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JUVENILE HORMONE ACTIVITY OF FOUR PHOSPHONIUM COMPOUNDS IN *AEDES AEGYPTI* (L). LARVAE

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Four organophosphorus compounds were tested for their biological activity on the 4th instar larvae of *Aedes aegypti* (L). Longer exposure to higher doses of these compounds (0.21 to 4 ppm) produced morphogenetic effects commonly observed after the treatment of insect growth regulators. The activity of these test compounds, however, cannot be equated with the standard juvenoids such as dimilin and methoprene and therefore making these compounds marginal in term of use as insect growth regulators.

INTRODUCTION

The chemosterilizing activity of HEMPA (Hexamethyl phosphoric triamide) [1] against insects led to the synthesis of substituted phosphor amides, thiophosphoramides and related organophosphorus compounds for biological activity in insects. Tris (dimethylamino ethyl phosphonium iodide) has been reported as a chemosterilant by Terry and Porkovec [2].

Phosphonium salts have been evaluated for their activities as insecticides, bactericides, fungicides, anti-tumor agents and as plant growth regulators. Their biological effects against the 4th instar larvae of the yellow fever mosquito, *Aedes aegypti* (L.) has not been reported in the literature. The present paper evaluated the toxicity and morphogenetic effects induced by three phosphonium salts and one phosphorane compound on larvae of *Aedes aegypti* (L.)

MATERIAL AND METHODS

The four organophosphorus compounds tested were 1-substituted 2-ethoxy-2 alkoxyvinyl triphenyl phosphonium tetrafluroborate (P_1), carbomethoxy methylene triphenyl phosphorane (P_2), methyl (P_3) and ethyl triphenyl phosphonium iodides (P_4). Their biological activity was compared with methoprene (isopropyl 11 methoxy-3,7,11 trimethyl-2,4-dodeca-dienoate) and dimilin TH–6040 (1-(4-chlorophenyl)-3(2,6 diflurobenzoyl)- urea).

The technical samples of phosphonium compounds were serially diluted with acetone and 1 ml was then pipetted into 249 ml of water to obtain the desired concentration. The formulations of methoprene (65% a.i) and dimilin (25% WP) were diluted similarly with distilled water. Untreated controls were maintained under parameters that were identical to the treated larvae.

For each replicate twenty, 2 day-old 4th instar larvae were placed in 250 ml beakers containing the desired concentration of the chemical. The experiments were replicated four times. The larvae were left in the beakers until the adults emerged or died. All the experiments were conducted at 26±4° and 70±5% R.H. In each case total mortality of larvae, pupae, larval–pupal intermediates and pupal–adult intermediates as well as abnormal adults were recorded at each 24 hr observation period. The overall activity was assessed as percent inhibition of emergence corrected for control by the formula:

$$\% \text{ inhibition of emergence (EI)} = 100 - \frac{T}{C} \times 100$$

Where T = percent emergence in treated and C = percent emergence in control. The corrected percent values were plotted in log probit scale against concentration and LC_{50} values in ppm were determined.

RESULTS

The test compounds produced delayed effects on the metamorphosis of larvae and the continuous exposure to these compounds until the emergence of adults produced morphogenetic effects that were similar to those produced by some insect development inhibitors or juvenoids. However, the data presented in the Table 1 indicate that the

dosages required for control are considerably higher than dimilin and methoprene making these compounds marginal in terms of their use as IGR's.

Morphogenetic effects included formation of intermediates with larval characters in the head and posterior region and pupal characteristics such as free respiratory trumpets and palmate hair on the thorax. In other larvae metamorphosis was inhibited in various ways ranging from cessation of development at the pupal stage to fully formed adults which did not eclose. In many cases, death occurred after the eclosion as the adults lacked the ability of flight. They died on the surface of water after various intervals of time. Some authors have described such adults as feeble adults.

In some cases pupae that shed the larval cuticle remained unmelanised, except for the presence of pigmentation in the eye. Fig. 1 summarizes the data on mortality in larval and pupal stages treated, while the LC_{50} values, and the percent inhibition of emergence are given in Table 1.

Inhibition of Emergence. All the compounds tested resulted in delayed mortality occurring in late larval or pupal stages (Fig. 1). At higher doses of phosphonium salts (2 to 4 ppm) 70 to 80% larvae died during the larval stage, with no initiation of pupation.

Of the series of the test compounds, phosphorane (P_2) proved less effective than phosphonium salts (P_1, P_3, P_4), while ethyl triphosphonium iodide showed the maximum activity. The inhibition of emergence was in the order of: $P_4 > P_3 > P_1 > P_2$. The compounds having I_2 in their molecule proved more effective than the one having fluorine. Dimilin and methophrene, on the other hand, produced fifty percent inhibition of adult emergence at the 0.0002 and 0.065 ppm doses respectively. The mortality induced by dimilin was approximately 125 to 400 times higher than that of methoprene.

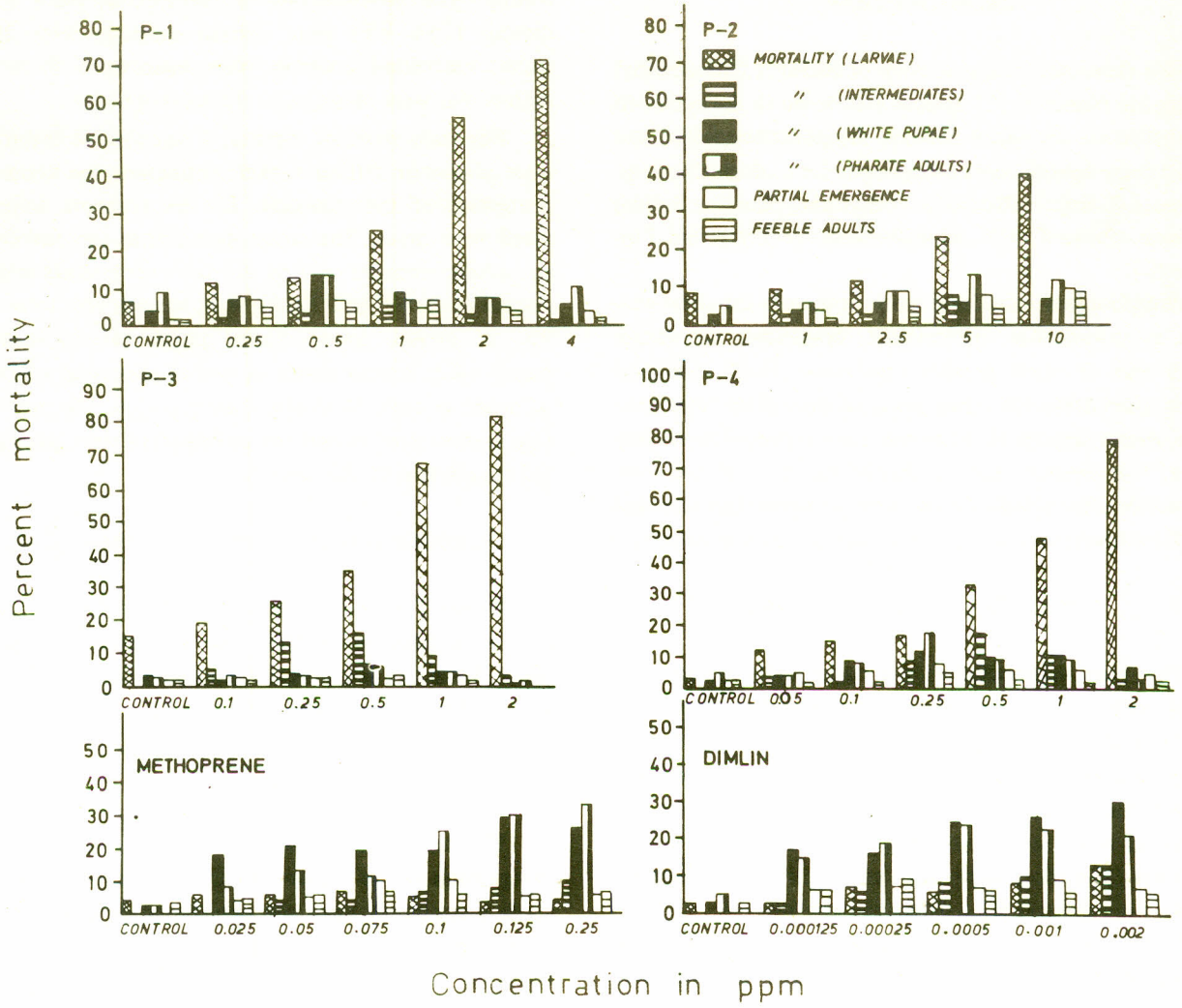

Fig. 1. Type of morphogenetic changes exhibited by organo-phosphorus compounds (P_1 – P_4), dimilin and methoprene. Continuous treatment of larvae till their emergence resulted in mortality at larval, pupal and adult stages due to morphogenetic defects.

Table 1. Effects of various organophosphorus compounds and two insect growth regulators on the percent emergence of *Aedes aegypti* (L).

	Chemicals	Inhibition of Emergence (EI) LC_{50} (ppm)
P–1	(1-substituted 2 ethoxy-2-alkoxyvinyl triphenyl phosphonium tetrafluroborate)	0.7
P–2	(Carbo methoxy methylene triphenyl phosphorane)	4.2
P–3	(Methyl triphenyl phosphonium iodide)	0.34
P–4	(Ethyl triphenyl phosphonium iodide)	0.21
	Methoprene (isopropyl-11-methoxy-3,7,11 (65% a.i) trimethyl(-2,4-dodecadienoate)	0.065
	Dimilin (1-(4-chlorophenyl-3(2,6 difluro- (25% WP) benzoyl)-urea.	0.0002

DISCUSSION

Of the series of compounds tested, the halogenated compounds (P_1, P_3, P_4) were more effective than the non-halogenated compound (P_2). Longer exposure to these compounds appear to inhibit the development of larvae into pupae and adult emergence, and some of the effects were similar to those produced by juvenoids [7] and other agents like petroleum oils [8], organophosphorus compounds [9], saturated and unsaturated fatty acids [10,11] and other lipids as well as certain antibiotics.

It may be possible that some of the compounds reported above may be causing local toxicity to the epidermal cells which subsequently produce pseudo-juvenilizing effects. Salama [12] obtained some non specific juvenile hormone effects with colchicine and some non esterified fatty acids and some long chain alcohols which had toxicity to epidermal cells. Although phosphonium compounds have no similarity with juvenoids or derivatives of farnesenic acid, the morphogenetic effects produced suggest that either these compounds are inhibitor of oxidative processes, or their biodegradation into metabolites in insect body may interfere with oxidative processes during the metamorphosis resulting in apparent JH-like activity.

When the data on mortality of larvae and pupae and the percentage of morphogenetic effects produced were compared with dimilin and methoprene, it appears that the delay in production of lethal effects by the test compounds is concomitant with the mode of action of juvenoids. However, the dose level at which the morphogenetic effects are noticed clearly indicate that virtually none of the test compounds can be equalized in their action with standard juvenoids both quantitatively and qualitatively.

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