



شبكة المعلومات الجامعية

بِسْمِ اللَّهِ الرَّحْمَنِ الرَّحِيمِ



شبكة المعلومات الجامعية
@ ASUNET



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

تحفظ هذه الأفلام بعيدا عن الغبار

في درجة حرارة من ١٥-٢٥ مئوية ورطوبة نسبية من ٢٠-٤٠%

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

بعض الوثائق الأصلية تالفة

بالرسالة صفحات لم ترد بالاصل

Cairo University
Faculty Of Veterinary Medicine
Department Of Physiology

Syehmar

**EFFECT OF SOME ENVIRONMENTAL
STRESSORS ON GROWTH, DEVELOPMENT
AND BEHAVIOUR OF EXPERIMENTAL ANIMALS**

22.7.98

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ACKNOWLEDGMENT

I wish to express my deepest gratitude to **Professor Dr. ESMAT A. SEIF EL NASR**, the head of department of physiology , Faculty of Veterinary Medicine , Cairo University , for her constant encouragement and supervision as well as her highly appreciated advice throughout the work .

Grateful thanks are extended to **Professor Dr. NABIL A. HEMEIDA** , Professor of Theriogenology , Faculty of Veterinary Medicine , Cairo University for his valuable suggestions , constant supervision and encouragement throughout the work .

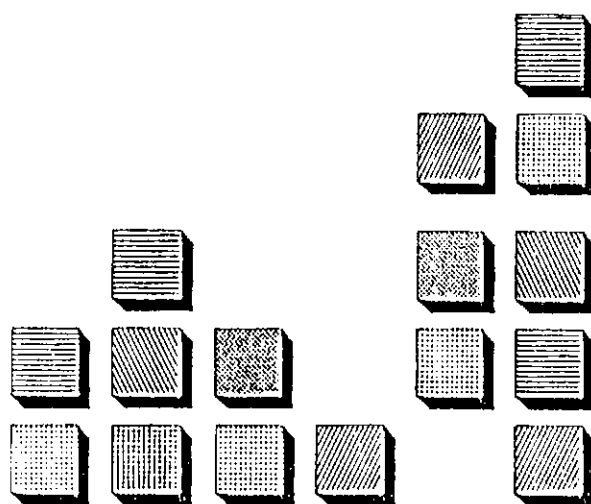
Deep appreciation is expressed to **Professor Dr. YOUSSEF L. AWAD** Professor of Biochemistry , Animal Health Research Institute , Dokki - Cairo , for his valuable help, valuable suggestions and assistance during the work .

My thanks are extended to **Dr. NADIA A. TAHA** , Asst. Professor of physiology, Faculty of Veterinary Medicine , Cairo University, for her help, kindness and encouragement during the work .

It gives me a great pleasure to record my great thanks to **Professor Dr. AHMED A. EL. KARIM** , the head of Air Pollution Department in the National Research Center, and to **Dr. OMA YMA M. KANDEEL**. Senior Researcher at Animal Reproduction Department in the National Research Center.

My appreciation is also being expressed to all colleagues in Animal Health Research Institute specially to **Dr. GEHAN GAMEL** , Senior Researcher in the Pathological Department, Thanks are also to **Dr. GAMAL A. EL HAMID** Asst. Professor in Department of Pharmacology , Faculty of Veterinary Medicine, Cairo University .

INTRODUCTION



by autoexhaust or insecticides .

Insecticides cause a marked environmental pollution particularly in the farm vegetables ; residues may be toxic to livestock , in addition they cause many toxic effects to many farm workers and to general public. Manifestations of this toxicity are represented by delayed neurotoxicity, mutagenicity, teratogenesis and carcinogenesis (Durham and Williams,1972; Gupte et al .,1985 and Courtney et al.,1986). Insecticides may be carried in many tissues of human and animals ,which cause many troubles affecting growth ,development and pregnancy (Uphous et al.,1985 and Simmon ,1986).

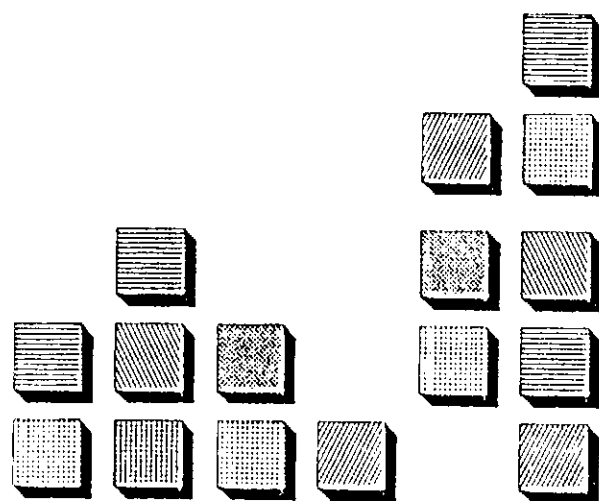
The exhaust gas induces stressing action similar to the classical activation of the pituitary adrenal axis with increase of relative adrenal weight and the rate of corticosteroid production . After chronic exposure, deep metabolic changes appear which reflect accentuated state of exhaustion . Moreover, interruption of spermatogenesis with azoospermia is noted . Vianna et al.(1984) reported that exposure of fathers to autoexhaust may be of etiologic importance for leukaemia in children . Thus, exhaust gas could be considered as a very potent toxic agent (El Feki et al.,1984) .

The present investigation was conducted to study the effect of insecticides and autoexhaust on rats. In order to record their effects the following physiological monitors were used :

- * Clinical symptoms and mortalities.
- * Growth .

- * Maternal behaviour .
- * Haemogram and hormonal assays.
- * Reproductive and developmental toxicity.
- * Gross and microscopic examination of visceral and sexual organs as well as some endocrine glands.

REVIEW OF LITERATURE



REVIEW OF LITERATURE

I. PYRETHROID INSECTICIDE

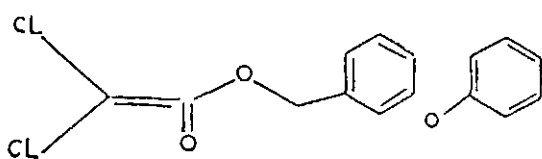
Pyrethrum is a contact insecticide obtained from the flower head of *Chrysanthemum cinerariae* Folium. The varieties grown in high lands of Kenya yield the highest proportions of active ingredient. It is also grown in Caucasus, Iran, Japan, Ecuador and New Guinea (Harley and West, 1974). Pyrethrum was produced as insecticide since 1950. Jacobson and Cresby (1971), and Woods (1974) reported that it has a rapid toxicity to flying insects combined with a very low mammalian toxicity due to its ready metabolism to non toxic compounds. Martin and Worthing (1974) and Schreck et al. (1978) reported that domestic pests such as cockroaches, mosquitoes, biting flies and house flies are very susceptible to Pyrethroids. Also Nolan et al. (1979), found that Pyrethroids have considerable potential for veterinary pests as *Boophilus*, *Microcephalus* *Stomoxys*, *Calcitrans*, *Haemotobia* Irritants and *Musca* *Autummlis*.

Natural pyrethrins were modified in order to obtain derivatives with a better biological performances. The production of synthetic pyrethroids started with allethrin and cylothrin around 1950 (Brent, 1967; Wouters and Van Den Bercken, 1978). These early compounds, however, still lacked sufficient stability and they are less effective against many species of insects than the natural products. The total yearly production of allethrin

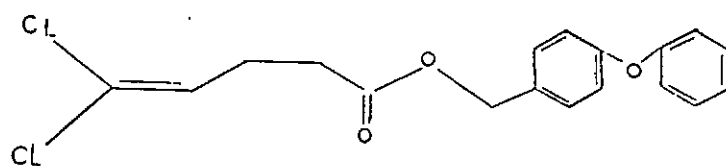
is very high specially with the development of more widespread application of pyrethroids.

Synthetic pyrethroids act directly on the axon through interference with sodium channel gating mechanism that reduce the generation and conduction of each nerve impulse. Pyrethroids only appeared to affect the sodium current during depolarization. However, once the sodium channel is opened, the activation gate was restrained in the open position by the pyrethroid molecule (Flannigan and Tuckpr, 1985). Lund and Narahashi (1983) reported that alfa cyanopyrethroids primarily affect the sodium channels in the nerve membrane and cause a long lasting prolongation of the transient increase in sodium permeability of the membrane during excitation.

In rats cis - permethrin is more stable than trans - permethrin, cis- compound yields 4 fecal ester metabolites in the fat and liver that retained the highest radiocarbon levels (Loretta et al., 1977), and in hens the peak permethrin equivalent was found in the skin.



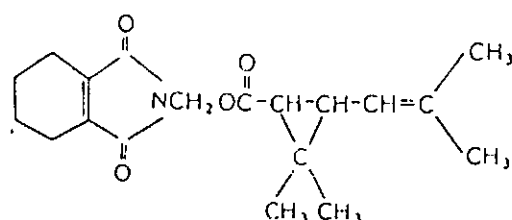
Trans-permethrin



Cis-permethrin

Tetramethrin is less toxic than cis-pyrethrins. All necessary toxicological data support

their safety and have been obtained at world leading research institutes. Its toxicity is 4640 mg/kg (L D 50) in rats (Hazelton laboratories , Inc., USA) .



Tetramethrin

Pyrethroids and behaviour:

Casida (1973) noticed that rats suffered from pneumonia after spraying cages with pyrethrins sesam oil solution . Garratt and Bigger (1923) and Ramiriz (1930) found that some individuals suffered from asthma due to exposure to pyrethrum mixture . Pyrethrins caused muscular fibrillation in addition to signs of toxicity of other insecticides , from excitation to convulsion and tetanic paralysis (Chevalier and Ripert,1927).Martin and Leog (1966) noticed that when rats were exposed to 1% pyrethrin (2mg/ liter) for one hour , no animal died although they appeared hyperexcitable and exhibited extreme hostility towards each other . These signs disappeared in 24 hrs, gross examination showed that tissues were normal except for pin point hemorrhages in the lungs.Wallwork et al. (1974) reported that when groups of mature female mice received oral doses of permethrin in corn oil daily at 0,200,400,800 or 1600 mg / kg body weight for 10 days, signs of acute toxicity such as spasm and convulsions were noticed in the highest dose group , half of which died after the initial dose .

Blair and Roderick (1976) concluded that male and female rats exposed to cypermethrin as 400 g/liter by inhalation for 4 hrs and thoroughly soaked during exposure, the female showed abnormal gait with urinary incontinence. Butterworth and Hend (1976) observed persistent tremors in rats fed permethrin at 3000 mg/kg body weight. Hend and Butterworth (1976) observed that when male and female rats were fed cypermethrin at a dietary concentrations of 0,25,100,400 or 1600mg/kg body weight for 3 months , signs of intoxication as hypersensitivity and abnormal gait were observed in the 1600 mg/kg body weight group,during the first 5 weeks one died .There were increased plasma concentrations of urea ,alkaline phosphatase activity and RBCs count in females , no effect was found in the 100 mg /kg body weight group.Killeen and Rapp(1976)noticed that when male and female rats were fed permethrin at doses of 0, 20, 100 or 500 mg/kg body weight diet for 90 days showed no mortality and tremors at the highest dose level in the first week. Meteker (1978) concluded that when rats were fed permethrin in diet for 12 days at dose levels of 54, 108, 216 , 432 , 864 or 1728 mg/kg body weight per day, there were muscle tremors in all animals at 432 mg/kg body weight but dose of 216mg/kg or less caused no toxic signs in either male or female.

When male and female rats inhaled permethrin in concentrations of 125 , 250 or 500 mg/m³ 6 hr / day for 13 week , tremors and convulsions occurred in rats at 500mg/m³ during the first week of exposure, but disappeared in the second week (Meteker,1978). Gombe and Odourokelo (1983) reported that when young dogs (6-12 months) were fed for 90 days a diet to which pyrethrum (10, 25,50 or 100 mg/kg body weight) were added, resulted ataxia,