

STUDIES ON RESISTANCE OF COSTON LEAFWORM
TO CERTAIN INSECTICIDES

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to study the phenomenon of resistance in the cotton leafwor from different points of view namely :-

- a) The relative toxicity of some organophosphorus insecticides recommended by the Egyptian Ministry of Agriculture
  to control this insect, to resistant, field and susceptible strains.
- b) The enzymatic activity of the cholinesterase (ChE),
  Aliesterase (AliE), Alkaline and acid phosphatase in the
  previously mentioned insect strains.
- c) The effect of the organophosphorus insecticides on in vivo enzymatic activity in the same strains.
- d) The penetration of certain organophosphorus insecticides in resistance, field and susceptible strains.
- e) Comparative studies of qualitative and quantibative amino acid contents in different strains.

#### II. REVIEW OF LITERATURE

# A. Toxicity and cross resistance of organophosphorous insecticides.

Darrow and plapp (1960), found that a colony of <u>Culex</u> tarsalis Coq., 60 times resistant to Malathion was found to be resistant to Malaoxon and the diethyl homology of Malathion, although to a lesser degree. Slight resistance to Co-Ral was also observed, but no resistance was found to any other of Demeton, Dipterex, Delnav, Parathion and Paroxon. Two-three fold resistance to DDT and Dieldrin was also found.

Hanna (1964), studied the development of resistance of the cotton leafworm <u>Spodopters littoralis</u> to Toxaphene in Egypt. He found that Giza strain was the most susceptible, while Behera strain proved to be the most tolerant one.

Heavel and Cochran (1965), found that dosage mortality data, obtained by a topical application method, for 8 organophosphorus compounds given for 2 laboratory selected strains of <u>Blatella germanica</u> L., revealed that development of resistance to Malathion was highly specific. Imparting essentially no cross resistance to any of the other 7 organophosphorus compounds evaluated. Selection with

Diazinon induced a low level of tolerance to all the organophosphates studied.

Tabano and Brown (1966) concluded that selection with Dieldrin increased the LD<sub>5C</sub> of the Rangoon, Douala and Fretown strains of <u>Gulex pipiens Matigans</u>, up to 5 p.p.m. marking a 40 fold increase for the initially more susceptible Freetown strain. When examined for cross resistance, the first three strains were found to have increased there LC<sub>5C</sub> to -BHC by 3-10 times. There was no change in the LC<sub>5C</sub> levels to Diazinon, Fenthion and was an increase in susceptibility to Malathion, Parathion and Methyl parathion.

Atkisson (1968) found that the tobacco budworm, Heliothis virescens F., in Texas developed a high degree of resistance to Endrin and Company: during the period 1963-65. During the period 1961-65 the LD<sub>50</sub> value for Endrin was increased from 0.06 mg/g of larva to 12.94 mg/g (200 fold) and for Carbaryl from 0.3 mg/g to 54.47 mg (180 fold). He suggested that the development of resistance to Endrin might be a logical sequence to the earlier development of resistance to DDT. However, it might be possible that the usage of Endrin, although limited, over the 9-year period 1954-63 was sufficient to cause the development of resistance. But it seemed unlikely that the limited usage of Carbaryl during the 4-year period 1959-63 would be sufficient to cause the obtained high degree of resistance

to that compound. More likely, Carbaryl was a victim of the circumstance that the resistance to carbaryl probably was an example of cross-resistance between the carbamate and chlorinated hydrocarbon insecticides.

Carter and Fhillips (1968) determined Methyl parathion resistance capabilities of a laboratory population of Heliothis zea (Boddie). Results indicated that the response of the bollworn to Methyl parathion had progressed through vigor tolerance, periods of increases in LD<sub>50</sub> and decreases in slope of the ld-p line, to a period showing a sharp increase toward homogeneity and a marked shift to the ld-p line to the right.

Champ et al. (1970) compared the responses of susceptible and resistant strains of Tribolium castaneum (Duv.) found in peanuts and cereals in storage when exposed to insecticide-impregnated filter paper. They found that the resistant strains from peanuts showed tolerance to Dichlorovos (X 3.3), Fenitrothion (X 3.8), Gardona (X 227), Diazinon (X 11.4), Dursban (X 4.7), DDT (X 2.2) and Carbaryl (X 12.5). There was no significant change in tolerance to Bromophos, and a significantly lower tolerance to Iodofenphos (X 0.7).

Hassan et al. (1970) found that there was a seasonal variation in the sensitivity of the different strains of

They found that the cessation of the use of Toxaphene caused relaxation of resistance to both Toxaphene and Endrin, but few generations of chemical pressure produced highly tolerant strains to both insecticides. They also mentioned that examination of the tolerance levels to various organophosphoses indicated a greater toxicity for compounds with diethyl group as compared with the diethyl group compounds.

El-Guindy (1973) found that selection for Endrin resistance to S. littoralis was associated with cross resistance to Toxaphene, Dieldrin and Zectran. No cross resistance was observed to DDT, Methyl parathion, Dipterex, Nuvacron, Cyolane and Lannate. Selection for Fenitrothion resistance while confered cross resistance to DDT, Dipterex, Methyl parathion and Zectran. No cross resistance was observed to Toxaphene, Endrin, Dieldrin, Nuvacron, Cyolane and Lannate.

Moustafa et al. (1974 a) found that the response of different strains of the cotton leafworm, collected from different provinces of Lower Egypt during September 1967, to Cyolane, Sumithion, Dipterex, Matacil and Endrin varied from one province to another according to the back history of insecticides application in each province.

Moustafa et al. (1974b) stated that resistance to insecticide was likely to be developed by a cotton leafworm

insecticides. Field strains in Gharbia and Dakahlia provinces showed resistance to Endrin following extensive use of Endrin late in 1965 season and during 1966. The resistant strain of Gharbia showed very high increase in its tolerance amounting to 350 fold over that of the susceptible strain. The same behaviour was noticed in both Kafr El Sheikh and Sharkia provinces in 1966 and 1967 seasons following the use of Endrin and Dipterex where an increase of 170 and 200 fold respectively were obtained.

# B. Cholinesterase, Aliesterase, Acid and Alkaline phosphatase activity in organophosphorus susceptible and resistant strains.

Alexander et al. (1958) measured the differences in the phosphatase activity of susceptible and DDT-resistant house flies, susceptible and Chlordane-resistant cockroaches. They found that no significant differences were obtained in the acid and alkaline phosphatase activity between resistant and susceptible strains.

Bigley and Plapp (1960) studied the cholinesterase and aliesterase activity in organophosphorus susceptible and resistant houseflies. They concluded that the levels of ChE activity were found to be nearly identical in each

strain, but aliesterase activity was much lower in resistant strain than in the susceptible strain, indicating a possible relationship between this esterase and resistance.

Bigley and Plapp (1961) determined the ChE and AliE activity of different stages in the life cycle of susceptible and organophosphorus resistant strains of the house fly Musca domestica L. They found that, as in the adult, level of AliE activity were markedly less when the resistant flies were in immature stages. ChE levels were similar for all fly strains at all stages tested.

Menzel et al. (1963) studied the properties of esterases from susceptible and resistant strains of Musca domestica. They stated that their results supported the theory that resistance to organophosphate insecticides such as Malathion was related to a higher phosphatase activity.

Dresden (1965) stated that ChE activity in resistant flies was less than its activity in the susceptible ones.

Busvine (1968) reported that resistance to organophosphorus insecticides in the field produced confusing variations in intensity and cross resistance. Organophosphorus resistance in <u>Musca domestica</u>, <u>Culex tarsalis</u>, <u>Chilo suppressalis</u>, <u>Blatella germanica</u> and <u>Tribolium castaneum</u>, did not depend mainly or change of cuticular penetration or on potentiation

of phosphorethiomates; cholinesterases were not involved. Resistance was due to detoxication systems.

Whitten and Bull (1970) found that the resistant and susceptible bud worms strains appeared to have similar levels of cholinesterase activity while homogenates of resistant larvae demonstrated greater Aliesterase activity.

Abdallah et al. (1973) found that the Methyl parathion R-strain of S. littoralis contained lower AliE activity than the S-strain. The activity in the R-strain was approximately 75% of that in S-strain. On the other hand, the ChE activity was higher in the R-strain than that in the S-strain, where it was 1.45 times as high as that in the S-strain.

El-Guindy (1973) found that the enzymatic activity of Ali-E was lower in the resistant Fenitrothion strain than that in the susceptible strain of <u>S. littoralis</u>.

# G. Enzymatic inhibition in different insect strains.

Oppenounth and Van Asperen (1960) stated that a mutant gene which produced an altered ali-esterase in all of six phosphate-resistant strain of Musca domestica L. The modified enzymes were no longer irreversibly inhibited by the oxygen analogs of the insecticides to which the strains were resistant but could slowly convert them. In five of the strains the resistance was caused by this gene only.

Forgash et al. (1962) found that aliesterase activity of the resistant strains of <u>Musca domestica</u> L. was inversely proportional to the level of Diazinon resistance, this fact confirmed previous reports on other GP-resistant house flies. There were only slight differences among the strain in cholinesterase level and sensitivity to <u>in vitro</u> inhibition.

Smissaert (1964) provided evidence that organophosphate resistance in a strain of spider mites was due to decreased sensitivity of its cholinesterase to organophosphates. The cholinesterase activity of the susceptible strain in vitro was three times that of the resistant strain of mites.

Voss and Matsumura (1964) found that the Chk of two Leverkusen resistant strains of spider mites were different from the ChE of Leverkusen susceptible (Liv), Bluvelt (USA) resistant and Niagara (USA) susceptible. The last three strains did not differ significantly in their sensitivity to Paraoxan (2 x 10<sup>-3</sup> M), whereas the first two strains had a definite tendency to be insensitive. They also stated that Blauvelt resistant strain might have developed another effective defence mechanism such as detoxication and/or reduced absorption.

Smissaert (1965) reported that his work on resistant and susceptible strains of the spider mite, <u>Tetranychus</u> <u>urticae</u>, showed that the ChE of resistant mites was considerably less sensitive to Diazoxon and Paraoxon inhibition than that of susceptible mites using -naphthyl acetate as substrate.

Mansingh (1965) found correlation between the <u>in vivo</u> cholinesterase activity in the head of Malathion-poisoned German cockroaches, <u>Blatella germanica</u> L., and the resistance to the insecticides.

Jarezyk (1966) stated that the mid-gut of lepidop-terous caterpillars contained at least 2 highly active enzymes which catalyze the hydrolysis of insecticidal E605 (Parathion) related compounds in vitro at alkaline pH. The enzymes degraded esters of phosphoric and phosphorothioic acids at different rates. These were detexifying enzymes and very abundant in E 605-resistant houseflies.

Morallow and Sherman (1967) studied the toxicity, the in vitro anticholinesterase activity, and the correlation between toxicity and anticholinesterase activity of four organophosphate insecticides to four species of flies. The results of their study indicated that the most toxic compounds were not always the most effective cholinesterase