

## Toxicity and Rodenticidal Potency of Zinc Phosphide

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Zinc phosphide has been in use as a rodenticide since 1919 (SHEPARD 1951). The recommended concentration in the bait varies from 0.5 to 20% throughout the world (SCHOOFF 1970, BROM 1968). Today's major concern in applying this rodenticide is to use it effectively and safely for alleviating rodent damage problems. The reported LD<sub>50</sub> values range from 25 (GARLOUGH 1941) to 47 mg/kg body weight (SPECTOR 1955). Its usage at higher concentration is hazardous to humans and non-target species leading to direct and secondary poisoning. Hence evaluation of toxicity and bioefficacy of this compound is imperative. This paper discusses the toxicity of zinc phosphide to albino rats in addition to its efficacy and safe concentration for rodent control in fields.

### MATERIALS AND METHODS

Samples of zinc phosphide (Technical grade) were obtained from different industries and named as A (Swadeshi Pvt. Ltd); B (Excel Industries); C (Tata Fison); and D (Analytical grade). The purities were 87.0; 83.0; 94.0 and 99.9% respectively.

#### A. Forced feeding trials:

1. Animal and diet: Individually caged female albino rats (*Rattus norvegicus*) of CFT-Wistar Strain weighing 200 to 250 g were statistically grouped. For each dosage ten animals were used. Rats were maintained in the animal house of this Institute and were fed with basal diet (KRISHNAKUMARI et al. 1979) and tap water. Suspensions of zinc phosphide were prepared using peanut oil to give the concentration of 5 and 10 mg of active ingredient/mL of oil.

2. Method: Prior to oral intubation rats were partially starved. Calculated dosages of zinc phosphide (Table 1) in peanut oil were orally intubated. Rats fed with peanut oil alone served as controls. Treated rats were observed for symptoms and mortality. Food consumption

was recorded for seven days and the survivors were observed for a further period of three weeks while being fed with the basal diet and water. Weekly body weights were recorded. Before autopsy, blood samples were collected and subjected to haematological studies (viz., haemoglobin, RBC, WBC, PCV, ESR, and differential counts) as described by HEPLER 1950. The weights of liver, kidney, heart, spleen, lungs and ovary were recorded. The tissues were processed for histopathological studies. The dose response was determined and the LD<sub>50</sub> and LD<sub>90</sub> values were calculated by probit analysis. Data on body weights and organ weights were subjected to statistical analysis. For convenience, the results of zinc phosphide for sample A are presented in detail. However for other samples LD<sub>50</sub> and LD<sub>90</sub> values are reported (Table 1).

TABLE 1. Comparison of LD<sub>50</sub> Values for Albino Rats After Intragastric Administration of Zinc Phosphide

Sample	Chemical Purity (%)	Dosage mg/kg	Death time (h)	mg/kg body weight	
				LD <sub>50</sub>	LD <sub>90</sub>
A*	87.0	0	0	54	120
		21	18	(95% confidence limits: 33 to 120)	-
		28	18		
		38	12		
		51	24		
		68	(10-36) 22 (8-40)		
B	83.0			56	120
C	94.0			44	100
D	99.9			43	97

\* Details are reported for sample A only. For B, C and D LD<sub>50</sub> and LD<sub>90</sub> are presented.

## B. Ad Libitum Trials:

1. Animals and diet: Rattus rattus (roof rat) and Mus musculus (house mouse) were collected from various localities by trapping. Bandicota bengalensis (mole rat) and Tatera indica (Indian gerbil) were collected in fields by digging. Rats collected were brought to the laboratory and released in conditioning cages (96 x 76 x 56 cm)

for seven days for acclimatization. Healthy adult rats of known weight and sex were released into the individual experimental cages (23 x 23 x 23 cm). Zinc phosphide (Sample C) was mixed thoroughly with standard bait (KRISHNAKUMARI et al. 1963) at 2% concentration (KRISHNAKUMARI 1968) and was evaluated against the selected rodents.

2. Method: The standard bait (10 g/rat) was fed to the rats for two nights and was replaced with 2% zinc phosphide baits, on the third night. The poison bait was left in cages for 16 h, the residues were collected next morning and intakes were calculated for each rat. The symptoms and mortality were noted and death time was recorded. Survivors were observed for 15 days while being fed with basal diet and water.

## RESULTS

### A. Forced feeding trials:

The five different dosages tested (for Sample A) showed that only 10% of the rats succumbed to the first two dosages within 18 h of intubation (Table 1). However, the percent mortality increased to 20 and 60% with the increased dosages. Generally the rats succumbed to zinc phosphide within 40 h. From the regression line, the LD<sub>50</sub> and LD<sub>90</sub> values determined were 54 and 120 mg/kg body weight. However, the LD<sub>50</sub> values determined for other samples ranged from 43 to 56 mg/kg body weight.

Irrespective of the dosage of zinc phosphide, symptoms appeared within 5 min. The immediate symptoms were heavy breathing, ataxia, restlessness, coma and paralysis of hind limbs. Convulsions occurred prior to death. These symptoms continued in animals which succumbed.

Food intake: Excluding the last two doses there was no significant difference between the treated and untreated animals in food consumption rate. Consumption was decreased initially in the animals which survived the higher dosage and resumed to normalcy after 48 h.

Body weights: There was no significant difference in body weights after treatment. Even at the highest dosage (68 mg/kg) the weights were almost on par with the controls.

Haematological studies: Results obtained on various blood tests indicated that there were no abnormalities. Irrespective of the dosage all the values were comparable to control.

TABLE 2. Ad Libitum Trials of Zinc Phosphide (2%) to Various Rodent Pests Under Captivity

Species	Av. body weight (g)	Av. poison bait consumption (g) (in 16 h)	AI Intake (mg/kg)	No. tested/		No. dead (h)		
				No. dead		16	24	48
<u>Rattus norvegicus</u> (albino)	160	1.4 (0.5-2.5)	240	8/8		7	0	1
<u>Rattus rattus</u>	100	1.4 (1.0-3.0)	504	8/8		6	1	1
<u>Mus musculus</u>	14	0.6 (0.2-1.0)	638	8/8		4	2	2
<u>Bandicota bengalensis</u>	245	3.0 (1.5-4.0)	230	8/8		3	3	1*
<u>Tatera indica</u>	180	2.2 (1.0-2.5)	216	8/8		6	1	1

AI: Active Ingredient

\* Out of two one rat died after 48 h.

Relative organ weights: There were no differences in weights of vital organs as a result of zinc phosphide intubation. The values were normal and comparable to control.

Histopathology: Among the organs examined, kidney of some rats fed with different dosages showed slight haemorrhage in glomeruli and tubules. Mild cellular infiltration around bronchioles was discernible in lungs. There were no histological changes in the liver, spleen, ovary and heart of treated animals.

#### B. Ad Libitum trials:

Zinc phosphide at 2% in the bait was lethal to all rodents tested viz., roof rat, house mouse, albino rat, mole rat and Indian gerbil. The death occurred within 48 h in all that species except in B.bengalensis where one animal took more than 48 h (Table 2). However, the minimum time to death was noticed in roof rats (2.5 h) while the maximum was taken by B.bengalensis (56 h). Zinc phosphide at 2% was acceptable to all the species of rats tested as evident by the average ingestion of poison bait (0.6 to 3.0 g/rat). However the ingestion of active ingredient varied from 59 to 1140 mg/kg.

### DISCUSSION

The results of our investigation revealed that zinc phosphide when forced fed to adult female rats, the LD<sub>50</sub> value varied from 43 to 56 mg/kg (Table 1). It is well established that some impurities present in technical grade samples potentiate the oral toxicity of the compound (PELLERGINI & SANTI 1972). However in our studies it was observed that as the purity of the sample increased, the LD<sub>50</sub> values decreased. This suggests that the impurities like bauxite, zinc oxide and trace elements (Fe, Cu, Ni, SiO<sub>2</sub>) present in zinc phosphide did not influence the toxicity of zinc phosphide. The variation in toxicity could also be due to the age, sex, species, strain, diet (KRISHNAKUMARI et al. 1963) & BOYD 1972) and vehicle used (STAREK & ZABINSKI 1970). However in the present study the difference obtained could be attributed to the purity of samples as the other conditions remained same.

The quick appearance of symptoms in zinc phosphide poisoned animals with higher doses finally ending in death within 40 h indicated that zinc phosphide monitors its toxicity on mammals quickly due to the release of phosphine in the gastrointestinal tract. Zinc phosphide when comes in contact with water or acid released phosphine which enters the blood stream and gets distributed

throughout the body resulting in haemolysis and blocking of the kidney (PEOPLES 1970). Though phosphoric acid and zincchloride are breakdown products of zinc phosphide whose toxic nature cannot be ruled out, it seems that phosphine is the main toxicant in causing death of animals poisoned with zinc phosphide. However, monitoring the release of phosphine from zinc phosphide in vivo was difficult since it depended on the availability of acid or water within the system.

The variation in mortality depended on the rate of release of phosphine in the system. Zinc phosphide ingestion causes both fatty degeneration and necrosis of the liver and pulmonary hyperemia and edema in humans (DREISBACH 1964). The results of histopathological, haematological and relative organ weights in the survival rats indicate that zinc phosphide is not retained in the tissues for a longer time to exert any local/permanent damage to the cells. Absence of any change in either food consumption or body weight also confirmed that rats could recover completely from zinc phosphide intoxication within a period of four weeks.

The fact that symptoms appeared within 5 min in forced fed rats while it took more than 30 min when it was orally ingested in bait could be attributed to the time taken for the compound to reach the stomach and thereby the site of action. It is of interest to observe that although the symptoms appear immediately, the rats succumb to it only after 3 to 6 h of ingestion points out that there is a 'lag' period for manifesting the toxicity. This might be correlated to the time taken for phosphine gas to build up the lethal concentration in the animal system. NEUBERT & HOFFMEISTER (1960) have also observed that the animals took one h to succumb even though they were exposed to phosphine as high as 450 ppm.

This was further confirmed by MUTHU et al (1980) who established the acute  $LC_{50}$  of phosphine on albino rats (28 ppm for 5.2 h). The fact that zinc phosphide at 2% level in the bait was acceptable by all rodents ending in lethality suggests that this compound is an effective single dose rodenticide. Nevertheless, with its low  $LD_{50}$  value, it warrants careful handling and application. When judiciously used, it is one of the useful poisons among many acute rodenticides currently employed for rodent control.

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