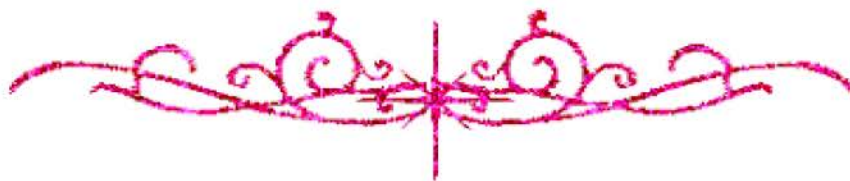


بسم الله الرحمن الرحيم





شبكة المعلومات الجامعية التوثيق الالكتروني والميكروفيلم



جامعة عين شمس

التوثيق الإلكتروني والميكروفيلم

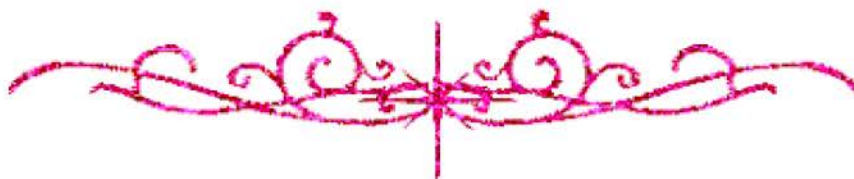
قسم

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



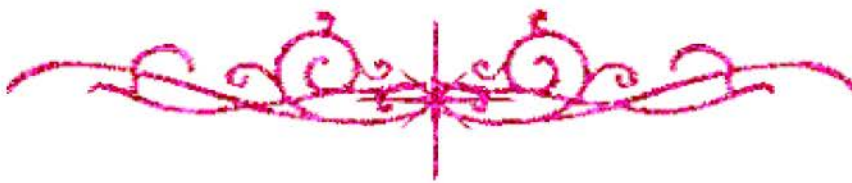
يجب أن

تحفظ هذه الأقراص المدمجة بعيدا عن الغبار



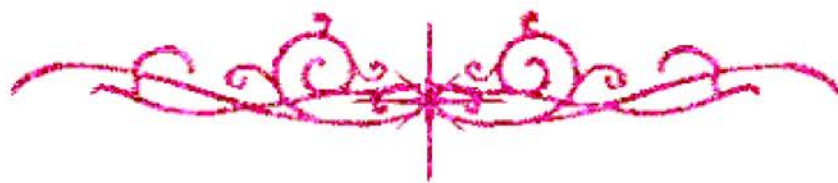


بعض الوثائق الأصلية تالفة





بالرسالة صفحات
لم ترد بالأصل



B1WCA5

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

Thesis

Submitted to the Faculty of Science

Zoology Department

Alexandria University

For

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INTRODUCTION

INTRODUCTION



*T*he 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 filament movement so blood could not

drive through gill capillaries and sooner or later death followed from a combination of anoxemia and CO₂ 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. Sangalang 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 Stickney 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 lesions 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 tributyltin 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.