بسم الله الرحمن الرحيم

شبكة المعلومات الجامعية

التوثيق الالكتروني والميكروفيلم

شبكة المعلومات الجامعية

جامعة عين شمس

التوثيق الالكتروني والميكروفيلم

قسم

نقسم بالله العظيم أن المادة التي تم توثيقها وتسجيلها
علي هذه الأفلام قد أعدت دون أية تغيرات

يجب أن

:حفظ هذه الأفلام بعيدا عن الغبار

في درجة حرارة من 15-25 مئوية ورطوبة نسبية من 20-40%

To be Kept away from Dust in Dry Cool place of 15-25- c and relative humidity 20-40%

EFFECT OF TANNING PROCESSING WASTE WATER ON PHYSIOLOGICAL CHARACTERISTICS OF *SOLEA* SP.

Thesis

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By

Olfat Malik Wahbi Sedrak

National Institute of Oceanography and Fisheries

SUPERVISED BY

Prof.Dr Ismail A.Sadek

Professor of Experimental Zoology

Head of Department of Zoology

Faculty of Science

Alexandria University

Prof. Dr. Soheir E. M. Khadre

Professor of Cytology and Histochemistry

Faculty of Science

Alexandria University

Prof. Dr. Magda I. Zaki

Professor of Fish Reproduction

National Institute of Oceanography and Fisheries, Alexandria

Prof. Dr. Farag. K. Aziz

Professor of Experimental Embryology

Faculty of Science

Alexandria, University.

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INTRODUCTION

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The rapid development of industry and especially chemical industry has created serious problems of water pollution. The more industrial development the more wastes produced and the more destruction of environment. In most instances these toxic wastes command high price when properly packaged, yet they are discharged into sewer outlet and dumped into the sea.

When chemical pollutants were dumped into sea they entered a dynamic system. They were not only diluted and dispersed by storms, winds and currents, but became involved in the complexities of the biological food web of the sea and may be reconcentrated by the marine biomass. Microorganisms may act directly on various chemical constituents in the water. Marine animals feed on the plants and may concentrate these chemical substances still further, predatory animals can concentrate these chemical constituents. If the final species were eaten by man who received the combined chemical concentrates at the end of the food chain.

Most of these toxic agents affect all living things nutritional, reproductive, respiratory, genetic and a variety of other metabolic activities of the organisms may be seriously altered or destroyed.

The position of aquatic animals living in water containing a toxic substance was different instead of receiving by mouth or ingestion a

certain absolute quantity of this substance. It is continually exposed to a certain concentration of the poison rather than absolute dose required to harm or kill. Animals had the power to eliminate poisons at least in some degree or to destroy them and whether they can do this sufficiently fast to survive, this must depend on the concentration of toxic substances to which they were exposed and to the animal's metabolic processes. If it was not metabolically destroyed or eliminated, it will accumulate in by time. A fatal dose will be build up, it may be as long as the animals normal life span.

The toxic substances pose special problems in environmental toxicology many of them were highly lipophilic and were not adversely affected by water. These substances accumulate in fish fatty tissues or became protein bound, thus when finally were eaten by man he will receive a dosage which may be thousands of time greater than that present in environment. There is a growing belief by a great number of medical investigators that chronic degenerative diseases of man may be caused by environmental intoxicants.

Action of pollutants on fish

(1) Pollutant may kill therefore by a combination of chemical and physical injuries rather than by true toxic action. Pollutant combined with the mucous secreted by the fish skin, mouth and gills forming insoluble compounds that block interlamellar spaces, death supervenes very rapidly from the respiratory failure. This respiratory failure resulted from either preventing the blood from reaching respiratory cells of the gills, the aeration of the blood with its accompanying gas exchanges was prevented or impairment of fillament movement so blood could not

drive through gill capillaries and sooner or later death followed from a comoination of anoxemia and CO_2 retention.

Other pollutants which did not form precipitates with mucous secretion on gills produced either direct damage to gill filament cells which in turn cause stasis of blood in gill capillaries and death followed from anoxemia and circulatory failure or impairment of other functions specially those of salt balance and excretion as gills are regarded as an excretory organ in addition to its respiratory function.

(2) Pollutants may enter the body of the fish and exerting true toxic action, absorbed through the gills, the mouth lining, the skin to the gastrointestinal tract damaging its lining or may be absorbed from the gastrointestinal tract and gain access to internal organs.

I. Histological study on the effect of pollutant on fish organs

It has been established by various workers that histological investigations were of great importance in estimating toxic influence of pollutants on fish. Sangalong and O'Halloran(1972), Oronsaye (1989) working on effect of cadmium on brook trout and stick leback *Gasterosteus aculeatus* found cytological breakdown of kidneys, gill tissue and testicular injury. Ammonia toxicity studies had presented histological evidence of gill epithelial damage including hyperplasia and fusion of secondary lamellae, in *Tilapia aurea*, (Redner and Stickeney 1979). Köhler and Holzel (1980) investigated the health condition of flounder and smelt by inspecting liver, kidney, spleen and gastro-intestinal tract, they recorded the influence of toxic industrial compounds on the health of both species. Goodman *et al.*, (1982) recorded pathological

changes in hepatic cells attributed to Kepone poisoning in sheepshead minnow. Zaki and Saad (1982) studied the lethal and sublethal effects of raw and treated tannery wastes using fish bioassay. They found that exposed fish had swollen and dark red gills. Mitchell and Cech (1983) studied the mixed effect of ammonia and monochloramine on channel catfish *Ictalurus punctatus* and found gill hyperplasia with fusion of secondary lamellae. Chevalier et al.,(1985), Norragren et al.,(1986) and Leino et al., (1987) working on brook trout *Salvelinus fontinalis* pearl dace and brown trout *Salmo trutta* stated that acidification disclosed gill lesions characterized by hyperplastic primary lamellar epithelia, diminished respiratory lamellar surface area and chloride cells hyperplasia in secondary lamellar epithelium. Hamza *et al.*, (1985) studied the lethal and sublethal effects of industrial effluents containing high levels of heavy metals on *Tilapia zillii* organs. Experimentally exposure of English sole *Parophrys vetulus* to interactive effects of cadmium, polychlorinated biphenyls and fuel oil caused hepatocellular necrosis including Karyomegaly and necrosis of intestinal musoca with mucous cell hyperplasia were recognized by Rhodes *et al.*, (1985). Disrupted gonadal maturation has been reported in conjunction with exposure to chlorinated and organophosphate pesticides (Saxena and Mani 1985). Cross *et al.*, (1986) in their work on contaminated sites near Los-Angeles California reported increased atresia of early stages oocytes in white croaker *Genyonemus lineatus*, also, Susani (1986) discussed the effects of contaminants on teleost reproduction and found that exposure to xenobiotic compounds was believed to have a detrimental effect on ovarian development and spawning, increased follicular atresia of both yolked and previtellogenic oocytes, abnormal yolk deposition and yolk formation within oocytes. Brule (1987), Mazhar et al.,(1987a), Stott et al.,

(1988) Abdel Wahab (1990) and Khadre (1990), Shabana (1991) reported histopathological lessions in gills, extensive ova atresia and low gonadosomatic index, fatty infiltration of liver parenchyma cells, and necrotic foci in intestinal mucosa and musculosa as effect of oil exposure. A retardation in gonadal development was reported by Khan (1987) after chronic exposure to petroleum hydrocarbons on two species of marine fish. Johnson *et al.*, (1988) carried out a histological observation on contaminant effects on ovaries of English sole *Parophrys vetulus* from Puget sound, Washington and they found ovarian atresia, lowered mean gonadosomatic index and reduced gonadal recrudescence. Braunbeck *et al.* (1989) recorded pathological changes attributed to nitrophenol poisoning in liver of zebra fish. Byrne *et al.* (1989) showed that as zephiran concentration and or time exposure increased gills were severely spongiotic and necrotic and there were much lamellar fusion in rainbow trout *Salmo gairdneri*. Light and scanning electron microscopic studies on the effect of plant sap on gills of hill stream fish *Garra lamta* (Ham.) was carried out by Ojha *et al.* (1989). Acute and chronic exposure of *Fundulus heteroclitus* to tributylin compounds revealed hypertrophy of lamellar epithelium in acute concentrations of toxicants with no pathological changes in gills of fish exposed to sublethal concentrations in chronic test (Pinkney *et al.*. 1989).

Injection of sublethal dose 500 mg/kg of hexachlorobutadiene (HCBD) caused cytoplasmic vacuolation and necrosis in renal tubules (Reimschuessel *et al.*,1989). Sulaiman *et al.* (1989) carried out histopathological studies by light microscope on gill, liver, and gut. Malathion exposed fish indicated abnormalities such as hyperplasia, hypertrophy and degeneration of hepatocytes.

Brown *et al.*, (1990) also Jagoe and Haines (1990) dealt with the influence of acid and lime water on gills of Atlantic salmon *Salmo salar* and found hyperplasia and hypertrophy of chloride and mucous cells as a response reaction for increasing ionic uptake to offset the losses of ions during low pH stress.

Aflatoxin B_1 at acute and chronic toxicity in channel catfish caused histological lesions included sloughing of intestinal mucosa and necrosis of haematopoietic tissues, and hepatocytes and pancreatic acinar cells, (Jantrarotai *et al.*, 1990 and Lovell& Jantrarotai 1990). Different combinations of pH and aluminium caused white blood cell infiltration of the lymphatic space of gill tissue, and presence of dense cells in lamellar epithelium in brook trout as mentioned by Ingersoll *et al.*, (1990) and Tietge *et al.*, (1988).

Khadre (1990) studied the structural changes in Tilapias culture due to copper toxicity.

Examination of gill histology in Atlantic salmon and brook trout from acidic streams of southwest Nova Scotia showed localized epithelial hypertrophy and hyperplasia with clubbing tips of secondary lamellae (Lacroix *et al.*, 1990). The same result was detected by Freda et al., (1991) on exposing rainbow trout *Oncorhynchus mykiss* to different levels of acidity. Kjesbu *et al.*, (1991) carried out a histological observation of atresia resulted from captivity in Atlantic cod *Gadus morhua* a number of different phases were distinguished based on the shape and location of the chorion (zona radiata), proliferation of the follicle cells and breakdown of the yolk granules. The central area of the oocytes showed degenerative

process, the yolk was resorbed and there were large numbers of different vesicles present in the cytoplasm.

Khadre (1991) studied the cytochemical localization of heavy metals in some tissues of *Tilapia zillii* inhabiting lake Mariut, also, Khadre (1992) investigated the cytology and cytochemistry of liver of *Tilapia zillii* surviving in Lake Mariut, and proved a degenerative changes. Munkittrick *et al.*, (1991), Adams *et al.*,(1992), Munkittrick *et al.*, (1992) studied the response of fish and communities to bleached Kraft Mill effluent (BKME) and found that it caused reproductive disfunction and recruitment failure, fish had reduced gonad sizes, delayed age to maturation, lower fecundity, ovaries contained a large number of atretic oocytes, kidney showed distention of Bowman's space, glomerular swelling and distention due to deposits of hyaline material. Roy and Munshi (1991) showed the harmful effect of malathion concentrations on gills secondary lamellae of *Cirrhinus mrigala* (Ham.), causing inflammatory alterations and hyperplasia.

Examination of skin of air-breathing catfish *Saccobranchus fossilis* after exposure to 5.6 mg/L chromium for 7 days revealed increased number of mucous cells having a flask or cylindrical shape instead of polygonal normal shape (Khangarot and Tripathi 1992). Marlasca *et al.*, (1992) subjected rainbow trout to sublethal effects of synthetic dyes and found hyperplasia of primary lamellae and fusion of secondary lamellae.

Wahbi (1992) recorded the acute effect of different concentrations of Abou Qir industrial effluents on developmental stages of *Sparus auratus* and found it caused severe lesions in gills, liver, intestine and kidney.