

University of Nebraska - Lincoln

DigitalCommons@University of Nebraska - Lincoln

Proceedings of the 5th Vertebrate Pest Conference
(1972)

Vertebrate Pest Conference Proceedings collection

March 1972

ZINC PHOSPHIDE—A NEW LOOK AT AN OLD RODENTICIDE FOR FIELD RODENTS

Glenn A. Hood

Bureau of Sport Fisheries and Wildlife, Denver Wildlife Research Center

Follow this and additional works at: <http://digitalcommons.unl.edu/vpc5>



Part of the [Environmental Health and Protection Commons](#)

Hood, Glenn A., "ZINC PHOSPHIDE—A NEW LOOK AT AN OLD RODENTICIDE FOR FIELD RODENTS" (1972).

Proceedings of the 5th Vertebrate Pest Conference (1972). 16.

<http://digitalcommons.unl.edu/vpc5/16>

This Article is brought to you for free and open access by the Vertebrate Pest Conference Proceedings collection at DigitalCommons@University of Nebraska - Lincoln. It has been accepted for inclusion in Proceedings of the 5th Vertebrate Pest Conference (1972) by an authorized administrator of DigitalCommons@University of Nebraska - Lincoln.

ZINC PHOSPHIDE--A NEW LOOK AT AN OLD RODENTICIDE FOR FIELD RODENTS

GLENN A. HOOD, Research Biologist, Bureau of Sport Fisheries and Wildlife, Denver Wildlife Research Center, Denver Federal Center, Denver, Colorado¹

ABSTRACT: Of the many toxicants tested to control field rodents, compound 1080 (sodium monofluoroacetate), strychnine alkaloid, and zinc phosphide are the only effective single-dose rodenticides currently available. Considering the federal requirements for use in food and feed crops, zinc phosphide is the toxicant most likely to be registered for field rodent control. It is generally well accepted by rodents, is relatively safe for nontarget species, and does not seriously contaminate the environment. It is already registered, with an established tolerance, for use in one food crop (Hawaiian sugarcane). The Bureau of Sport Fisheries and Wildlife is conducting research, some in cooperation with other agencies, to register zinc phosphide for controlling: prairie dogs (*Cynomys ludovicianus*) in shortgrass rangeland; jackrabbits (*Lepus californicus*) along cropland-rangeland borders; cotton rats (*Sigmodon hispidus*), rice rats (*Oryzomys palustris*), black rats (*Rattus rattus*), and Florida water rats (*Neofiber aileni*) in Florida sugarcane; ground squirrels (*Spermophilus* spp.) and meadow voles (*Microtus* spp.) in alfalfa, sugarbeets, artichokes, and rangeland.

Considerable literature has been published on the role of rodents in crop losses and disease transmission. These problems are virtually world-wide, with very serious economic and health implications. "Biological" methods for controlling rodents, such as diseases, predators, and habitat modifications, have been attempted; but rodenticide-treated baits are more extensively used because they produce quick and more controllable results and are usually economical. Historically, many toxicants, including arsenic, phosphorus, endrin, and thallium sulphate have been used as rodenticides. Thousands of other compounds have been screened for rodenticidal activity. For various reasons, compound 1080, strychnine alkaloid, and zinc phosphide have evolved as the only effective single-dose rodenticides currently available.

In 1964, the Leopold Committee recommended that 1080 be banned as a rodenticide because of secondary hazards and replaced with "strychnine or other chemicals which are not readily transmitted to scavenging animals" (Leopold 1964). Strychnine is not a general-purpose rodenticide; it is poorly accepted by many rodents and its use poses hazards to humans and nontarget wildlife (Rudd and Genelly 1956; Gleason, et al. 1969). Our studies with the desert kit fox (*Vulpes macrotis arsipus*) indicate that strychnine may also be hazardous secondarily to canids. We had high hopes for DRC-714 (Gophacide)* as a replacement general-purpose rodenticide (Richens 1967; Ward, et al. 1967; Schroeder 1967; Hoffer, et al. 1969). Unfortunately, after several years of research, the parent company cancelled further development. DRC-3492 (6-aminonicotinamide) is another promising rodenticide, but there are some questions concerning its registration--furthermore, its release would probably be several years away.

Replacement rodenticides are difficult to come by. We feel that, in order to be considered for registration, a toxicant must conform or be adaptable to a majority of the following criteria: (1) well accepted by target species; (2) selectively toxic to target species, or usable in a manner minimizing primary hazards to nontarget species; (3) safe to handle by humans; (4) causing no secondary hazards; (5) relatively slow-acting to minimize bait shyness; (6) causing painless and nonviolent death; (7) noncumulative;

¹H. Wayne Hilton of the Hawaiian Sugar Planters' Association, Honolulu, Hawaii; James Evans, Richard E. Griffith, Jr., Roger D. Nass, William H. Robison, Frank Schitoskey, Jr., and Howard P. Tietjen of the Bureau's Denver Wildlife Research Center, and other cooperators should have been listed as coauthors of this paper. Since this was not editorially practical, their contributions to our knowledge of zinc phosphide are gratefully acknowledged.

*Trade name of Farbenfabriken Bayer for 0, 0 bis (p-chlorophenyl) acetimodoylphosphoramidothioate. Reference to trade names does not imply endorsement of commercial products by the Federal Government.

(8) not translocated into vegetation; (9) capable of rapid decomposition into harmless products to reduce hazards and environmental contamination; (10) counteracted by an antidote; (11) economical; and (12) registerable by the Environmental Protection Agency (EPA) and Food and Drug Administration (FDA). It is readily apparent that none of the known rodenticides meet all of these criteria. In the past, emphasis was placed on efficacy but has since shifted to safety, a major consideration for registration.

Considerably more data are now required for federal registration of new toxic agents and reregistration of those currently used. Generally, if the area produces a food crop or is utilized by livestock, it is considered a "food crop" use. Areas used only by wildlife may also be classified as a "food crop" use, especially if wildlife are harvested for food. Under this definition, most rodent control is or will be occurring on "food crops." Therefore, data are needed on short- and long-term toxicology to target and non-target species, residues to establish tolerances for the pesticide and its metabolites in the raw food crop, and the impact and fate of the pesticide in the environment. Within this framework, it will be difficult to establish tolerances and demonstrate acceptable safety standards for the 1080 and strychnine formulations currently registered.

However, we believe that the chances for registration of zinc phosphide for field rodent control are good. To the best of our knowledge, it is the only rodenticide federally registered, with an established tolerance, for use in a food crop--sugarcane in Hawaii. It is also registered, without tolerances, for control of field and orchard mice (primarily *Microtus* spp.), nutria (*Myocastor coypus*), pocket gophers (*Geomyidae*), and rats (*Rattus* spp., *Sigmodon* spp., etc.) with some in-crop uses permitted. Reregistration for these latter uses is questionable without additional residue and environmental impact information.

HISTORY

Zinc phosphide was first used in 1911-12 to control field rodents in Italy, and later in other European countries (Chitty and Southern 1954; Schoof 1970). Its use increased substantially during World War II, when thallium and strychnine were in short supply. The popularity of zinc phosphide decreased during the mid-1940's and early 1950's when 1080 and the anticoagulants first appeared. Because of the emphasis on 1080, zinc phosphide was never fully developed. However, in recent years, as problems associated with the use of 1080 and strychnine have been recognized, interest in zinc phosphide has again increased.

PROPERTIES AND MODE OF ACTION

Technical grade (94 percent purity) zinc phosphide is a grayish-black, fine, crystalline powder, essentially insoluble in water and alcohol, slightly soluble in alkalis and oils. Although quite stable in air and water of pH-7, it decomposes in the presence of acids and alkalis to produce zinc oxide or salts and phosphine (PH_3), a highly toxic, colorless gas with a "garlic" odor. Zinc phosphide and phosphine residues are of concern as environmental contaminants. Zinc compounds occur naturally, and the minute quantities added by baiting rodents are of less concern.

Upon ingestion, zinc phosphide reacts with dilute acids in the gastrointestinal tract and produces phosphine, which enters the blood stream. Chronic exposure to phosphine may cause nausea, vomiting, diarrhea, tightness of chest, coughing, headaches, and dizziness. Acute symptoms also include thirst, back pains, feeling of coldness, and stupor or periodic fainting. Death, from asphyxia, takes somewhat longer than with 1080 and strychnine poisoning and usually occurs after terminal symptoms of mild convulsions, paralysis, and coma.

Zinc phosphide is not readily absorbed through intact skin, but it can enter the blood stream through cuts or breaks (Anon. 1967). It is toxic if inhaled as a dust, as it liberates phosphine in the lungs. Based on human experiences, the maximum phosphine concentration in air that can be tolerated for several hours without symptoms is 7 ppm (Jacobs 1967). The odor threshold is 1.4 to 2.8 ppm, and the maximum continuous allowable concentration is 0.05 ppm. I could locate no data indicating that zinc phosphide caused eye or skin irritations.

A National Pest Control Association release (Anon. 1967) cites J.B.P. Stephenson (Zinc Phosphide Poisoning, Archives of Environmental Health, 15:83-88, July 1967) as follows: "chronic poisoning is not a problem with zinc phosphide. To be effective as a rodenticide, zinc phosphide must be consumed in a relatively short period of time." However, Kilmer (1969), in studying the toxicology of phosphine, found that repeated inhalation of relatively

low concentrations (5-10 ppm) resulted in subacute and possibly lethal accumulative poisoning of cats, rabbits, guinea pigs, and rats. He concluded that phosphine does not act by physical accumulation, but by accumulation of effects. In his opinion, no exposure below 5 ppm will result in chronic poisoning of experimental animals. (However, it should be noted that 5 to 10 ppm PH₃ are levels often used for fumigating insects.)

ACCEPTANCE AND EFFICACY

In general, zinc phosphide is less toxic than 1080 or strychnine, but is usually better accepted than strychnine. At concentrations of 0.75 to 2.0 percent on grain, fruit, or vegetable baits, it has been used against meadow voles, pine voles (*Microtus pinetorum*), ground squirrels, prairie dogs, Norway rats (*R. norvegicus*), black rats (*R. rattus*), Polynesian rats (*R. exulans*), cotton rats, kangaroo rats (*Dipodomys* spp.), nutria, jack-rabbits, and house mice (*Mus musculus*). Efficacy is somewhat less than that obtainable with 1080, but better than with strychnine. Prebaiting is usually recommended. In California, zinc phosphide has been recommended for controlling California ground squirrels (*Spermophilus beecheyi*), Belding ground squirrels (*S. beldingi*), meadow voles, and rats (Anon. 1968). During 1970, approximately 393,000 acres in California were baited with about 2,149 lb of toxicant (Anon. 1971).

TOXICITY AND PRIMARY HAZARDS

Rodents show large variations in response to zinc phosphide. The LD₅₀ ranges from a low of 5.6 mg/kg for nutria to 40 mg/kg for Norway rats and 55.5 mg/kg for white rats (Table 1). Zinc phosphide is relatively toxic to pheasants, ducks, and geese (LD₅₀) 7-5 to 35.7 mg/kg) and is considered a definite hazard to these species and to domestic fowl. It is less toxic than chlorinated hydrocarbon pesticides to fish, which are generally more susceptible to the zinc itself than the phosphine. In our studies, crayfish, shrimp, and gobies tolerated concentrations of zinc phosphide in water from 10 to 50 ppm; and crayfish readily consumed 1.88 percent zinc phosphide-oat groat bait and survived.

Zinc phosphide must be used with care--it is toxic to most forms of animal life. Its emetic properties and disagreeable odor may make it unattractive to some nontarget animals, but this cannot be depended on. Instances of primary hazards to livestock have been documented (Chitty and Southern 1954). In these cases, poisoning was accidental and caused through careless handling and misuse. Many of you are familiar with the accidental poisoning of 455 geese at Tule Lake, California, in 1963. Barley fields were treated with a zinc phosphide-oat groat bait in July or early August to control voles. Although prior agreement was made to delay burning treated fields, a 90-acre field was burned about 3 months after baiting. Keith and O'Neill (1964) concluded: "Burning of a treated barley field was undoubtedly the factor that made lethal quantities of the bait available to geese." In this case, improper management of a baited area contributed to the problem.

Zinc phosphide has no specific antidote. Treatment of poisoning is symptomatic, by evacuation of the stomach and intestinal tract, administration of oxygen, treatment with cardiac and circulatory stimulants, and neutralization of gastric acids with sodium bicarbonate. Von Oettingen (1947) recommended gavage with 0.1 percent potassium permanganate solution and Gleason et al. (1969) suggested 3-5 percent sodium bicarbonate.

TOXICOLOGY AND SECONDARY HAZARDS

Rudd and Genelly (1956) reported that several days are required for complete breakdown of zinc phosphide inside the stomach, with the possibility of secondary poisoning existing during that time. Since zinc phosphide is not assimilated into tissues or bones, secondary poisoning is apparently a form of primary poisoning. Chitty and Southern (1954) reported secondary hazard when cats were fed rats killed by zinc phosphide. They used 5 percent zinc phosphide in sugar-meal or bread-mash baits, and rats consumed 72 to 192 mg of toxicant. Cats that ate rats containing less than 37 mg/kg of toxicant vomited and survived. Cats consuming 44 and 96 mg/kg of toxicant vomited but died the next day. Storer and Jameson (1965) stated that dogs were killed by secondary poisoning in ground squirrel control programs. Doty (1945) reported that cats and mongooses (*Herpestes auro-punctatus*) were not affected when fed rats killed with zinc phosphide. According to Przygodda (1961), raptors are not affected by secondary poisoning from zinc phosphide-killed rodents.

In studies by Evans (1970), feeding nutria killed by zinc phosphide to bald eagles (*Haliaeetus leucocephalus*), black vultures (*Coragyps atratus atratus*), mink (*Mustela vison*),

Table 1. Oral toxicity of Zinc phosphide to various animals. Dose responses are expressed in terms of LD₅₀, median lethal dose (MLD), lethal dose (LD), approximate lethal dose (ALD), or lethal concentration (LC₅₀).

Animal	Source of data	Test type	Toxicity (mg/kg)
<u>Hares:</u>			
Jackrabbit (<u>Lepus californicus</u>)	DWRC*	LD ₅₀	8.2
<u>Rodents:</u>			
Calif. ground squirrel (<u>Spermophilus beecheyi</u>)	DWRC	LD ₅₀	33.1
Prairie dog (<u>Cynomys ludovicianus</u>)	DWRC	LD ₅₀	18.0
Northern pocket gopher (<u>Thomomys talpoides</u>)	DWRC	LD ₅₀	6.8
Northern pocket gopher (<u>T. t. quadratus</u>)	DWRC	ALD	28.0
Kangaroo rat (<u>Dipodomys spectabilis</u>)	DWRC	ALD	8.0
Deer mouse (<u>Peromyscus maniculatus</u>)	DWRC	ALD	42.0
Muskrat (<u>Ondatra zibethica</u>)	DWRC	LD ₉₀	29.9
Meadow vole (<u>Microtus pennsylvanicus</u>)	DWRC	LD ₅₀	18.0
Meadow vole (<u>M. californicus</u>)	DWRC	LD ₅₀	15.7
Black rat (<u>Rattus rattus</u>)	DWRC	LD ₅₀	21.0
Black rat (<u>R. r. mindanensis</u>)	DWRC	LD ₅₀	28.5
White rat	DWRC	LD ₅₀	55.1
Norway rat (<u>R. norvegicus</u>)	DWRC	LD ₅₀	27.0
	Schoof (1970)	LD ₅₀	40.0
Polynesian rat (<u>R. exulans</u>)	DWRC	LD ₅₀	23.0
Ricefield rat (<u>R. argentiventer</u>)	DWRC	LD ₅₀	35.0
Nutria (<u>Myocaster coypus</u>)	DWRC	LD ₅₀	5.6
<u>Carnivores:</u>			
Dog	DWRC	ALD	40.0
Cat	DWRC	ALD	40.0
<u>Ungulates:</u>			
Cow	Anon. (1967)	ALD	50.0
<u>Birds:</u>			
White-fronted goose (<u>Anser albifrons</u>)	Calif.**	LD ₅₀	7.5
Snow goose (<u>Chen hyperborea</u>)	Calif.	LD ₅₀	8.8
Mallard duck (<u>Anas platyrhynchos</u>)	DWRC	LD ₅₀	35.7
	Calif.	LD ₅₀	13.0
Partridge (<u>Perdix perdix</u>)	Janda & Bosseova (1970)	LD ₅₀	26.7
Quail (<u>Lophortyx californica</u>)	Calif.	LD ₅₀	13.5
Pheasant (<u>Phasianus colchicus</u>)	Hayne (1951)	MLD	8.8
	DWRC	LD ₅₀	16.4
	Calif.	LD ₅₀	8.8
	Janda & Bosseova (1970)	LD ₅₀	26.7

Table 1. Continued.

Animal	Source of data	Test type	Toxicity (mg/kg)
Chicken	Blaxland & Gordon (1945)	MLD	20-30
	Robertson et al. (1945)	LD	7-17
Mourning dove (<u>Zenaidura macroura</u>)	DWRC	LD ₅₀	34.3
Sparrow (<u>Passer domesticus</u>)	DWRC	ALD	20-50
Red-winged blackbird (<u>Agelaius phoeniceus</u>)	DWRC	LD ₅₀	23.7-178
Tricolored blackbird (<u>A. tricolor</u>)	DWRC	ALD	75-316
<u>Fish:</u>			
Rainbow trout (<u>Salmo gairdnerii</u>)	DWRC	LC ₅₀	0.5
Carp (<u>Cyprinus carpio</u>)	DWRC	LC ₅₀	0.3
Channel catfish (<u>Ictalurus punctatus</u>)	DWRC	LC ₅₀	0.5
Black bullhead (<u>I. melas</u>)	DWRC	LC ₅₀	0.4
Bluegill (<u>Lepomis macrochirus</u>)	DWRC	LC ₅₀	0.8
Yellow perch (<u>Pecca flavescens</u>)	DWRC	LC ₅₀	0.6

*DWRC = unpublished data on file with the Bureau of Sport Fisheries and Wildlife, Denver Wildlife Research Center, Denver, Colorado 80225.

**Calif. = California Department of Game and Fish, Wildlife Investigations Laboratory. 1962. Economic Poisons (Pesticides) Investigations. Job Completion Report, Pittman-Robertson Wildlife Restoration Project No. W-52-B-6. 10 pp.

dogs, and cats resulted in minimal secondary poisoning. Tests showed that the toxicant was in the nutria stomachs. Although one dog and one cat were killed by eating stomach contents (Evans, pers. comm.), hazards to free-roaming dogs, cats, and mink were considered negligible. Golden eagles (Aquila chrysaetos canadensis), great horned owls (Bubo virginianus), and coyotes (Canis latrans) receiving multiple feedings of poisoned jackrabbits showed no visible symptoms of secondary intoxication (Evans et al. 1970). In other studies by Denver Center personnel, mink fed poisoned prairie dogs for 30 days showed no ill effects. Kit foxes fed poisoned kangaroo rats vomited, then reconsumed the rats and survived.

These data indicate that a potential for secondary poisoning exists but varies according to the zinc phosphide residues in the primary target animals, the food habits of the secondary species, and their susceptibility to zinc phosphide. In general, hazards are considered minimal to all nontarget species tested, except perhaps cats and dogs, which may succumb if they eat stomachs and intestines.

ENVIRONMENTAL ASPECTS

Zinc phosphide and phosphine residues in sugarcane and their fate in soils and water were studied by Hilton et al. (1971) and Robison and Hilton (1971). They found that: (1) free phosphine does not exist in, and is not adsorbed on, cane, and residues were from surface contamination with zinc phosphide; (2) amount of residues was influenced by rainfall (3) phosphine in contact with sugarcane reacted to form water-soluble, nonvolatile forms of phosphorous; (4) recoveries of phosphine from analysis of zinc phosphide in sugarcane, sugar, molasses, and soils were always less than theoretical, indicating transformation; (5) small traces of phosphine could be detected in sugarcane 3 months after the last of four aerial bait applications at above-normal rates; (6) for normal application rates, residues were within the tolerance limits (0.01 ppm); and (7) zinc phosphide decomposed

quite rapidly in soils--faster in moist soils than in dry soils. In laboratory experiments with soil, phosphine was reabsorbed and oxidized to phosphate ions almost as fast as it formed; oxidation rates differed among soil types. Studies with radioactive phosphine indicated that: (1) it decomposed slowly in water; (2) was absorbed by roots and leaves and translocated as $^{32}\text{P}0_4$ ions; (3) was absorbed rapidly and completely by soils; and (4) in contact with oat bait, formed considerable amounts of nonvolatile phosphorous compounds. Van Wazer (1958;123-131, 179-219) describes other reaction properties of phosphine useful in determining its fate in the environment.

Tests by Hilton et al. (1971) also showed that weathering of toxicant from bait in Hawaiian sugarcane fields was primarily a physical process caused by rainfall. In one test, about 60 percent of the toxicant was removed by an inch of rain. We found that baits applied in humid sugarcane fields become moldy and disintegrated after about 3 weeks, reducing environmental contamination and potential primary hazards. Under other less severe weathering conditions, zinc phosphide baits have remained toxic for at least 9 months (Elmore and Roth 1943; Guerrant and Miles 1969).

RISKS

The "ideal" toxicant does not exist, and we must recognize that some risks are inherent with the use of those available--including zinc phosphide. Probably the greatest risks of using zinc phosphide cereal baits are primary hazards to gallinaceous birds and waterfowl. A problem analysis should be made for each proposed use to determine if risks can be held to an acceptable level. The analysis should include an evaluation of primary and secondary hazards, environmental impact, and the need for control. Careful attention should be given to proper bait formulation, methods and rates of application, and when, where, and how treatments are made. It is unrealistic to make blanket recommendations as to how rodent control can be safely achieved.

CURRENT RESEARCH FOR REGISTRATION

The registration of a rodenticide for use in food crops is difficult, time consuming, and costly--estimates range from 1/4 million to 1 million dollars. The studies necessary to provide data supporting the establishment of tolerances and registration are too numerous to discuss here. In addition, the data required by the FDA and EPA are not always clearly defined because each compound and its uses are unique in some aspects and judgments are based on test results. If data turn out to be inadequate, additional studies are required, delaying registration and increasing costs.

Basically, there is a better chance of registering zinc phosphide than 1080 or strychnine for field rodent control because: (1) it has a long history of use, and a minimum of efficacy data is required; (2) it is now registered, with a tolerance, for use in Hawaiian sugarcane; (3) considerable data on phosphine are available; and (4) suitable analytical techniques for residues have been developed. The Bureau is conducting research, some in conjunction with other cooperating agencies, to extend the registration of zinc phosphide to other situations. We are concentrating on registrations for controlling ground squirrels and voles in alfalfa, sugarbeets, artichokes, and rangeland (California); four species of rodents in sugarcane (Florida); jackrabbits along cropland-rangeland borders (Idaho); prairie dogs in rangeland (Colorado); and possibly three species of rats in macadamia nuts (Hawaii). Typically, evaluations involve:

1. Toxicology studies--to determine the LD_{50} 's for the target species and for the nontarget species of greatest concern for each proposed use.
2. Efficacy studies of bait formulations and methods of bait application--to develop and evaluate operational recommendations and instructions for the proposed label.
3. Chemical and translocation studies--to determine residues in plants and soil. Data must be obtained for all proposed uses if crop types, soil types, and climatic conditions differ from the current food crop registration. For example, analyses for phosphine are run on samples collected on days 1, 15, and 30 after baiting at a normal and two exaggerated application rates. Such data are used to establish tolerances and baiting cut-off periods before harvest.

4. Bait weathering and hazard studies - to determine hazards of the proposed use. These studies include bait acceptance trials and surveys for occurrence and mortality of nontarget species.

In addition, studies on the effects of zinc phosphide on stream fauna and water quality are planned in Hawaii.

It is possible to register a compound for multiple uses in agricultural crops by submitting one application and appropriate data. The various uses are then stated on the label or labels accompanying the registration. Most of the work covering the proposed uses of zinc phosphide is in progress and we hope to begin preparing petitions early next year. At best, registration could be issued as early as mid-1973. Even after registration (and we hope that we are not overoptimistic), additional research will be required to extend registrations to other pest rodent situations and to develop techniques to improve efficacy and minimize hazards.

LITERATURE CITED

- ANON. 1967. Zinc phosphide. Nat. Pest Control Ass., Tech. Release 17-67. 6 pp.
- ANON. 1968. Vertebrate pest control handbook. Calif. Dep. Agr. 95 pp.
- ANON. 1971. Pesticide use report--1970. Calif. Dep. Agr. 107 pp.
- BLAXLAND, J. D., and R. F. GORDON. 1945. Zinc phosphide poisoning in poultry. Vet. J. 101:108-110.
- CHITTY, D., and H. N. SOUTHERN. 1954. Control of rats and mice, vol. 1. Oxford Univ. Press, London. 305 pp.
- DOTY, R. E. 1945. Rat control on Hawaiian sugarcane plantations. Hawaiian Planters Record 49:71-239.
- ELMORE, J. W., and F. J. ROTH. 1943. Analysis and stability of zinc phosphide. J. Ass. Official Anal. Chem. 26:559-564.
- EVANS, J. 1970. About nutria and their control. USDI, Bureau of Sport Fisheries and Wildlife, Resource Pub. 86. 65 pp.
- _____, P. L. HEGDAL, R. E. GRIFFITH, JR. 1970. Methods of controlling jackrabbits, pp. 109-116. In Proc. 4th Vert. Pest Conf., West Sacramento, Calif., March 3-5.
- GLEASON, M. N., R. E. GOSSELIN, H. C. HODGE, and R. P. SMITH. 1969. Clinical toxicology of commercial products. Williams & Wilkins Co., Baltimore, 3rd ed., 1428 pp.
- GUERRANT, G. O., and J. W. MILES. 1969. Determination of zinc phosphide and its stability in rodent baits. J. Ass. Official Anal. Chem. 52:662-666.
- HAYNE, D. W. 1951. Zinc phosphide: its toxicity to pheasants and effect of weathering upon its toxicity to mice. Michigan Agr. Exp. Sta. Quart. Bull. 33(4):412-425.
- HILTON, H. W., R. D. NASS, W. H. ROBISON, and A. H. TESHIMA. 1971. Zinc phosphide as a rodenticide for rats in Hawaiian sugarcane. In Proc. 14th Congress, Int. Soc. Sugar Cane Technol., New Orleans, La., November. (in press)
- HOFFER, M. C, P. C. PASSOF, and R. KROHN. 1969. Field evaluation of DRC-714 for deer-mouse control in a Redwood habitat. J. Forestry 67(3):158-159.
- JACOBS, M. B. 1967. The analytical toxicology of industrial inorganic poisons. Intelscience Publ., New York. 943 pp.
- JANDA, J., and M. BOSSEOVA. 1970. The toxic effects of zinc phosphide baits on partridges and pheasants. J. Wildl. Manage. 34 (1):220-223.
- KEITH, J. O., and E. J. O'Neill. 1964. Investigations of a goose mortality resulting from the use of zinc phosphide as a rodenticide. Unpub. Rep., U.S. Bureau of Sport Fisheries and Wildlife, Denver, Colorado. 7 pp. (Mimeo)
- KLIMMER, O. R. 1969. Beitrag zur Wirkung des Phosphorwasserstoffes (PH₃) . Archiv fur Toxikologic 24(2/3):164-187.(Translation)
- LEOPOLD, S. A. 1964. Predator and rodent control in the United States, pp. 27-49. In Trans. 29th N. American Wildl. Natur. Res. Conf., Las Vegas, Nevada, March 9-11.
- PRZYGODDA, W. 1961. Feldmausbekämpfung und Vogelwelt. Deutsche Vogelwelt 72:106-111.
- RICHENS, V. B. 1967. The status and use of Gophacide, pp. 118-125. In Proc. 3rd Vert. Pest Conf., San Francisco, Calif., March 7-9.
- ROBERTSON, A., J. G. CAMPBELL, and P. GRAVES. 1945. Experimental zinc phosphide poisoning in fowls. J. Comp. Pathol. Therapeutics 55:290-300.
- ROBISON, W. H., and H. W. HILTON. 1971. Gas chromatography of phosphine derived from zinc phosphide in sugarcane. Agr. Food Chem. 19(5):875-878.
- RUDD, R. L., and R. E. GENELLY. 1956. Pesticides: their use and toxicity in relation to wildlife. Calif. Dep. Fish and Game, Bull. 7. 209 pp.
- SCHOOFF, H. F. 1970. Zinc phosphide as a rodenticide. Pest Control 38(5):38, 42-44.
- SCHROEDER, M. H. 1967. Gophacide, a candidate for control of Ord's kangaroo rat. J. Wildl. Manage. 31(2):339-341.

- STORER, T. I., and E. W. JAMESON, JR. 1965. Control of field rodents in California. Div. Ag. Sci., Univ. Calif., Circ. 535. 50 pp.
- VAN WAZER, J. R. 1958. Phosphorus and its compounds, vol. I. Interscience Publ., New York.
- VON OETTINGEN, E. W. 1947. The toxicity and potential dangers of zinc phosphide (phosphine). Public Health Rep. 203:1.
- WARD, A. L., P. L. HEGDAL, V. B. RICHENS, and H. P. TIETJEN. 1967. Gophacide, a new pocket gopher control agent. J. Wildl. Manage. 31(2):332-338.