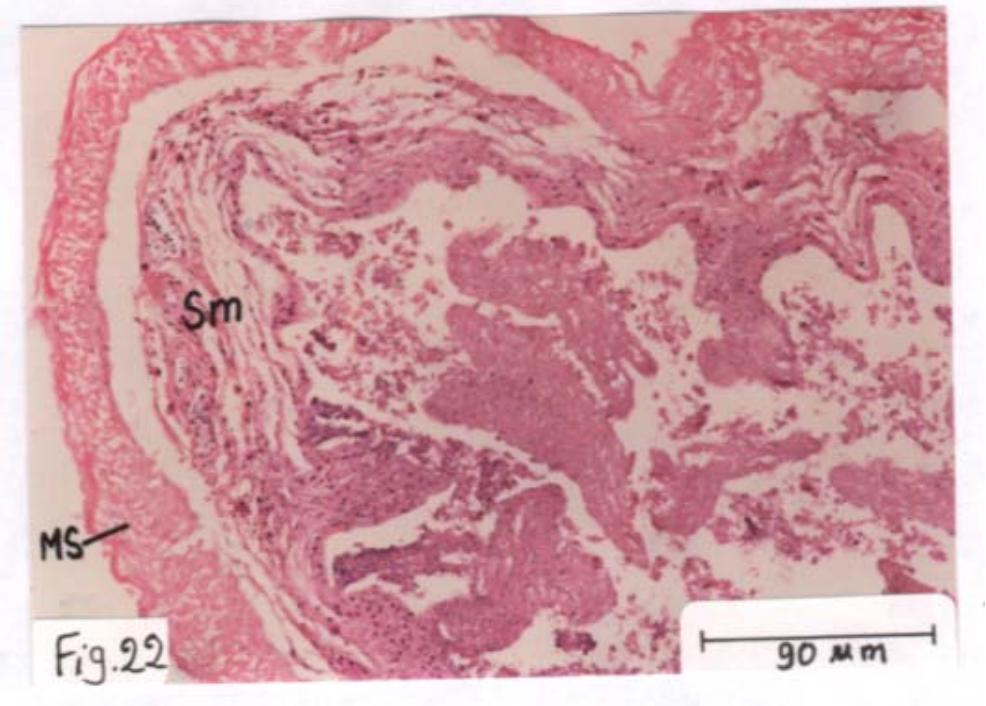


Fig.21
200 µm


Sm
MS
Fig.22
90 µm

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EXPLANATIONS OF FIGURES

Fig. 1: a - Map showing the five Sampling stations of the study area of Jeddah fish market.
Fig. 2: Secondary Lamellae with central Lamellar blood sinuses (LS) and respiratory epithelium (RE). Note: mucous cells (mu); chloride cells (C) (X 7500 μm).
Fig. 3: Electron micrograph (E.M.) of secondary Lamella showing respiratory epithelial cell (E) with large nucleus (N); Pillar cell (P) with arms encircling blood Sinus (BS) with red blood cells (X 7500 μm).
Fig. 4: E.M. of secondary Lamellae. Notice: mucous cell (mu) filled with secretary vesicles stretched pillar cell (P); RBCs in blood sinus (BS) (X 7500 μm).
Fig. 5: E.M. of primary lamellae showing chlorid cells Loaded with pleomorphic mitochondria (M) (X 2800 μm).
Fig. 6: High power E.M. from (Fig. 5) showing mitochondria (M) with tubular & reticular cristae; smooth endoplasmic reticulum (SER) (X 7500 μm).
Fig. 7: Gill filament showing cruling and twesting of secondary Lamellae; proliferation of chloride cells & separation of respiratory epithelia (RE) at basilamellar region. Notice. Lamellar swelling in the other gill filameut (arrow) (T.B.; bar = 100 μm).
Fig. 8: Swelling and contact of adjacent secondary Lamellae; Notice: Lamellar aneurysm (arrow), separation of respiratory lamellae (R E) chloride cells proliferation and Spreading (C) (T. B.: bar = 20 μm).
Fig. 9: Fusion of the adjacent secondary Lamellae. Notice atrophy of lamellar sinuses (arrow); accumulation of mucous cells at fused surface (mu) (T. B.; bar = 20 μm).
Fig. 10: Lamellar clubbing (arrows); sloughing and lysis of respiratory epithelium ; Leukocyte infilterate (Li) ; chlorid cells damage (C) (H.& E., bar = 35 μm).
Fig. 11: Fused secondary Lamellae showing atrophy and lysis of Lamellar blood sinuses (arrows); disintegration of respiratory epithelium, intact chlorid cells (C). Leukocyte infilteration (Li). Note: proliferation of mucous cells (mu) on fused surface; cartilaginous supporting axis (Ca) in primary lamella (H.& E., bar = 20 μm).
Fig. 12: Fused secondary Lamellae . Note : Complete lysis of Lamellar blood sinuses & respiratory epithelium ; damaged and intact chloride cells (C); heavy Leukocyte infilteration ; mucous cells proliferation (mu) at margin of fused Lamellae & fibrosis of the blood vessel (BV) (H.& E., bar = 20 μm).
Fig. 13: Parasitic infected gill showing parasitic colonies (arrow)on tope of damaged and congested gill filaments (H.& E., bar= 600 μm) .
Fig. 14: Part from Parasitic infected gill filament. Notice: Lamellar damage; heavy Lymphocytic (Li) & eosinophilic (Ei) infilteration (H & E., bar = 50 μm).
Fig. 15: Coagulative necrosis in parasitic infected gill tissue. Notice: blood vessel fibroses (H.& E., bar = 50 μm).
Fig. 16: Hypertrophy of the intestinal villi . Notice submucosal vacuolation (Sm); vacuolar degenetration of mucosal epithelial cells (mu); lymphocytic infilteration (Li); damaged serosa and outer muscularis (arrow) (PAS: bar = 50 μm).
Fig. 17: Cloudy swelling of the mucosal epithelial cells (arrow) and cellular infilteration in lamina properia (LP) (PAS: bar= 50 μm).
Fig. 18: Notice: Mucosal epithelial cells with abnormal long hair like microvilli (arrow); lymphatic sinuses (Ls) & heavy lymphocyte infilteration (Li) in mucosal and submucosal layers (H. E.; bar = 20 μm).
Fig. 19: Parasitic infected intestine. Notice. Parasitic warm (P) and sloughed intestinal tissue in lumen (H. E.; bar = 200 μm) .
Fig. 20: A high power from fig 19 showed: heavy eosinophilic infilteration (Ei) in the parasitic infected intestinal tissue (H. E.; bar = 50 μm).
Fig. 21: Parasitic infected intestine. Notice: damaged and sloughed intestinal villi, hemorrhage; parasitic warms (arrow) in intestinal lumens (H. E.; bar = 200 μm).
Fig. 22: Necrosis and massive desquamation of mucosal epithelium. Notice: separation of submucosa (Sm) from vacuolated muscularis; (Ms); (H. E.; bar = 90 μm).