Results

Results

Physical Analysis:.

1. Water temperature (°C):

Table (1): indicates that the maximum temperature was recorded in the south drain in summer of 1996 as 32.5° C, at the same time its temperature of the north drain was 30.6° C. Only in autumn, the temperature of the north drain was higher than that of the south drain. During the rest of the year, the temperature of the south drain was always higher with about 2 °C than that of the north drain.

Temperature of the wastewater of the fertilizer plant (FP): south Unit in summer of 1997 was 30.5° C. The south drain showed the highest level of temperature all the year around except in November1996. Wastewater of Misr-Iran showed the lowest level of temperature most of the year rounds except April.

Table (1): Seasonal variations of the Temperature °C of different wastewater

SEASONS LOCATIONS	SUMMER 1996	AUTUMN 1996	WINTER 1997	SPRING 1997	SUMMER 1997
FP (N. Unit):	**29.9**	**25.2**	**20.9**	**24.6**	**30.6**
FP (S. Unit):	**30.1**	**26**	**21**	**26**	**30.5**
Misr-Iran	**28**	**25**	**18.3**	**25**	**29**
North drain	**30.6**	**30.2**	**20.9**	**27.9**	**32**
South drain	**32.5**	**25.6**	**22**	**29.9**	**32**

N=north S=south

2. Hydrogen ion concentration (pH):

The pH values in table (2): show a considerable variation from one season to another. In autumn, the north drain wastewater showed maximum pH value (10.7):. The minimum values of pH for the south drain and FP north unit (7.7): were recorded in summer, 1997. On the other side, the maximum values of the south drain as 8.8 and Misr-Iran as 11.9 were recorded in Summer 96, while the wastewater of the south unit showed its highest pH value as 8.9 in autumn of 1996 and winter of 1997.

Table (2): Seasonal variations of the pH of different wastewater drains.

SEASONS LOCATIONS	SUMMER 1996	AUTUMN 1996	WINTER 1997	SPRING 1997	SUMMER 1997
FP (N. Unit):	**9.7**	**8.4**	**9.5**	**11.7**	**8.1**
FP (S. Unit):	**7.9**	**8.9**	**8.9**	**7.2**	**7.7**
Misr-Iran	**11.9**	**8.5**	**10**	**11.5**	**11.6**
North drain	**9.68**	**10.7**	**10.04**	**9.5**	**8.7**
South drain	**8.8**	**7.96**	**8.28**	**8.7**	**7.7**

Chemical analysis :.

1. Dissolved Oxygen (DO):

Results in table (3): show the dissolved oxygen concentration of the different industrial wastewater samples collected during the study period. Maximum value of DO was 3.5 ml O_2 /L recorded for Misr- Iran of Textile during winter of 1997, and the minimum value was 0.32ml O_2/L during summer of 1997. On the other hand, in south drain wastewater, the maximum DO value was 2.6 ml O_2/L in winter 1997, and the minimum value was 2.0 ml O_2/L in summer 1996.It is obvious from present data that the levels of DO in different effluents are inadequate for aquatic life.

Table (3): Seasonal variations of the DO of different wastewater drains.

SEASONS LOCATIONS	SUMMER 1996	AUTUMN 1996	WINTER 1997	SPRING 1997	SUMMER 1997
FP (N. Unit):	**2.8**	**2.2**	**3.0**	**1.2**	**2.2**
FP (S. Unit):	**2.9**	**3.1**	**3.0**	**2.4**	**2.0**
Misr-Iran	**3.2**	**3.3**	**3.5**	**2.9**	**0.32**
North drain	**2.3**	**2.3**	**2.5**	**2.4**	**2.3**
South drain	**2.0**	**2.5**	**2.6**	**2.4**	**2.2**

3. Alkalinity

The seasonal variations of the alkalinity are given in table (4):. In the wastewater of north drain, maximum value of alkalinity was 445 mg ($CaCO_3$):/L during spring 1997, and the minimum value was 220 mg/L in autumn of 1996, while in wastewater of the south drain the maximum value was recorded as 376 mg/L in winter, its the minimum value was 220 mg/L during summer 1997. On the other hand, in the wastewater of the Textile Company (Misr-Iran):, the alkalinity values were found to be 721.4 mg/L, during summer 1996, and 341.2 mg/L, in winter 1997. The results of alkalinity in FP north unit of Semad company varied between 593.5 mg/L in summer 1996 and 150 mg/L in winter 1997. Similarly, maximum value of alkalinity recorded in the wastewater of south unit was 320 mg/L in spring 1997 and the minimum value was 110 mg/L during winter 1997.

Table (4):. Seasonal variations of the alkalinity of different wastewater drains.

SEASONS LOCATIONS	SUMMER 1996	AUTUMN 1996	WINTER 1997	SPRING 1997	SUMMER 1997
FP (N. Unit):	**593.5**	**238.8**	**150**	**520**	**340.4**
FP (S. Unit):	**190**	**250**	**110**	**320**	**264.5**
Misr-Iran	**721.4**	**582.3**	**341.2**	**690.3**	**531.7**
North drain	**370**	**220**	**366.5**	**445**	**336**
South drain	**302.5**	**239.5**	**376**	**296**	**220**

4. Chemical Oxygen Demand (COD):

Table (5): shows the COD values of the industrial wastewater during the present study period. The results showed high values of COD in different industrial wastewater in all seasons. In the north drain wastewater, the COD values ranged between 166.7 mg O_2/L in spring 1997 and 145.5 mg/L in winter 1997. For south drain wastewater values of COD varied from 161.8 mg/L in summer 1997 to 148.8 mg/L in winter. On the other hand, the wastewater of Misr-Iran Textile exhibited a high value of COD (437.1 mg /L): in summer of 1996 and a low value (105.6 mg/L): in spring 1997, while maximum value of COD in the wastewater of FP north unit was 210 mg/L in summer 1996 and the minimum value was 72 mg/L in spring 1997. The maximum value of COD in south unit (187 mg/L): recorded in summer 1997 and the minimum value was 65.2 mg/L during the spring.

Table (5): Seasonal variations of the COD of different waste - water drains.

SEASONS LOCATIONS	SUMMER 1996	AUTUMN 1996	WINTER 1997	SPRING 1997	SUMMER 1997
FP (N. Unit):	**210**	**100.8**	**166.4**	**72**	**162**
FP (S. Unit):	**140**	**160**	**110.4**	**65.2**	**187**
Misr-Iran	**437.1**	**192**	**240.5**	**105.6**	**339**
North drain	**158.8**	**158.4**	**145.5**	**166.7**	**160**
South drain	**161.1**	**152.8**	**148.8**	**158**	**161.8**

5.Ammonium Salts:

Table (6): shows the ammonium concentrations in the wastewater of the two main drains and in the wastewater of the different industrial plants in the area of study. The concentration of ammonia was always higher in summer than any other season. This general character was observed in 1996 and 1997 in all the five wastewater drains studied. The higher values were recorded in the north drain in summer 1996 as 781.0 and 369.3 mg/L in summer of 1997 for the same drain. The wastewater of the FP south unit showed relatively high value in summer 1996 (497.0): comparing with that of summer 1997 (269.5 mg/L):. In winter, the lowest value 1.49 was recorded in the wastewater of the Misr-Iran of Textile and the highest value was recorded for the south unit of fertilizer plant as 217.4 mg/L.

Table (6): Seasonal variations of the Ammonia salts of different wastewater drains.

SEASONS LOCATIONS	SUMMER 1996	AUTUMN 1996	WINTER 1997	SPRING 1997	SUMMER 1997
FP (N. Unit):	**271.9**	**229.6**	**100.9**	**131.8**	**179.8**
FP (S. Unit):	**497**	**151.4**	**217.4**	**177.5**	**269.5**
Misr-Iran	**3.99**	**1.17**	**1.49**	**1.91**	**3.0**
North drain	**781**	**153**	**131.8**	**77.5**	**369.3**
South drain	**271.7**	**131.1**	**100.9**	**131.8**	**269.5**

6. Phosphate Salts:

Table (7): shows the seasonal variations in the concentration of inorganic phosphorous dissolved in the discharged industrial wastewater. The highest concentration of inorganic phosphorous was recorded in spring in the wastewater of Misr-Iran of Textile (3.2 mg/L): followed by the north drain in the same season (1.86 mg /L):. The lowest concentration of inorganic phosphorous was recorded for the wastewater of the FP south unit 0.13 mg/L in spring 1997. Also, the lowest concentration for the other wastewater was recorded in autumn 1996 for the north drain and Misr-Iran (0.5 mg/L each):. In summer 1996 the south drain showed (0.31 mg/L): and finally in winter 1997, the FP north unit showed 0.26 mg /L.

Table (7): Seasonal variations of the Phosphorus salts of different wastewater drains.

SEASONS LOCATIONS	SUMMER 1996	AUTUMN 1996	WINTER 1997	SPRING 1997	SUMMER 1997
FP (N.Unit):	**0.32**	**0.53**	**0.26**	**0.65**	**1.38**
FP (S. Unit):	**1.2**	**0.49**	**0.67**	**0.13**	**0.9**
Misr-Iran	**1.32**	**0.5**	**1.26**	**3.2**	**0.9**
North drain	**0.94**	**0.50**	**0.93**	**1.86**	**0.88**
South drain	**0.31**	**0.57**	**1.26**	**0.52**	**0.61**

Toxicity tests:

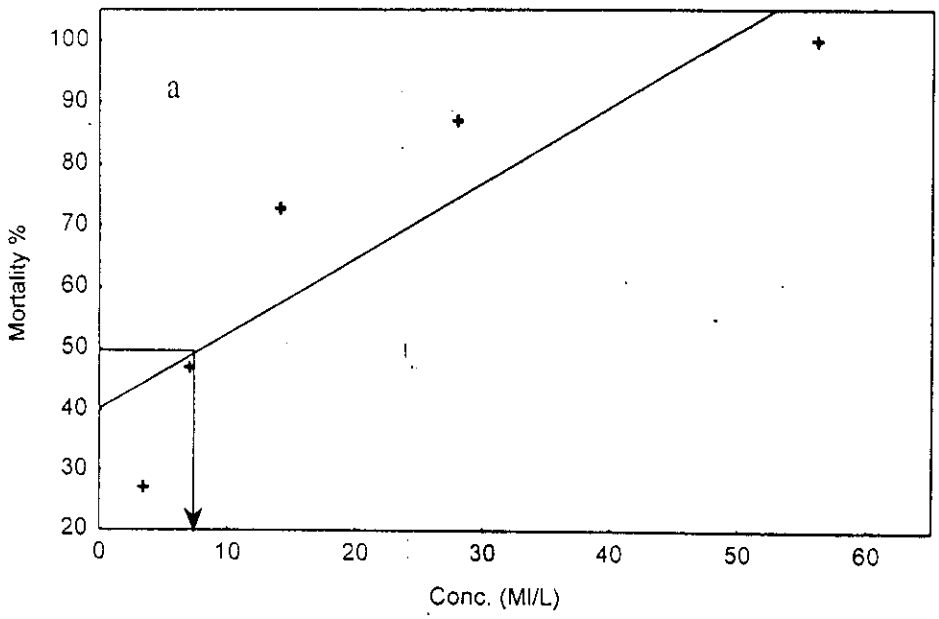
In the present study, Bioassay tests were carried out to identify the effect of these effluents and their lethality.

1- Fertilizer plant (N. Unit): 96 h. LC_{50}.

The results of these experiments are given in table (8): and illustrated in Figure (10 a.):. The highest mortality rates were observed in the concentration 56.0 ml/L with no survivals after 48 h and the least effect was observed in the concentration 3.5 ml/L where only 8 fishes were dead after 96 h. According to the data, the observed 96 h LC_{50} of the wastewater of the FP north unit was 7.4 ml/L and the calculated one was 9.3 ml/L (Fig. 10 b):.

Table (8): Observed and corrected mortality of *Mugil seheli* fingerlings after 24, 48, 72, and 96 h of exposure to plant fertilizer (North unit): effluents.

CONC. ML/L	No. OF LIVING FISHES	NO. OF DEAD FISHES AT				OBSERVED M %	CORRECTED M %
		24 h	48 h	72 h.	96 h.		
56	30	27	28	29	30	100	100
28	30	22	23	24	26	87	87
14	30	14	17	20	22	73	72
7	30	10	12	13	14	47	45
3.5	30	6	7	7	8	27	25
Cont.	30	1	1	1	1	3	
The observed 96 h LC_{50} A B R		7.4 ml/L 40.21 1.22 0.88					
The calculated 96 h LC_{50} A B R		9.3 ml/L 38.33 1.27 0.88					



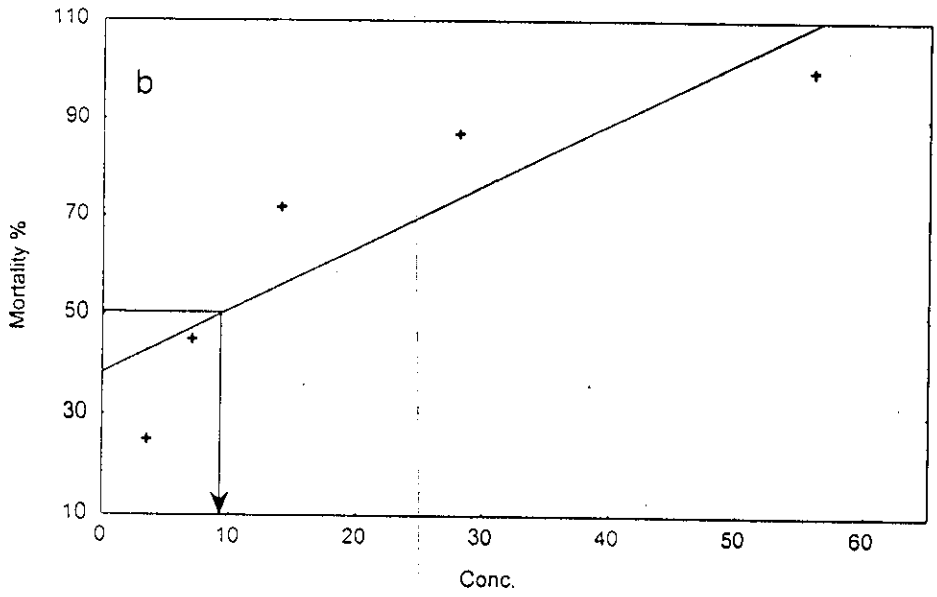


Figure (10). The observed (a) and corrected (b) mortality of fingerlings of *Mugil seheli* and the 96h LC_{50} of different concentrations of north unit effluent.

2- Fertilizer plant (S. unit): 96h LC_{50} :

The results of this experiment were given in Table (9): and illustrated in figure (11 a):, according to the data 25 fishes died in the first 24 h in the highest concentration (56.0 ml/L): while only 5 fishes died at concentration 3.5 ml/L. The percentage of mortality tends to increase gradually in all concentrations. By the end of the experiment, it was found that 43 % of the fishes died in the concentration 7.0 ml/L compared with 27 % in concentration 3.5 ml/L. The observed 96 h LC_{50} for the effluent was 7.6 ml /L and the calculated one was 9.0 ml/L (Fig. 11 b):.

Table (9): Observed and corrected mortality of *Mugil seheli* fingerlings after 24, 48, 72, and 96 h of exposure to fertilizer plant (South unit): effluents.

CONC. ML/L	No. OF LIVING FISHES	NO. OF DEAD FISHES AT				OBSERVED M %	CORRECTED M %
		24 h	48 h	72 h.	96 h.		
56	30	25	26	28	30	100	100
28	30	22	22	23	37	90	90
14	30	19	20	22	23	77	76
7	30	10	11	12	23	43	41
3.5	30	5	6	7	8	27	25
Cont.	30	-	-	1	1	3	
The observed 96 h LC_{50} A B R		7.6 ml/L 40.25 1.25 0.85					
The calculated 96 h LC_{50} A B R		9.0 ml/L 38.38 1.29 0.88					

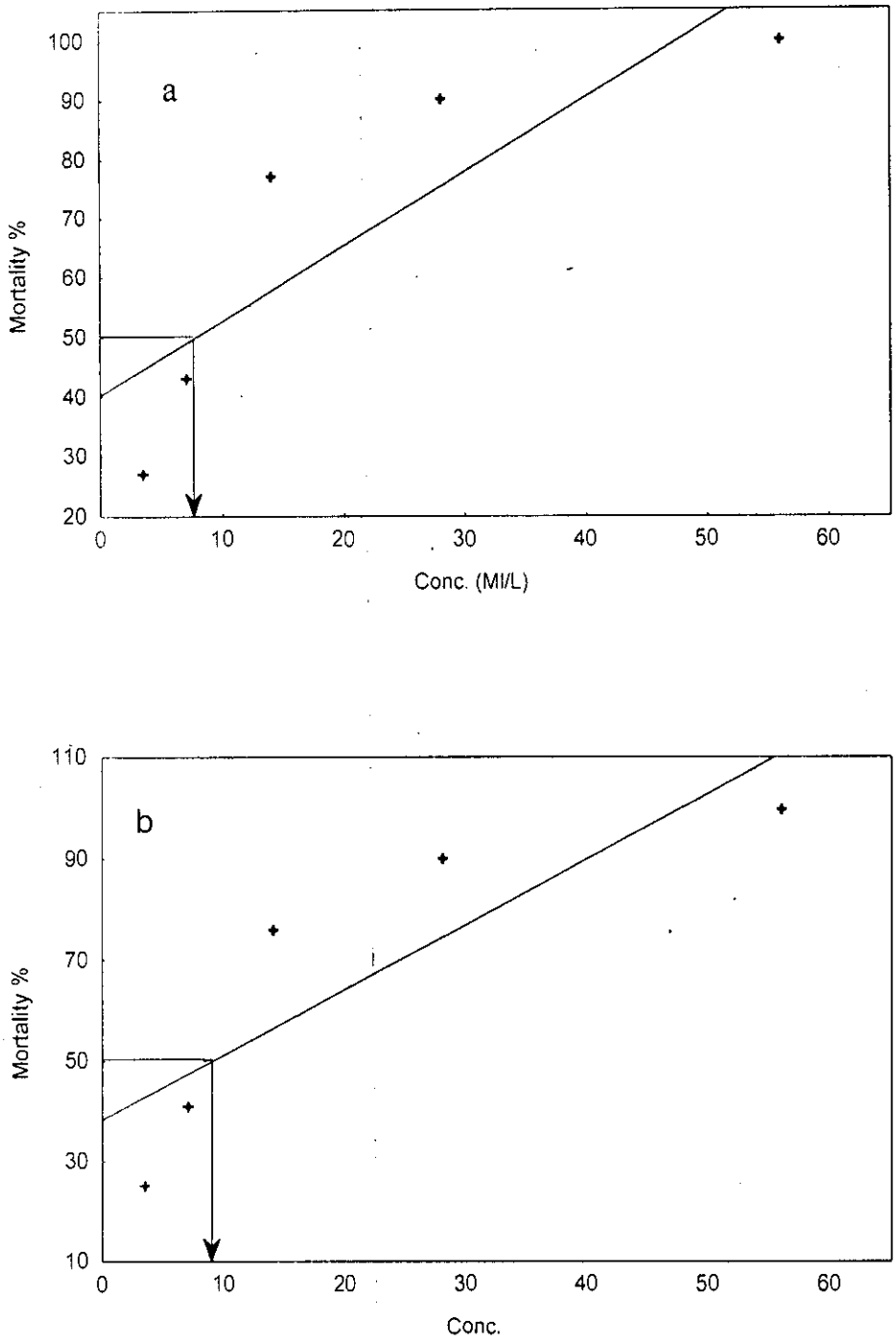


Figure (11). The observed (a) and corrected (b) mortality of fingerlings of *Mugil seheli* and the 96h LC_{50} of different concentrations of the South Unit effluent.

3. Misr-Iran of Textile 96h LC_{50}:

Table (10): shows the details of the bioassay experiment of LC_{50} for Misr-Iran of Textile effluent with fingerlings of *Mugil seheli*. The results of this experiment are illustrated in figure (12 a):. According to table (10):, 93 % of the fishes in concentration 250 ml/L died before the end of the experiment. It was observed that the percentage of mortality in the concentrations 200, 150 and 100ml/L did not increase in the last 24 h of the experiment. However, after the first 48 h only 7 fishes dead at concentration 50 ml/L were counted. Application of the linear regression equation revealed that the observed 96 h LC_{50} for Misr-Iran of Textile raw effluent was 118.0 ml/L. and the calculated one was 122.0 ml/L (Fig. 12 b):.

Table (10): Observed and corrected mortality of *Mugil seheli* fingerlings after 24, 48, 72, and 96 h of exposure to Misr-Iran of Textile effluents

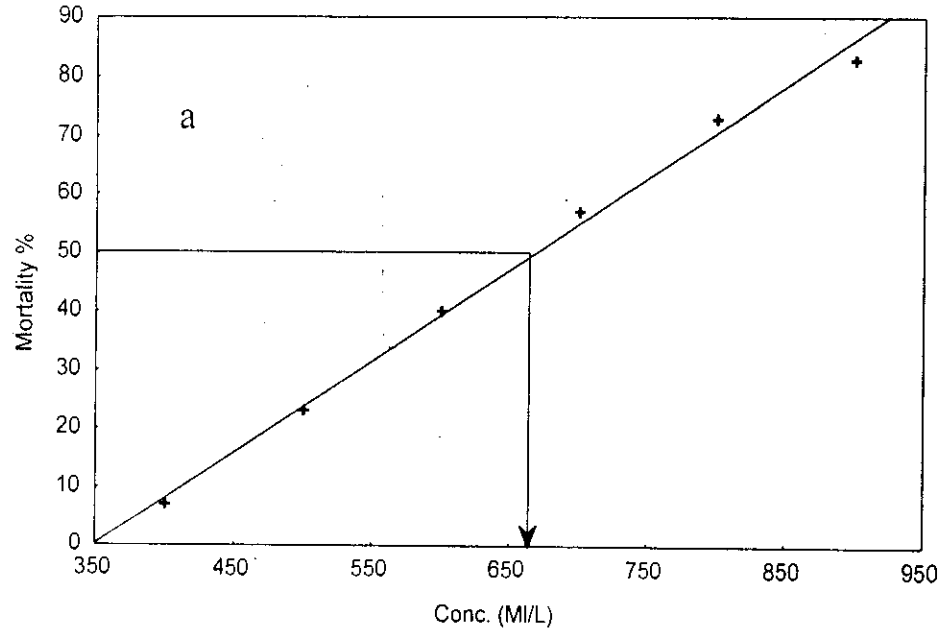
CONC. ML/L	No. OF LIVING FISHES	NO. OF DEAD FISHES AT				OBSERVED M %	CORRECTED M %
		24 h	48 h	72 h.	96 h.		
250	30	23	26	28	30	100	100
200	30	19	21	24	25	83	82
150	30	14	16	18	19	63	62
100	30	6	10	13	13	43	41
50	30	4	7	7	7	23	21
Cont.	30	1	1	1	1	3	
he observed 96 h LC_{50} A B R		118 ml/L 4.20 0.39 0.99					
The calculated 96 h LC_{50} A B R		122 ml/L 1.53 0.39 0.99					

4. Slaughterhouse effluents 96 h. LC_{50} : -

The results are graphically represented in figure (10):, which indicate that, there was a limited effect on the survival of fishes within the first 24 h in concentrations 400 and 500 ml/L. The mortality percentage increased gradually with increase of the concentration and reached 83 % at 900m/L after 96h of exposure. The observed LC_{50} (table, 11 and Figure 13 a): was 664 ml/L after 96h of exposure while the calculated one was found to be also 664 ml/L (Fig.13 b):.

Table (11): Observed mortality of *Mugil seheli* fingerlings after 24, 48, 72, and 96 h of exposure to Slaughterhouse effluents.

CONC. ML/L	No. OF LIVING FISHES	NO. OF DEAD FISHES AT				OBSERVED M %	CORRECTED M %
		24 h	48 h	72 h.	96 h.		
900	30	20	20	23	25	83	83
800	30	17	19	20	22	73	73
700	30	11	14	16	17	57	57
600	30	6	10	11	12	40	40
500	30	2	5	6	7	23	23
400	30	-	-	2	2	7	7
Cont.	30	-	-	-	-	-	-
The observed 96 h LC_{50} A B R		664 ml/L -57.42 0.16 0.99					
The calculated 96 h LC_{50} A B R		664 ml/L -57.42 0.16 0.99					



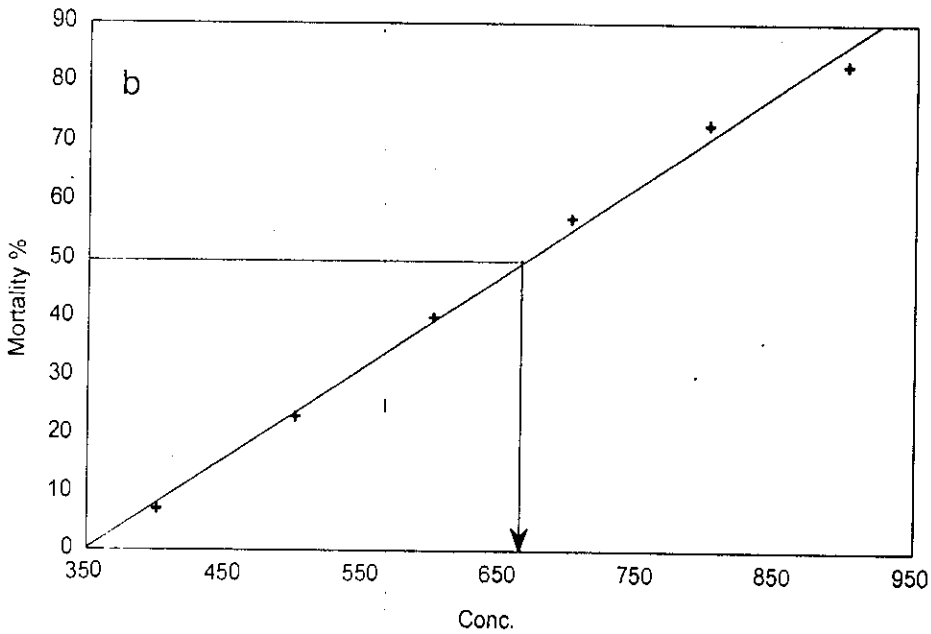


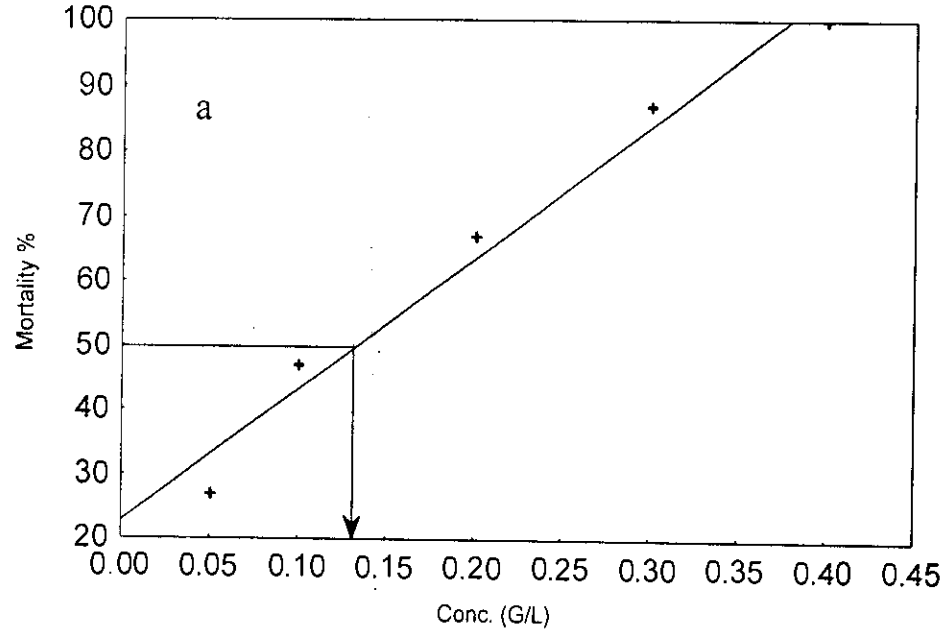
Figure (13). The observed (a) and corrected (b) mortality of fingerlings of *Mugil seheli* and the 96h LC_{50} of different concentrations of the Slaughter House effluent.

5. Ammonium Chloride 96 h. LC50:

The results of this experiment are given in table (12): and graphically represented in figure (14):. Toxicity of ammonia is not a matter of discussion, however, in dilution such as 0.05 g/L, fishes could survive with few mortalities. Table (12): shows that, 27 % of the fishes were dead after 96 h in low concentration, the observed LC_{50} after 96 h was found to be 0.13 g/L Ammonium Chloride and the calculated one was 0.14g/L (Fig.14 a & b):

Table (12): Observed and corrected mortality of *M. seheli* fingerlings after 24, 48, 72, and 96 h. of exposure to Ammonium Chloride.

CONC. ML/L	No. OF LIVING FISHES	NO. OF DEAD FISHES AT				OBSERVED M %	CORRECTED M %
		24 h	48 h	72 h.	96 h.		
0.4	30	30	30	30	30	100	100
0.3	30	20	23	25	26	87	87
0.2	30	14	16	17	20	67	66
0.1	30	8	11	13	14	47	45
0.05	30	3	5	7	8	27	25
Cont.	30	1	1	1	1	3	
The observed 96 h LC_{50} A B R		0.13 g/L 22.91 203.29 0.98					
The calculated 96 h LC_{50} A B R		0.14 g/L 20.50 210.00 0.98					



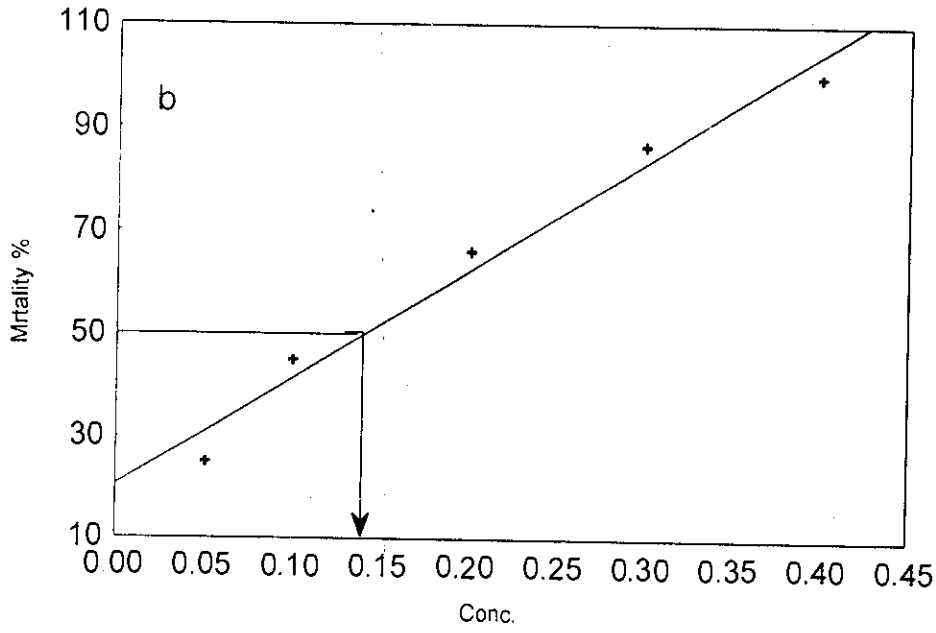


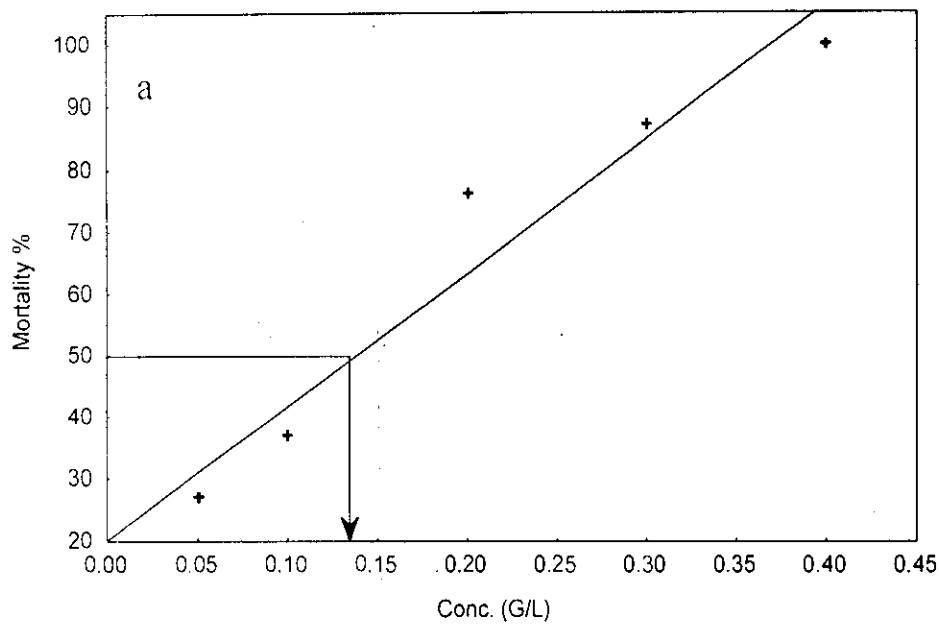
Figure (14). The observed (a) and corrected (b) mortality of fingerlings of *Mugil seheli* and the 96h LC_{50} of different concentrations of the Ammonium Chloride.

6. Sodium Hydroxide 96h LC_{50}:

The results in Table, 13 and Figure 15 show that, as concentration 0.4 g/L has the highest effect as 93% of the fishes were died in the first 24 h. In other concentrations, the mortality increased gradually every 24 h. The concentration 0.05 g/L showed the lesser mortality rate as only 27% of the fishes were dead after 96h. The results revealed the observed LC_{50} for NaOH was 0.13g/L after 96 h and the calculated one was 0.14g/L (Fig.15 a&b):.

Table (13): Observed and corrected mortality of *Mugil seheli* fingerlings after 24, 48, 72, and 96 h of exposure to Sodium Hydroxide.

CONC. ML/L	No. OF LIVING FISHES	NO. OF DEAD FISHES AT				OBSERVED M %	CORRECTED M %
		24 h	48 h	72 h.	96 h.		
0.4	30	28	29	30	30	100	100
0.3	30	22	23	25	26	87	87
0.2	30	13	17	19	22	73	76
0.1	30	8	11	11	11	37	35
0.05	30	5	6	7	8	27	25
Cont.	30	1	1	1	1	3	
The observed 96 h LC_{50} A B R		**0.13 g/L** **20.31** **212.81** **0.98**					
The calculated 96 h LC_{50} A B R		**0.14 g/L** **17.94** **222.19** **0.97**					



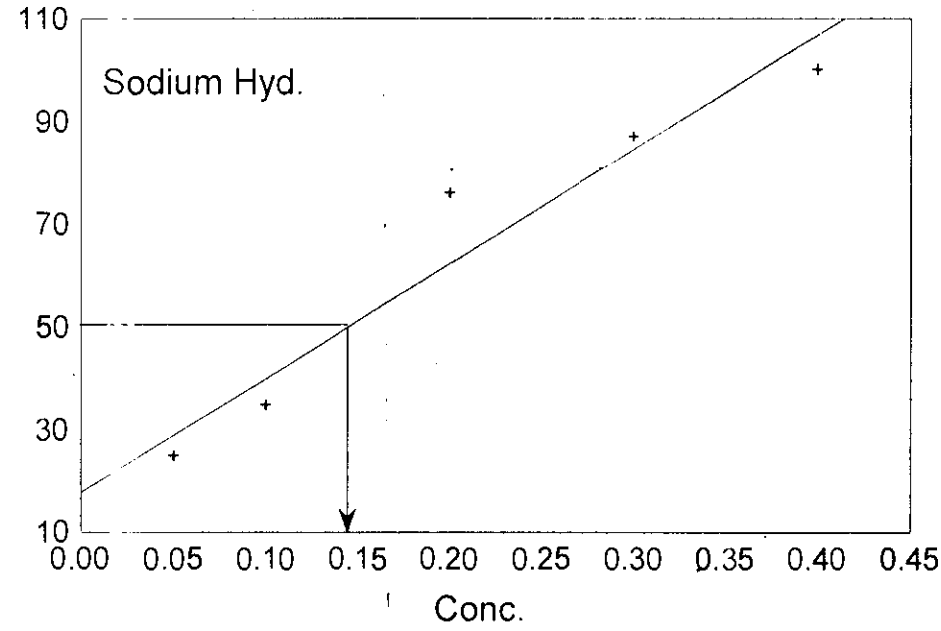


Figure (15). The observed (a) and corrected (b) mortality of fingerlings of *Mugil seheli* and the 96h LC_{50} of different concentrations of the Sodium Hydroxide.

7. North drain effluents 96h LC_{50}:

The results are given in table (14): and figure (16):. Both the table and graph are showing the relationship among mortality percentage, effluent concentration and exposure time, The highest mortality percentage was 100 % in concentration 56.0 ml/L after 48 h of exposure. In concentration 7.0 ml/L, the mortality was 27% within the first 24 h of exposure and 43% after 96 h of exposure. The observed LC_{50} was 9.13 ml/L after 96 h of exposure and the calculated one was 10.5 ml/L (Fig. 11 a & b):.

Table (14): Observed and corrected mortality of *Mugil seheli* fingerlings after 24, 48, 72, and 96 h of exposure to (North drain): effluents.

CONC. ML/L	No. OF LIVING FISHES	NO. OF DEAD FISHES AT				OBSERVED M %	CORRECTED M %
		24 h	48 h	72 h.	96 h.		
56	30	29	30	30	30	100	100
28	30	19	22	24	25	83	82
14	30	14	16	19	22	67	66
7	30	8	11	12	13	43	41
3.5	30	3	7	8	9	33	31
Cont.	30	1	1	1	1	3	
The observed 96 h LC_{50} A B R		**9.13 ml/L** **38.96** **1.211** **0.93**					
The calculated 96 h LC_{50} A B R		**10.5 ml/L** **36.96** **1.25** **0.93**					

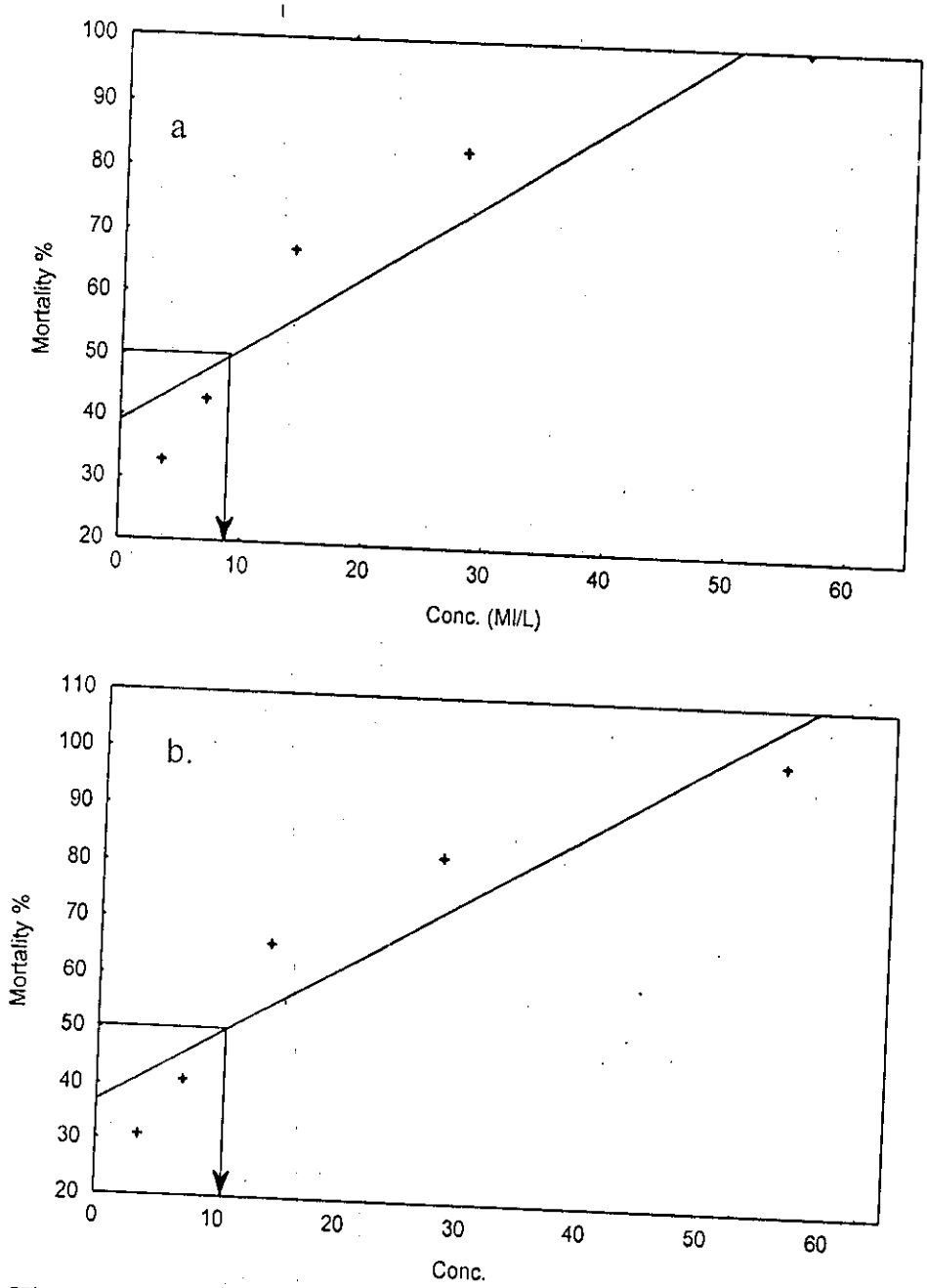


Figure (16). The observed (a) and corrected (b) mortality of fingerlengs of *Mugil seheli* and the 96h LC_{50} of different concentrations of the North Drain effluent.

8. South drain effluents 96h LC_{50}:

According to the results of this experiment (Table, 15 and Figure 17): the toxicity of the south drain seems to be less severe than that of the north drain. The concentration 3.5 ml/L showed less mortality rate, as only 20% of the fishes were dead after 96 h. The final result after calculation of the observed and calculated mortalities, the observed LC_{50} was 10.69 ml/L and the calculated was 12.70 ml/L respectively for the south drain effluents (Fig.17 a & b):.

Table (15): Observed and corrected mortality of *Mugil seheli* fingerlings after 24, 48, 72 and 96 h of exposure to (South drain): effluents.

CONC. ML/L	No. OF LIVING FISHES	NO. OF DEAD FISHES AT				OBSERVED M %	CORRECTED M %
		24 h	48 h	72 h.	96 h.		
56	30	29	29	29	30	100	100
28	30	20	23	25	26	87	87
14	30	14	17	18	19	63	62
7	30	7	10	11	12	40	37
3.5	30	3	4	6	6	20	17
Cont.	30	-	-	-	1	3	
The observed 96 h LC_{50} A B R		**10.69 ml/L** **31.75** **1.39** **0.91**					
The calculated 96 h LC_{50} A B R		**12.70 ml/L** **29.08** **1.46** **0.90**					

Histological Observations.

I. Gills.

Normal gills.

The gills of control specimen of *Mugil seheli* fingerlings show normal characteristics as seen in Figures (18 & 19):. One layer of epithelial cells surrounding the lamellae. The epithelial cells are joined together by desmosomes and light junction. The inner surface of the epithelial cells sits on basement membrane of pillar cells. Each pillar cell is shaped like a spool, where the flanges of each cell touch those of the adjacent pillar cells. This arrangement creates a space between the two surfaces of the pillar cells function as blood capillaries. Beneath the epithelium, the chloride cells, the goblet cells and undifferentiated cells are located at the base of the secondary lamellae, which are supported by blood capillaries and the pillar system (Fig.19):.

Treated Gills.

1.Effect of the north drain effluents.

The gills of the fingerlings exposed to 7.0 ml/L of north drain effluents for 48 h displayed different abnormalities as shown in Figures (20&21):. Gill filaments lost its normal histological appearance. The epithelial layer manifested irregular shape and was desquamated in some areas. Subepithelial oedema was focally seen. Proliferation of epithelium in interlamella spaces causes deformation of pillar system. Some chloride cells appeared degenerated. The blood vessels in some lamellae showed congestion.

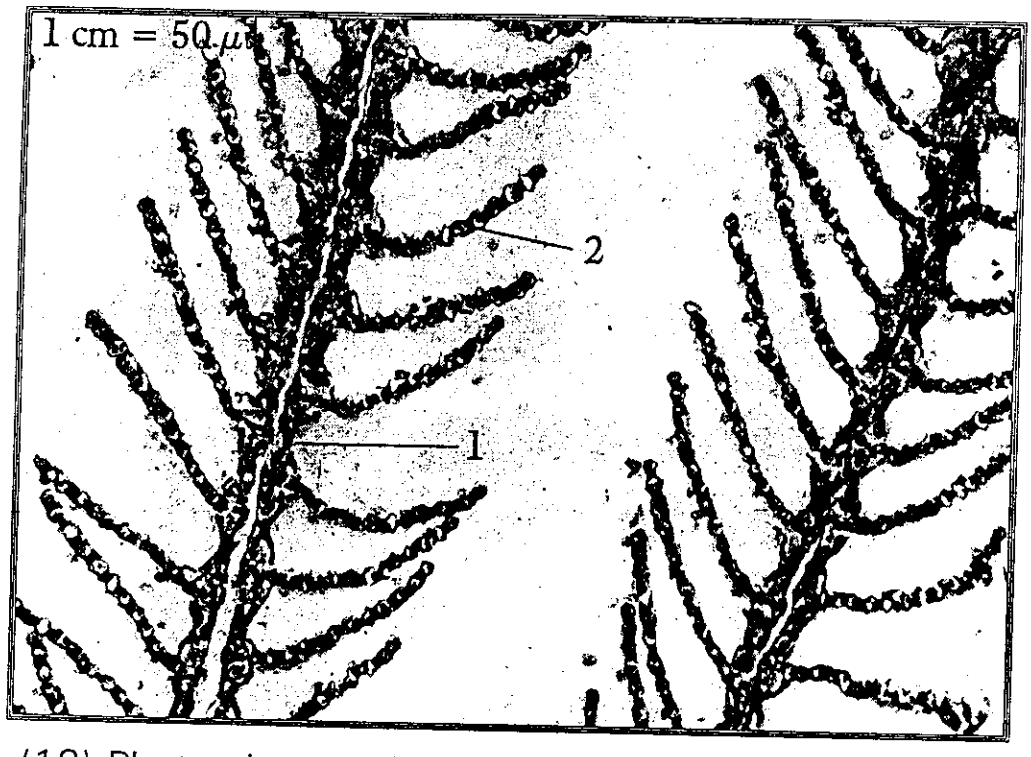


Fig (18) Photomicrograph of a section through the gill of *Mugil seheli* fingerlings showing the normal architecture of teleosts gills {primary lamella (1) & secondary lamella (2)}. The section stained with H & E.

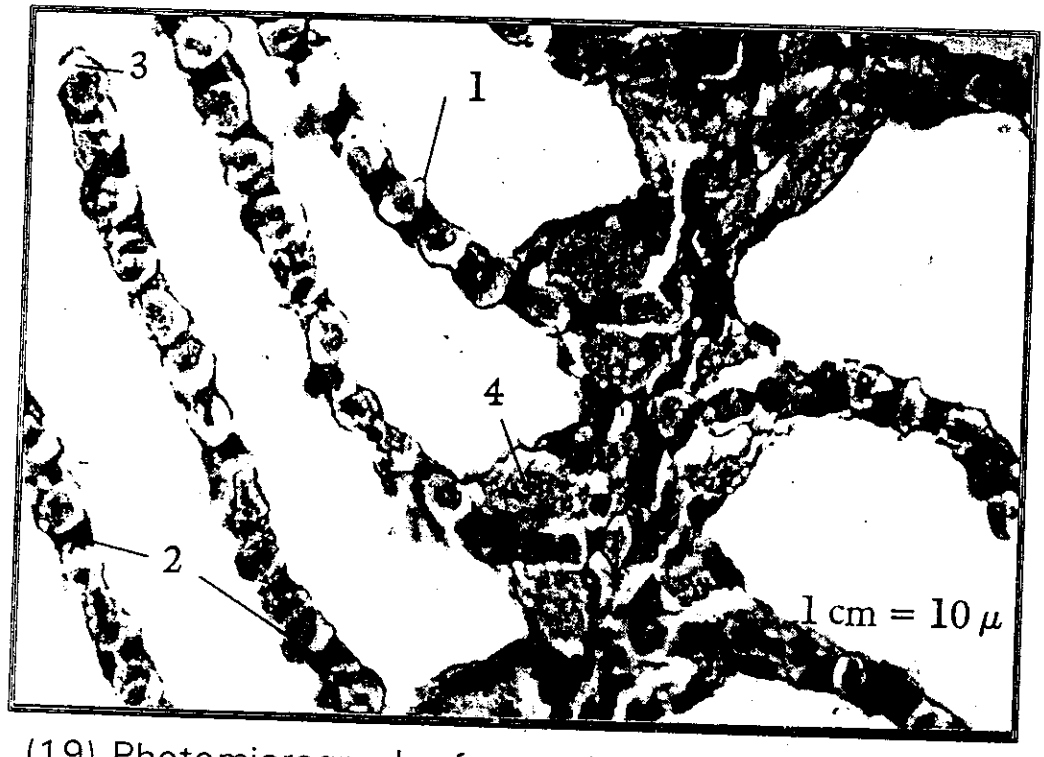


Fig (19) Photomicrograph of a section through the gill of *Mugil seheli* fingerlings showing a healthy primary lamella and secondary lamella with thin relatively thin epithelium (1) covers well-organized pillar system (2), blood capillaries (3) and chloride cells (4). The section stained with H & E.

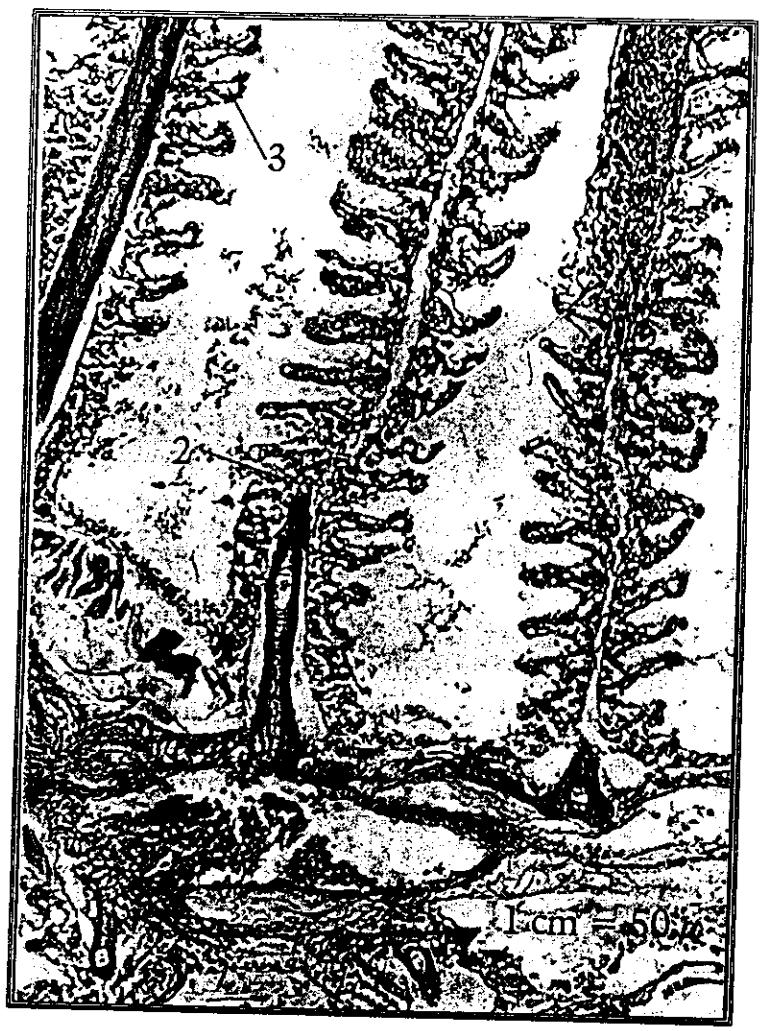


Fig (20) Photomicrograph of a section through the gill of treated *Mugil seheli* fingerlings exposed to 7.0 ml/L of north drain effluents for 48 h showing dilatation and congestion of blood vessels (1), hyperplastic proliferation of epithelial cells lining the secondary lamellae (2) and subepithelial oedema (3). The section stained with H & E.}

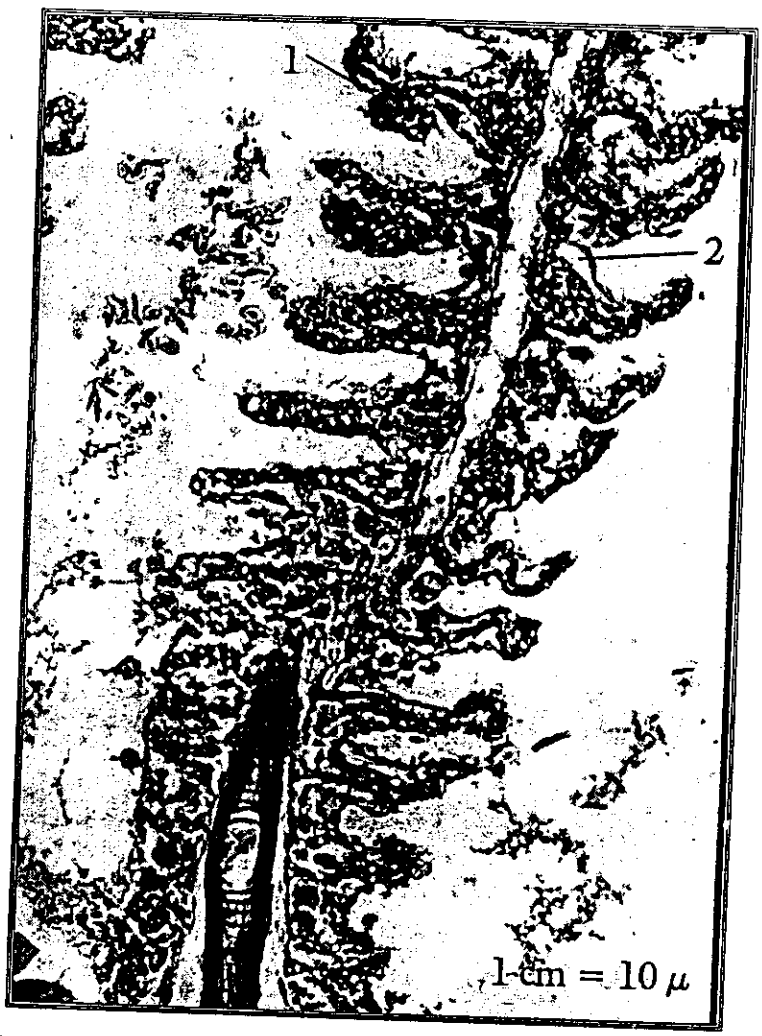


Fig (21) Photomicrograph of a section through the gill of treated *Mugil seheli* fingerlings exposed to 7.0 ml/L of north drain effluents for 48 h showing slight ;lamellar hyperplasia (1) which appeared focally sloughed and desquamated subepithelial oedema (2) was also seen. The section stained with H & E.}

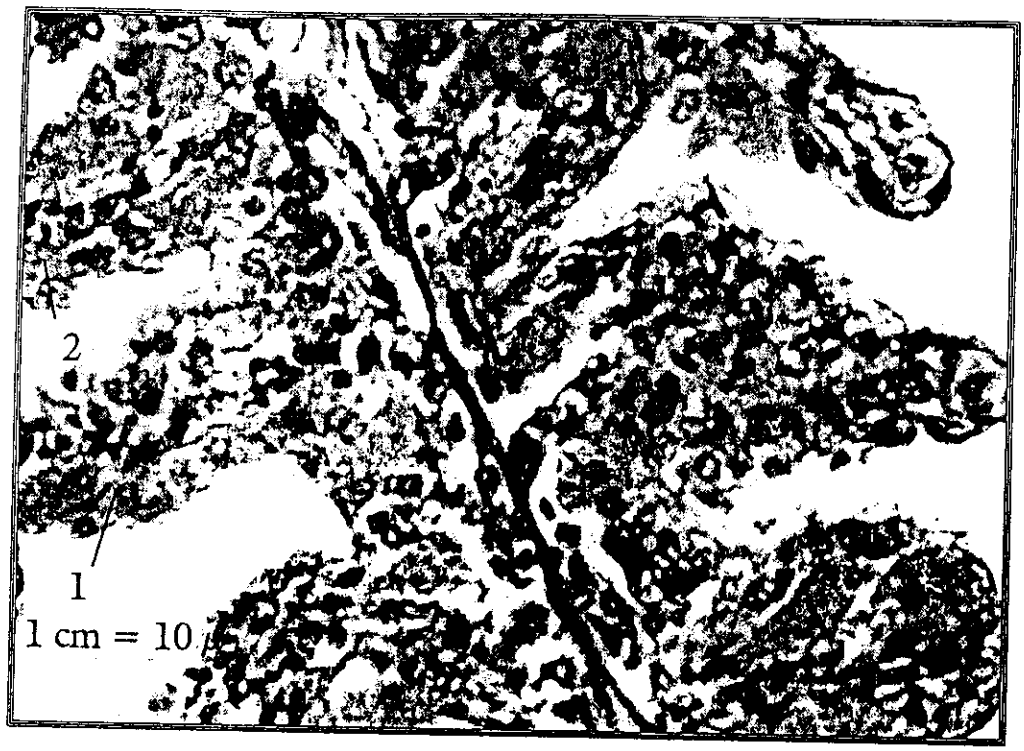


Fig (22) Photomicrograph of a section through the gill of treated *Mugil seheli* fingerlings exposed to 7.0 ml/L of north drain effluents for 96 h showing extensive hyperplastic proliferation of epithelial cells (1) lining secondary lamellae and the chloride cells migrate to the tip of the lamellae (2). The section stained with H & E.

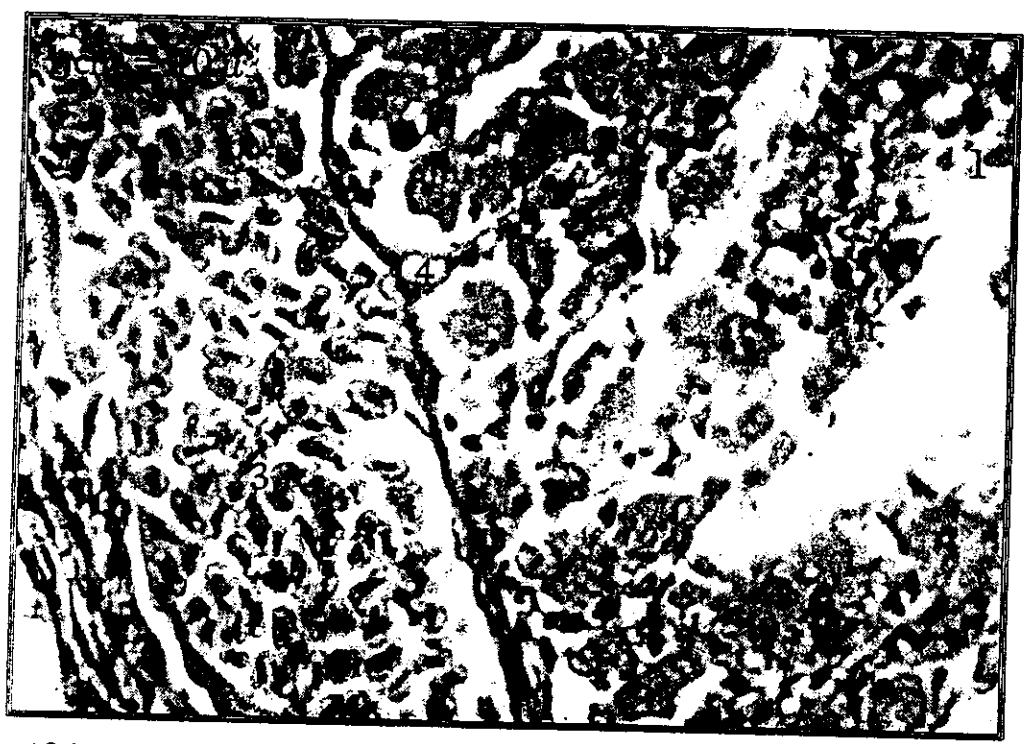


Fig (23) Photomicrograph of a section through the gill of treated *Mugil seheli* fingerlings exposed to 7.0 ml/L of north drain effluents for 96 h showing hyperplastic proliferation of epithelial cells (1). Hypertrophy and hyperplasia of both chloride and mucous cells (2) and congestion of blood vessel (3). Haemorrhage (4) was also detected . The section stained with H & E.

Mugil seheli fingerlings exposed to 7.0 ml/L of the north drain effluents for 96h showed severe damage as seen in figures (22 & 23):, extensive lamellae hypertrophy (Fig., 22): and proliferation of the epithelial cells around the secondary lamellae. The original structure of the lamellae was completely deformed. Hyperplasia and hypertrophy of chloride cells were observed, and also some chloride cells were migrated to the distal ends of the secondary lamellae. The cytoplasm of some cells showed fine red granules. Pyknotic nuclei were frequent. Blood vessel congestion could be observed in secondary lamellae capillaries (Fig.23):.The normal structure of the epithelial layer almost disappeared. The blood vessels were dilated in the apical region of primary lamellae.

2. Effect of south drain effluents.

The gills of treated *Mugil seheli* fingerlings exposed to 7.0 ml /L of south drain effluents for 48 h., showed deterioration of both primary and secondary lamellae as shown in (Fig 24):. Secondary lamellae lost their normal structure and became irregular in shape. The epithelial layer was separated from the lamella and appeared to be degenerated with oedema underneath. Also, the pillar cells lost their normal structure, which led to the curvature of the lamella.

Fig (24) Photomicrograph of a section through the gill of treated *Mugil seheli* fingerlings exposed to 7.0 ml/L of South drain effluents for 48 h showing marked subepithelial oedema (1). Dislocation of chloride cells (2) and separation of epithelial lining (3). The section stained with H & E.}

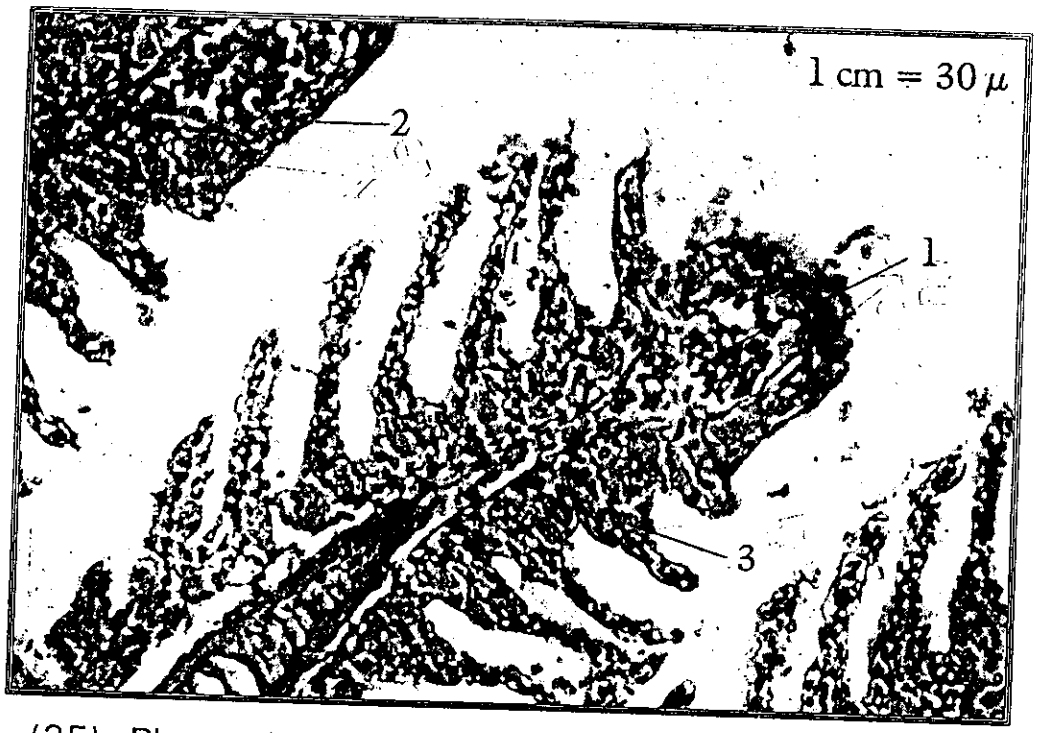


Fig (25) Photomicrograph of a section through the gill of treated *Mugil seheli* fingerlings exposed to 7.0 ml/L of South drain effluents for 96 h showing congestion of blood vessel (1), hyperplasia of lamellar epithelium (2) and proliferation of mucous cells, migration of chloride cells (3)). The section stained with H & E.}

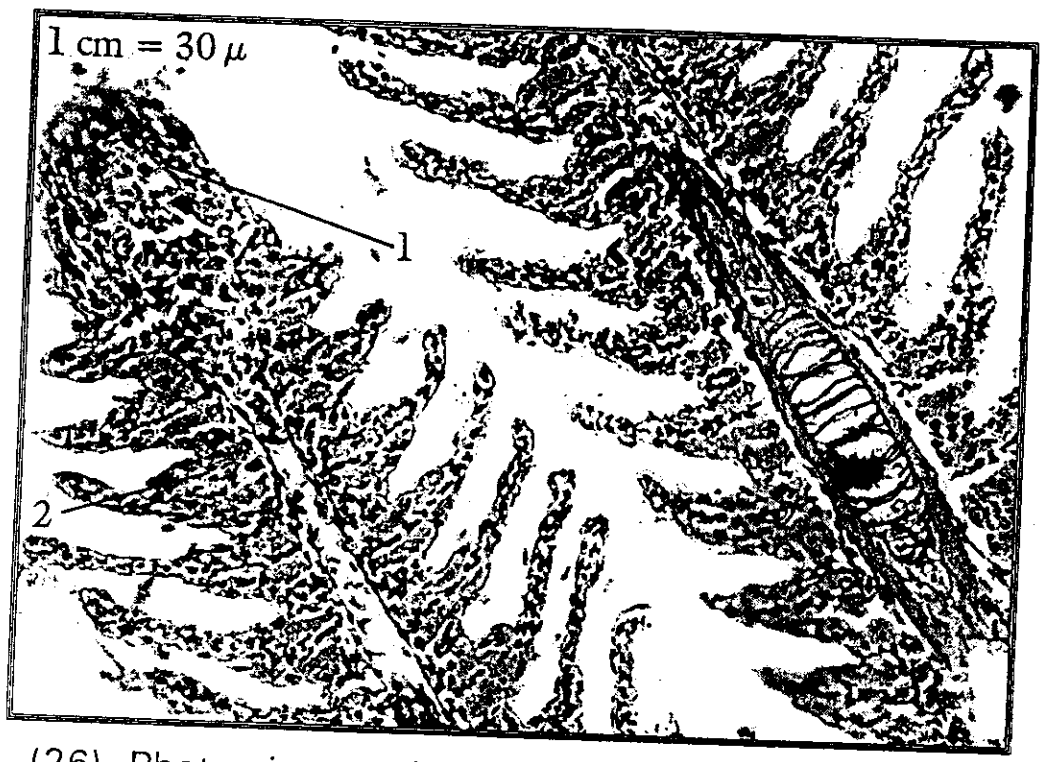


Fig (26) Photomicrograph of a section through the gill of treated *Mugil seheli* fingerlings exposed to 7.0 ml/L of South drain effluents for 96 h showing dilatation and congestion of the main branchial blood vessel (1) and hyperplasia of chloride cells (2). The section stained with H & E.}

After 96 h. of exposure to 7.0 ml /L of south drain effluents, the gills of treated *Mugil seheli* fingerlings, showed extensive histological changes (Figs.25 & 26):. Concerning the secondary lamella, more reliable lesion and distortion of lamellae were apparent. Clubing, blunting and fusion of the lamellae, were concentrated at the tips of the filaments. Also, hyperplasia of lamellar epithelium was combined with migration of chloride cells toward the distal ends of lamellae. In addition, a significant increase in size and numbers of chloride cells was observed. The pillar cells also were reduced in size, causing reduction of circulatory capacity.

3. Effect of Misr- Iran of Textile effluents.

Fingerlings of *Mugil seheli* exposed to concentration of 100 ml/L of textile effluents for 48 h displayed mild tissue reaction (Fig. 27): which was mainly subepithelial oedema in which the oedematus fluid separated the epithelial cells from the underlying capillaries. Hyperplastic proliferation of epithelial cells were apparent predominantly at the apical portion of the filaments.

The gills of the treated *M. seheli* fingerlings exposed to 100 ml /L of Textile mill for 96 h. revealed severe lamellar subepithelial oedema which separated the epithelial cells from the underlying interterstitial layer and pillar system. Leucocytic infiltration was detected at the base of secondary lamellae (Figs. 28 & 29):.

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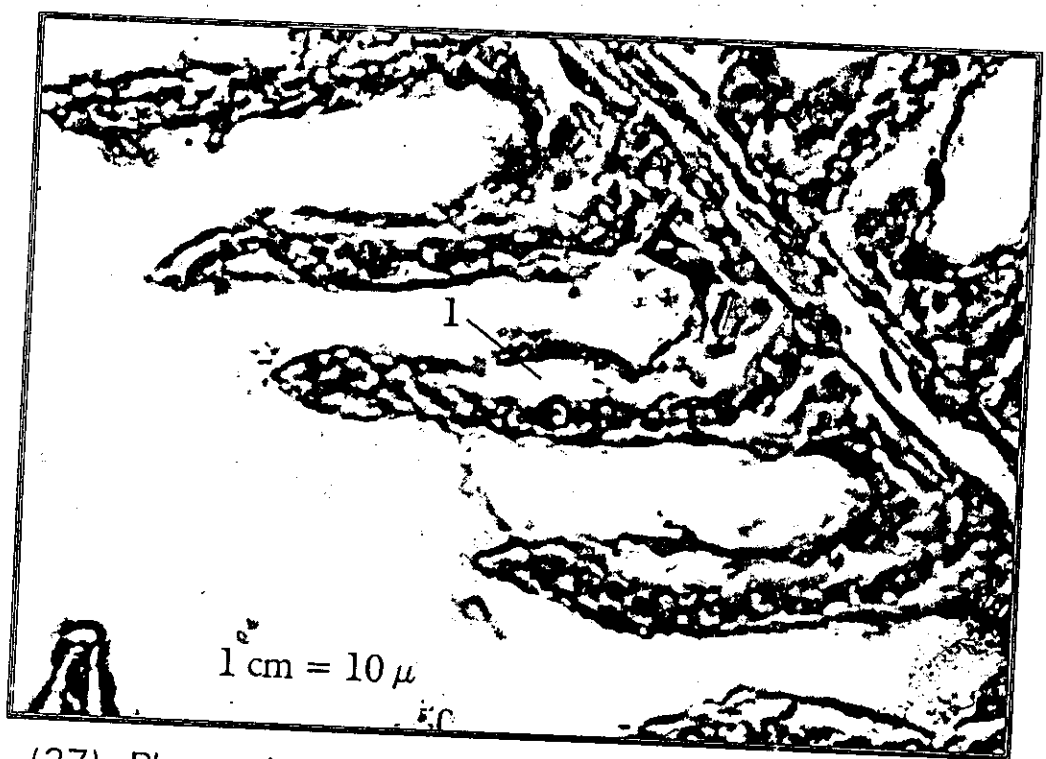

Fig (27) Photomicrograph of a section through the gill of treated *Mugil seheli* fingerlings exposed to 100 ml/L of Misr-Iran of Textile effluents for 48 h showing subepithelial oedema (1) The section stained with H & E.

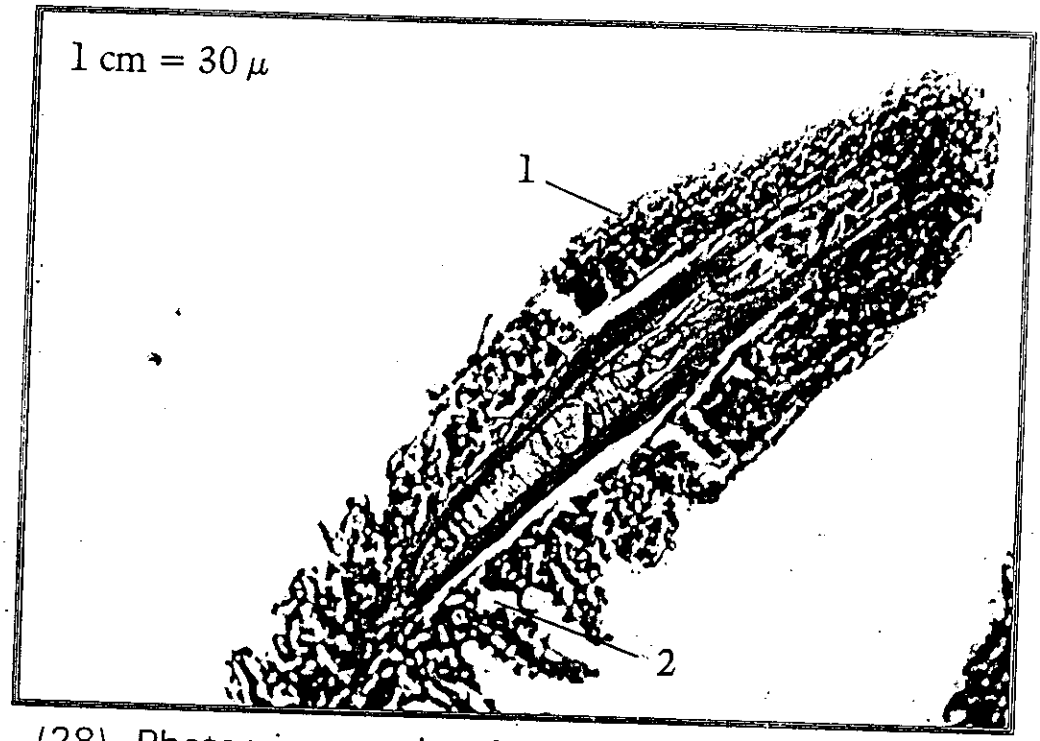

Fig (28) Photomicrograph of a section through the gill of treated *Mugil seheli* fingerlings exposed to 100 ml/L of Misr-Iran of Textile effluents for 96 h showing hyperplastic proliferation of epithelial cells (1) which appeared predominant at the apical portion of the filament. Subepithelial oedema (20 was focally seen. The section stained with H & E.

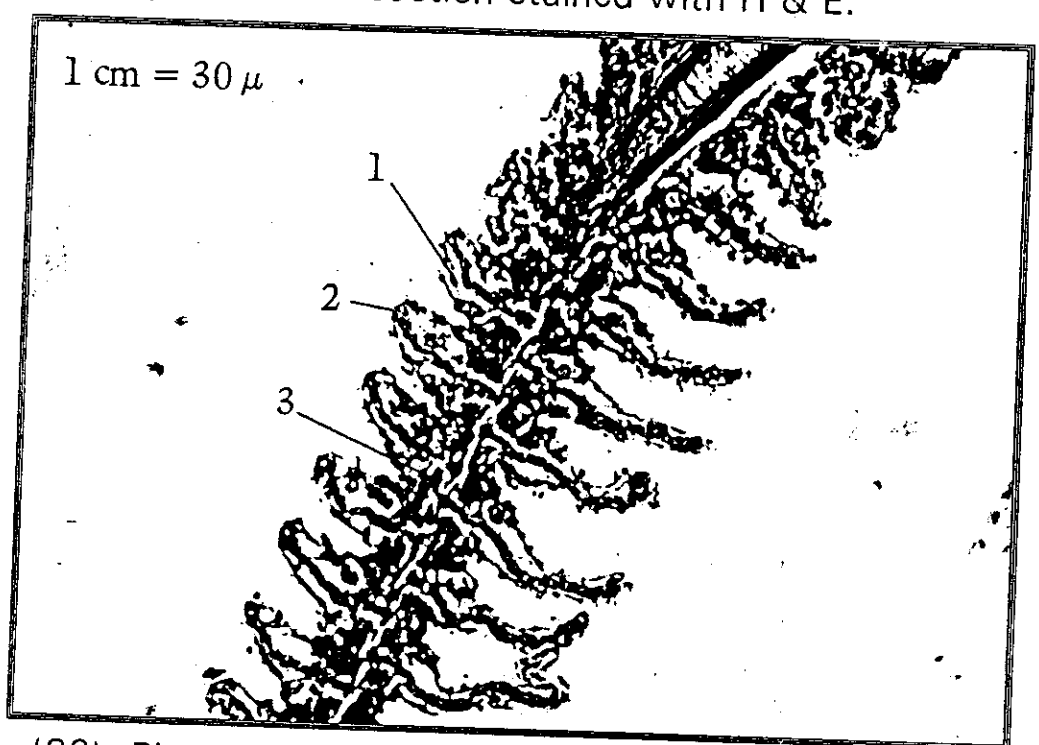

Fig (29) Photomicrograph of a section through the gill of treated *Mugil seheli* fingerlings exposed to 100 ml/L of Misr-Iran of Textile effluents for 96 h showing subepithelial oedema in the secondary lamellae (1), the oedematous fluid separated the epithelial cells from the underlying blood sinuses (2). Leucocytic infiltration were detected at the base of secondary lamellae (3). The section stained with H & E.

4. The effect of Slaughterhouse effluents.

Microscopic examination of fingerlings of *Mugil seheli* exposed to 500 ml/L of slaughterhouse effluents for 48 h revealed the hypertrophy of gill filaments and hyperplasia of the epithelial surface of respiratory lamellae. Unilateral fusion of secondary lamellae and congestion of blood vessels were appeared (Fig. 30):. In addition, the salt cells migrated and increased in their numbers and size. This appeared clearly in the distal end of lamellar and in spaces (Fig.31):.

The gills of treated *M. seheli* fingerlings after 96 h exposure to 500 ml /L of Slaughter house effluents showed severe damage. In general, complete destruction of the gills was very clear. Hyperplastic proliferation of epithelial cells especially at the apical portion of the secondary lamellae with subepithelial oedema at the basal portions is shown in Fig. 32.

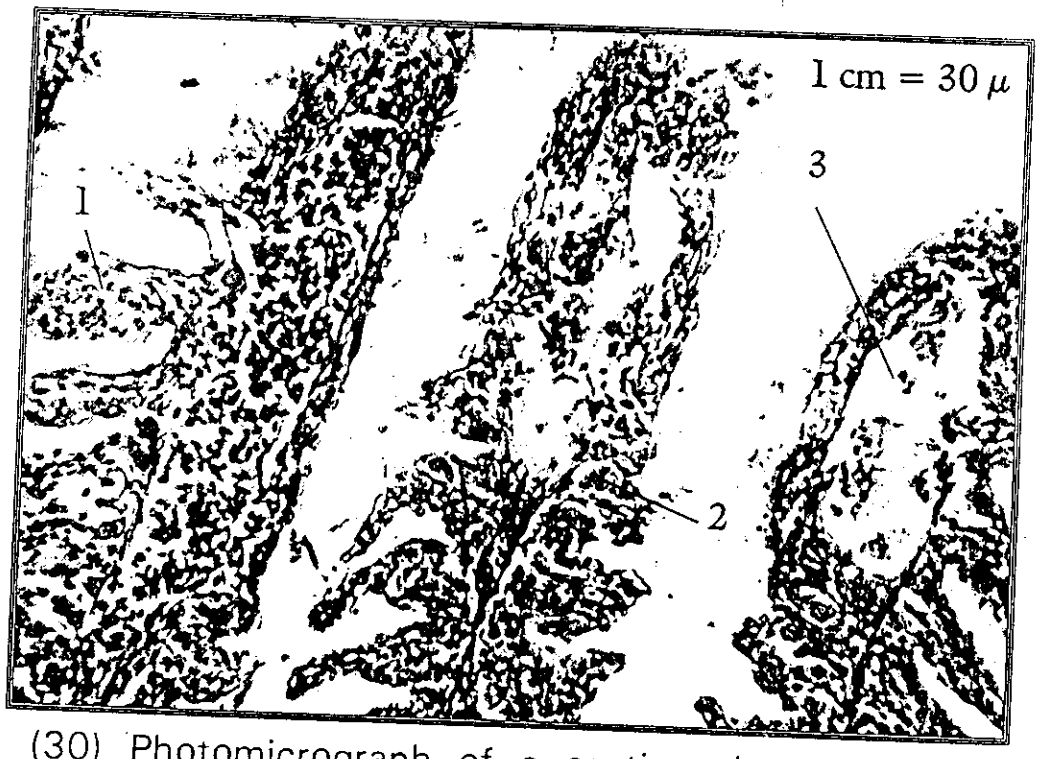

Fig (30) Photomicrograph of a section through the gill of treated *Mugil seheli* fingerlings exposed to 500 ml/L of Slaughter-House effluents for 48 h showing hyperplasia of lamellar epithelial (1),unilateral fusion of secondary lamellar (20, dilation and congestion of blood vessels (3). The section stained with H & E.

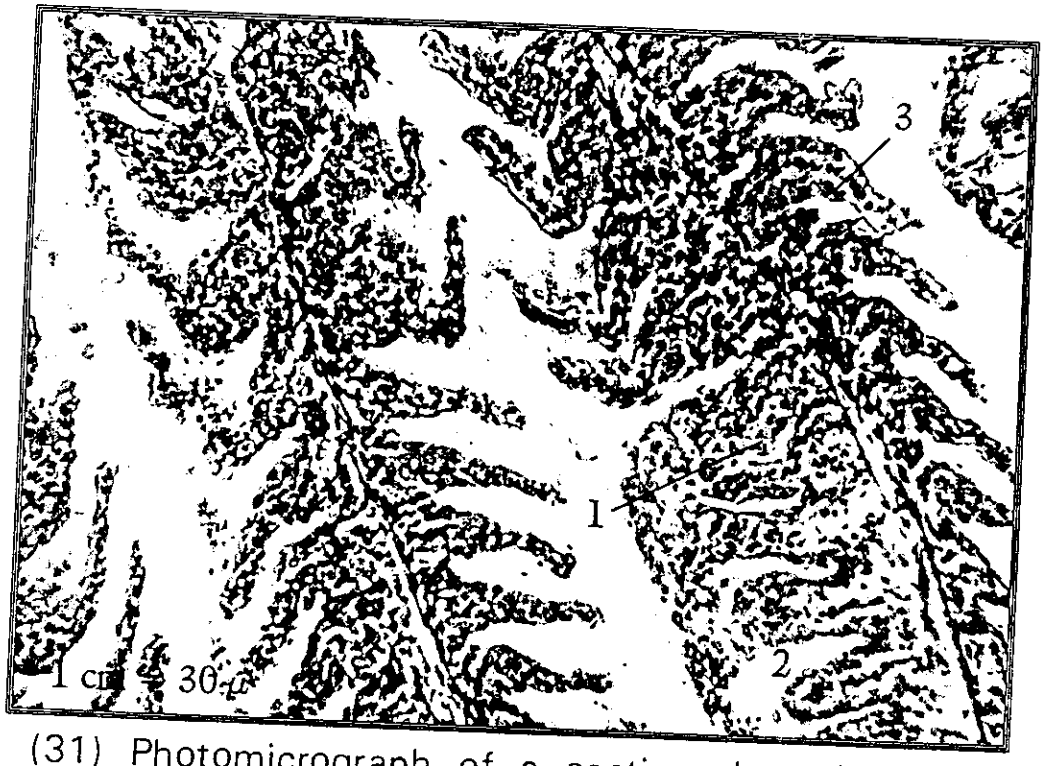

Fig (31) Photomicrograph of a section through the gill of treated *Mugil seheli* fingerlings exposed to 500 ml/L of Slaughter-House effluents for 48 h showing focal lamellar fusion (1) and subepithelial oedema (2) associated with migration of chloride cells occupying interlamellar spaces (3), focal leucocytic infiltration were seen The section stained with H & E.

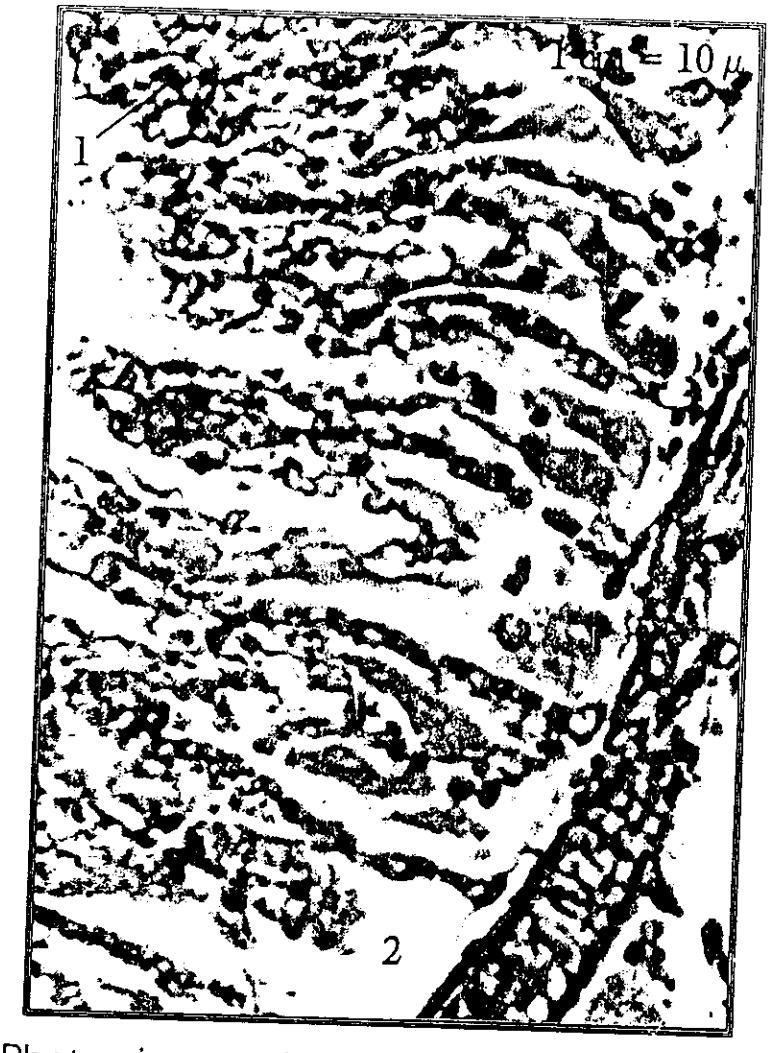

Fig (32) Photomicrograph of a section through the gill of treated *Mugil seheli* fingerlings exposed to 500 ml/L of Slaughter-House effluents for 96 h showing hyperplastic proliferation of epithelial cells (1) especially at the apical portion of secondary lamellae with subepithelial oedema (20 at the basal portions. The section stained with H & E.

II. Liver

Normal liver.

Histological examinations of *Mugil seheli* fingerlings showed healthy features (Figs., 33 & 34):. The hepatocytes are polygonal in shape with a centrally located dark blue nuclei, each containing one nucleolus. The hepatocytes were arranged as spools with narrow light areas of sinusoides containing blood. The pigment cells of dark stain were scattered in the whole tissue. Morever, the central blood vessel filled with erythrocytes and its wall consisted of epithelial cells that revealed a moderate fatty vacuoles (Fig. 34):.

Treated liver

1. Effect of the north drain effluent.

The liver after 96h exposure to 7.0 ml /L of north drain effluents reaveled diffuse necrobiotic changes in hepatic parenchymal cells and severe dilation of hepatic sinusoids (Fig. 35):. Large numbers of hydropic vacuoles were noticed throughout the cytoplasm of hepatic cells. Hepatic necrosis was seen especially around the remains of the central vein. Under the high power, the hepatocytes in (Fig. 35): generally appeared with a single large vacuole with little cytoplasmic remain and nucleus was eccentric. The bile canalicule was distended with bile secretion, interlobular spaces showed fibrolysis

Fig (33) Photomicrograph of a section through the liver of *Mugil seheli* fingerlings (Control) showing healthy polygonal hepatic cells (1) and central vein structure (2). The section stained with H & E.

Fig (34) Photomicrograph of a section through the liver of *Mugil seheli* fingerlings (Control) showing narrow blood sinusoids (1) moderate vacuoles (2) and well developed nucleus (3). The section stained with H & E.

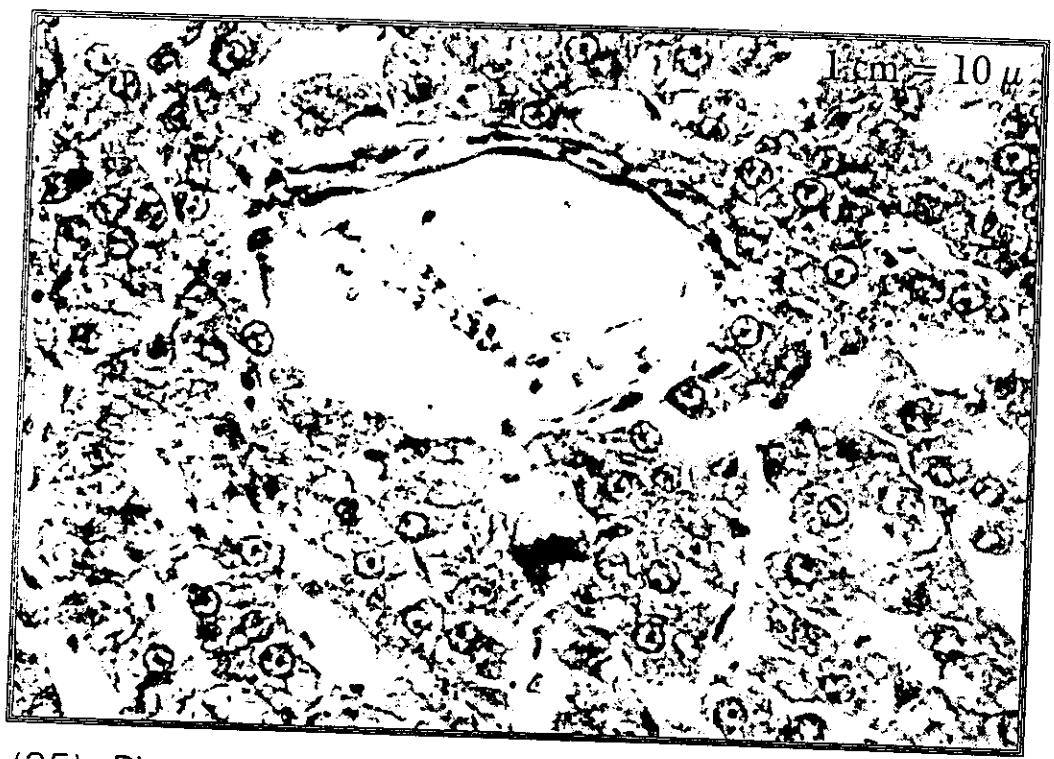

Fig (35) Photomicrograph of a section through the liver of treated *Mugil seheli* fingerlings exposed to 7.0 ml/L of north drain effluents for 96 h showing diffuse necrobiotic changes in hepatic parenchymal cells and severe dilatation of hepatic sinusoides. The section stained with H & E.

2. Effect of the south drain effluent.

The histological photomicrograph of treated *Mugil seheli* fingerlings liver after 96 h. of exposure to 7.0 ml/L of the south drain effluents showed fatty accumulation in the liver hepatocytes (Fig., 36) Large intracellular hydropic vacuoles were common among hepatocytes. A considerable number of the hepatic cells showed different types of degeneration and the normal outline of most of the hepatic cells no longer exists (Fig. 36).

3. Effect of Misr-Iran of Textile effluents.

The liver of *M. seheli* fingerlings exposed to 100 ml/L of Textile for 96h showed severe damage (Fig. 37):. Fatty accumulation was observed in the hepatocytes. Hydropic vacuoles were common in most hepatocytes; the hepatocytes membrane (cell wall): were not well detectable. Degenerated cells were frequent and diffuse hydropic degeneration was also seen (Fig., 38):. The normal arrangement of hepatocytes was disturbed. The blood vessels showed abnormal appearance as well as the blood cells.

4. Effect of the Slaughterhouse effluent.

The liver of *Mugil seheli* fingerlings exposed to 500 ml /L of Slaughterhouse effluents for 96 h. revealed hydropic vaculation, fatty degeneration of hepatocytes and aggregation of melanomacrophage cells was seen in which the melanosomes appeared between hepatic cells. Congestion of blood sinuses and central vein (Fig. 39):.

Fig (36) Photomicrograph of a section through the liver of treated *Mugil seheli* fingerlings exposed to 7.0 ml/L of south drain effluents for 96 h showing vacuolar degeneration changes of hepatocytes and dilatation of hepatic sinusoides. The section stained with H & E.

Fig (37) Photomicrograph of a section through the liver of treated *Mugil seheli* fingerlings exposed to 100 ml/L of Misr-Iran of Textile of effluents for 96 h showing Intercellular oedema (1), intercellular hydropic vacuoles (2) and aggregation of blood cells (3) with less distinct features. The section stained with H & E.

Fig (38) Photomicrograph of a section through the liver of treated *Mugil seheli* fingerlings exposed to 100 ml/L of misr-Iran of Textile of effluents for 96 h showing diffused degenerative changes accompanied with diffuse necrobiotic reactions and the hepatic sinusoides appeared severely dilated (1). The section stained with H & E.

Fig. (39) Photomicrograph of a section through the liver of treated *M.seheli* fingerlings exposed to 500 ml/L of Slaughter-House effluents for 96 h showing hydropic vaculation, fatty degeneration of hepatocytes and aggregation of melanomacrophage cells (1) was seen in which the melanosomes appeared between hepatic cells. Congestion of blood sinuses (2) and central vein. The section stained with H & E.

III. Intestine.

Normal intestine.

The histological examination of the intestinal structure of *Mugil seheli* fingerlings manifested normal features (Fig. 40):. The intestine formed of four layers, mucosa, submucosa, muscularis and serosa. In a higher magnification the mucosal layer consisted of simple columnar cells. In figure (41):, normal brush border and bright eosinophilic spherical nuclei were present in the mucosa layer. The submucosal layer contained eosinophilic granular cells and a reticular connective tissue with blood sinus.The serosal layer appeared as one layer of epithelial cells, covering the subserosa (muscularis): layer (Fig., 35):.

Treated Intestine.

1-Effect of the north drain effluent.

After 96h exposure to 7.0ml /L of north drain effluents, the mucosa layer showed some abnormalities. The goblet cells became more active (Fig., 42):. The basement membrane of the columnar cells were not detectable and the basal side of the columnar cells extended inward, clear Oedema was present between the mucosa and submucosal layers. The submucosal layer became less compact as seen in (Fig., 43):.

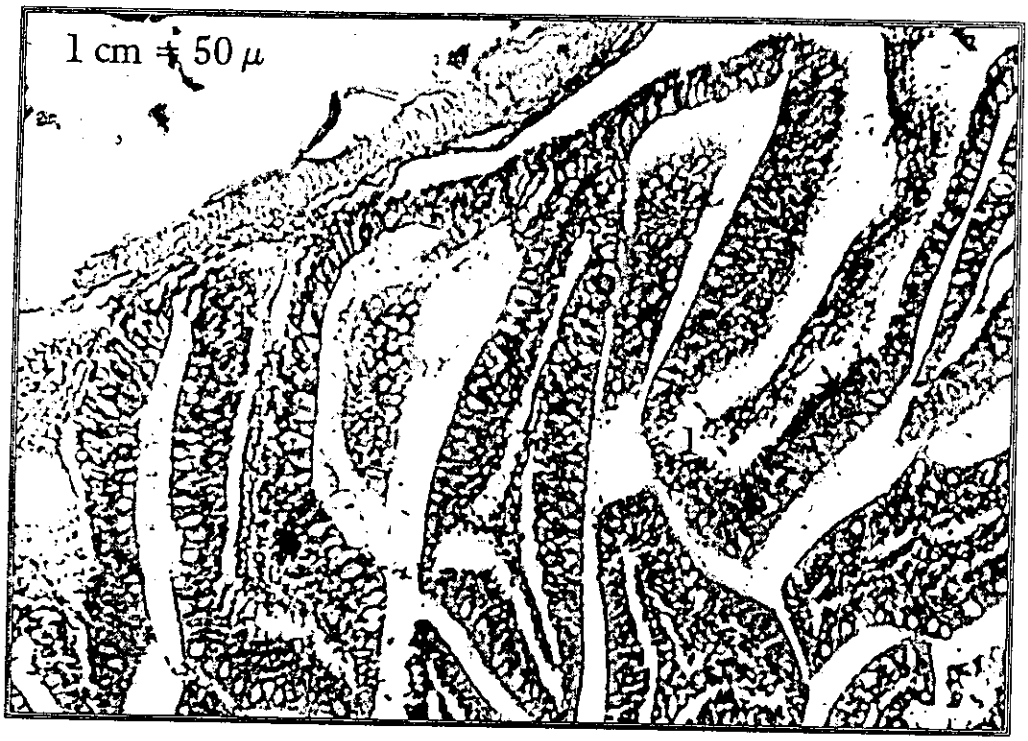

Fig. (42) Photomicrograph of a section through the intestine of treated *M. seheli* fingerlings exposed to 7.0 ml/L of north drain effluents for 96 h showing separation of epithelial mucosal layer from fibrous tissue and clear oedema (1) in between the mucosal and submucosal layers. section stained with H & E.

Fig. (43) Photomicrograph of a section through the intestine of treated *M. seheli* fingerlings exposed to 7.0 ml/L of north drain effluents for 96 h showing Reduction of submucosal layer and water accumulation (1) between mucosa and submucosa layers , leucocytic infilteration (2) was markedly seen. The sections stained with H & E.

2-Effect of the south drain effluent

The intestine of *Mugil seheli* fingerlings exposed to 7.0 ml /L of south drain effluents for 96 h. revealed some clear changes in their histological appearance (Fig., 44):. The most obvious feature was the extreme reduction in the size of submucosa, while the mucosal layer seemed to have its normal size. Such situation caused complete separation between the two layers. The basement membrane of the columnar cells became undetectable and also the nuclei of such cells lost their normal arrangement in the base of the cells. Higher magnification revealed lytic forthy vacuolation of mucosal epithelium and degeneration of submucosal connective tissue beside congested of submucosa blood vessels. Hypertrophic and hyperplastic proliferation of mucosa cells (goblet metaplasia): were detected. Leucocytic infiltration also seen in (Fig. 45):.

3-Effect of Misr-Iran of Textile effluent.

The intestine of *M. seheli* fingerlings exposed to 100ml/L mill of Textile for 96 h. revealed some changes in their histological appearance. In fig. (46): the most obvious feature was the reduction of the core villi which cause complete separation between the submucosa and mucosa layers. Lost of brush border hairs, hyperplasia and focal necrosis of mucosal cells were noticed (Fig.47):.

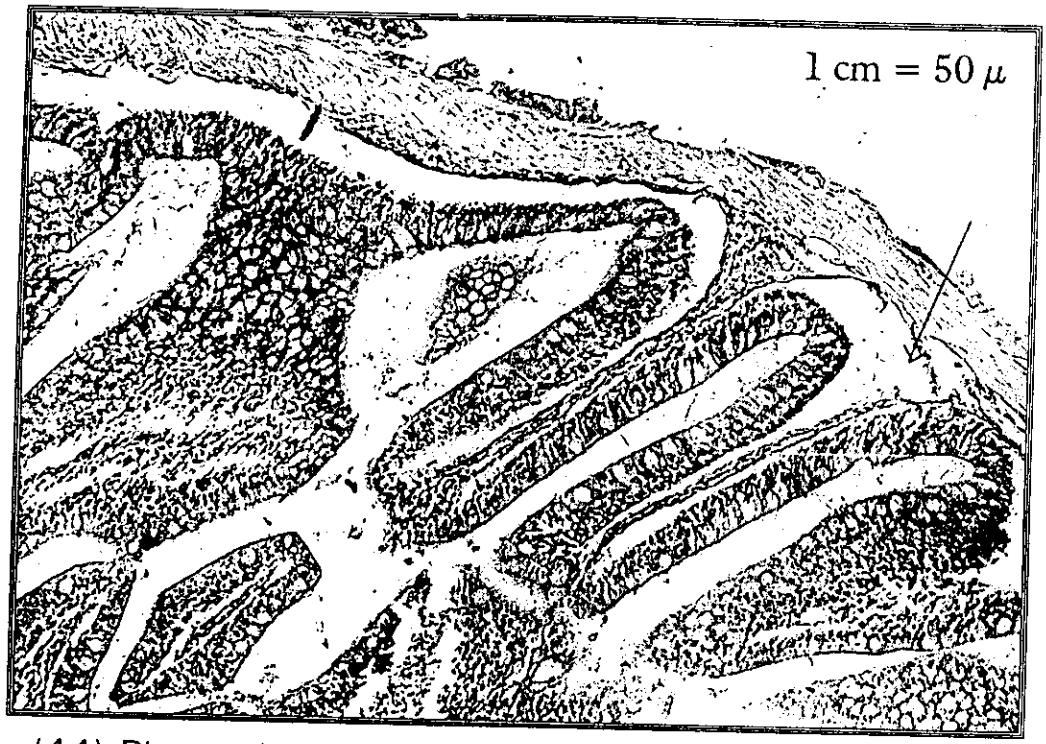

Fig. (44) Photomicrograph of a section through the intestine of *M. seheli* fingerlings exposed to 7.0 ml/L of south drain effluents for 96 h showing focal necrosis and pyknosis of the epithelium cells of the intestinal mucosa and complete separation of mucosal layer from the mucosal layer (arrow). The sections stained with H & E.

Fig. (45) Photomicrograph of a section through the intestine of treated *M. seheli* fingerlings exposed to 7.0 ml/L of south drain effluents for 96 h showing lytic forthy vacuolation of mucosal epithelium (1) and degneration of submucosa connective tissue and congestion of submucosa connective tissue (2) and hypertrophic and hyperplastic proliferation of mucosa (3) cells (goblet metaplasia), leucocytic infilteration also seen. The sections stained with H & E.

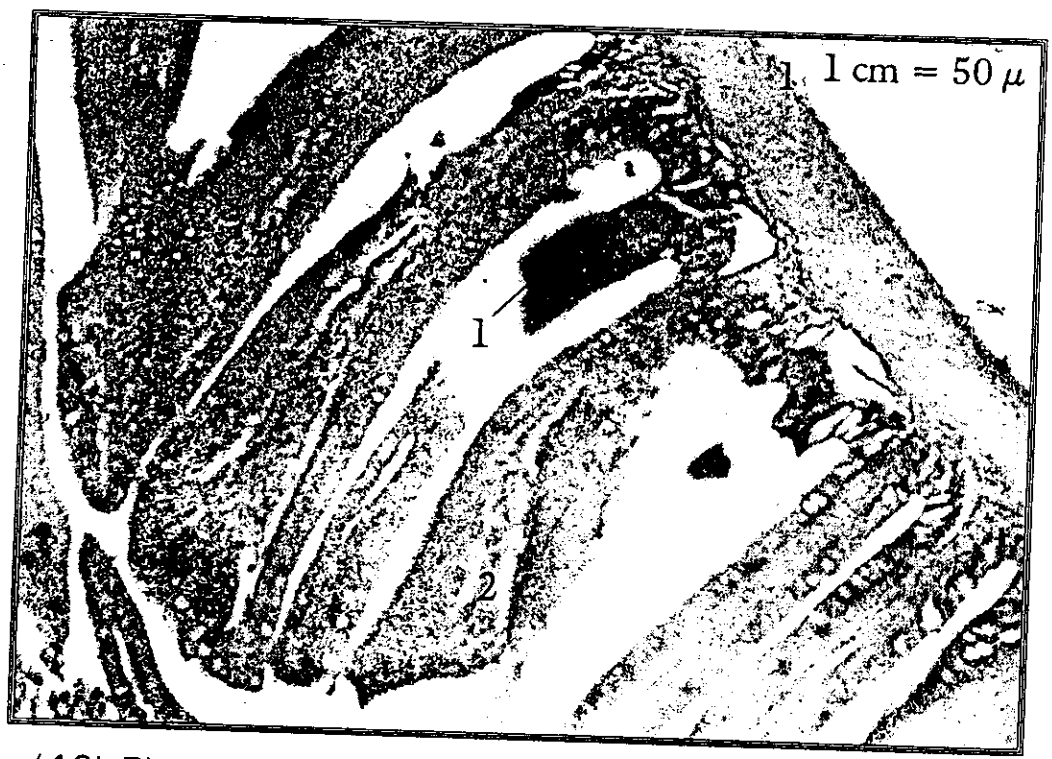

Fig. (46) Photomicrograph of a section through the intestine of *M. seheli* fingerlings exposed to 100ml/L of Misr-Iran of Textile effluents for 96 h showing sloughing of mucosal villi (1) of reduction the submucosa connective tissues (2). The sections stained with H & E.

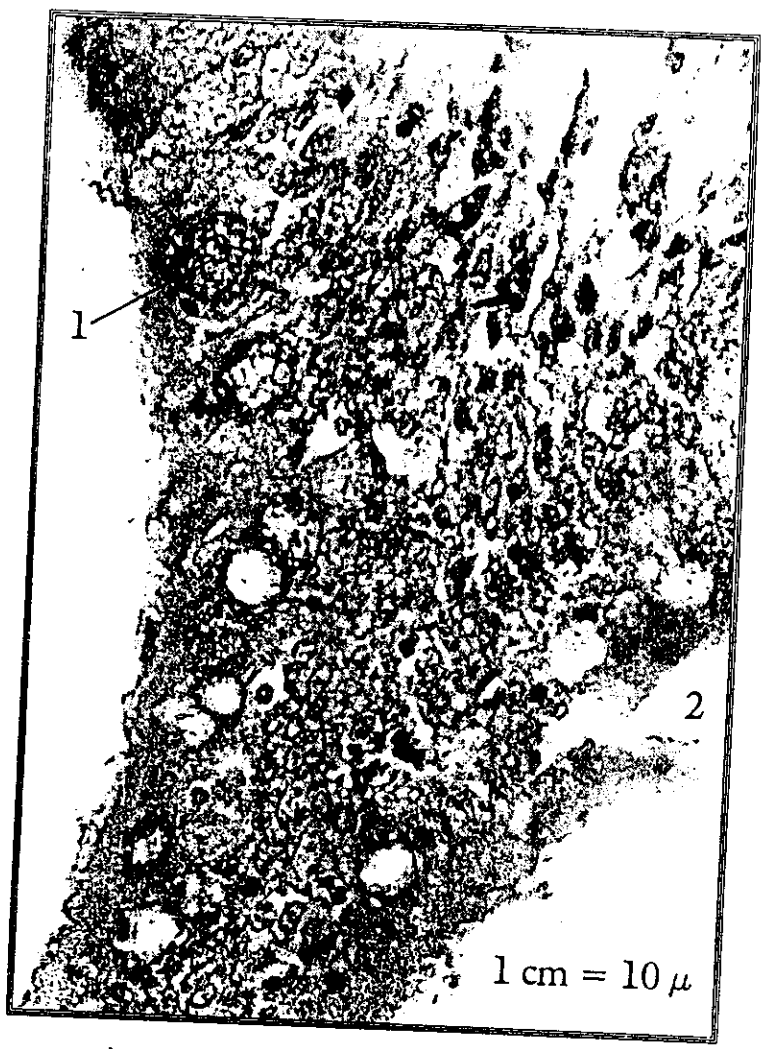

Fig. (47) Photomicrograph of a section through the intestine of *M. seheli* fingerlings exposed to 100ml/L of Misr-Iran of Textile effluents for 96 h showing lost of brush border hairs and hyperplasia of mucous cells (1) and focal necrosis of columnar cells (2). The sections stained with H & E.

4. Effect of the Slaughterhouse effluent.

The intestine of *M. seheli* fingerlings exposed to 500 ml /L of Slaughterhouse effluents for 96 h. showed slight deformity in the mucosa as the collumnar cells became longer than normal. The basement membrane became less straight and moved inward, also slight degree of necrosis was appeared in the submucosal layer. Under the high power, the brush border not continuous and appeared in bad shape as shown in figure (48):.

IV. Spleen

Normal Spleen.

General histology of *Mugil seheli* spleen collected from the control tests did not markedly differ from the published description of normal (Ferguson, 1989):.The section (Fig.49): revealed that, an activation of melanomacrophage centers were clearly observed as dark brown patches scattered in the tissue.

Treated Spleen.

1. Effect of the north drain effluents.

The histological examinations of *Mugil seheli* spleen after 96 hours of exposure to 7.0 ml/L of wastewater revealed congestion of the red bulbs and focal aggregation of melanin carrying cells. Dilatation of white bulbs was observed. In addition, splenic lymphocytic cell depletion, congestion and necrosis of ellpisoid sheaths were observed. (Fig. 50):.

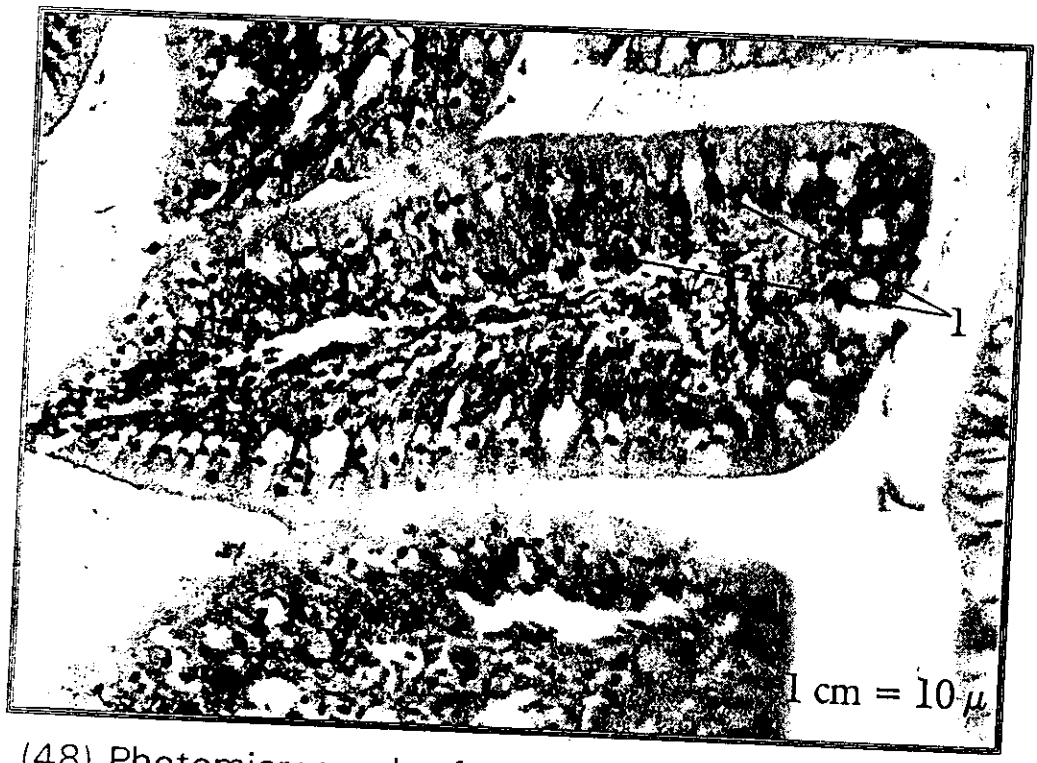

Fig. (48) Photomicrograph of a section through the intestine of treated *M. seheli* fingerlings exposed to 500 ml/L of Slaughter-House effluents for 96 h showing early stage of sloughing the mucosal villi and lose of bursh border hairs and inflammatory cell reactions (1) were seen. The sections stained with H & E.

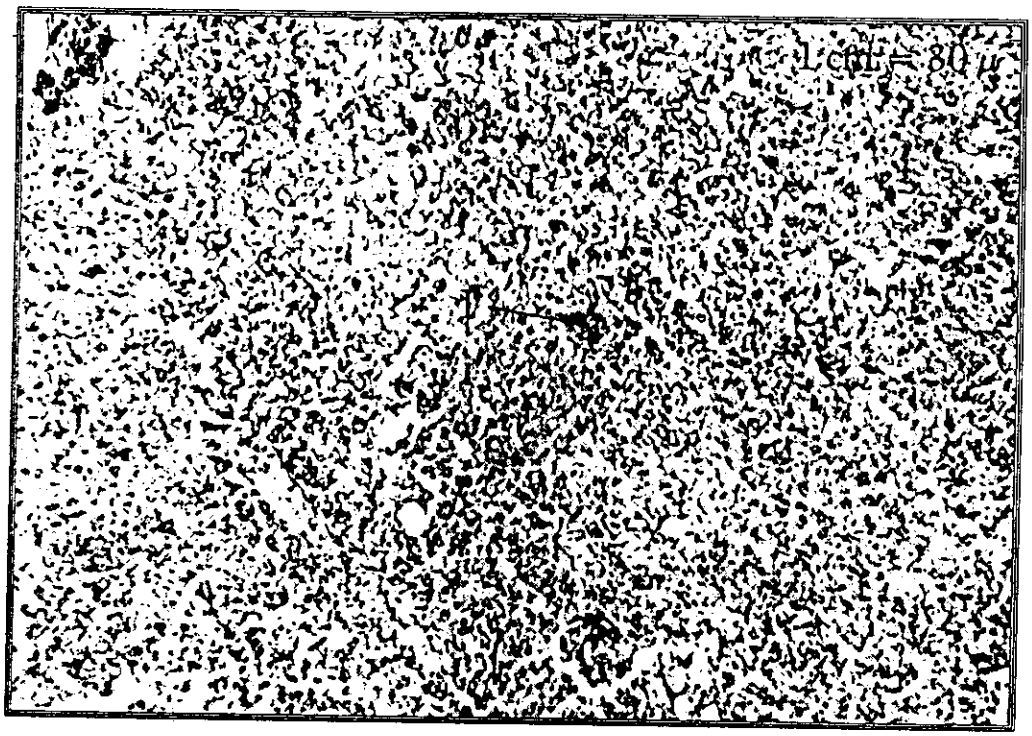

Fig. (49) Photomicrograph of a section through the spleen of control *M. seheli* fingerlings 96 h showing the normal structure of splenic capsule and the melanomicrophage centers (1) appeared activated and speared allover the splenic tissue. The sections stained with H & E.

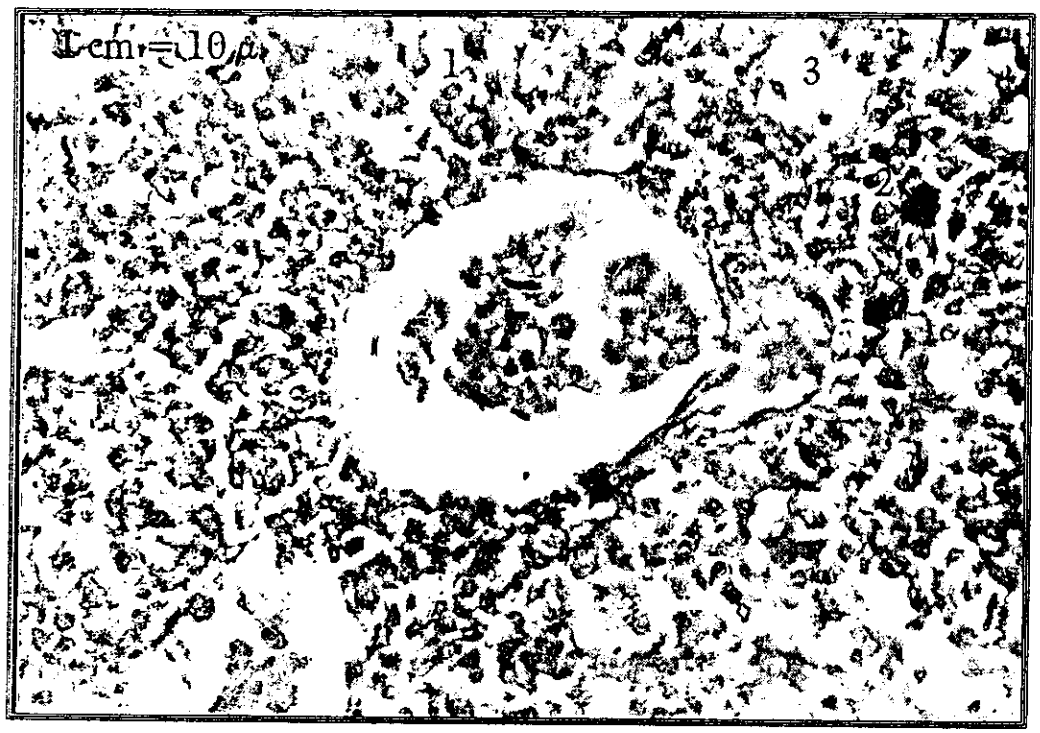

Fig. (50) Photomicrograph of a section through the spleen of treated *M. seheli* fingerlings exposed to 7.0 ml /L ol north drain effluents for 96 h showing congestion of red bulbs (1) and focal aggregation of melanin carrying cells (2). Dilatation of white bulbs (3).The sections stained with H & E.

2. Effect of the south drain effluent

The microscopic examination for the spleen of the fish subjected to diluted industrial wastewater in concentration 7.0 ml/L from the south drain for 96 hours revealed unhealthy characteristics and severe damage. An abundance of darker macrophages pigments with variable sizes and thyroid similar follicles contained hyaline casts occurred close to blood vessel were observed in the splenic tissue. The interstitial spaces was increased and revealed fibrosis and the ellipsoids were swollen (Fig. 51):.

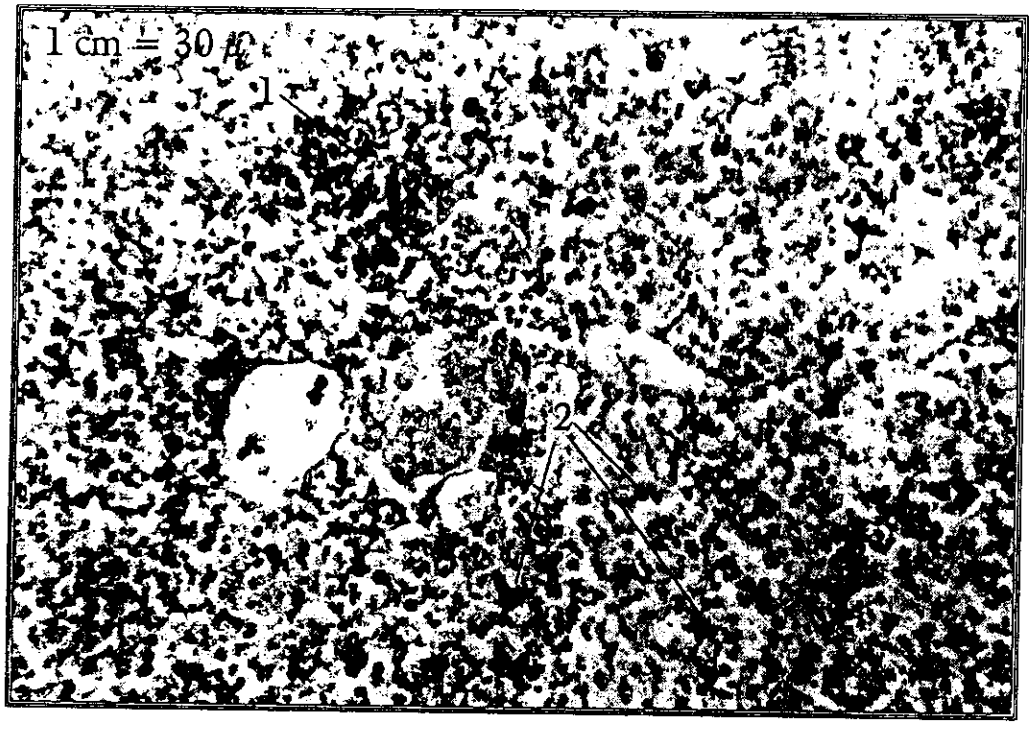

Fig. (51) Photomicrograph of a section through the spleen of treated *M. seheli* fingerlings exposed to 7.0 ml /L of south drain effluents for 96 h showing diffuse ellpsoidal necrosis (1) and focal aggregation of melanin carrying cells (2). The sections stained with H & E.

Discussion

Suez Bay is an important natural resource for the fish juveniles needed as a stock for the northern part of Gulf of Suez. Its shallowness, wideness and flat bottom nature may fit to be a nursery ground. In addition, Suez Bay is considered the main source for fish market of Suez City. However, this condition no longer exists, may be as a result of increasing pollution made by human activities in the area. Such activities include construction of new industrial activities and power stations besides to the increasing of sewage due to the increasing in population density.

Unfortunately, the wastewater of such various activities is directly dumped into the Suez Bay. The total amount of the wastewater entering the Suez Bay was estimated in the 1960s as 24,000 m^3/h (Meshal, 1970). This amount increased in 2000 to be 97,328 m^3/h (Environmental Affairs Authority, EAA 2000). Meshal (1970) estimated the values of flow in and out of the Suez Bay during the tidal cycle to be 13.5×10^6 m^3/h. The ratio of the flow water to the drainage water was 1 to 0.0017 in 1970. This ratio increased in 1998 to be 0.0072, i.e. four times greater than before. Hence, the influence of the drained water on the Bay environment increased.

At present, the present study was carried out on the area between Suez city and El-Ataka where most industrial and urban activities were concentrated and the wastewater drains of many factories are discharged.

Seasonal samples of wastewater are collected from four drains: -

1- North drain effluents.

2- South drain effluents.

3- Misr-Iran of textile effluents.

4- Slaughterhouse effluents.

The thesis includes three types of studies: -

I- Physico-chemical analysis.

II- Toxicity.

III- Histological studies

Physico – Chemical analysis

In the present study, the physico-chemical parameters of the effluents from FP (N & S units), Misr-Iran Textile, north and south drain revealed higher values than those reported by Hamed (1992).

The present results indicated that, all physical and chemical parameters determined for wastewater drained in Suez Bay showed high values at the different seasons except the low values of DO. Also, The results confirmed limited variation in wastewater temperature; highest temperature (32.5° C) was recorded in south drain effluent during summer 1996 while lowest temperature (28° C) was recorded in Misr-Iran of Textile effluents at the same time. The temperature of south and north drains

was always higher than Textile, FP (N & S units). This elevation in the effluent temperature is most probably due to the mixing and ensuing reactions of the different effluents. Higashi *et al.* (1992) similarly reported that wastewater from acid, alkaline, chlorine oxidant, and other chemically diverse processes are sewered together to form the whole-mill discharge. Mixing of these streams leads to a final effluent with high temperature. Also, the presence of organic matter disposal along with the sewage increases the heat emission, while Shriaidah (1982): attributed the increase in water temperature to the kinetic energy of the large amounts of organisms adapted to organic materials and its high rate of oxidation. On the whole, the prevailing climate conditions, the relative shallowness of drains, its semi-closed shape and the chemical reaction of the effluents of each industry plant in the area may be the cause of such temperature elevation reported here.

The highest values of pH recorded in the effluents of Misr-Iran of Textile ranged between 8.5 in autumn 1996 and 11.9 in summer 1996. During the rest of the year, the pH values were 10, 11,5 and 11.6 in winter, spring and summer, respectively. This may be due to the presence of the bleaching agents used in Mis-Iran of Textile. Jaber (1995) reported that the company consumes nearly 982 ton/year of NaOH, and 197 ton/year of NaOH were discharged. On the other hand, the effluents of FP south unit showed a minimum value of pH (7.2)

during spring 1997, this might be attributed to the presence of acid traces in the effluents. Where effluents of FP south unit contain 540 m^3/year nitric acid and 480 m^3/year sulphuric acid (Environmental Affairs Authority, Suez, 1999). Low values of pH in the south drain effluents may be resulted from liberation of H_2S from anaerobic bacteria or decomposition of the accumulated decayed organic matter discharged from Ataqa power station through the drain. This result coincides with Hutuhinso (1957) and Nessim and Tadros (1986), though, Abd El- Azim (1996) attributed the low pH value to the remarkable decrease in concentration of salinity at this effluent.

Variations in the seasonal DO values in each season and in each effluent match the type of effluent and the climatic conditions. Highest DO concentration was recorded in Misr-Iran of Textile. This elevation in DO content of Misr-Iran of Textile may be due to the continuous reaction between NaOH and chlorine in the discharged sewage with the industrial effluents and the highest DO concentration in winter related to the decrease of water temperature. On the other hand, the lowest DO content in the rest of the drains probably is due to the elevation in the effluents temperature. El-Deep (1977), Abdallah (1979) and El-Nady (1981) reported that the slight rise of the surface water temperature cause such decrease in the surface dissolved oxygen values.

The disposal of untreated industrial effluents affects the DO level of inshore water. Hamed (1992) reported that continuous disposal of polluted water in addition to the biological

decomposition of raw organic matter in the water are the main cause of oxygen depletion in Suez Bay.

Skirrow (1965), and Shaker & Khan (1983) pointed out *that* water owes its alkaline properties due to the presence of bicarbonate, carbonate, and borate. Sodium bicarbonate is formed as a result of NaOH reaction in the industrial effluent and CO_2 liberated from the decomposition of organic matter in sewage. This may explain the high alkalinity of Misr-Iran reported Textile. Hutuhinso (1957) attributed that the increasing rate of photosynthetic activity and pH values is a result of CO_2 consumption. The results in the present study reflect a relative seasonal relationship between the pH values and the alkalinity values. While, alkalinity is actually more related to the oxygen content of the effluent and the chemicals used in the industry. FP industrial activity increased during spring and summer causing increasing discharge, which in turn is reflected as high alkalinity.

The present results showed high values of COD in different industrial wastewater during all seasons. Misr-Iran of Textile effluent exhibited high values of COD, 437.1, 192, 240.5,105.6 and 339 mg/L, in summer 1996, autumn 1996, winter 1997, spring 1997 and summer 1997, respectively. While, the maximum value of COD in industrial wastewater of FP north unit was 210 mg/L only in summer 1996, the minimum value was 72 mg/L in spring 1997. The maximum value of COD in south unit (187 mg/L) was recorded in summer 1997 and the minimum value was 65.2 mg/L during spring 1997. The

detected elevation may be due to the reactions taking place in different chemical components in each effluent and the oxidation of organic matter in the sewage. This finding agrees with Higashi *et al.* (1992) for the chemical analysis of paper mill effluents and the industrial wastewater that exhibited high values of COD as a result of reactions of such chemicals in the effluents. El- Awady (1977) in his study on the western harbor of Alexandria mentioned that the increase in COD values was due to the high concentration of organic matter in the discharged sewage.

Ammonia is considered the most stable and predominant inorganic nitrogen compound in seawater (Kandil, 1982). It appears to be the most essential limiting major nutrient for phytoplankton growth (Ewins & Spencer, 1967). The maximum and markedly high values of NH_3-N at north drain effluent studied here may be due to the formation of free ammonia as a result of the mixing between the NaOH from Misr-Iran Textile, and Nitrogenous compounds from FP, and the decayed organic material discharged from the slaughterhouse and sewage. This observation agrees with Nessim and Tadros (1986) in the western harbor of Alexandria and Sedrak (1992) on the effluents of plant fertilizers of Abu-Qir.

The high concentration of inorganic phosphate in the present study that recorded in the effluents of Misr-Iran Textile may be due to the presence of detergents and the decaying of organic matter in the discharged sewage with the industrial wastewater. This remark agrees with Saad (1973) who stated

that the high phosphate concentration attributed principally to the discharge of domestic wastes. Also, the increase in phosphate content is related to the decay of organic matter (Cooper, 1958; Kramer *et al.*, 1972), excretion of large amounts of phosphate by aquatic organisms and release of phosphate from the bottom sediments into the overlying water (Kramer *et al.*, 1972).

Toxicity tests

Toxicity tests were designed to provide information on the potential harmfulness of the chemical effluents (FAO, 1993). Industrial wastewater from acid, alkaline, chlorine oxidant, and other chemically diverse processes are sewered together to form the final effluent that is highly complex. This condition is getting to be more pronounced as polluted streams become dangerous for aquatic life having a considerable effect on fish production, fish fertility and biochemical composition of these organisms (Smart. 1975).

Regarding the components of Suez FP effluent, Jaber (1995) reported that the effluent of north unit contains 3800 m^3/day liquid ammonium salts, while the south unit discharges about 3620 m^3/day liquid ammonium salts and both nitric and sulphuric acids. Fertilizer factories are considered a source of air and water pollution such as Talkha fertilizer factory (Hussein *et al.*, 1999). In general, pollutants of Suez plant fertilizer factories are characterized by ammonia gas and nitrogenous oxides liberated into the atmosphere at a rate of 0.49 mg/L NH_3

and 0.73mg/L nitrogenous oxides and liquid effluents at about 12 ton/day suspended solids, 50kg/L day copper, 53.2 ton /day ammonia, 4.5 ton/ammonia nitrite, 20ton/day urea. This discharge causes a change of water quality and creates a large problem for fish production (Saad, 1984).

The effect of ammonia on fish behavior and fish survival have been determined and documented in many studies (Tarzwell, 1957; Smart 1975; Bower and Bidwell 1978; Sedrak 1992; FAO 1993; Hussien *et al.*, 1999) among others.

In the present study, the toxicity of wastewater FP north unit on *Mugil seheli* fingerlings recorded 100% mortality at 56.0 ml/L after 96 h. The least effect was recorded at 3.5 ml/L where only 8 fishes only decreased after 96h. The observed LC_{50} of the FP north unit was 7.4 ml/L after 96h, while for FP south unit, 25 fishes were dead at the first 24-h due to the highest concentration (56.0 ml/L) and 5 fishes were dead at 3.5 ml/L. By the end of the experiment, it was found that 43% of fishes died in the concentration 7.0 ml/L compared with 27% at concentration 3.5 ml/L The observed 96 h LC_{50} for FP south unit effluents was 7.6 ml/L.

The high mortality of *Mugil seheli* fingerlings in the first 24- h may be attributed to the high toxicity of free ammonia and to the pH. Tarzwell (1957) stated that free ammonia is a powerful organic poison that is lethal in small doses. Significantly, free ammonia (unionized ammonia) is more toxic to fish than ammonium ion due to the increasing pH (Tabat, 1962). The increasing in ammonia toxicity with increasing pH is

due to the shift of solution equilibrium towards the formation of free ammonia, whereas the toxicity of the wastewater is due to the unionized form of ammonia (Smart 1975). High ammonia concentration is not necessary required for acute ammonia toxicity problems to occur for *Salmo gairdneri* (Smart, 1978).

The toxicity of ammonia depends actually on the concentration of unionized ammonia in the environment. Bower and Bidwell (1978) mentioned that the lethal effect depend on the concentration of unionized ammonia. Ball (1967) found that the 96-h LC_{50} values of unionized ammonia ranges from 0.4 - 3.1 mg/L for some fresh water fish species, as channel catfish *Ictalurus punctatus*. The present findings agreed also with those of Sedrak (1992) who studied the effect of FP effluents of Abu-Qir factory at Alexandria on deferent stages of *Sparus auratus*. She found that in concentration 15.58 mg/L (NH_3 -N), 100 % of fishes died after 24-h. Hamed (1992) concluded that the 96 h LC_{50} of the Suez FP effluents generated from north and south units was 2.33% for the fries of *Mugil* species. Hussien *et al.* (1999) tested the toxicity of Talkha fertilizer plant effluent upon *Tilapia Zilli*. They found that the dilutions brought about 50% fish survival were 4.1, 3.45, 3.25, 3.1, and 2.6 % after 24, 48, 72, and 96, 128-h of exposure, respectively.

The effluents of north and south drains are mixture of several compounds. All these compounds are sewered together to form the final effluent. In such conditions it is difficult to refer the toxic effect to one compound, but it is more reasonable to accept that the toxic effect of the wastewater is a result of the

combined action of all these compounds altogether.

In the present study, the observed LC_{50} of 96-h for Misr-Iran of Textile raw effluent was 118.0 ml/L. However, at concentration of 50 m/L only 23 % of fishes were found dead after 48-h; the difference may be due to the high pH values. Laboratory studies on the effect of alkaline solution at high pH have indicated that fish damage generally begins at pH 9.0 (Hartwell *et al.*, 1986). Jordan and Lloyed (1964) reported fish mortality at levels of pH up to 10 and they attributed it to pH and salinity. The concentration of unionized ammonia depends not only on total ammonia concentration but also on pH, temperature, and salinity. Values concentration of unionized ammonia increases with increasing temperature and pH, and decreases with higher salinity (Bower and Bidwell, 1978).

There are other parameters of combined sewage emission such as chlorine and insecticide. Chlorine is a poison for respiration as it gets easily to the fish. Hermann & Klinke (1965) found that at low temperatures, 0.2 mg/L chlorine can kill carp within 20 days and 4mg/L chlorine within 8 h.

Effluent of slaughterhouse contains a mixture of substances of varying degrees of toxicity. Ghittino (1961) reported that slaughterhouse effluents contain organic matter, raw meat, detergent, and parasites.

The results of the present study indicated that their limited effect on survival of fishes within the first 24-h at concentrations of 400 and 500 ml/L. The mortality percentage had increased gradually with increasing concentration reached 83 % at 900

ml/L after 96-h of exposure. The observed 96-h LC_{50} was 664 ml/L. High mortality under high concentrations may be attributed to the appearance of the harmful effect of each one of the organic and inorganic materials in the sewage or of the interference combined effect of different components. The final effect of pollutant is not only a matter of concentration, but it also the new environmental conditions created in the drain. The oxygen deficiency intensifies the poisonous effect, and several investigations indicated that, mortality of fish stocked in sewage stabilization ponds was attributed to deficiency of dissolved oxygen rather than ammonia toxicity (Henderson, 1979). Alaboster and Llyed (1980) reported that the sensitivity toward dissolved oxygen varies within species, life stage, and life process. They added that minimum constant value of 5 mg /L is satisfactory in most cases.

Mallat (1985) showed that high pH levels, above 9.0, could damage fish surface tissue leading to general stress reaction such as excessive secretion of mucous on the gills. The dilution of the raw organic effluent decreases its harmful effect. To this effect, Jayangoudar and Canapali (1965) applied a simple technique by diluting the sewage after primary sedimentation with river water in a ratio of 1:3 in autumn and 1:6 in summer to be used in fish farming.

Ammonia was detected in high concentration in the effluents of the FP, both north and south units, and was also detected in the north and south drains. So, ammonium chloride was chosen as one of the ammonium salts to be tested against

the fingerlings of *Mugil seheli* in the present study. Toxicity of ammonia is not a matter of discussion; however, in low dilution such as 0.05 g/L fishes can generally survive, only 27% of the fishes died after 96-h in low concentration.

For ammonium chloride, the observed LC_{50} after 96-h, in the present study, was found to be 0.13 g /L. This result agrees with Ball (1967) who worked on channel catfish and Fils (1968) who worked on *Cyprinus carpio L.*, exposed to 4.9 mg/L of ammonia for 35 days. While, FAO (1993) reported that the maximum admissible ammonia concentration is 0.05 mg/L for Cyprinids and 0.0125 mg/L for Salmonids. According to Hermann and Klinke (1965) at low temperature 0.2 mg/L chlorine can kill carp within 20 days and 4mg/L chlorine kill carp within 8 hours. Sedrak (1992) reported that the 96-h LC_{50} of exposure to ammonia effluents was 8.9 mg/L for Juvenile, 7.5 mg/L for fingerlings and 4.5 mg/L for fries of *Sparus auratus*.

Sodium Hydroxide as a bleaching agent is usually used in the Textile process. The present results indicated that the concentration 0.4 g/L had the highest effect as 93% of the fishes were dead in the first 24-h. In other concentrations, the mortality increased gradually every 24-h. The concentration 0.05 g/L showed the least mortality rate, as only 27% of the fishes were dead after 96-h. The observed LC_{50} was 0.13 g/L after 96-h. The mortality may be due to the high alkalinity of NaOH. The mode of toxic action of NaOH concentration to fish has not been extensively studied. Mercer *et al.* (1997) found several species of fish, which had been affected by alkaline

effluent, discharged after bleaching of wood for the production of pulp and paper. Higashi *et al.* (1992) Suggested that the bleaching Kraft mill effluent is the combined aqueous waste of chlorine, alkaline hypochlorite and Chlorine dioxide. However, the alkaline wastewater pH has a significant influence on the toxic action of ammonia on fish.

Concerning the toxicity of the north drain effluents, the detected results showed a relationship between mortality, effluent concentration and exposure time. Highest mortality percentage was 100% at concentration 56.0 ml/L after 96-h of exposure. While the concentration 7.0 ml/L showed 27.5% mortality rate within the first 24-h of exposure and 37% after 96-h of exposure. The observed LC_{50} was 9.13 after 96 h of exposure. Compared with the south drain effluents, the toxicity of the north drain effluents was lower. The concentration 3.5 ml/L showed a lower mortality rate as only 20% of the fishes dead after 96h. The observed LC_{50} was 10.69 ml/L for the south drain effluents.

In the present study, the increasing of mortality as a result of increasing of time may be attributed to the hydrolyzing and / or oxidizing of the nitrogenous compounds from the fertilizer effluents bringing more toxic derivatives which effect *Mugil seheli* fingerlings. National Academy of Sciences (1973) reported that LC_{50} of unionized ammonia attributed to several species of fish, as determined by short time test, lies between 0.3 and 3 mg/L. Moreover, the unionized ammonia produces a disturbance in the cellular metabolism so that the tissue exhibits

an increased requirement for oxygen. This observation may explain the high mortality rate at 72-h and 96-h of exposure in 56.0 and 14.0 ml/L of Suez fertilizer factories where hypoxia may share in increasing the mortality.

Spargue (1971) found that when the concentration of a toxicant, such as ammonia in the mixture is very low, its share in the toxicity would be less than other additives. Also, active chlorine is very toxic to fish; its toxicity depends on the water temperature, where an active chlorine concentration of 3.5 mg/L has a sublethal effect on Carp at a water temperature of 3 –7° C (FAO, 1993).

The data of the present toxicity study revealed that the combination of different effluents is responsible for the toxicity of the fingerlings of *Mugil seheli*.

Histological observations

Gills are considered the first delicate tissue facing the polluted water and consequently they are the first affected tissues. The gills of *Mugil seheli* fingerlings that exposed to 7.0 ml/L of north drain effluents for 48-h were examined. Compared with a control group; different stages of disorders such as histopathological deformations were observed. The microscopic investigation showed separation of epithelial layer from the pillar system and degeneration of some chloride cells. Moreover, a relative proliferation of mucous cells at the distal region between the secondary lamellae was observed. All these deformations reduce the efficiency of gills for gas exchange and ion

regulation. These changes in gill structure may be due to oxygen deficiency as a result of dissolved oxygen consumption in the oxidation of ammonia and decomposition of organic matter in wastewater.

Presence of alkaline wastewater discharged from Misr-Iran Textile in the north drain may cause destruction of gill respiratory epithelium junctions and dry the surface of gill lamellae. This may lead to water infiltration between the epithelial layer and supporting pillar cells causing an oedema.

Separation of squamous respiratory epithelium from the supporting pillar cells would generally increase the diffusion distance from water to blood. Several publications discussed pathological changes in gills of fish such as Herbert *et al.* (1965) who reported that many substances become more toxic in wastewater leading to oxygen deficiency. They added that the reaction of fish to an insufficient oxygen supply is to increase the volume of water passing over its gills, this may increase the amount of poison reaching the surface of gill epithelium. Burrow (1964) found similar changes in the gills after exposure of salmonids to subtoxic ammonia concentration. Fils (1968) found separation and destruction of the cellular structure of the respiratory epithelium in the gills of *Cyprinus carpio l.* induced by ammonia at a concentration of 4.9 mg/L for 35 days. Smith and Piper (1975) found the same changes in gills of rainbow trout *Salmo gairdneri* after 6-12 months exposure to 0.017ppm NH_3. Smart (1978) found that the primary toxic action of unionized ammonia is gill damage that results in reduced gas

exchange. Sedrak (1992) found separation of the outer cellular structure of the gill respiratory epithelium from pillar cells in *Sparus auratus* due to subtoxic levels of ammonia after 96-h. Mahmoud (1994) reported desquamation of respiratory epithelium in gills of *Mugil seheli* treated with 8% of diluted raw sewage for 96-h.

In the present investigation the microscopic investigation of the gills of *Mugil seheli* that exposed to 7.0 ml/L of north drain effluent for 96-h revealed hyperplasia of epithelial cells, proliferation of both chloride and mucous cells and compressed supporting pillar cells. These observations have been reported for cadmium stress on *Fundulus heteroclitus* exposed to 50 ppm (Gardner and Ycvich, 1970) and for ammonia stress on rainbow trout (Larmoyeux and Piper, 1973; Smart, 1978). Redner and Stickney (1979) described gill hyperplasia in *Tilapia aurea* as a common feature of ammonia stress. The same result was reported using chlorine on *Leiostomus xanthurus* (Middaugh *et al.*, 1980); low levels of monochloro amine combined with ammonia on channel catfish (Mitchell and Cech, 1983); and using 3.90 mg/L of ammonia for 96-h on different stages of *Sparus auratus* (Sedrak, 1992). While, Doaoust and Ferguson (1985) described unique form of proliferate gill disease in marine-cultured Atlantic salmon at high water temperature. Karlsson-Norrgern *et* al. (1986) worked on rainbow trout and described that the gill hyperplasia was coincided with the acidification of water supply by input of anthrogenic acids and subsequent increase in solubility of soil aluminum.

A significant increase of ammonia in wastewater causes an increase in the activity of chloride cells in the lamellar epithelium and compact mass of debris in the interlamellar space of the gills (FAO, 1993). Bucher and Hofer (1993) reported proliferation of chloride cells of rainbow trout gills due to the exposure to concentrations between 16.7-37.5% of treated sewage. Teh *et al.* (1997) reported that clubbing, blunting, and fusion of lamellae were concentrated in the tips of the gill filaments due to a combination and hyperplasia of squamous epithelial, chloride, and mucous cells.

In the present study, Histopathological biomarker lesions were identified in gills of fishes that treated with 7.0 ml/l of south drain effluents. Such lesions are similar to those observed in gills that exposed to the same concentration of north drain effluents for 48-h and 96-h. Such pathological reactions may be attributed to the ammonium effect.

Separation of squamous respiratory epithelium from the supporting pillar cells is a common feature in the gills of *M. Seheli* fingerlings exposed to 100 ml/L of the Textile effluent for 48- to 96-h. In addition, depletion of the mucous cells of the distal regions of the gill lamellae was observed. These injuries may be attributed to the high alkalinity of Textile effluent and / or due to the insufficient dissolved oxygen in the water. Insufficient oxygen supply increases the volume of water passing over fish gills and this may increase the amount of poison reaching the surface of gill epithelium causing destruction of the cellular structure of the gill (FAO, 1993).

Gills of *Mugil seheli* fingerlings reacted strongly to the effluents of the slaughterhouse. After exposure to 500 ml /L for 48-h to effluents of the slaughterhouse, the gills revealed hypertrophy of gill filaments and hyperplasia of the epithelial surface of respiratory lamellae and interlamellar epithelium. Moreover, exposure for 96-h to the same concentration caused complete destruction and sloughing of the gill lamellae. All these deformations may be due to the hypoxia and ammonia. Several publications discussed pathological changes in gills of fish intoxicated with ammonia. Burrow (1964) described histopathological changes in gills of salmonids treated with subtoxic ammonia concentrations and according to his observations complete destruction of the respiratory epithelium occurred. Smart (1978) reported that the primary toxic action of unionized ammonia is gill damage that result in reduced gas exchange. Moreover, the unionized ammonia actually produces a disturbance in cellular metabolism so that the tissue exhibits an increased requirement for oxygen. Also, similar histopathological changes in gills are recorded with other stimuli such as protozoan parasite infection, especially *Ichthrybada necator* (Goldes *et al.*, 1988). Similar intestine injuries of rainbow trout exposed to chronic aluminum and acid rain toxicity was detected (Karlsson-Norrgern *et al.*, 1986).

The microscopic investigations of the liver of the treated fishes after 96 h exposure to 7.0 ml/L of north drain effluent, revealed moderate to severe fatty infiltration causing large spaces between the cellular structures of the liver. Large

vacuoles with smooth membranes were formed inside the hepatocytes. These vacuoles represent a stress on the nucleus and the cell components. Many authors described lipoid liver as a result of combined wastewater components or sometimes of one component. Klinke (1965) stated that the lipoid liver is exacerbated by fatty offal from the Slaughterhouse and concentration of food in the sewage water.

Mazher *et al.* (1987) reported that the fatty infiltration of parenchymal cells in liver of *Claris lizera* was formed due to the exposure to three concentrations of crude oil for 9 days. Sedrak (1992) reported lipid accumulation in the liver of *Sparus auratus* due to the exposure to subtoxic levels of ammonia in concentration 3.3 mg/L for 96-h. Bucher and Hofer (1993) found the same observation in the liver of rainbow trout exposed to 24% of treated sewage for 95 days. Mahmoud (1994) recorded similar features in liver of *Mugil seheli* exposed to 8% of raw sewage for 96-h. Steher *et al.* (1999) found that, the liver of three species of starry flounder caught from US west coast, exhibit hydropic vacuoles in association with hydrocarbons contaminants.

Liver cell necrosis is a common finding in the present study after exposure to 7.0 ml/L of south drain effluents. Small clusters of necrotic hepatocytes (Focal necrosis): may be isolated within the hepatic tissue. These reported symptoms have been discussed as a result of using aflatoxin poisoning on winter flounder by Wales, (1970). Focal necrosis in the liver of Lemon sole in American rivers may be associated with

polychlorinated biphenyl levels (Pierce *et al.*, 1978). Teh *et al.* (1997) found similar observations in the liver of freshwater fish due to the exposure to bleached Kraft mill effluent.

Exposure to 100 ml/L of Misr-Iran Textile for 96-h showed fatty degeneration and hydropic vacuoles inside the hepatic cells, besides deterioration of blood vessels in the liver tissue. Among the vacuolated cells, necrotic hepatocytes with pyknotic nuclei and degenerative islands, composed of smaller basophilic hepatocytes were frequent. This result leads to the belief that bleached Textile mill effluents may plays a role in development of these lesions. Similar results were observed in the liver of sunfish collected from the Pigeon River, impacted by bleached Kraft mill effluents (Teh *et al.*, 1997) and in winter flounder collected from the US west coast contaminated by hydrocarbons (Steher *et al.*, 1999).

Although the gills of *Mugil seheli that* exposed to slaughterhouse effluents reacted strongly but the liver reacted weakly as observed.

The intestinal structure also showed some pathological changes after to exposure to 7.0 ml/L of north drain effluents for 96-h. These lesions were concentrated in the mucosa layer and appeared in reduction of columnar cells, hyperplasia of mucous cells and rupture of brush border hair. In addition, fatty deposits were accumulated between the mucosa. and submucosa. Similar changes were induced by exposure of sheep head minnow fries to Hepatochlor (Goodman *et al.*, 1976), Juvenile

Mugil cephalus to crude oil (Khadre and Shabana, 1991) and Sparus *auratus* to 2.5mg/L of ammonia (Sedrak, 1992).

Rupture of brush border hairs, dilation of the intestinal folds and hyperplasia of mucous cells were observed in the intestine of *Mugil seheli* fingerlings when treated with 7.0 ml/L of south drain effluents for 96-h. Similar intestinal injuries were reported after exposure of sheep head minnow fries to Hepatochlor (Goodman *et al.*, 1976), Juvenil *M. cephalus* exposed to crude oil (Khadre and Shabana, 1991) and fries of *Sparus auratus* to 2.5 mg/L of ammonia Sedrak (1992).

Degenerating changes of intestinal folds and destruction of the mucous cells were observed in the intestinal tissue of treated fish with 100 ml/L for 96-h of Textile effluent. Similar intestinal injuries were discussed by other researchers using different toxic substances. DiMichele and Taylor (1978) described mucosal necrosis in the intestine of *Fundulus heteroclitus* due to naphthalene. Also, Mazhar *et al.* (1987) found degenerative alterations and destruction of cellular structure of intestine in *Claris lazera* due to crude oil.

Ferguson (1989) reported that a wide array of different matters including inorganic, organic chemicals, bacteria and parasites have divergent effects on the fish intestine. The incidence of intestine infections was very high in all the industrial wastewater and virtually independent of wastewater concentrations (Heath, 1989). Roberts (1989) reported extensive haemorrhage and oedema of the intestine submucosa in channel catfish due to the virus infections, the gut

mucosa in acute toxic conditions of bacterial, viral or chemical origin or in toxic algal blooms. All can cause extensive haemorrhage and oedema of mucosa (Molnar, 1982).

Slight degree of sloughing and necrosis of intestinal mucosa were observed in the *M. seheli* fingerlings exposed to 500 ml/L of Slaughterhouse effluents for 96-h. The same pathological alterations were described by in salmonids intestine as a result of contamination with fungal disease (Sis *et al.*, 1979). Also, Ferguson (1989) found that the fungus *Phoma herbarum* and the yeast *Candida sake* have been associated with sloughing mucosal intestine in *Chinook salmon*. Carballo and Munoz (1991) demonstrated that the sublethal concentration of unionized ammonia and nitrate increased the susceptibility of rainbow trout juvenils to Suaprolegnic parasitic infection in the intestinal mucosa. Mahmoud (1994) reported similar findings after applying 8% of raw sewage on *Mugil seheli* fingerlings.

The spleen in teleosts fishes represents one of the major filters in the vascular system removing circulating antigens and effete blood cells (Heath, 1989). Infections of hematopoietic necrosis as a result of destruction of splenic red pulp and resembling thyroid follicles were formed within lumens of blood vessels. These alterations of spleen were observed following exposure to 7.0 ml/L of north drain effluents for 96-h. Findings of thyroid follicles in spleen and particularly follicles within splenic vessels was unusual and unexpected. Leatherland (1994) presented a history of the field of fish thyroidology

reviewing the microscopic appearance and location of this vertebrate structure. First, most orders of teleosts fishes lack the encapsulated glandular form of thyroid tissue. Rather thyroid follicles are scattered through connective tissue of the isthmus from cranial-most regions of the heart to rostral tip of mandible with aggregation generally around the ventral aorta. Second, in teleost fishes it is not unusual to find ectopic thyroid, most commonly in the head kidney, but also in eye, brain, or spleen particularly in animals affected by goiter. Third, epizootics of thyroid enlargement in fishes collected from the wild are rare with exception of the Pacific salmon introduced to the Great Lakes. Since only gill, liver, intestine and spleen were examined in this study, we don't know whether other sites of ectopic thyroid were present. However, Hoover (1984) reported ectopic thyroid following exposure to various contaminants. Our original interpretation was that the follicles within spleen represented ectopic thyroid as in goiter, but the finding of these follicles in blood vessels may indicate that they have reached the spleen via hematogenous route thereby fulfilling criteria for a malignant neoplasia (Baker-Cohen, 1969). Finally, our findings in the present study on the spleen are in agreement with Teh-Swee *et al.* (1997) who reported that spleens of two sunfish collected from the Pigeon River, contaminated by bleached Kraft mill effluent, contained similar thyroid follicles.

On the other hand, an example of splenic alteration was shown in fishes exposed for 7.0 ml/L of south drain effluents for 96-h. The spleen tissue had abundant white pulp of various

sizes and shapes. In addition, splenic erythroblasts, lymphocytes decline, congestion and necrosis of reticuloendothelial cells lining the ellipisoids were observed. These lesions may be due to the decreasing of spleen size as a result of the large volume of circulating blood. Our findings are not unusual as a result of the stress of chemical pollutants in the effluent. Similar alterations were reported for the spleen of bass fish that collected from East Fork Poplar Creek, receiving point source discharges of mixed contaminants from a nuclear weapons facility and exposed to radiation (Takashi, 1982; The-Swee *et al.*, 1997).

In general, the present study concluded that the industrial effluents in present form have detrimental effects on fish life either killing or destroying most of the vital organs and affecting the marine environment of the Suez Bay. In order to save the northern and western coasts of the Bay Late Meshal (1970) had suggested digging a canal to be digging crossing the Adabyia head to facilitate the out flow of the Bay water into Suez Gulf. On the other hand, treatment of the wastes is urgently needed and the cost of this treatment will be much less than its harmful effect.