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PHYSIOLOGICAL ACTION OF CERTAIN INSECTICIDES AND THEIR TOXINS ON ISOLATED COCKROACH HEART

A. PURUSHOTHAM RAO

Department of Zoology, Post-Graduate Centre, Vidyaranyapuri, Warangal (A. P.), India 506009

and

M. B. NAIDU

Entomology Section, Regional Research Laboratory, Hyderabad (A. P.), India 500009

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The physiological action of endrin, malathion and neurotoxins liberated by them was studied on isolated cockroach heart. In the action of malathion acetylcholine is involved and it does not paralyse the cardiac ganglia. Contrary to this malathion neurotoxin acts by paralysing cardiac ganglia and acetylcholine is not involved in its action. Endrin and its neurotoxin act at the cardiac ganglia by paralysing them and acetylcholine is not involved in their action.

INTRODUCTION

It has been shown that during DDT poisoning, a toxic substance is liberated into the blood of *Periplaneta americana* L., which by chemical analyses proved to be neither DDT nor its metabolite (STERNBURG & KEARNS, 1952; SHANKLAND & KEARNS, 1959). The neurotoxin released by TEPP, malathion and pyrethrum into the blood of cockroach, when applied to an isolated nerve cord, produced increased spontaneous activity and also an increase in the heart beat frequency (COLHOUN, 1958; SUDERSHAN & NAIDU, 1967). A recent report (PURUSHOTHAM RAO & NAIDU, 1976) has shown that site of action for neurotoxins and nicotine appears to be the same. The present investigation was undertaken to study and compare the physiological action of malathion, endrin and the neurotoxins released by their action.

MATERIALS AND METHODS

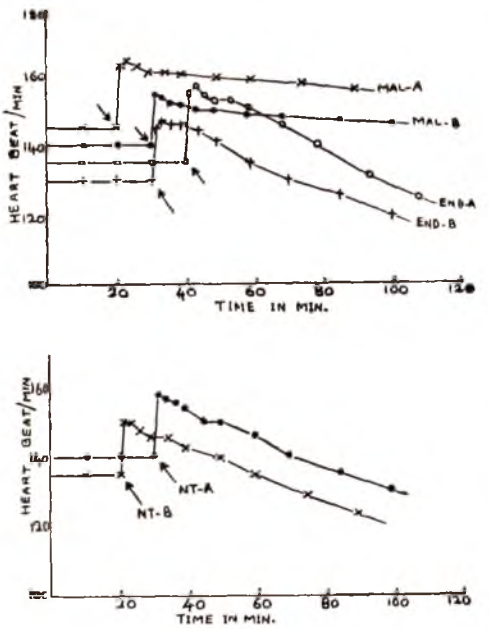
The isolated cockroach heart technique described earlier (KRIJGSMAN *et al*, 1950; NAIDU, 1955) was employed in the present studies. The frequency of heart-beat was taken as a measure of the action of insecticide or neurotoxin and each experiment was carried out on at least five different heart preparations. The mean result of five tests provides the value for each point on the graph. Arrows in the figures indicate the point of addition of test solution to the heart preparation. Laboratory reared male adults of *Periplaneta americana* L. were used for the experiments.

The concentration equivalent to LD_{90} of malathion and endrin dissolved in ethanol was injected intraperitoneally to *P. americana*. Four hours after the treatment blood was collected by centrifugation and the toxic neuroactive substance was isolated as described by STERNBURG *et al.* (1959). Test solutions of insecticides and neurotoxins were prepared in ethanol (wt/vol) while drugs in distilled water and incorporated into the physiological solution. The concentration of ethanol used was found to be not detrimental to the isolated cockroach heart.

RESULTS

Effect of malathion and endrin (Fig. 1)

Malathion (A, 2×10^{-6} μg/ml) caused an immediate increase in the frequency of the heart beat (FHB) which continued for sometime, later a steady decline was observed. At lower concentration (B,



Figs. 1 and 2. Effect of malathion (MAL) and endrin (END) on the isolated cockroach heart (upper) and Effect of malathion—neurotoxin (NT) on the isolated cockroach heart (lower).

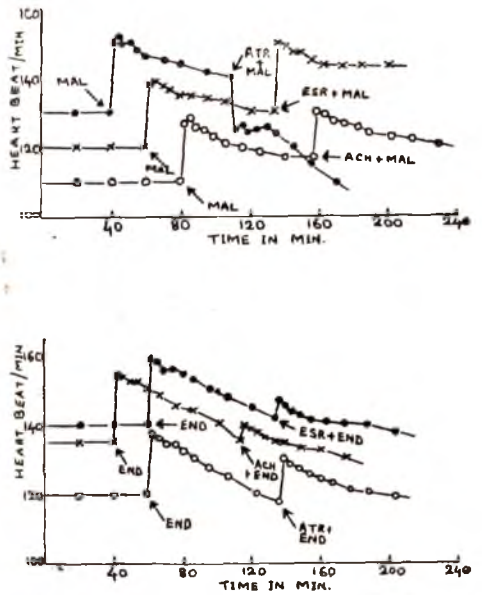
5 x 10^{-7} μg/ml) a similar effect was produced except that the initial increase caused was slightly less. A slight increase in the amplitude was seen. Endrin (A, 5 x 10^{-7} μg/ml) induced an immediate increase in the frequency followed by a rapid decline, this was accompanied by slightly reduced amplitude. At lower dilution (B, 3 x 10^{-8} μg/ml) the initial increase caused was slightly less.

Effect of malathion and endrin toxins (Fig. 2)

Neurotoxin released by the action of malathion (A, 5 x 10^{-3} μg/ml) caused an immediate increase in the FHB which was sustained for some time followed by rapid decline. No apparent change in the amplitude of the heart beat was observed. At lower concentration (B, 2 x 10^{-4} μg/ml) a similar effect on the FHB was seen except that the initial increase caused was less. An identical action was seen with endrin–neurotoxin on the isolated cockroach heart.

Effect of cholinergic drugs on the activity of insecticides

The anticholinesterase, eserine (1 x 10^{-8} μg/ml) by itself had little or no action on the FHB when added to a heart preparation, but potentiated the activity of malathion (5 x 10^{-7} μg/ml). Similar potentiation of malathion was also seen with very low concentration of acetylcholine (2 x 10^{-9} μg/ml) but atropine (5 x 18^{-5} μg/ml) inhibited the action of malathion (Fig. 3).



Figs. 3 and 4. Effect of eserine (ESR), acetylcholine (ACH) and atropine (ATR) on the action of malathion (MAL) (upper) and Effect of eserine (ESR), acetylcholine (ACH) and atropine (ATR) on the action of endrin (END) (lower).

To a heart preparation previously treated with endrin (5 x 10^{-7} μg/ml) addition of eserine (1x 10^{-8} μg/ml) or acetylcholine (2 x 10^{-9} μg/ml) did not cause significant change in the heart beat rate. Atropine (5 x 10^{-5} μg/ml) a cholinergic blocker did not interfere with the action of endrin (Fig. 4). After prolonged treatment of nicotine (5 x 10^{-5} μg/ml) for 3hrs addition of endrin did not cause any significant change in the frequency

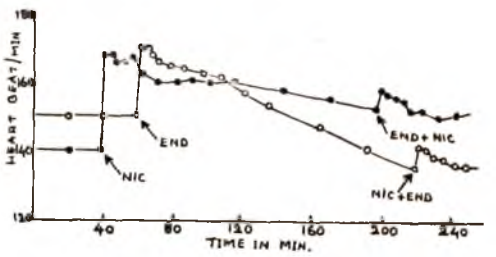


Fig. 5. Effect of nicotine (NIC) on the action of endrin (END) and *vice versa*.

of the heart beat. When the treatment was reversed the action of nicotine was reduced significantly (Fig. 5).

DISCUSSION

The changes produced in the FHB by malathion, endrin and their neurotoxins are similar, suggesting similarity in the site and mode of action of the insecticides and their neurotoxins. However, reaction with cholinergic drugs gave contrary results.

The potentiation of malathion by low concentrations of eserine or acetylcholine suggests the involvement of acetylcholine in its action, which is further supported by the fact that atropine a cholinergic blocker inhibits its action. Earlier findings of NAIDU (1965) showed that nicotine was able to manifest its normal action after prolonged treatment with malathion. If the treatment was reversed, the action of malathion was not seen. It was concluded that malathion does not produce its action by causing paralysis of cardiac ganglia as nicotine does, but by stimulating them.

Eserine or acetylcholine in low concentration do not potentiate the action of endrin and atropine does not antagonise its action. It is reasonable to believe that acetylcholine is not involved in the action of endrin. Further, its action on the cardiac ganglion is evident from the fact that after prolonged action of nicotine, endrin does not cause any change in the FHB.

The authors have recently reported (PURUSHOTHAM RAO & NAIDU, 1976) that after prolonged treatment of neurotoxin released by malathion and endrin, nicotine did not produce any change in the heart beat frequency. The activity of neurotoxins on the heart was neither antagonised by atropine nor potentiated by eserine. It was concluded that neurotoxins act at the cardiac ganglia and acetylcholine is not involved in their action. The present findings on endrin appear to be in agreement with the above contention.

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