

Rodenticidal Poisoning of Non-Target Animals: Acute Oral Toxicity of Zinc Phosphide to Poultry

T. Shivanandappa, H. P. Ramesh, and M. K. Krishnakumari

Discipline of Infestation Control & Pesticide Central Food Technological Research Institute, Mysore 570013, India

Zinc phosphide has been extensively used as an effective rodenticide because of its wide spectrum of activity against all types of rodents (CHITTY 1954). During the rodent control program, often secondary poisoning of non-target animals particularly poultry has caused concern. Reports on acute oral toxicity of zinc phosphide to poultry birds are scanty. Hence investigations were undertaken to establish the acute oral LD₅₀ and LD₉₀ on the domestic fowl, Gallus domesticus.

MATERIALS AND METHODS

Animals and diet: Individually caged female birds of 13-wk age weighing between 600 to 750g were statistically grouped and maintained individually in the cages placed in the well aerated animal house and were fed with grower mash and tap-water ad libitum.

Preparation of Zn₃P₂ capsules: Zinc phosphide obtained commercially (87% purity) was mixed with corn starch to give two stock concentrations, 10% and 20%. The amount of Zn₃P₂ per individual bird body weight was calculated and filled in gelatin capsules. Four dosages viz., 14, 31.5 and 47.2 mg/kg body weight were used.

Treatment: After one wk of maintenance, the birds were partially starved and fed with Zn₃P₂ capsules. The mouth of the bird was opened and the capsules were placed into the gullet with the help of a blunt forceps and the birds swallowed the capsules easily. Eight birds were used for each dosage. One group received empty gelatin capsules alone to serve as carrier controls and the other group was maintained as untreated controls. They were under observation for symptoms and mortality for 4 wk. Food and water were available ad libitum.

Autopsy: At the end of the 4th wk, birds were anaesthetised with chloroform and autopsied. Weights of liver, kidney, gizzard, heart, spleen, brain, thyroid, adrenal and ovary were recorded. The tissues were fixed

in 10% neutral formalin. 8-10 μ m thick paraffin sections stained with haematoxylin and eosin were observed for histopathological changes. The data were statistically analysed and LD₅₀ and LD₉₀ values were calculated by probit analysis from a dose-response curve.

RESULTS

Symptoms and Mortality: There was mortality in all the four dosages. The mortality rate in different groups is presented in Table 1. There was only one death in the initial dose (12% mortality) and 100% mortality in the

TABLE 1
Results of feeding zinc phosphide to pullets

Dosage (mg/kg body weight)	Morta- lity %	Average death time (h)	mg/kg body weight	
			LD ₅₀	LD ₉₀
Untreated	0	-	-	-
Gelatin	0	-	-	-
14.0	12	< 40	-	-
21.0	87	< 40	25	31
31.5	87	< 20	(24 to 26)*	
47.2	100	< 20		

*95% Confidence limits

highest dose. All the birds died within 18h in the 21 mg dosage group except one which died within 40h; only one survived. In the next dose group (31.5 mg/kg) all the birds but one died within 40h. In the highest dose group (47.2 mg/kg) all the birds died within 6-18h. The LD₅₀ and LD₉₀ values determined were 25 mg/kg and 31 mg/kg body weight respectively (Table 1).

Birds which succumbed to Zn₃P₂ were dull and showed heavy breathing and tremors. Paralysis of the legs was invariably noticed and convulsions preceded death.

Body weights: Body weights of control and zinc phosphide treated groups are depicted in Figure 1. The surviving animals in the initial dosage group showed no significant weight gain in the first week but they soon resumed normal rate of weight gain in the following weeks like gelatin and untreated controls.

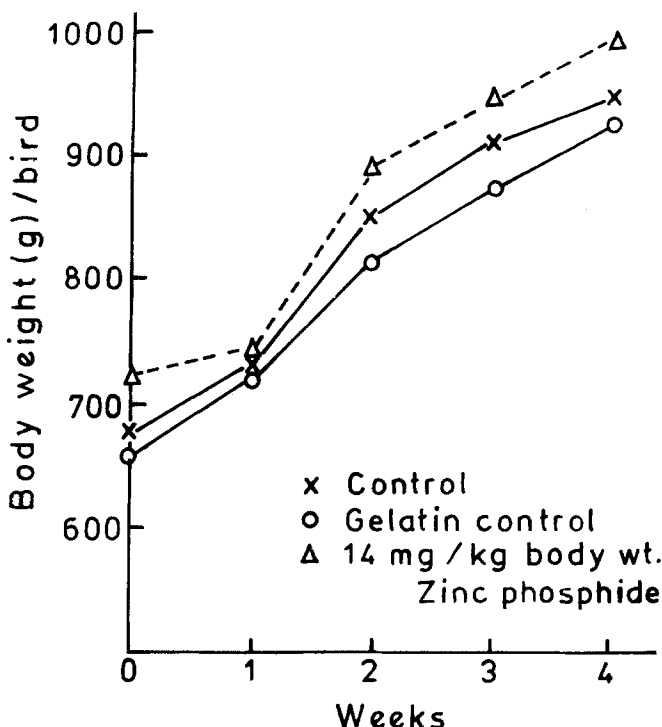


Fig.1. Comparison of body weight gain between zinc phosphide treated and control birds

Organ weights: There were no statistically significant differences in the relative organ weights of liver, lung, kidney etc., among Zn_3P_2 treated and control birds.

Histopathology: The histology of the liver and kidney of treated birds did not show any discernible changes from that of controls. In the gizzard of treated birds, there was mild to severe cellular infiltration and in some cases, slight erosion of the mucosal layer. In the lungs also cellular infiltration was commonly noticed. The histological picture of all the other organs *viz.*, brain, heart, spleen, ovary, thyroid and adrenal was normal.

DISCUSSION

LD₅₀ values obtained in the present study which fall between 24 to 26 mg/kg indicate high susceptibility of the domestic fowl to zinc phosphide. Water is not recommended as a medium or carrier for intubation of Zn_3P_2 as in the earlier studies since evolution of PH_3 gas occurs when Zn_3P_2 comes into contact with water. Feeding zinc phosphide in gelatin capsules seems to be the efficient method for

birds since there was neither leakage nor spillage.

The manifestation of symptoms preceding death is relatively quick and is probably due to the release of phosphine gas when zinc phosphide comes in contact with the secretions of the gastric tract. The phosphine enters the blood circulation which may cause haemolysis and finally resulting in death. The cause of severe cellular infiltration and damaged mucosal layer observed in the gizzard may be associated with the release of phosphine gas acting as the local irritant.

The absence of any significant histopathological signs in various organs of the zinc phosphide treated birds might indicate that the zinc which later enters the circulation may not be toxic at the acute doses used in this study and similar results have been obtained in the rat (KRISHNAKUMARI et al. 1978). The observed body weight loss in the surviving birds during the first week may be due to the after effects of zinc phosphide poisoning rather than zinc accumulation, since zinc is not known to affect growth or weight gain in poultry (ROBERTSON & SCHAIBLE 1960, KINCAID et al. 1976).

The LD₅₀ value for the domestic fowl is significantly less when compared to that of rat which is reported to be between 40-55 mg/kg (CHITTY 1954, SPECTOR 1955, KRISHNAKUMARI et al. 1978). These results indicate that zinc phosphide is more toxic to the domestic fowl and should be employed with great care in rodent control.

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REFERENCES

- CHITTY, B. : Control of rats and mice. Vol.1, pp 305 Clarendon Press, Oxford (1954).
KINCAID, R.L., W.J. MILLER, JENSEN, L.S., D.L. HAMPTON, M.W. NEATHERY and R.P. GENTRY: Poultry Sci. **55**, 1955 (1976).
KRISHNAKUMARI, M.K., K.MUKTHA BAI and S.K.MAJUMDER: Pesticides (In press).
ROBERTSON, R.H. and P.J. SCHAIBLE.: Poultry Sci. **39**, 893 (1960).
SPECTOR, W.S.: Hand Book of Toxicology, Vol.1, pp 353, United States Air Force, Wright-Patterson Air Force Base, Ohio (1955).