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HISTOPATHOLOGICAL STUDIES ON SOME ORGANS OF *OREOCHROMIS NILOTICUS, TILAPIA ZILLII* AND *SYNODONTIS SCHALL* FROM EL-SALAM CANAL, EGYPT

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ABSTRACT

In the present study, the histological structures of the gills, liver, kidneys and gonads of *Oreochromis niloticus, Tilapia zillii* and *Synodontis schall* from El-Salam canal were studied for a period extending from spring 2000 to winter 2001 (4 successive seasons). Several histopathological changes were observed in the gills, liver, kidneys and gonads of the studied fish during the four seasons.

In the gills, proliferative changes in the epithelium of gill filaments and secondary lamellae, degenerative and necrotic changes in gill filaments, separation of the epithelium of the secondary lamellae from the lamellar supporting cells in gill filaments, intravascular haemolysis and dilation in the blood vessels of gill filaments, haemorrhage between gill filaments, edema in secondary lamellae and mucus accumulation between gill filaments were seen.

In the liver, vacuolar degeneration, focal areas of coagulative necrosis, focal areas of necrosis, destruction of hepatoportal blood vessels and haemorrhage between the hepatocytes were observed. Besides, intravascular haemolysis and dilation were seen in hepatic and hepatoportal blood vessels. Also, dilation and congestion were noticed in blood sinusoids.

In the kidneys, the histopathological changes included vacuolar degeneration in the epithelium of renal tubules, focal areas of necrosis between the renal tubules, depletion in the haemopoietic areas, haemolysis between the renal tubules and edema in Bowman's capsules. Moreover, intravascular haemolysis in renal blood vessels and over population of glomeruli were observed.

In the testis, degenerative and necrotic changes in the cellular elements of seminiferous tubules, with inhibition of spermatogenesis (some seminiferous tubules appeared lucent or with a lesser number of sperms, indicating lack of active spermatogenesis), focal areas of necrosis and fibrous capsules around some seminiferous tubules were observed. Besides, malformation and distortion of the architecture of some seminiferous tubules were seen.

In the ovary, degenerative and necrotic changes (atresia) in the oocytes, proliferative changes in the granulosa of the oocytes, resulting sometimes in adhesion of the cellular coat of the oocytes, haemorrhage between the oocytes and intravascular haemolysis in some ovarian blood vessels were seen. Moreover, focal areas of necrosis, aggregations of haemosiderin between the oocytes and separation of the follicular layers from the oocytes were observed.

These histopathological alterations showed marked seasonal variations, where, they were more severe during winter. The results indicated that *Oreochromis niloticus* is more sensitive to pollution than *Tilapia zillii* and *Synodontis schall.*

INTRODUCTION

The aquatic environment is subjected to different types of pollutants which enter water bodies with industrial, domestic and agricultural waste waters and affect severely the aquatic organisms. The problems of environmental pollution and its deleterious effects on aquatic organisms received focused interest during the last decades.

El-Salam canal is one of the most important projects in Egypt, since it transports the Nile water to Sinai. The canal runs to the south of Lake Manzalah towards the northern area of Sinai. It is intended to supply enough irrigation water for about 450,000 feddans as a potential area for agriculture development in northern Sinai. The canal is carrying the Nile water from Damietta branch upstream of Faraskour Barrage (a total amount of 2110 million m^3/year) in addition to drainage water from El-Serw and Hadous drains (2050 million m^3/year drainage water) (Sabae and Abdel-Satar, 2001).

Previous studies reported that El-Salam canal is contaminated with heavy metals, bacteria indicative of sewage pollution and ammonia (Abdel - Baky, 2001, Bahnasawy, 2001, Rabeh, 2001 and Sabae and Abdel-Satar, 2001). Therefore, the increasing amount of agricultural, domestic and industrial drainage water which

discharged into El-Salam canal may exert considerable changes in the histological structures of the different organs of the fish.

Several histopathological changes have been reported in the gills, liver, kidneys and gonads of fish in response to agricultural, sewage and industrial pollutants. Histopathological changes in the gills were observed in *Fundulus heteroclitus* exposed to cadmium (Gardner and Yevich, 1970), in channel catfish, *Ictalurus punctatus*, experimentally and naturally infected with channel catfish virus disease (Major *et al.*, 1975), in *Thymallus arcticus* infected with ectoparasite monogenetic trematode, *Tetraonchus rauschi* (Wobeser *et al.*, 1976), in blue gill, *Lepomis macrochirus*, exposed to monochloramine (Bass *et al.*, 1977), in mummichogs, *Fundulus heteroclitus*, exposed to naphthalene (DiMichele and Taylor, 1978), in steelhead trout, *Salmo gairdneri*, exposed to nitrite (Wedemeyer and Yasutake, 1978), in immature rainbow trout exposed to crude oil (Enghelhardt *et al.*, 1981), in *Ictalurus punctatus* exposed to a combination of ammonia and low levels of monochloramine (Mitchell and Cech, 1983), in *Sarotherodon mossambicus* exposed to $HgCl_2$ (Naidu *et al.*, 1983), in fathead minnows, *Pimephales promleas*, experimentally exposed to acidified Canadian Lakes (Leino *et al.*, 1987), in *Tilapia nilotica* exposed to lead acetate, mercuric chloride and cadmium chloride (Balah *et al.*, 1993), in carp, *Cyprinus carpio*, exposed to 20 and 50% sewage (Kakuta and Murachi, 1997), in *Tilapia zillii* exposed to phenol (Marie *et al.*, 1997 a&b), in *Salmo trutta* exposed to iron sulphate (Dalzell and Macfarlane, 1999), in *Oreochromis niloticus* exposed to pesticides, reldan, roundup and lannate (Mohamed, 1999) and in *Tilapia zillii* exposed to lindane (Mourad *et al.*, 1999).

Histopathological changes in the liver of fish as a result of exposure to toxicants have been studied by several authors (Couch, 1975, Major *et al.*, 1975, Li *et al.*, 1978, Sastry and Malik, 1979, Dixon and Leduc, 1981 and Desai *et al.*, 1984). Several histopathological changes in the liver were also observed in ruffe, *gymnocephalus cernua*, collected from Elbe Estuary contaminated by domestic, industrial and agricultural pollutants (Heidemarie and Peters, 1985), in Atlantic tomcod, *Microgadus tomcod*, collected from contaminated Hudson River (Cormier, 1986), in *Tilapia nilotica* exposed to diazinon (Issa and Gabr, 1989), in *Tilapia nilotica* exposed to fluorine and sulphur emitting from factory of fertilizer (Aly *et al.*, 1992), in *Clarias lazera* exposed to bayluscide (Hamza *et al.*, 1996), in *Tilapia zillii* exposed to lindane (Mourad *et al.*, 1999)

and in *Oreochromis niloticus* and *Tilapia zillii* collected from the southern region of Lake Manzalah contaminated with domestic, industrial and agricultural pollutants (Mohamed, 2001b).

The histopathological alterations in the kidneys of fish induced by exposure to different toxicants have been reported by several authors (Gardner and Yevich, 1970, Wolf *et al.*, 1972, Major *et al.*, 1975, Li *et al.*, 1978, Kumar and Srivastava, 1980 and Sastry and Sharma, 1981). Histopathological changes in the kidneys were also observed in plaice, *Pleuronectes platessa*, exposed to crude oil (Haensly *et al.*, 1982), in European eels, *Anguilla anguilla*, infected with the parasite *Myxidium giardi* Cépède (Ventura and Paperna, 1984), in *Tilapia nilotica* exposed to pyrethroid insecticide (neopybuthrin) (Gabr, 1990), in *Tilapia nilotica* exposed to fluorine and sulphur emitting from factory of fertilizer (Aly *et al.*, 1992), in *Oreochromis niloticus* infected by *Streptococcus* sp. (Chang and Plumb, 1996), in carp (*Cyprinus carpio)* exposed to 20 and 50% sewage (Kakuta and Murachi, 1997), in *Tilapia zillii* exposed to phenol (Marie *et al.*, 1997b) and in *Oreochromis niloticus* exposed to pesticides, reldan, roundup and lannate (Mohamed, 1999).

Histopathological changes were seen in the testis of *Salmo gairdneri* exposed to cyanide (HCN) (Sylvia *et al.*, 1979), in the ovary of rainbow trout exposed to cyanide (HCN) (Lesniak and Ruby, 1982), in the ovary of *Puntius conchonius* exposed to zinc (Kumar and Pant, 1984), in the ovary of *Oreochromis mossambicus* exposed to malathion (Shukla *et al.*, 1984), in the gonads of winter flounder and cod infected with the hemoflagellate *Trypanosoma murmanensis* and then exposed to Venezuelan crude oil (Khan, 1987), in the gonads of *Monopterus albus* exposed to cadmium (Singh, 1989), in the testis of catfish, *Clarias batrachus* L., exposed to mercuric chloride, methyl mercuric chloride and emisan 6 (an organic mercurial fungicide) (Kirubagaran and Joy, 1992), in the ovary of *Oreochromis niloticus* infected by *Streptococcus* sp. (Chang and Plumb, 1996) and in the gonads of *Tilapia zillii* exposed to phenol (Mohamed, 2001a).

The present study aims to illustrate the harmfull effects of pollution of El-Salam canal on the histological features of four organs (gills, liver, kidneys and gonads) of the commercially important fish *Oreochromis niloticus, Tilapia zillii* and *Synodontis schall.*

MATERIAL AND METHODS

Samples of *Oreochromis niloticus* (150-200g), *Tilapia zillii* (150-200g) and *Synodontis schall* (100-150g) were collected seasonally during a period extending from spring, 2000 to winter, 2001 from El-Salam canal. During the same period, control samples of the studied fish were collected from the River Nile, at Al-Kanater Al-Khairya station.

Fish were dissected and pieces of gills, liver, kidneys and gonads were immediately isolated and fixed in Bouin's fluid for 24-48 hrs. After fixation, the tissues were washed in 70% ethyl alcohol to get rid of excess fixative and then dehydrated through ascending grades of ethyl alcohol (70% 1 hr, 80% 1 hr, 90% 1 hr and 100% changed twice during 1 hr). The specimens were cleared in xylene for 15-20 min and infiltrated with and embedded in paraffin wax. The paraffin wax block was sectioned at the thickness 4-6 μm. Sections were mounted on clear glass slides and were stained with Harris' haematoxylin and eosin.

RESULTS

I. Gills:

The histological investigations indicated that the gills of control *Oreochromis niloticus, Tilapia zillii* and *Synodontis schall* -- collected from the River Nile, at Al-Kanater Al-Khairya station – have a normal architecture (Fig. 1A).

Several histopathological changes were seen in the gills of the studied fish – collected from El-Salam canal – during the four seasons (spring, summer, autumn and winter).

Spring:

During spring season, the gills of *Oreochromis niloticus* showed proliferative changes in the epithelium of some gill filaments and secondary lamellae, resulting sometimes in obliteration of the space between the secondary lamellae (Fig. 1B), degenerative and necrotic changes in some gill filaments (Fig. 1F) and separation of the epithelium of the secondary lamellae from the lamellar supporting cells in some gill filaments (Fig. 3A&C). Severe dilation and congestion in the blood vessels were seen in some gill filaments (Fig. 2D). Severe haemorrhage was also observed between the gill filaments (Fig. 3A&C).

The gills of *Tilapia zillii* showed slight degenerative and necrotic changes in some gill filaments and severe dilation and congestion in the blood vessels of some gill filaments (Fig. 2D).

Moreover, proliferative changes were seen only in the epithelium at the base of the secondary lamellae. However, the gills of *Synodontis schall* showed slight degenerative and necrotic changes in some gill filaments, slight haemorrhage between the gill filaments and dilation in the blood vessels of some gill filaments.

Summer and autumn:

During summer and autumn seasons, the gills of *O. niloticus* showed the same histopathological alterations observed in the gills of *O. niloticus* during spring season. Besides, accumulation of mucus was noticed between the gill filaments in some fish.

In the gills of *T. zillii*, proliferative changes were observed in the epithelium of the secondary lamellae resulting in nodular proliferation in some gill filaments (Fig. 2E). In other gill filaments, the proliferative changes were noticed only in the epithelium at the base of the secondary lamellae (Fig. 2E). Moreover, degenerative and necrotic changes were observed in some gill filaments (Fig. 1F). Also, slight haemorrhage was seen between the gill filaments during summer season.

The gills of *S. schall* showed the same histopathological changes observed in the gills of *S. schall* during spring season. Besides, slight proliferative changes in the epithelium of some gill filaments and secondary lamellae and separation of the epithelium of the secondary lamellae from the lamellar supporting cells in some gill filaments (Fig. 3B) were noticed.

Winter :

During winter season, the histopathological alterations in the gills of the three studied fish appeared more severe. The histopathological changes in the gills of *O. niloticus* included separation of the epithelium of the secondary lamellae from the lamellar supporting cells in most gill filaments and severe proliferative changes in the epithelium of some gill filaments and secondary lamellae, resulting in obliteration of the space between the secondary lamellae (Fig. 1D&E). Also, excessive mucus accumulation between the gill filaments (Fig.2A) and severe degenerative and necrotic changes in the gill filaments – the secondary lamellae appeared destructed and necrotized tissues aggregated in the mucus between the gill filaments – (Fig. 2A) were seen. Moreover, intravascular haemolysis and dilation were observed in the blood vessels of some gill filaments (Fig. 2F). Edema was also seen in the secondary lamellae (Fig. 2G).

However, in the gills of *T. zillii* and *S. schall*, severe degenerative and necrotic changes in the gill filaments (Fig. 2B) and separation of the epithelium of the secondary lamellae from the lamellar supporting cells in some gill filaments (Fig. 3D) were noticed. Besides, proliferative changes were observed in the epithelium of the secondary lamellae resulting sometimes in obliteration of the space between the secondary lamellae (Fig. 1C). Severe dilation and congestion in the blood vessels of some gill filaments (Fig. 2C) and excessive mucus accumulation between the gill filaments (Fig. 2B) were seen.

II- Liver:

The histological structures of the liver of control *O. niloticus*, *T. zillii* and *S. schall* are shown in fig. 3E. No abnormal histological features were observed.

Several histopathological alterations were observed in the liver of the studied fish – collected from El-Salam canal – during the four seasons.

Spring :

During spring season, the liver of *O. niloticus* showed focal areas of coagulative necrosis (Fig. 4A), small multiple vacuoles in the hepatocytes and sometimes this vacuolar degeneration appeared pronounced and the hepatocytes appeared empty (Fig. 6B) and focal areas of necrosis (Fig. 5B). Also, destruction of some hepatoportal blood vessels was seen (Fig. 5A). Besides, haemorrhage and aggregations of inflammatory cells were observed between the hepatocytes (Fig. 5D). Moreover, in some cases, dilation was noticed in the central veins (Fig. 4F).

The hepatocytes of *T. zillii* showed slight vacuolar degeneration and dissociation. Also, small focal areas of coagulative necrosis (Fig. 4A) were observed. In addition, destruction of some hepatoportal blood vessels was observed (Fig. 5A). Also, slight haemorrhage was noticed between the hepatocytes. Dilation and congestion were also seen in some blood sinusoids (Fig. 5F). Thrombosis formation was observed in some hepatoportal blood vessels (Fig. 4B).

In *S. schall*, the liver showed vacuolar degeneration in the hepatocytes (Fig. 6C), small focal areas of necrosis, haemorrhage between the hepatocytes and destruction of some blood vessels. Moreover, haemolysis was seen in some blood sinusoids (Fig. 6A).

Summer and autumn:

During summer and autumn seasons, the liver of *O. niloticus* showed focal areas of vacuolar degeneration in the hepatocytes (Fig. 6D), focal areas of coagulative necrosis, focal areas of necrosis (Fig. 5B) and haemorrhage between the hepatocytes. Besides, during summer season, thrombosis formation was seen in some hepatic (Fig. 4C) and hepatoportal (Fig. 4B) blood vessels.

The liver of *T. zillii* showed focal areas of vacuolar degeneration in the hepatocytes, focal areas of necrosis, destruction of some hepatoportal blood vessels and haemorrhage between the hepatocytes. Moreover, during summer, severe haemolysis and dilation were observed in some hepatoportal blood vessels of some fish (Fig. 4D).

In *S. schall,* the liver showed the same histopathological alterations observed in the liver of *S. schall* during spring season, however, the vacuolar degeneration was more pronounced.

Winter :

During winter, the histopathological changes in the liver of the three studied fish appeared more severe. In the liver of *O. niloticus,* extensive vacuolar degeneration in the hepatocytes (Fig.6E&F), focal areas of coagulative necrosis, multiple focal areas of necrosis (Fig. 5C) and severe haemorrhage between the hepatocytes (Fig. 5E) were seen. Moreover, severe intravascular haemolysis and dilation were observed in some hepatic (Fig. 4E) and hepatoportal (Fig. 4D) blood vessels.

The liver of *T. zillii* and *S. schall* showed the same histopathological changes observed in the liver of *O. niloticus*, however, the pathological changes appeared more pronounced in the liver of *O. niloticus.*

III. Kidney:

The histological investigations indicated that the kidneys of control *O. niloticus*, *T. zillii* and *S. schall* – collected from the River Nile, at Al-Kanater Al-Khairya station – have a normal architecture (Fig. 6G).

Several histopathological alterations were observed in the kidney of the studied fish – collected from El-Salam canal – during the four seasons.

Spring:

During spring season, the kidney of *O. niloticus* showed vacuolar degeneration in the epithelium of some renal tubules (Fig.

8C) and focal areas of haemolysis and aggregation of inflammatory cells between the renal tubules (Fig. 7E).

In the kidney of *T. zillii*, slight vacuolar degeneration in the epithelium of some renal tubules, depletion in the haemopoietic areas (Fig. 7H) and slight haemolysis between the renal tubules were seen. Moreover, dilation of the capillary tubes of some renal tubules was noticed (Fig. 7C).

The histopathological alterations in the kidney of *S. schall* included slight vacuolar degeneration in the epithelium of some renal tubules, aggregation of inflammatory cells (Fig. 6H) and slight haemolysis between the renal tubules and congestion of the capillary tubes of some renal tubules (Fig. 7D).

Summer and autumn:

During summer and autumn seasons, the kidney of *O. niloticus* showed the same histopathological changes observed in the kidney of *O. niloticus* during spring, however, the vacuolar degeneration in the epithelium of the renal tubules was more pronounced during summer and autumn seasons. Small focal areas of necrosis were also seen between the renal tubules (Fig. 8D). Moreover, during autumn, edema was observed in some Bowman's capsules (Fig. 7A).

In *T. zillii* and *S. schall*, the kidney showed the same histopathological changes observed in the kidney of these fish during spring.

Winter:

During winter season, as in the gills and liver, the histopathological alterations in the kidney of the three studied fish appeared more severe. In the kidney of *O. niloticus*, the pathological changes included extensive vacuolar degeneration in the epithelium of renal tubules (Fig. 7B), focal areas of necrosis (Fig. 8B), severe haemolysis between the renal tubules (Fig. 7F) and in some renal blood vessels (Fig. 8A), depletion in the haemopoietic areas (Fig. 7H), edema in most Bowman's capsules and dilation of the capillary tubes of some renal tubules (Fig. 7B).

The kidney of *T. zillii* and *S. schall* showed vacuolar degeneration in the epithelium of renal tubules and focal areas of necrosis (Fig. 8B). Moreover, intravascular haemolysis in some renal blood vessels (Fig. 8A) and severe haemolysis between the renal tubules (Fig. 7G) were seen. Besides, depletion in the haemopoietic areas (Fig. 7H) and over population of glomeruli (Fig. 8A) were observed in the kidney of *T. zillii*.

IV. Testes :

The histological structures of the testes of control *O. niloticus, T. zillii* and *S. schall* are shown in fig. 8E, no abnormal histological features were observed.

Several histopathological alterations were observed in the testes of the studied fish – collected from El-Salam canal – during the four seasons.

Spring:

During spring season, the testes of *O. niloticus* showed degenerative and necrotic changes in the cellular elements of some seminiferous tubules (Fig. 9D). Some seminiferous tubules revealed a lesser number of sperms or appeared lucent (without sperms) indicating lack of active spermatogenesis (Fig. 10A).

On the other hand, the testes of *T. zillii* and *S. schall* showed slight degenerative and necrotic changes in the cellular elements of some seminiferous tubules.

Summer and autumn:

During summer and autumn seasons, the testes of *O. niloticus* showed the same pathological changes observed in the testes of *O. niloticus* during spring. Besides, small focal areas of necrosis were seen in the testes (Fig. 9B). Fibrous capsule was observed around some seminiferous tubules (Fig. 10D).

In the testes of *T. zillii* and *S. schall,* degenerative and necrotic changes in the cellular elements of some seminiferous tubules (Fig. 9E) and small focal areas of necrosis (Fig. 9E) were seen. Moreover, some seminiferous tubules revealed a lesser number of sperms or appeared lucent (Fig. 10B). Malformations and distortion of the architecture of some seminiferous tubules were observed in the testes of *T. zillii* (Fig. 9E). In some cases, sperms were observed between the seminiferous tubules in the testes of *T. zillii* during summer (Fig. 9E).

Winter :

During winter, the histopathological alterations in the testes of the studied fish appeared more severe. In the testes of *O. niloticus*, the histopathological changes included multiple focal areas of necrosis, degenerative and necrotic changes in the cellular elements of most seminiferous tubules (Fig. 9C) and focal areas of coagulative necrosis (Fig. 9A). Some seminiferous tubules revealed a lesser number of sperms or appeared lucent. Fibrous capsule was seen around some seminiferous tubules (Fig. 10C).

The testes of *T. zillii* and *S. schall* showed the same histopathological changes observed in the testes of both fish during summer and autumn. However, the pathological alterations appeared more pronounced during winter.

V. Ovaries:

The ovaries of control *O. niloticus, T. zillii* and *S. schall* are shown in fig. 10E, no abnormal histological features were observed.

Several histopathological changes were seen in the ovaries of the studied fish – collected from El-Salam canal – during the four seasons.

Spring :

During spring season, the ovaries of *O. niloticus* and *T. zillii* showed degenerative changes (atresia) in some oocytes (Fig. 12B) and proliferative changes in the granulosa of some oocytes, resulting sometimes in adhesion of the cellular coat of the oocytes (Fig. 11D). Besides, some oocytes collapsed and became abnormally irregular in shape (Fig. 11A). Moreover, haemorrhage was seen between the oocytes (Fig. 11B). In addition, separation of the follicular layers from the oocytes was observed in the ovaries of *O. niloticus* (Fig. 10F).

On the other hand, the ovaries of *S. schall* showed degenerative changes in some oocytes (Fig. 12B) and slight haemorrhage between the oocytes. Also, some oocytes collapsed and became abnormally irregular in shape (Fig. 11A).

Summer and autumn:

During summer and autumn seasons, the ovaries of *O. niloticus* and *T. zillii* showed the same histopathological changes observed in the ovaries of both fish during spring season.

In the ovaries of *S. schall*, degenerative changes in some oocytes, separation of the follicular layers from the oocytes (Fig. 10G) and haemorrhage between the oocytes (Fig. 11B) were observed.

Winter:

During winter season, the histopathological alterations in the ovaries of the studied fish appeared more severe. In the ovaries of *O. niloticus*, the histopathological changes included degenerative and necrotic changes in the oocytes (Fig. 12A), proliferative changes in the granulosa of most oocytes, resulting sometimes in adhesion of the cellular coat of the oocytes (Fig. 11E), separation of the follicular layers from the oocytes and severe haemorrhage between the oocytes. Besides, focal aggregations of haemosiderin were observed between

the oocytes (Fig. 11C). Moreover, thrombosis formation was seen in some ovarian blood vessels (Fig. 12C).

The ovaries of *T. zillii* and *S. schall* showed the same histopathological changes observed in the ovaries of both fish during summer and autumn; however, the pathological changes appeared more severe during winter. Moreover, thickening of the ovarian wall was observed in the ovaries of *T. zillii* (Fig. 10H).

DISCUSSION

The results of the present study revealed that fish collected from El-Salam canal manifested histopathological changes in the gills, liver, kidneys and gonads. The histopathological alterations observed in the gill tissues of the studied fish throughout the study period included proliferative changes in the epithelium of gill filaments and secondary lamellae, degenerative and necrotic changes in gill filaments and separation of the epithelium of the secondary gill lamellae from the lamellar supporting cells. Also, intravascular haemolysis and dilation in the blood vessels of gill filaments, haemorrhage between gill filaments,. edema in secondary lamellae and mucus accumulation between gill filaments were seen.

The pathological changes may be a reaction to toxicants intake or an adaptive response to prevent the entry of the pollutants through the gill surface.

The observed dilation of the lamellar blood vessels and the presence of edematous fluid at the base of the secondary lamellae may be due to increased permeability induced by the prolonged exposure to the pollutants. This edematous fluid separated the respiratory epithelium from the underlying tissue and led to its desquamation as well as necrosis (Balah *et al.*, 1993).

Since gills are the respiratory and osmoregulatory organ of the fish, the cellular damage observed in the gills in terms of epithelial hyperplasia, necrosis and separation of the epithelial layer from supportive tissues might impair the respiratory function of the gills by reducing respiratory surface area, and this badly affects the physiology and may lead to the death of fish.

The present study showed several histopathological changes in the liver of the studied fish. The alterations included vacuolar degeneration, focal areas of coagulative necrosis, focal areas of necrosis, destruction of hepatoportal blood vessels and haemorrhage between the hepatocytes. Also, intravascular haemolysis and dilation

of the hepatic and hepatoportal blood vessels and congestion and dilation in blood sinusoids were seen.

The observed fatty degeneration in the liver of the studied fish may be due to an inability of the liver cells to synthesize proteins that are normally conjugated with triglycerides in the hepatic cells as a response to the pollutants (Kadry and Abdel Mageid, 1995 and Mohamed, 2001b), a decrease in the rate of utilization of energy reserve or an enhance in the synthesis of fats in the liver (Desai *et al.*, 1984 and Mohamed, 2001b).

The cellular degeneration observed in the liver of the studied fish may be due to the vascular dilation and intravascular haemolysis observed in the blood vessels with subsequent stasis of blood (Mohamed, 2001b).

The necrosis observed in the liver of the fish may be attributed to the destruction of the hepatoportal blood vessels which causes invasive infiltration of leucocytes and detrimental focal necrosis resulting in the complete dissolution of the hepatocytes (Ram and Singh, 1988), to a direct effect of the toxicants on the cell or to an accumulation of acetylcholine in the tissues (Desai *et al.*, 1984).

The results of the present work revealed that fish collected from El-Salam canal have shown histopathological changes in the kidney; these included vacuolar degeneration in the epithelium of renal tubules, focal areas of necrosis between the renal tubules, depletion in the haemopoietic areas, haemolysis between the renal tubules and edema in Bowman's capsules. Besides, intravascular haemolysis in renal blood vessels and over population of glomeruli were seen.

Yokote (1982) reported that the necrosis of renal tubules affects the metabolic activities and promotes metabolic abnormalities in fish.

The present study showed several histopathological alterations in the gonads of the studied fish. In the testes, degenerative and necrotic changes in the cellular elements of seminiferous tubules, development of focal areas of necrosis, focal areas of coagulative necrosis, formation of fibrous capsules around some seminiferous tubules and malformation and distortion of the architecture of some seminiferous tubules were seen. In the ovaries, development of degenerative and necrotic changes in the oocytes, focal areas of necrosis, proliferative changes in the granulosa of the oocytes resulting sometimes in adhesion of the cellular coat of the oocytes, haemorrhage between the oocytes, intravascular haemolysis in some

ovarian blood vessels, aggregations of haemosiderin between the oocytes and separation of the follicular layers from the oocytes were observed.

The observed histopathological alterations in the testes and ovaries of the studied fish may reduce the ability of fish to reproduce. It is well known that water pollution has a serious inhibitory effect on fish reproduction (Lesniak and Ruby, 1982, Chang and Plumb, 1996 and Mohamed, 2001a) resulting in a decrease in their abundance and, consequently, a decline in fish species diversity.

Along its course, the Nile receives sewage, agricultural and industrial drainage water from about 37 main drains (Aboul-Ela *et al.*, 1990) and the level of pollution increases upward from south to north. The Nile water from Damietta branch (2110 million m^3) mixed with the drainage water from El-Serw and Hadous drains (2050 million m^3) are the source of water of El-Salam canal.

In previous studies on El-Salam canal, Abdel-Baky (2001) reported that El-Salam canal is contaminated with Cu (0.018 mg/l), Pb (0.045 mg/l), Zn (3.09 mg/l) and Mn (0.07 mg/l). Also, he found that the concentrations of Pb and Zn in the liver, kidney, gonads and gills of *Clarias gariepinus* exceeded the maximum permissible limit. Similarly, Bahnasawy (2001) reported that El-Salam canal is polluted with Cu, Zn, Pb and Mn (0.04, 0.15, 0.09 and 0.09 mg/l, respectively). He found that the gills, kidney and gonads (especially ovaries) of *Clarias gariepinus* had high amounts of heavy metal accumulation (Cu, Zn, Pb and Mn). Rabeh (2001) showed a remarkable increase in the bacteria indicative of sewage pollution in El-Salam canal. Sabae and Abdel-Satar (2001) recorded high concentrations of ammonia (0.62 – 5.27 mg/l), Fe (0.33 – 4.76 mg/l), Mn (27.06 – 223.36 µg/l), Zn (6.92 – 56.71 µg/l), Cu (2.98 – 54.56 µg/l) and Pb (12.94 - 53.06 µg/l) in El-Salam canal during four successive seasons from autumn 1999 to summer 2000. They also recorded high bacterial counts (72 x 10^3 – 90x10^4 cells/ml) in El-Salam canal.

Therefore, the histopathological alterations observed in the gills, liver, kidneys and gonads of the studied fish may be attributed to the effects of the agricultural, industrial and sewage wastes discharge into El-Salam canal.

The histopathological changes observed in the gills of the studied fish, collected from El-Salam canal, are in agreement with those observed in *Fundulus heteroclitus* exposed to cadmium

(Gardner and Yevich, 1970), in channel catfish infected with channel catfish virus disease (Major *et al.*, 1975), in *Thymallus arcticus* infected with ectoparasite monogenetic trematode (Wobeser *et al.*, 1976), in steelhead trout, *Salmo gairdneri* exposed to nitrite (Wedemeyer and Yasutake, 1978), in immature rainbow trout exposed to crude oil (Enghelhardt *et al.*, 1981), in *Ictalurus punctatus* exposed to a combination of ammonia and low levels of monochloramine (Mitchell and Cech, 1983), in *Tilapia nilotica* exposed to lead acetate, mercuric chloride and cadmium chloride (Balah *et al.*, 1993), in carp (*Cyprinus carpio*) exposed to 20 and 50% sewage (Kakuta and Murachi, 1997), in *Tilapia zillii* exposed to phenol (Marie *et al.*, 1997 a&b), in *Salmo trutta* exposed to iron sulphate (Dalzell and Macfarlane, 1999) and in *Oreochromis niloticus* exposed to pesticides, reldan, roundup and lannate (Mohamed, 1999).

Also, the histopathological alterations observed in the liver of the studied fish are similar to those observed by Desai *et al.* (1984) in *Tilapia mossambica* exposed to monocrotophos, Heidemarie and Peters (1985) in ruffe, *Gymnocephalus cernua,* collected from Elbe Estuary contaminated by domestic, industrial and agricultural pollutants, Aly *et al.* (1992) in *Tilapia nilotica* exposed to fluorine and sulphur emitting from factory of fertilizer, Mourad *et al.* (1999) in *Tilapia zillii* exposed to lindane and Mohamed (2001b) in *Oreochromis niloticus* and *Tilapia zillii* collected from the southern region of Lake Manzalah contaminated with domestic, industrial and agricultural pollutants.

Moreover, the histopathological changes observed in the kidneys of the studied fish are similar to those observed in plaice, *Pleuronectes platessa*, exposed to crude oil (Haensly *et al.*, 1982), in European eels, *Anguilla anguilla*, infected with the parasite *Myxidium giardi* Cépède (Ventura and Paperna, 1984), in *Tilapia nilotica* exposed to fluorine and sulphur emitting from factory of fertilizer (Aly *et al.*, 1992), in carp, *Cyprinus carpio*, exposed to 20 and 50% sewage (Kakuta and Murachi, 1997), in *Tilapia zillii* exposed to phenol (Marie *et al.*, 1997b) and in *Oreochromis niloticus* exposed to pesticides, reldan, roundup and lannate (Mohamed, 1999).

Similarly, the histopathological alterations observed in the gonads of the studied fish are in agreement with those observed in *Salmo gairdneri* exposed to cyanide (Sylvia *et al.*, 1979), in *Puntius conchonius* exposed to zinc (Kumar and Pant, 1984), in *Oreochromis mossambicus* exposed to malathion (Shukla *et al.*, 1984), in winter flounder and cod infected with hemoflagellate *Trypanosoma*

murmanensis and then exposed to Venezuelan crude oil (Khan, 1987), in *Heteropneustes fossilis* exposed to textile-mill effluent (Murugesan and Haniffa, 1992), in *Oreochromis niloticus* infected by *Streptococcus* sp. (Chang and Plumb, 1996) and in *Tilapia zillii* exposed to phenol (Mohamed, 2001a).

The results indicated that the histopathological alterations were more severe during winter and this may be attributed to the types of pesticides in the agricultural wastes dumping to the water body of El-Salam canal during winter. Also, the histopathological changes were more severe in *Oreochromis niloticus* than *Tilapia zillii* and *Synodontis schall* and this may be attributed to the fact that *T. zillii* and *Synodontis schall* can adapt easily to variations in the ecological conditions of the canal.

It could be concluded that the environmental conditions of El-Salam canal induce histopathological changes in the gills, liver, kidneys and gonads of *Oreochromis niloticus, Tilapia zillii* and *Synodontis schall.* Consequently, it is recommend to subject the drainage waters discharged into El-Salam canal to technical treatment that fulfill its safety.

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EXPLANATION OF FIGURES

Fig. (1): Sections of gills of fish showing:

(A): control *Oreochromis niloticus, Tilapia zillii* and *Synodontis schall* (X100).

(B): proliferative changes in the epithelium of gill filaments and secondary lamellae (*O. niloticus*, spring 2000) (X 100).

(C): proliferative changes in the epithelium of gill filaments and secondary lamellae (*S. schall,* winter 2001) (X 400).

(D) & (E): proliferative changes in the epithelium of gill filaments and secondary lamellae (*O. niloticus,* winter 2001) (X100 & 400, respectively).

(F): degenerative and necrotic changes in gill filaments (*O. niloticus*, spring 2000) (X 400).

Fig. (2) : Sections of gills of fish showing:

(A) & (B): severe degenerative and necrotic changes in gill filaments and mucus accumulation between gill filaments (*O. niloticus* and *T. zillii,* respectively, winter 2001) (X400).

(C) & (D) : dilation and congestion in the blood vessels of gill filaments (*T. zillii*, winter 2001 and *O. niloticus*, spring 2000, respectively) (X 400).

(E): nodular proliferation in gill filaments (*T. zillii,* summer 2000) (X 100).

(F): intravascular haemolysis and dilation in the blood vessels of gill filaments (*O. niloticus*, winter 2001) (X 400).

(G): edema in secondary lamellae (*O. niloticus,* winter 2001) (X 100).

Fig. (3): Sections of gills and liver of fish showing:

(A) & (C): separation of the epithelium of the secondary gill lamellae from the lamellar supporting cells and haemorrhage between gill filaments (*O. niloticus,* spring 2000) (X 100 & 400, respectively).

(B) & (D): separation of the epithelium of the secondary lamellae from the lamellar supporting cells (*S. schall,* summer 2000 and *T. zillii,* winter 2001, respectively) (X 400).

(E): hepatocytes of control *Oreochromis niloticus, Tilapia zillii* and *Synodontis schall* (X 400).

Fig. (4): Sections of liver of fish showing:
(A): coagulative necrosis (*O. niloticus*, spring 2000) (X 400).
(B): thrombosis in hepatoportal blood vessels (*T. zillii*, spring 2000) (X 400).
(C): thrombosis in hepatic blood vessels (*O. niloticus*, summer 2000) (X400).
(D): intravascular haemolysis and dilation in hepatoportal blood vessels (*T. zillii*, summer 2000) (X 100).
(E): intravascular haemolysis and dilation in hepatic blood vessels (*O. niloticus*,, winter 2001) (X 100).
(F): dilation of central vein (*O. niloticus*, spring 2000) (X 400).

Fig. (5): Sections of liver of fish showing:
(A): destruction of hepatoportal blood vessels (*O. niloticus*, spring 2000) (X 400).
(B) & (C): necrosis between the hepatocytes (*O. niloticus*, spring 2000 and winter 2001, respectively) (X 400).
(D): haemorrhage and aggregation of inflammatory cells between the hepatocytes (*O. niloticus*, spring 2000) (X 400).
(E): haemorrhage between the hepatocytes (*O. niloticus*, winter2001) (X 400).
(F): dilation and congestion in blood sinusoids (*T. zillii*, spring 2000) (X 400).

Fig. (6): Sections of liver and kidneys of fish showing:
(A): vacuolar degeneration of the hepatocytes and haemolysis in blood sinusoids (*S. schall*, spring 2000) (X 400).
(B): vacuolar degeneration of the hepatocytes (*O. niloticus*, spring 2000) (X 400).
(C) & (D): vacuolar degeneration of the hepatocytes (*S. schall*, spring 2000 and *O. niloticus*, autumn 2000, respectively) (X 400).
(E) & (F): extensive vacuolar degeneration of the hepatocytes (*S. schall* and *O. niloticus*, winter 2001, respectively) (X 400).
(G): kidneys of control *Oreochromis niloticus, Tilapia zillii* and *Synodontis schall* (X 400).
(H): aggregation of inflammatory cells between the renal tubules (*S. schall*, spring 2000) (X 400).

Fig. (7): Sections of kidneys of fish showing:
(A): edema in Bowman's capsules (*O. niloticus*, autumn 2000) (X100).
(B): extensive vacuolar degeneration in the epithelium of renal tubules and dilation of the capillary tubes of renal tubules (*O. niloticus*, winter 2001) (X 400).
(C): dilaton of the capillary tubes of renal tubules (*T. zillii*, summer 2000) (X 400).
(D): congestion of the capillary tubes of renal tubules (*S. schall*, autumn 2000) (X 400).
(E): haemolysis and aggregation of inflammatory cells between renal tubules (*O. niloticus*, spring 2000) (X 400).
(F) & (G): severe haemolysis between renal tubules (*O. niloticus* and *S. schall*, respectively, winter 2001) (X 400).
(H): depletion in the haemopoietic areas (*T. zillii*, spring 2000) (X 400)

Fig. (8): Sections of kidneys and testes of fish showing:
(A): intravascular haemolysis in renal blood vessels and over population of glomeruli (*T. zillii*, winter 2001) (X 400).
(B): necrosis between the renal tubules (*O. niloticus*, winter 2001) (X 400).
(C): vacuolar degeneration in the epithelium of renal tubules (*O. niloticus*, spring 2000) (X 400).
(D): necrosis between the renal tubules (*O. niloticus*, summer 2000) (X400).
(E): testes of control *Oreochromis niloticus*, *Tilapia zillii* and *Synodontis schall* (X 400).

Fig. (9): Sections of testes of fish showing:
(A) : coagulative necrosis (*O. niloticus*, winter 2001) (X 400).
(B) : necrosis (*O. niloticus*, summer 2000) (X 400).
(C) & (D): degenerative and necrotic changes in the cellular elements of seminiferous tubules (*O. niloticus*, winter 2001 and spring 2000, respectively) (X 400).
(E): degenerative and necrotic changes in the cellular elements of seminiferous tubules, focal areas of necrosis and sperms between the seminiferous tubules (*T. zillii*, summer 2000) (X 400).

Fig. (10): Sections of testes and ovaries of fish showing:

(A) & (B): seminiferous tubules with a lesser number of sperms or lucent (*O. niloticus*, spring 2000 and *S. schall*, autumn 2000, respectively) (X400).

(C) & (D): fibrous capsules around seminiferous tubules (*O. niloticus*, winter 2001 and autumn 2000, respectively) (X400)

(E): ovaries of control *Oreochromis niloticus*, *Tilapia zillii* and *Synodontis schall* (X100).

(F) & (G): separation of the follicular layers from the oocytes (*O. niloticus*, spring 2000 and *S. schall*, summer 2000, respectively) (X400).

(H): thickening of the ovarian wall (*T. zillii*, winter 2001) (X400).

Fig. (11): Sections of ovaries of fish showing:

(A): collapsed and abnormal irregularly shaped oocytes (*O. niloticus*, spring 2000) (X400).

(B): haemorrhage between the oocytes (*T. zillii*, spring 2000) (X400).

(C): aggregation of haemosiderin between the oocytes (*O. niloticus*, winter 2001) (X400).

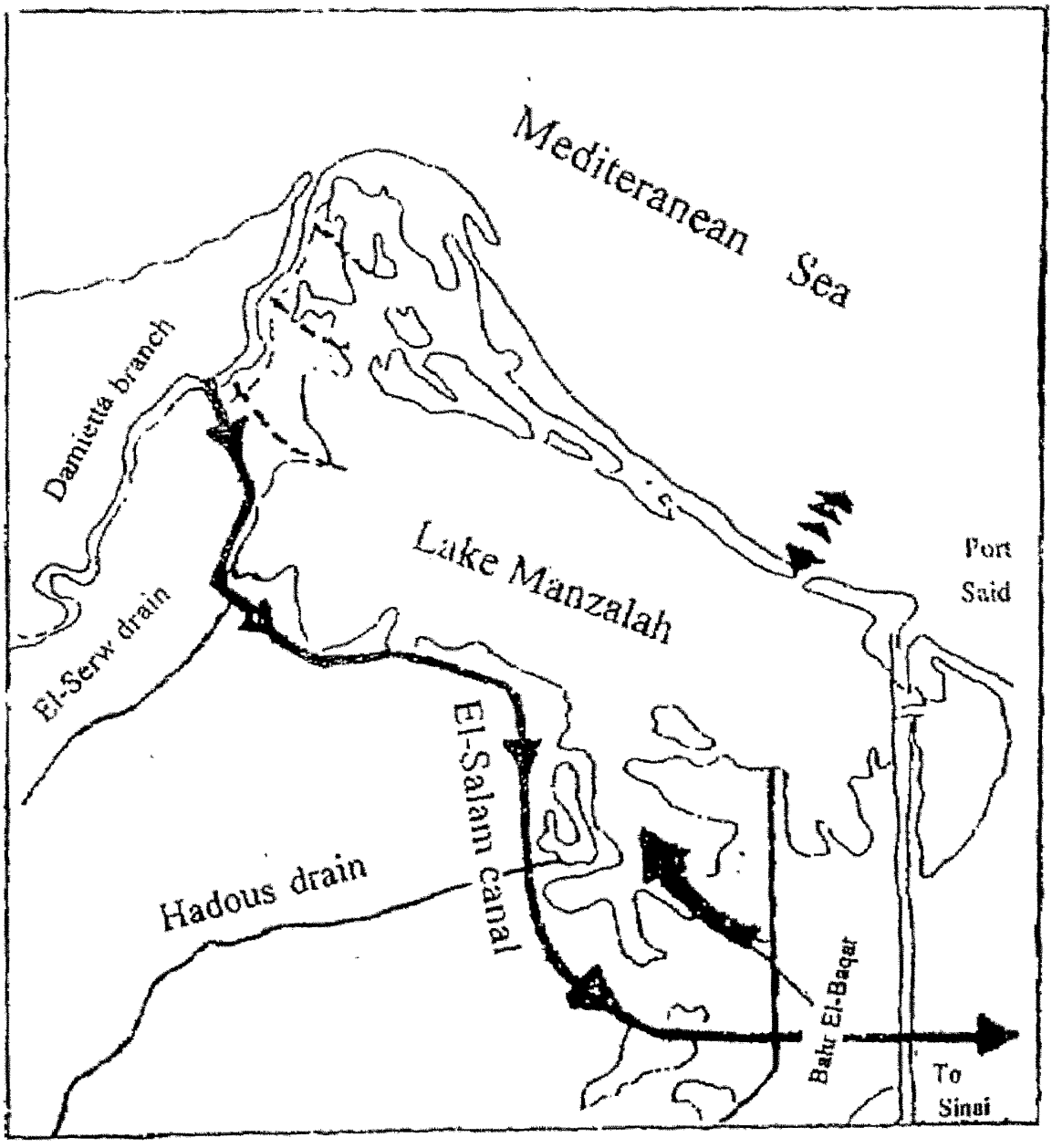
(D) & (E): proliferative changes in the granulosa of the oocytes, resulting in adhesion of the cellular coat of the oocytes (*T. zillii*, spring 2000 and *O. niloticus*, winter 2001, respectively) (X400)

Fig. (12): Sections of ovaries of fish showing:

(A): degenerative and necrotic changes in the oocytes (*O. niloticus*, winter 2001) (X400).

(B): degenerative changes in the oocytes (*O. niloticus*, spring 2000) (X400).

(C): thrombosis in ovarian blood vessels (*O. niloticus*, winter 2001) (X400).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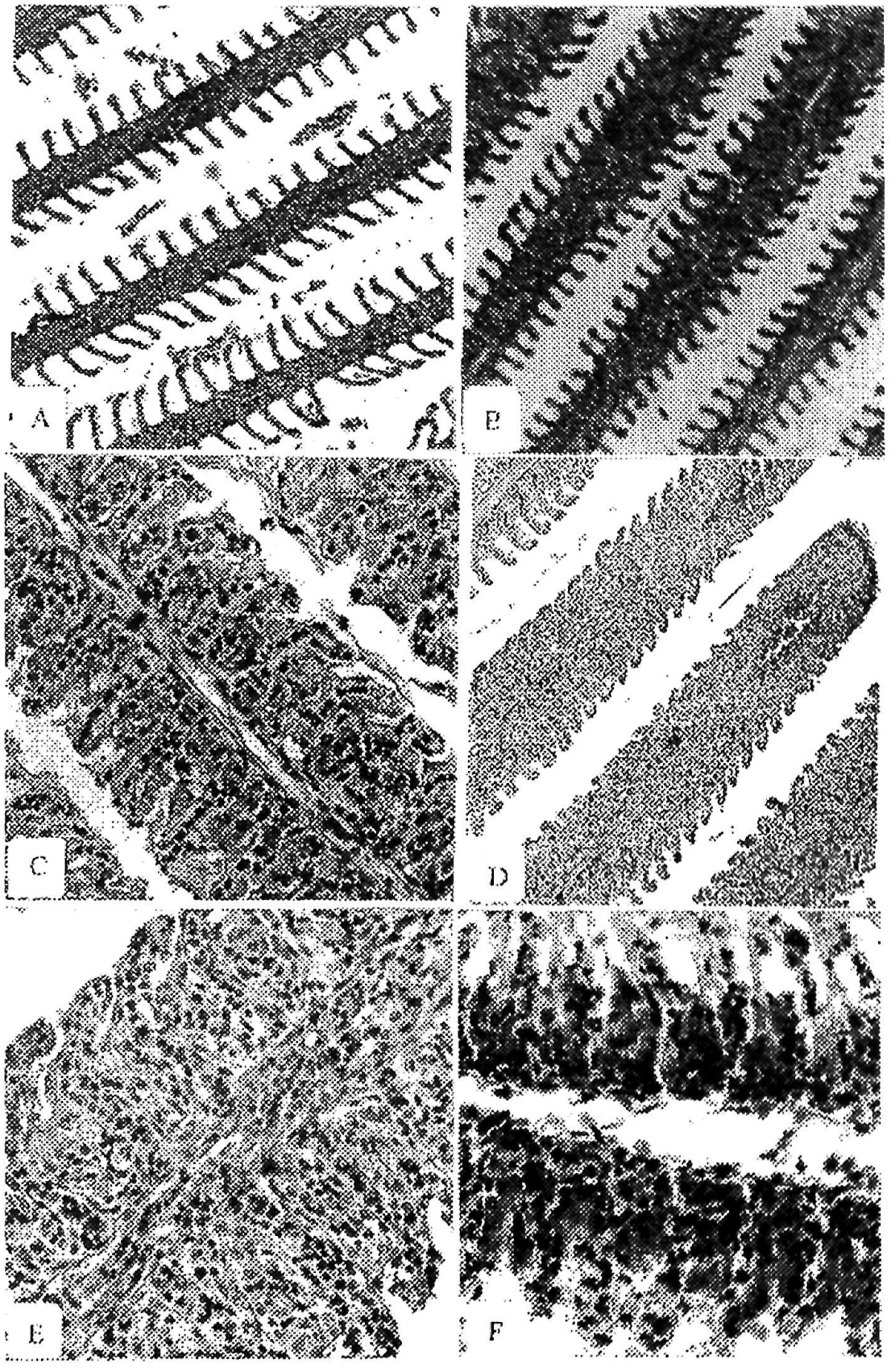

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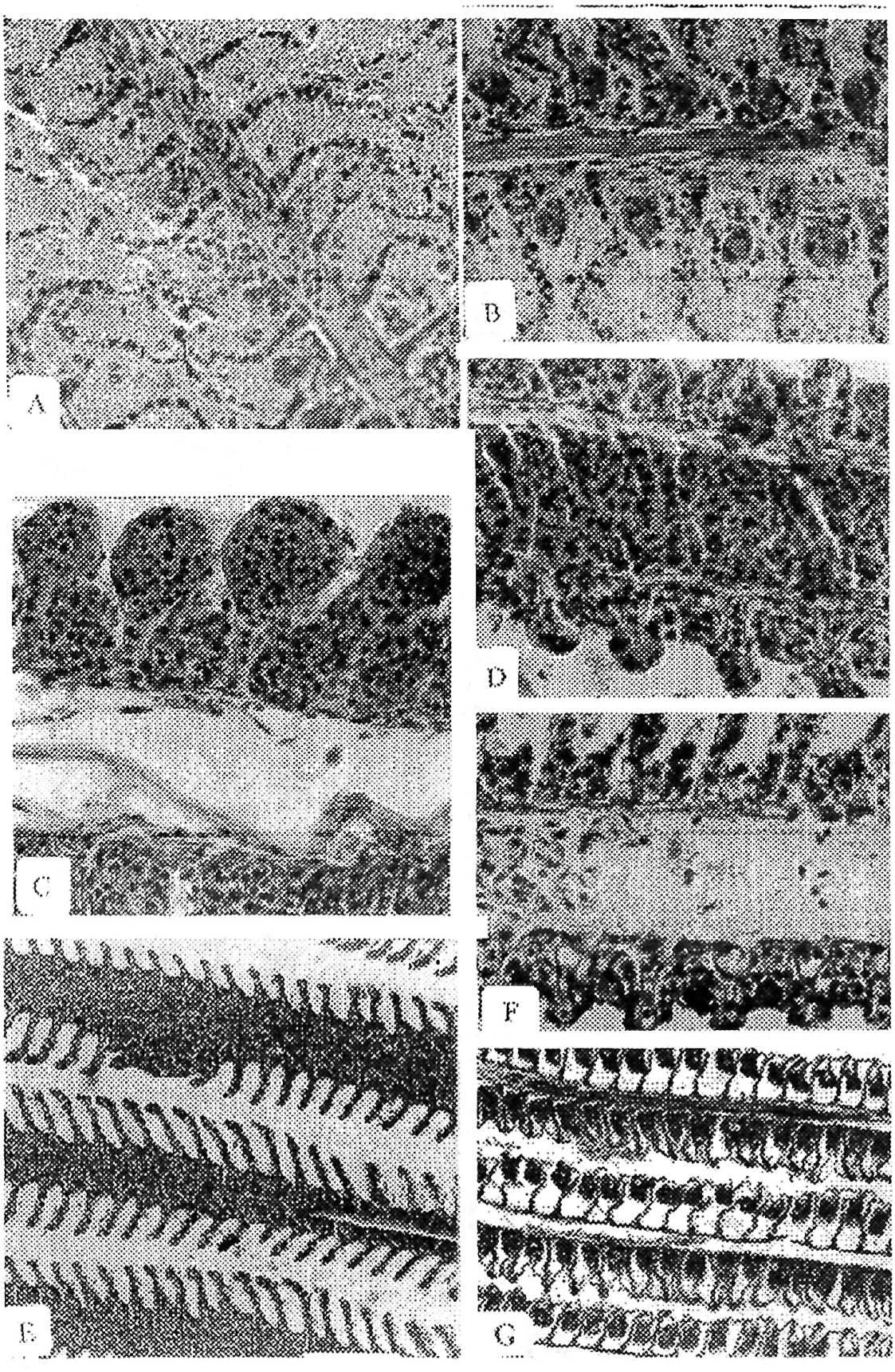

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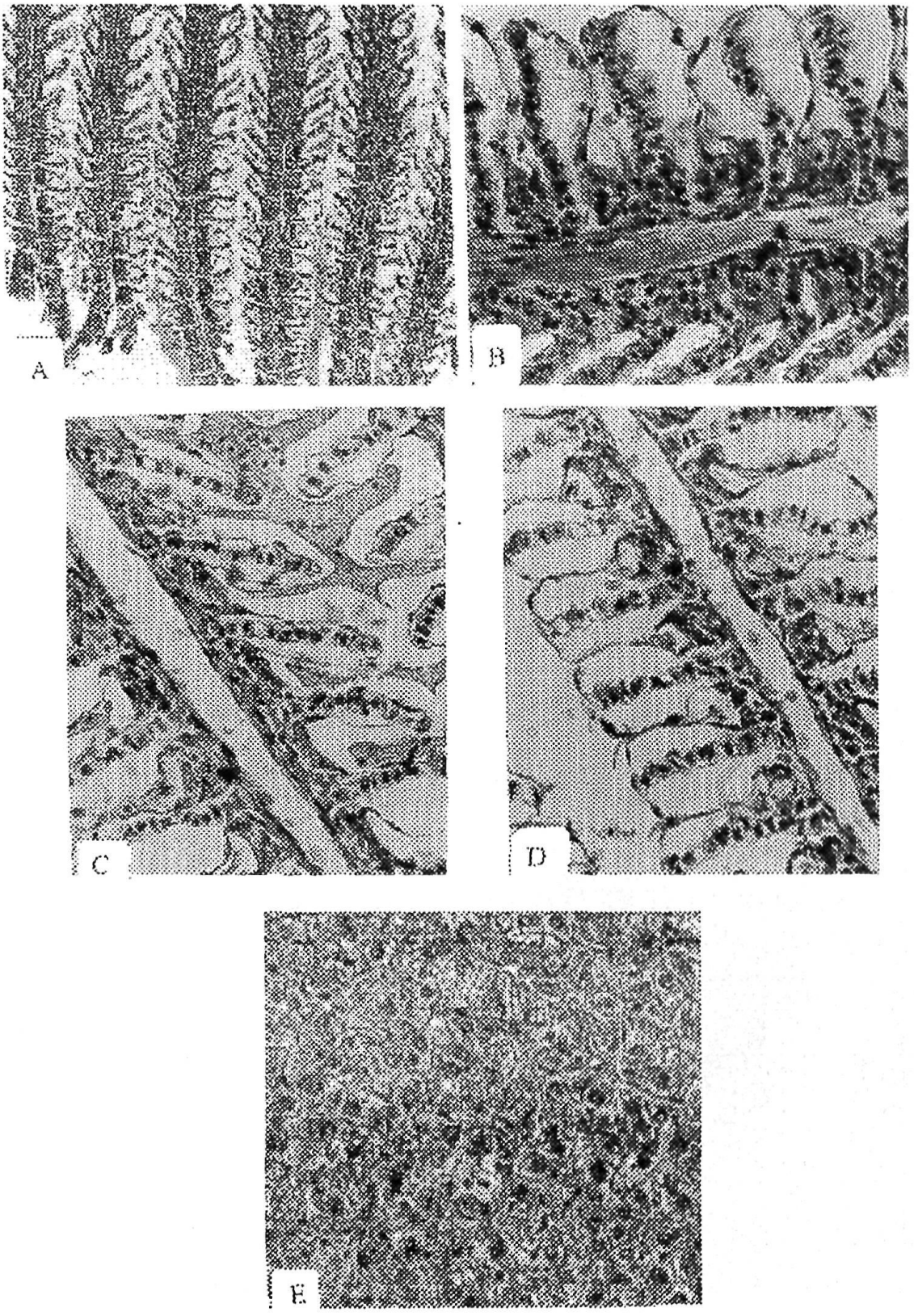

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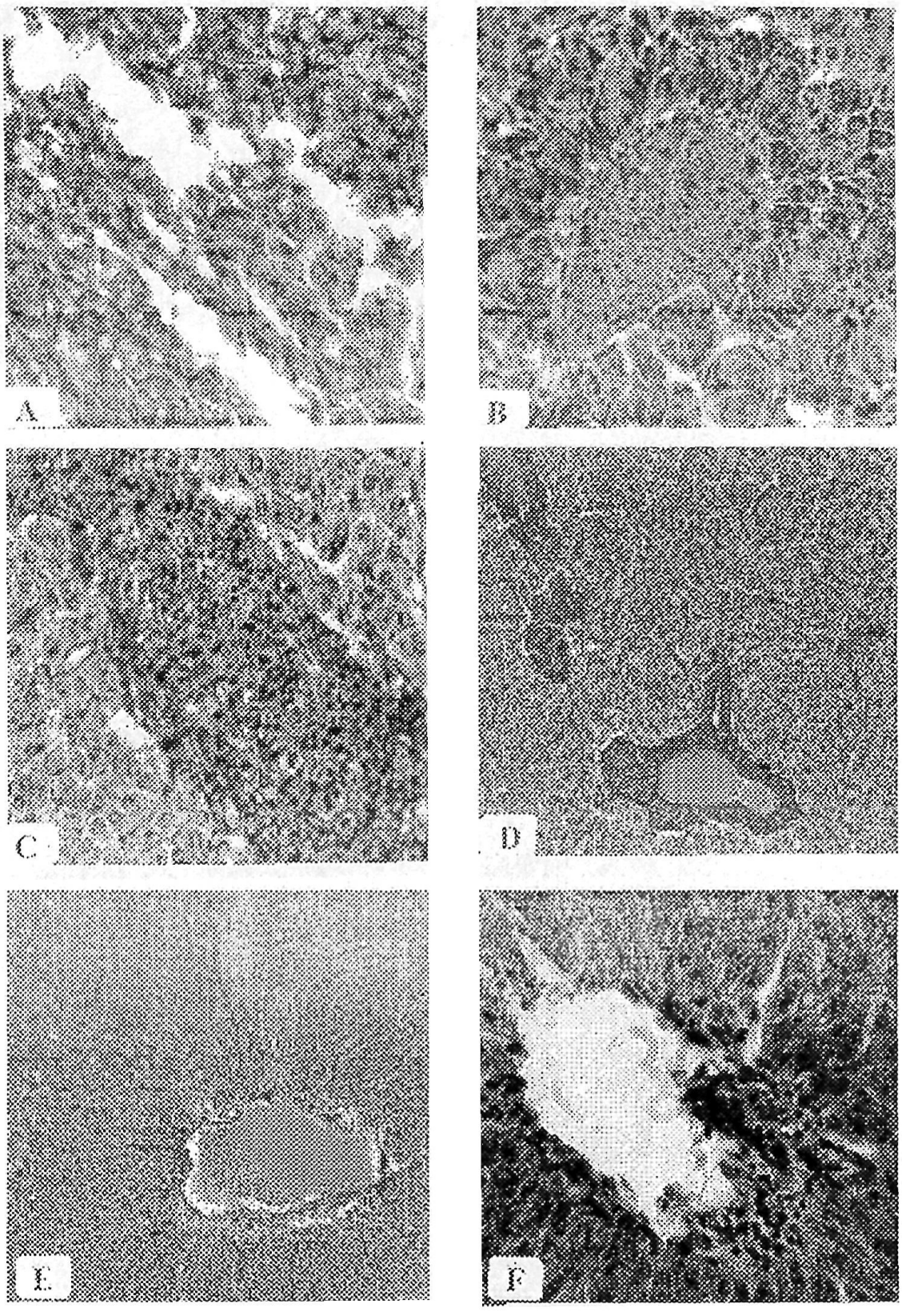

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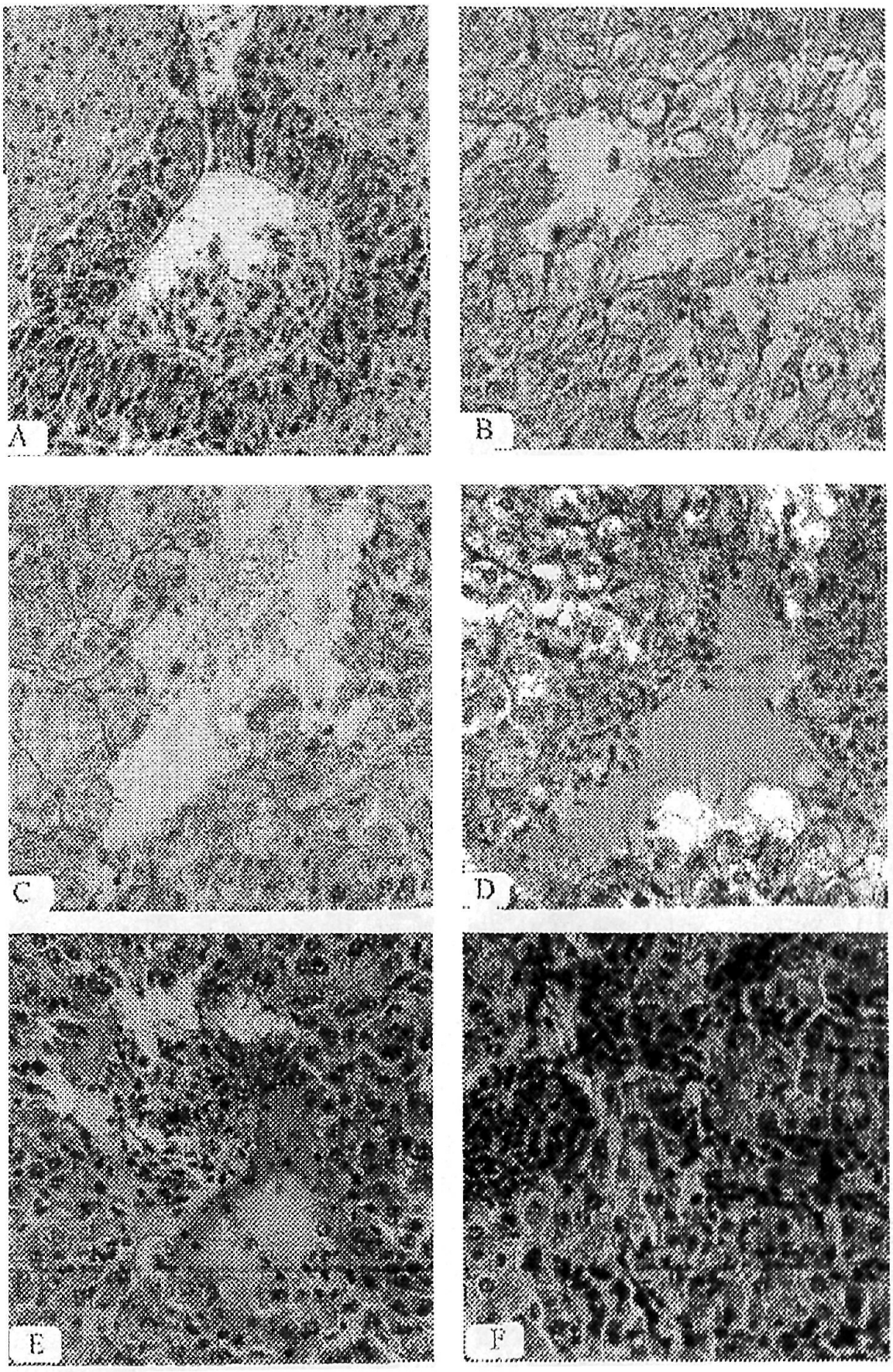

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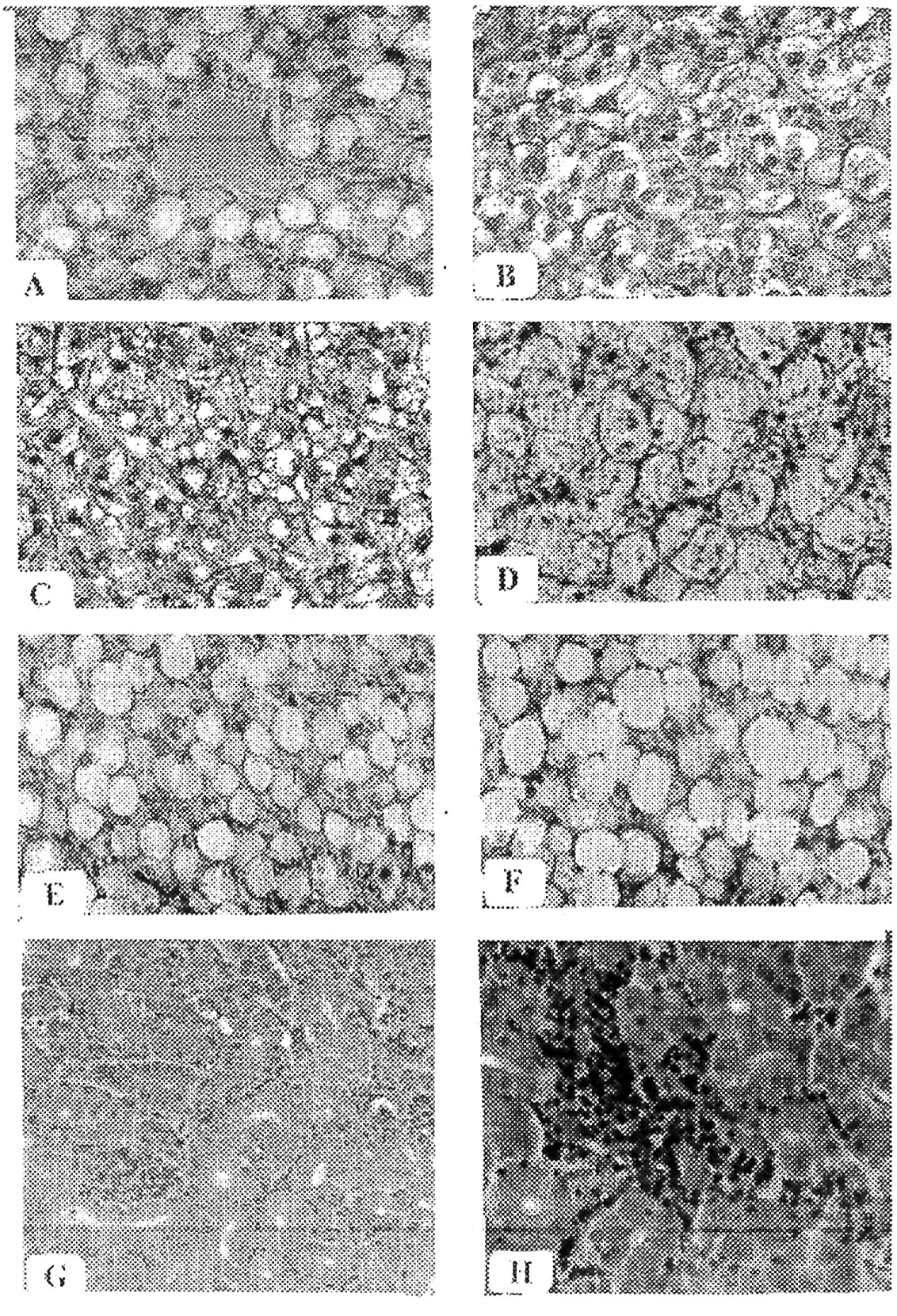

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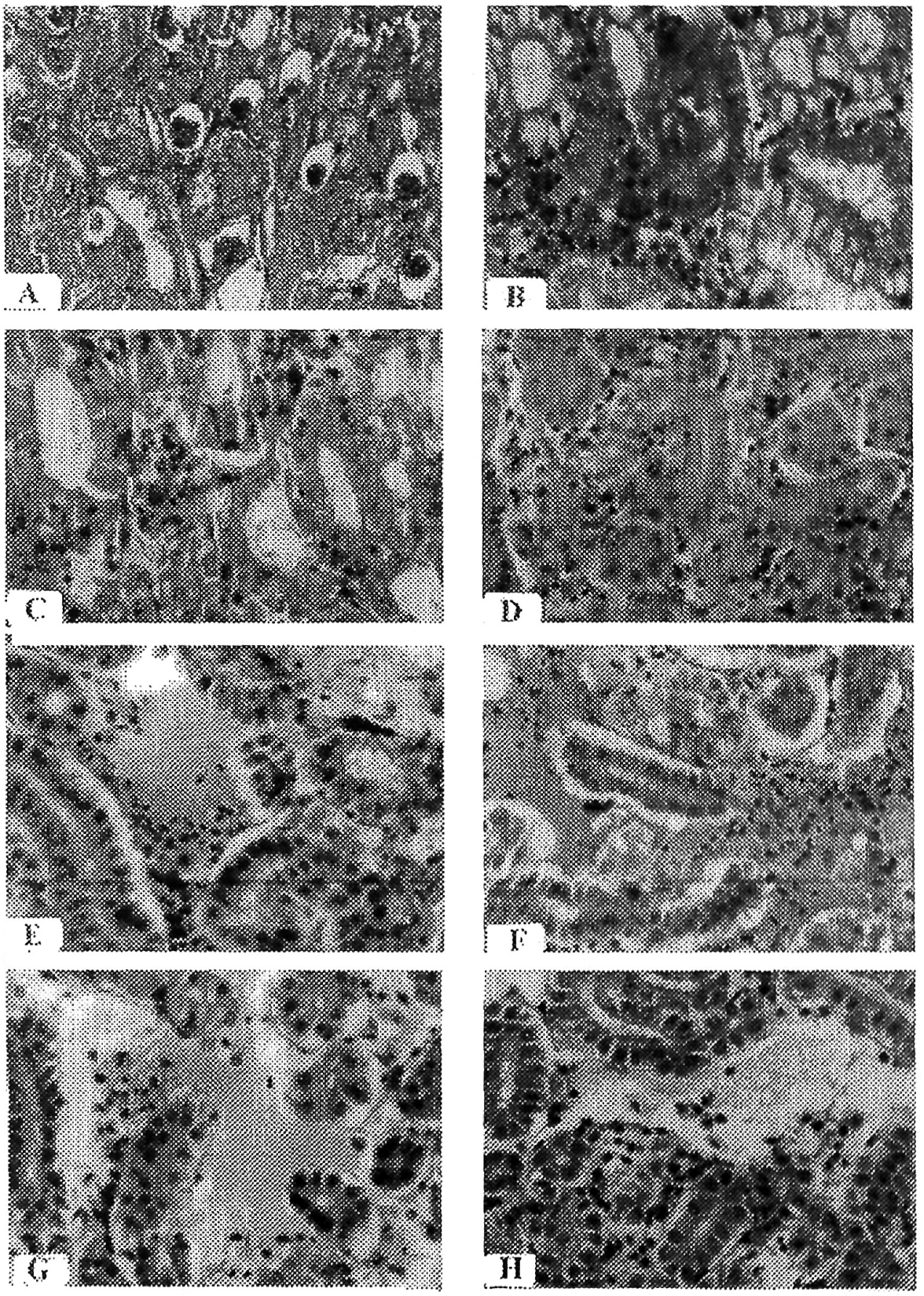

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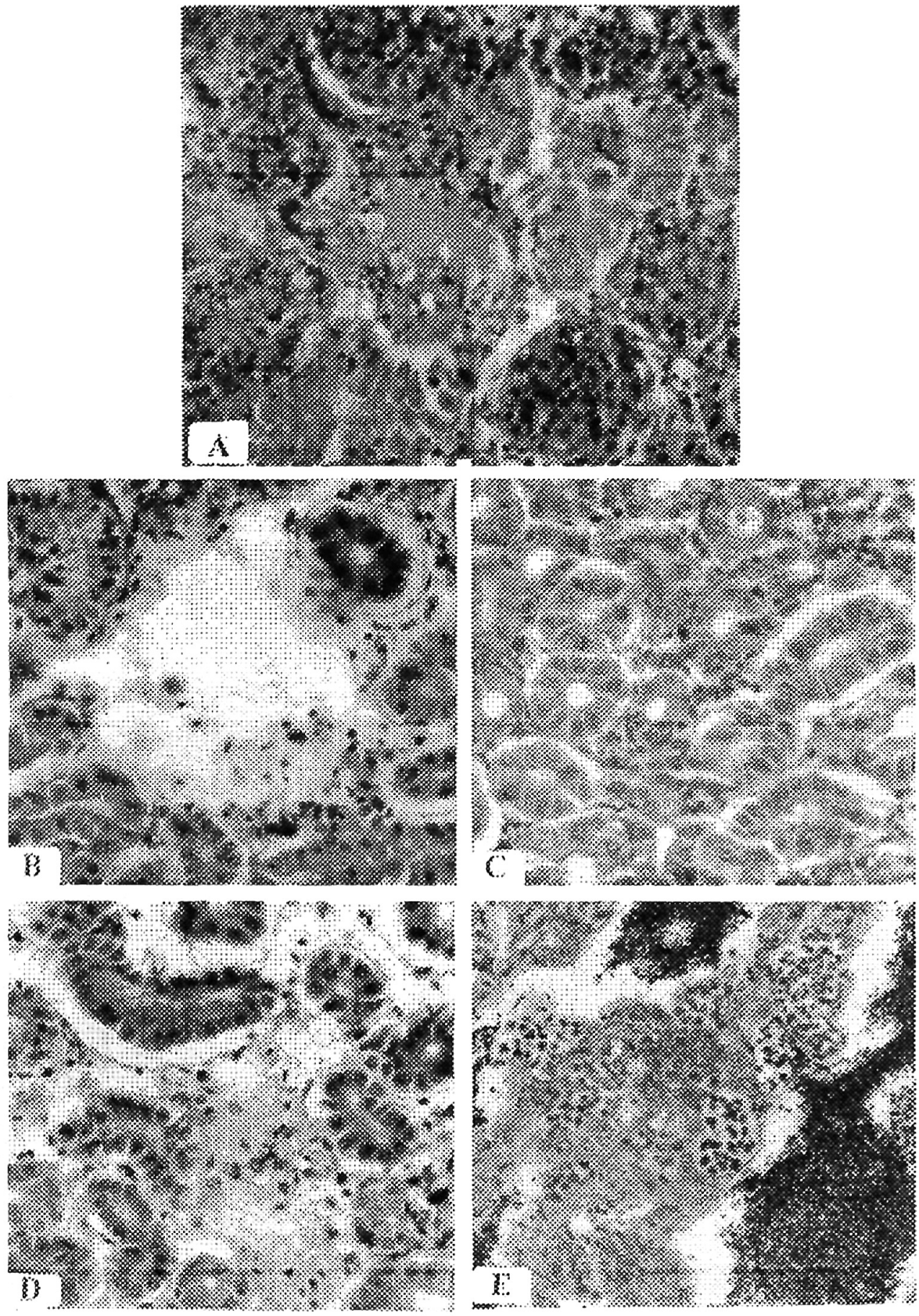

Fig.(8)

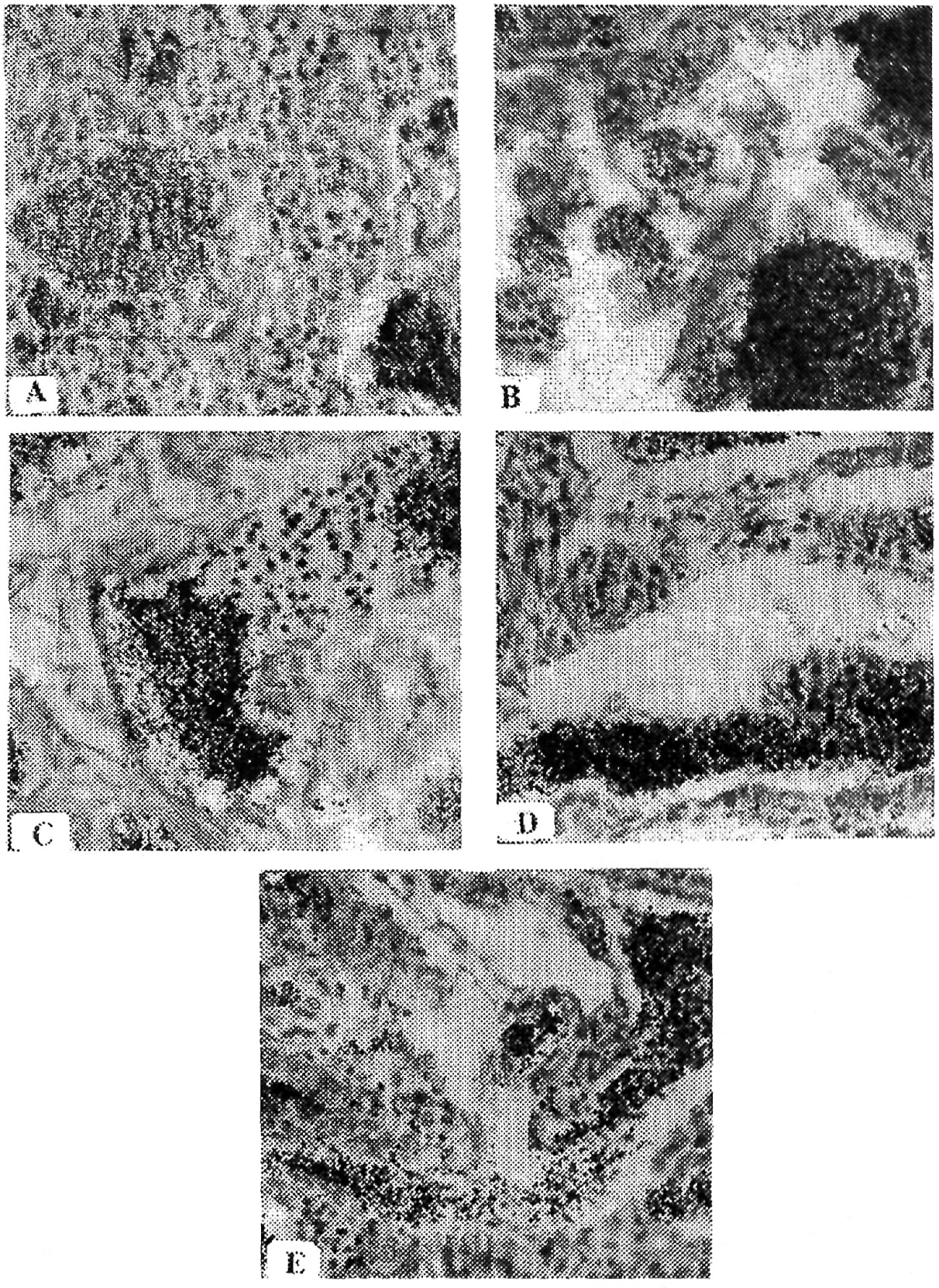

Fig.(9)

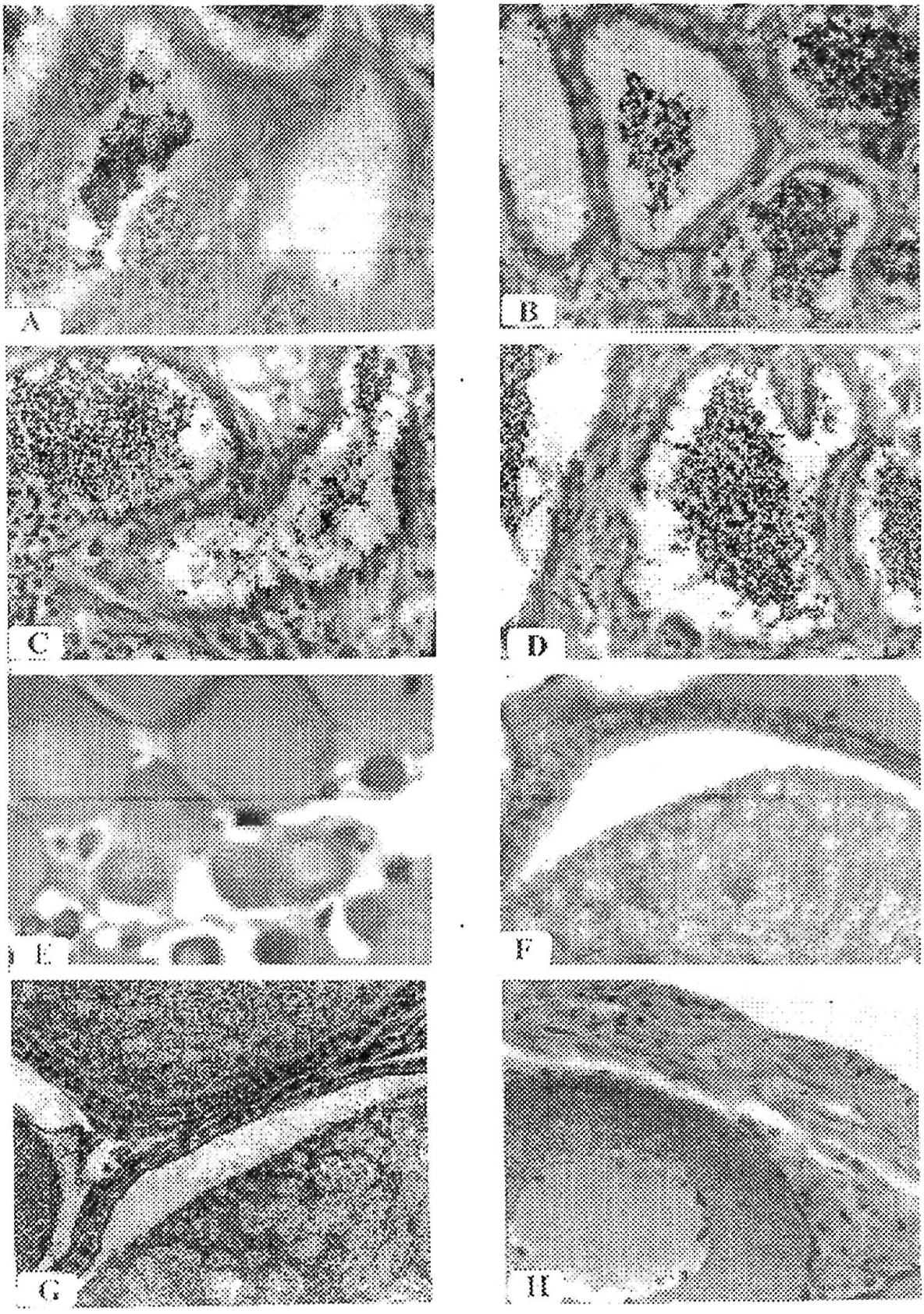

Fig.(10)

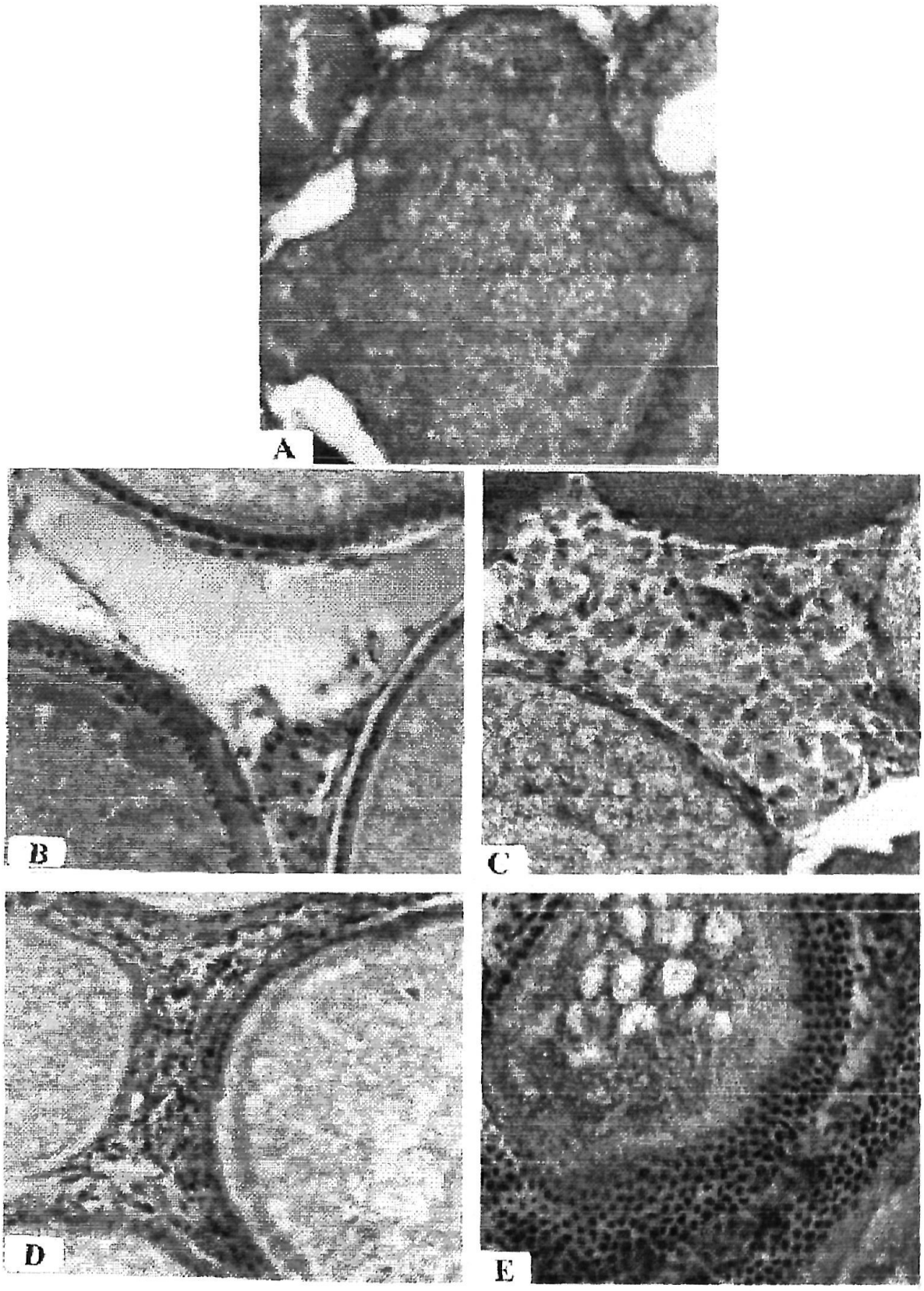

Fig.(11)

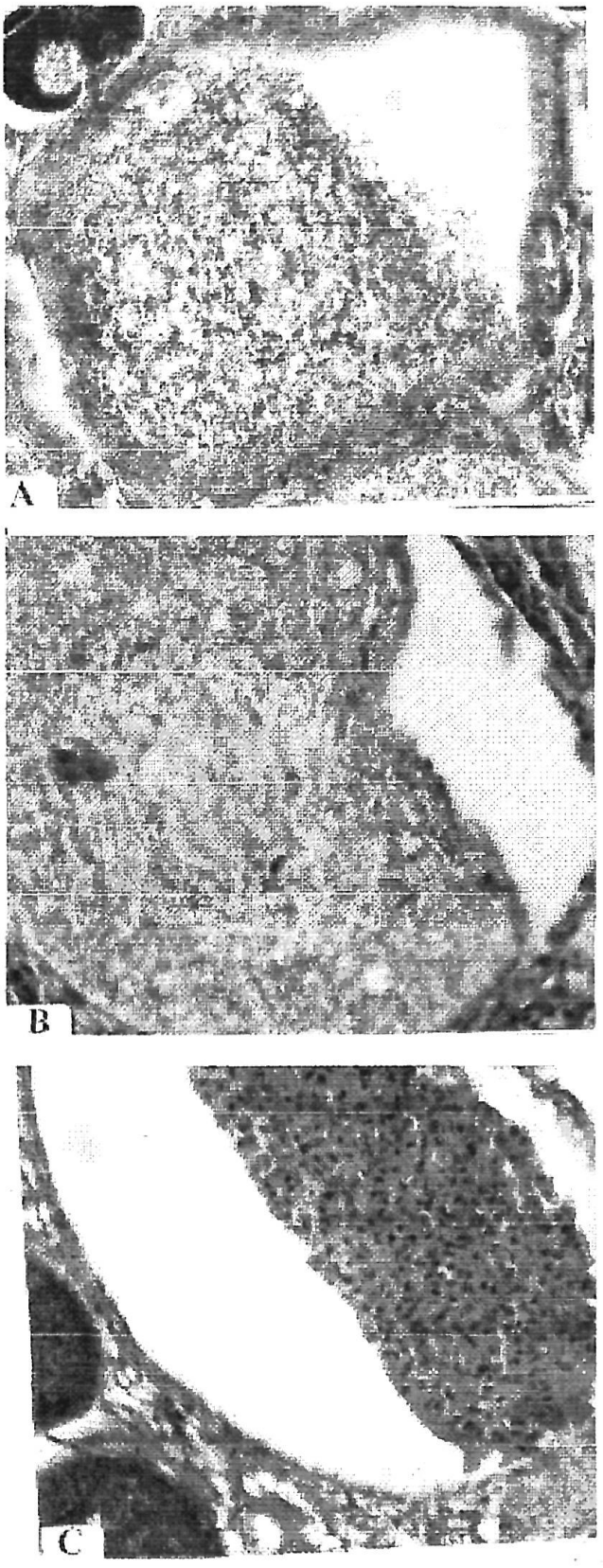

Fig.(12)