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Gas Chromatographic Evaluation of Phosphamidon Intoxication in Sparrows

Prabhu N. Saxena and Pradeep Bhatnagar

Department of Zoology, University of Rajasthan, Jaipur-302004, India

Organophosphorus (OP) insecticides are used extensively for the control of agricultural pests and disease vectors. Although these compounds have short half-lives in the environment and in the tissues of homeothermic animals, some (OP) insecticides viz. Phosphamidon have been observed to be highly toxic to birds, the non target species following application. Adequate information is available on the effect of organophosphorus compound on avain physiology (Saxena and Bhatnagar 1983). In the present investigation an attempt was made to correlate the effect of a sublethal dose of phosphamidon with the severity of physiological disorders in sparrows. The vitals (brain, liver and muscles) in the present investigation, have been observed retaining the chemical and strengthen the observations of (barnett et al 1983) suggested for vitals following fenthion treatment. The possibility of such linear relationship dose response etc. in bringing about physiological disorders has been discussed.

MATERIALS AND METHODS

Passer domesticus, the common house sparrow, trapped from suburban region of walled city Jaipur were placed in separate small cages and were individually weighed before placing in test cages. Thirty healthy birds (average weight 12..24 ± 1.64) were treated orally with 60 ug of phosphamidon in 0.5 ml acetone separately each time. Three such sets were run simultaneously.

Autospies were performed 1 and 2 days after a single oral dose. Brain, liver and muscles were taken out and phosphamidon was extracted from them after Stahr (1977). The extraction required the use of acetonitrile 3X. Further acqueous sodium sulfate (2%) was added and mixed with the aid of vortex mixter.

From the aqueous sodiumsulfate solution the phosphamidon was extracted in benzene. The benzene extract was concentrated and the interfering pigments were removed by eluting 200 ml of benzene in a florisil column (10 gm). The elutent was concentrated to 0.5 ml and analysed by gas Chromatograph model chemito 3800, detector ECD with Ni^{63} as electron source, column temperature 175°C, injection temp. 250°C column 3% SE 4' x 1/4" S.S., N_2 flowrate 40 ml/minute, retention time 225 seconds. The general behaviour of the animal was also taken into account for the period of experimentation. The controls were run simultaneously.

RESULTS AND DISCUSSION

The symptoms of intoxication developed after 1 hour of treatment include tail tremors, stumbling gait followed by general tremors, immobility and convulsions. Regurgiation, ataxia, dyspnea and prostration was more pronounced on the second day after the treatment. There was a general reduction in activity, fluffing of feathers followed by a general lethargy, the second day of treatment.

The quantity detected in the three vital organs (Fig. 1,2 and 3) is in the order of brain muscle liver (Table 1) irrespective of day after the treatment. The birds which served as control did not show any change in their general behaviour.

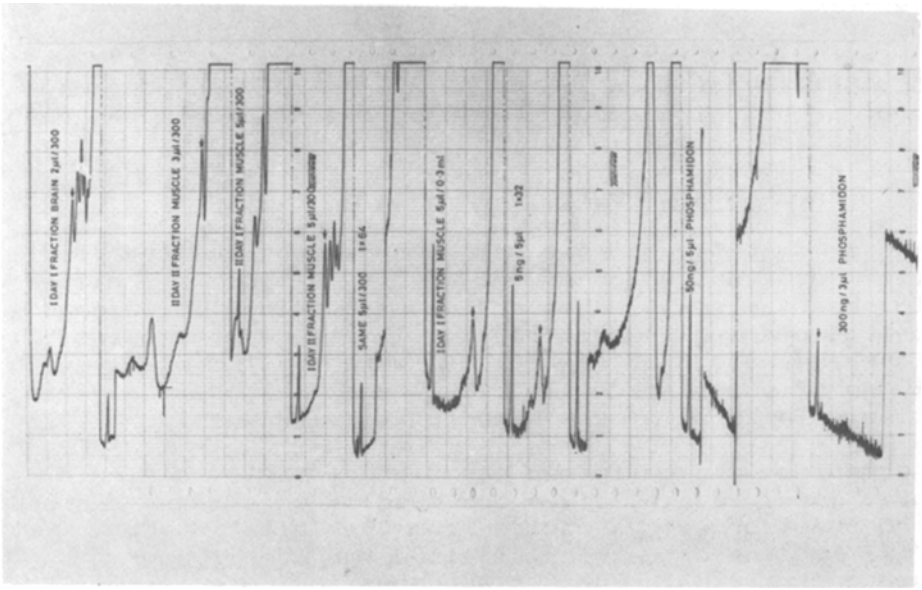

Figure 1. Shows peaks of phosphamidon in muscle and brain

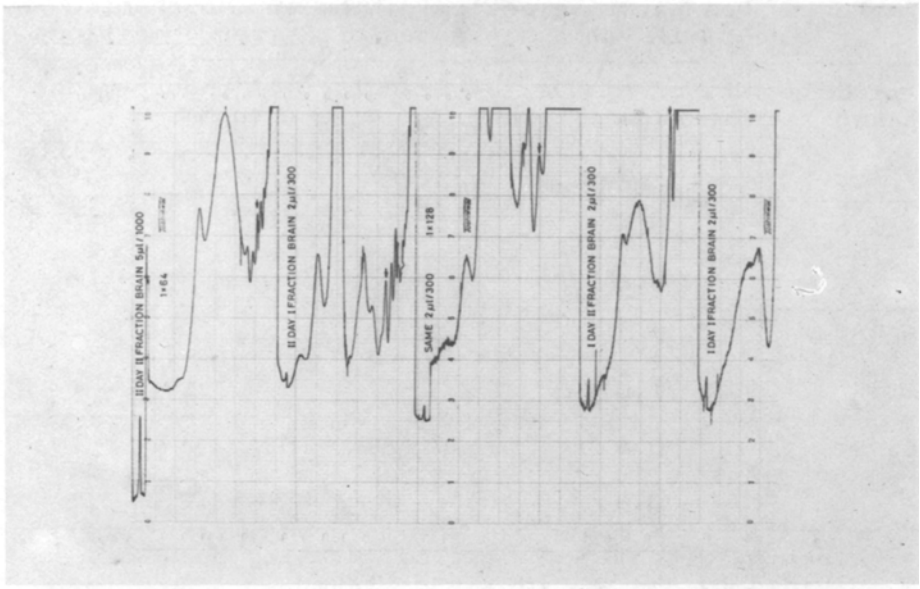

Figure 2. Peaks of Phosphamidon in brain

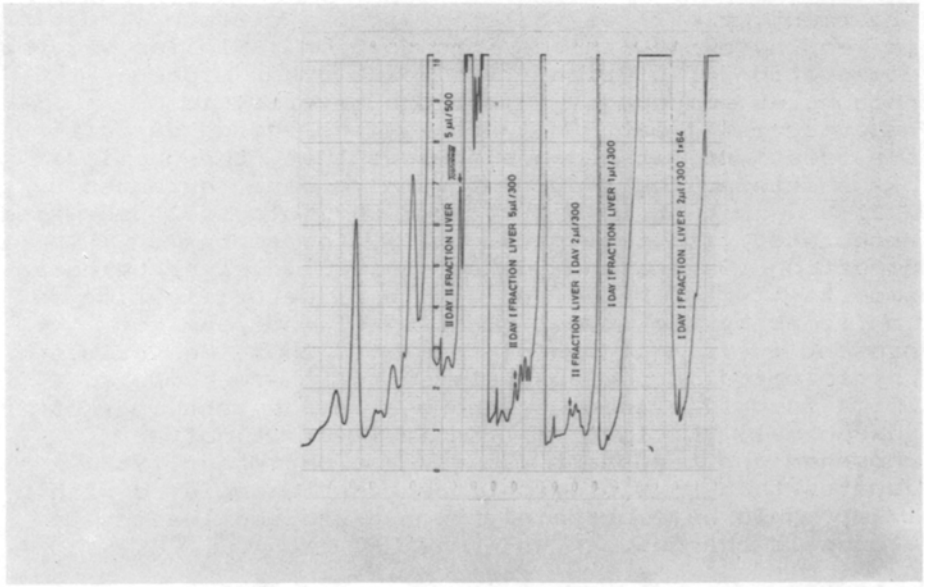

Figure 3. Peaks of phosphamidon in liver

Table 1: Quantitative determination of phosphamidon by GLC in various tissues of *Passer domesticus*.

Tissue taken	Wt. of tissue+ (gm)	Quantity* of phosphamidon (ppm)	
		1 day	2 day
Brain	0.8	12.176 ±.34	
	0.6		8.975 ±.08
Muscle	0.8	0.7008 ±.12	
	1.0		3.5496 ±.04
Liver	0.6	3.468 ±.07	
	0.8		3.210 ±.05

* Quantity determined after calculating peak area divided by the wt. of tissue taken.

\+ The quantity of the tissues viz. brain, muscle and liver taken for detection was fixed for thirty individuals of each set.

Since the quantity of phosphamidon is in a decreasing order irrespective of the tissues and the day after the treatment, clarifies that the process of detoxification is keeping pace with the intoxication. Similar was the observation of O'Brien (1960) with other organo-phosphorus compounds. Since the severity in behaviouristic pattern increases the second day after the treatment, it becomes obvious that the chemical has shattered the important vital organs (detected by GLC) and in turn to the various physiological processes associated with them particularly the anorexic condition (Saxena and Bhatnagar 1983). Liver being an important vital organ and a site of detoxification is confirmed by the lowest quantity of phosphamidon present in it. It can be concluded that the various physiological processes related to it are hampered which is well evident by changes in the behaviour of the animal. Earlier, a loss in liver wt. after phosphamidon treatment (Saxena and Bhatnagar 1983) denotes that physiological processes associated with liver would have certainly been disturbed leading to aforesaid changes.

Further, appreciable quantity of phosphamidon in muscles intervenes with the various biochemical processes (related to energy production for the

important avain event, the flight) and results in general lethargy, reduction in activity and fluffing of wings.

Again, a greater concentration of chemical in brain is indicative of the greater affinity of the organo-phosphorus compounds to the nervous tissue (O'Brien 1967). It is the higher residual amount which is responsible for tremors followed by convulsions, ataxia prostration and death. A chain of events lead to death after intoxication in brain which include - inhibition of cholinesterase, acetylcholine accumulation, disruption of nerve functions, respiratory failure and death (Saxena and Bhatnagar, 1984, unpublished data). Hence phosphamidon intoxication leading to various physiological disorders can be judged the best by Gaschromatograph.

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