



United States
Department of
Agriculture

Animal and
Plant Health
Inspection
Service

DWRC
Research Report
No. 11-55-005

Primary and Secondary Hazards of Zinc Phosphide to Nontarget Wildlife— A Review of the Literature

Abstract: When zinc phosphide, an inorganic acute rodenticide, reacts with water and hydrochloric acid in the gastrointestinal (GI) tract of an animal, it forms the highly toxic gas phosphine. This paper provides a review of the toxicity, primary hazards, and secondary hazards of zinc phosphide.

At least 61 acute oral toxicity studies, representing 28 species of mammals and 16 species of birds, have been conducted on zinc phosphide. Based on these toxicity data, zinc phosphide is toxic to both mammals and birds. It is several times more toxic to rodents than to carnivores. Of the bird species tested, waterfowl and gallinaceous birds appear the most sensitive, with geese being the most susceptible of all wild birds to primary zinc phosphide poisoning. Some passerines, such as red-winged blackbirds, also appear relatively sensitive. Several factors, including prior exposure to untreated bait, nutritional condition of the bird, availability of alternate food sources, and ability to regurgitate treated baits, influence the magnitude of primary hazards.

The many secondary toxicity studies conducted on mammalian predators, raptors, and reptiles generally indicate low risk of secondary toxicity. Because zinc phosphide does not accumulate in muscle tissue of poisoned animals, no true secondary poisoning occurs. Death caused by eating animals poisoned with zinc phosphide results from primary ingestion of zinc phosphide remaining in the GI tract. The low risk of secondary intoxication with zinc phosphide can also be attributed to the following factors: (1) Mammalian predators are less susceptible to zinc phosphide than other species; (2) the strong emetic action of zinc phosphide reduces secondary risks; and (3) most animals refuse to eat the GI tracts of poisoned animals.

Field studies to determine effects of zinc phosphide on nontarget wildlife have generally found no significant effects, but operational zinc phosphide applications have occasionally killed nontarget wildlife. Most of these incidents have involved misuse of zinc phosphide or application rates and concentrations that were much higher than current labelled rates.

Key words: zinc phosphide, rodenticide, primary hazards, secondary hazards, toxicity

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Issued September 1994

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Introduction

Zinc phosphide (Zn_3P_2) is an inorganic acute rodenticide. Toxicity occurs when this compound reacts with water and hydrochloric acid (HCl) in the GI tracts of poisoned animals to form the highly toxic gas phosphine (Chitty and Southern 1954, Henderson and Boggess 1979). Zinc phosphide is relatively nonselective and has been used to control a variety of rodent pests. Marsh (1988) described the history of zinc phosphide use. Synthesized in 1740, zinc phosphide was first used as a rodenticide in Europe in 1911. It was first used in the United States around 1939–40 for control of commensal rodents. Its use for control of field rodents began in the early 1940's and expanded after World War II.

Current labelled uses include control of rats (*Rattus* spp.), house mice (*Mus musculus*), voles (*Microtus* spp.), prairie dogs (*Cynomys* spp.), ground squirrels (*Spermophilus* spp.), deer mice (*Peromyscus* spp.), meadow jumping mice (*Zapus hudsonius*), pocket gophers (*Geomys* and *Thomomys* spp.), moles (Talpidae), kangaroo rats (*Dipodomys* spp.), nutria (*Myocaster coypus*), muskrats (*Ondatra zibethica*), and feral rabbits (Leporidae) (table 1). Zinc phosphide may be applied to sugarcane, macadamia nut orchards, no-till or minimum-till corn, orchards, groves, vineyards, ornamentals, nurseries, wheat, oats, barley, alfalfa, artichokes, sugarbeets, and Timothy hay. Many crop uses (e.g., no-till corn, Timothy hay, oats, barley) are registered as Special Local Needs under Section 24(c) of the Federal Insecticide, Fungicide and Rodenticide Act, and use is restricted to certain States (table 1). Zinc phosphide is also registered for use on noncrop areas, including lawns, golf courses, parks, highway medians, reseeded forest sites, and rangeland, as well as areas adjacent to wetlands where damage by nutria and muskrat is occurring. Zinc phosphide may also be applied in and around homes and industrial, agricultural, and commercial buildings for commensal rodent control.

For most noncommensal species, zinc phosphide is applied using grain (e.g., oats, wheat, cracked corn) as the carrier or as a pelleted formulation. A tracking powder formulation is registered for control of commensal rodents indoors, while treated apples, carrots, or sweet potatoes may be used for nutria and muskrat control. Currently labelled formulations or baits generally contain 1–2 percent zinc phosphide (Marsh 1988). Depending on the target species and area being treated, zinc phosphide can be broadcast from the air, from ground-driven devices (e.g., cyclone spreaders), or by hand. Zinc phosphide can also be placed in or around burrows, underground, in artificially created trails, and in tamperproof bait boxes.

Acute Oral Toxicity

Acute oral toxicity values have been determined for a wide variety of target mammals as well as for nontarget mammals and birds (table 2). LD_{50} values for mammals ranged from 5.6 mg of zinc phosphide per kilogram of body weight for nutria (Hood 1972) to 93 mg/kg for kit fox (*Vulpes macrotis*) (Schitoskey 1975). LD_{50} 's determined for a variety of birds ranged from 7.5 mg/kg for white-fronted geese (*Anser albifrons*) (California Department of Fish and Game 1962 unpubl.) to 67.4 mg/kg for mallards (*Anas platyrhynchos*) (Matschke and Higgins 1978a unpubl.).

Lethal dietary concentrations (LC_{50} 's) determined for northern bobwhite quail (*Colinus virginianus*) ranged from 468.5 parts per million (p/m) (Matschke 1978 unpubl.) to 849 p/m (U.S. Fish and Wildlife Service 1977 unpubl.). The LC_{50} for coturnix quail (*Coturnix japonica*) was 960 p/m (Hill and Camardese 1986), and for mallards, 2,885 p/m (U.S. Fish and Wildlife Service 1977 unpubl.).

Based on these toxicity data and on classifications in Smith (1987), zinc phosphide is highly to extremely toxic to both mammals and birds. Zinc phosphide is 2–15 times more toxic to rodents than to carnivores (Hill and Carpenter 1982). Of the bird species tested, waterfowl and gallinaceous birds appear the most sensitive. According to Marsh (1985), geese may be the most susceptible to primary zinc phosphide poisoning of all wild birds as shown by the low LD_{50} 's.

Table 1—Current labelled uses for zinc phosphide in the United States

| Target species | Area treated |
|--|--|
| Rats (<i>Rattus</i> spp.) | Sugarcane; macadamia nut orchards; homes; agricultural, industrial, and commercial buildings |
| House mice (<i>Mus musculus</i>) | Homes; agricultural, industrial, and commercial buildings; no-till or minimum-till corn (Ohio only) |
| Voles (<i>Microtus</i> spp.) | Orchards and groves; ornamentals; vineyards; wheat; no-till or minimum-till corn (Ohio and Indiana only); alfalfa (Washington only); Timothy hay (Washington only); lawns and golf courses; parks; nurseries; highway medians; forest reseedings |
| Prairie dogs (<i>Cynomys</i> spp.) | Rangeland, pastures (New Mexico only) |
| Ground squirrels (<i>Spermophilus</i> spp.) | Rangeland and noncrop areas. In Montana only: pastures, alfalfa, wheat, oats, barley. |
| Deer mice (<i>Peromyscus</i> spp.) | Vineyards, orchards, wheat; forest reseedings; highway medians |
| Meadow jumping mice (<i>Zapus hudsonius</i>) | Vineyards |
| Pocket gophers (<i>Geomys</i> and <i>Thomomys</i> spp.) | Alfalfa; noncrop areas; lawns and golf courses |
| Moles (Talpidae) | Alfalfa; noncrop areas; lawns and golf courses |
| Kangaroo rats (<i>Dipodomys</i> spp.) | Rangeland, noncrop areas |
| Nutria (<i>Myocaster coypus</i>) | Wetlands |
| Muskrats (<i>Ondatra zibethica</i>) | Wetlands |
| Feral rabbits (Leporidae) | Destruction Island (WA) only |

Table 2—Acute oral toxicity of zinc phosphide to vertebrates

| Species | Test | Toxicity | Source |
|---|------------------|--------------|--|
| | | <i>Mg/kg</i> | |
| Target mammals | | | |
| Black-tailed jackrabbit (<i>Lepus californicus</i>) | LD ₅₀ | 8.2 | Matschke and LaVoie (1976 unpubl.) |
| California ground squirrel (<i>Spermophilus beecheyi</i>) | LD ₅₀ | 33.1 | Hood (1972) |
| | LD ₅₀ | 22.1 | U.S. Fish and Wildlife Service (unpubl.) |
| Richardson's ground squirrel (<i>S. richardsonii</i>) | LD ₅₀ | 62.0 | Matschke and LaVoie (1976 unpubl.) |
| Black-tailed prairie dog (<i>Cynomys ludovicianus</i>) | LD ₅₀ | 18.0 | Tietjen (1976) |
| Northern pocket gopher (<i>Thomomys talpoides</i>) (<i>T. t. quadratus</i>) | LD ₅₀ | 6.8 | Hood (1972) |
| | ALD | 28.0 | Hood (1972) |
| Plains pocket gopher (<i>Geomys bursarius</i>) | LD ₅₀ | 63.0 | U.S. Department of the Interior, Bureau of Indian Affairs (1975 unpubl.) |
| Kangaroo rat (<i>Dipodomys spectabilis</i>) | ALD | 8.0 | Hood (1972) |
| Deer mouse (<i>Peromyscus maniculatus</i>) | ALD | 42.0 | Hood (1972) |
| Meadow vole (<i>Microtus pennsylvanicus</i>) (<i>M. californicus</i>) | LD ₅₀ | 18.0 | Hood (1972) |
| | LD ₅₀ | 15.7 | Hood (1972) |
| Prairie vole (<i>M. ochrogaster</i>) | LD ₅₀ | 16.2 | Bell (1972) |
| House mouse (<i>Mus musculus</i>) | LD ₅₀ | 25.8 | Bell (1972) |
| White rat (<i>Rattus</i> sp.) | LD ₅₀ | 55.1 | Matschke and LaVoie (1976 unpubl.) |
| | LD ₅₀ | 41.3 | Chitty and Southern (1954) |
| Black rat (<i>R. rattus</i>) (<i>R. r. mindanensis</i>) | LD ₅₀ | 21.3 | Pank (1972 unpubl.) |
| | LD ₅₀ | 28.5 | Matschke and LaVoie (1976 unpubl.) |
| Grey-bellied rat (<i>R. r. alexandrinus</i>) | LD ₅₀ | 27–47 | Tongtavee (1978) |
| White-bellied rat (<i>R. r. frugivorus</i>) | LD ₅₀ | 22–49 | Tongtavee (1978) |

Table 2—Acute oral toxicity of zinc phosphide to vertebrates (cont'd)

| Species | Test | Toxicity | Source |
|---|------------------|----------|---|
| | | Mg/kg | |
| Norway rat (<i>R. norvegicus</i>) | LD ₅₀ | 27.0 | Pank (1972 unpubl.) |
| | LD ₅₀ | 40.0 | Schoof (1970) |
| | LD ₅₀ | 40.5 | Dieke and Richter (1946) |
| Polynesian rat (<i>R. exulans</i>) | LD ₅₀ | 23.1 | Pank (1972 unpubl.) |
| Ricefield rat (<i>R. argentiventer</i>) | LD ₅₀ | 35.0 | Hood (1972) |
| Grass rat (<i>Arvicanthis niloticus</i>) | LD ₅₀ | 25–40 | Tongtavee (1978) |
| Cairo spiny mouse (<i>Acomys cahirinus</i>) | LD ₅₀ | 21–32 | Tongtavee (1978) |
| Gerbil (<i>Meriones hurrianae</i>) | LD ₅₀ | 35.0 | Tongtavee (1978) |
| (<i>Tatera Indica</i>) | LD ₅₀ | 35.0 | Tongtavee (1978) |
| Nutria (<i>Myocaster coypus</i>) | LD ₅₀ | 5.6 | Matschke and LaVoie (1976 unpubl.) |
| | LD | 20.0 | Evans (1965 unpubl.) |
| Muskkrat (<i>Ondatra zibethica</i>) | ALD | 29.9 | Evans et al. (1966a unpubl.) |
| Mongoose (<i>Herpestes auropunctatus</i>) | LD ₅₀ | 82.0 | Keith et al. (1987 unpubl.) |
| Nontarget mammals | | | |
| Dog (<i>Canis familiaris</i>) | ALD | 40.0 | Matschke and LaVoie (1976 unpubl.) |
| Cat (<i>Felis domesticus</i>) | ALD | 40.0 | Matschke and LaVoie (1976 unpubl.) |
| Kit fox (<i>Vulpes macrotus mutica</i>) | LD ₅₀ | 93.0 | Matschke and LaVoie (1976 unpubl.) |
| Cow (<i>Bos taurus</i>) | LD ₅₀ | 50.0 | National Pest Control Association (1967) |
| Sheep (<i>Ovis aries</i>) | LD | 60–70 | Nekrasova (1964) |
| Nontarget birds | | | |
| Canada goose (<i>Branta canadensis</i>) | LD ₅₀ | 12.0 | Glahn and Lamper (1983) |
| White-fronted goose (<i>Anser albifrons</i>) | LD ₅₀ | 7.5 | California Department of Fish and Game (1962 unpubl.) |
| Snow goose (<i>A. caerulescens</i>) | LD ₅₀ | 8.8 | California Department of Fish and Game (1962 unpubl.) |

Table 2—Acute oral toxicity of zinc phosphide to vertebrates (cont'd)

| Species | Test | Toxicity Mg/kg | Source |
|--|------------------|-------------------|---|
| Mallard (<i>Anas platyrhynchos</i>) | LD ₅₀ | 35.7 | Tucker (1969 unpubl.) |
| | LD ₅₀ | 13.0 | California Department of Fish and Game (1962 unpubl.) |
| | LD ₅₀ | 67.4 | Matschke and Higgins (1978a unpubl.) |
| Golden eagle (<i>Aquila chrysaetos</i>) | LD ₅₀ | >20 | Hudson et al. (1984) |
| Gray partridge (<i>Perdix perdix</i>) | LD ₅₀ | 26.7 | Janda and Bosseova (1970) |
| Northern bobwhite (<i>Colinus virginianus</i>) | LD ₅₀ | 12.9 | Matschke and Higgins (1978b unpubl.) |
| California quail (<i>Lophortyx californica</i>) | LD ₅₀ | 13.5 | California Department of Fish and Game (1962 unpubl.) |
| Coturnix quail (<i>Coturnix japonica</i>) | LD ₅₀ | 35.0 | Ikeda (1971) |
| Ring-necked pheasant (<i>Phasianus colchicus</i>) | LD ₅₀ | 8.8 | Hayne (1951) |
| | LD ₅₀ | 16.4 | Matschke and LaVoie (1976 unpubl.) |
| | LD ₅₀ | 8.8 | California Department of Fish and Game (1962 unpubl.) |
| | LD ₅₀ | 26.7 | Janda and Bosseova (1970) |
| Domestic chicken (<i>Gallus gallus</i>) | MLD | 20–30 | Blaxland and Gordon (1945) |
| | LD | 7–17 | Robertson et al. (1945) |
| | LD ₅₀ | 24–26 | Shivanandappa et al. (1979) |
| Mourning dove (<i>Zenaida macroura</i>) | LD ₅₀ | 34.3 | California Department of Fish and Game (1962 unpubl.) |
| House sparrow (<i>Passer domesticus</i>) | ALD | 20–50 | Matschke and LaVoie (1976 unpubl.) |
| Red-winged blackbird (<i>Agelaius phoeniceus</i>) | LD ₅₀ | 24–178 | Matschke and LaVoie (1976 unpubl.) |
| Tricolored blackbird (<i>A. tricolor</i>) | ALD | 75–316 | Matschke and LaVoie (1976 unpubl.) |
| Horned lark (<i>Eremophila alpestris</i>) | LD ₅₀ | 47.2 | Hudson et al. (1984) |

Laboratory and Pen Studies

Primary Hazards

Primary hazard studies with zinc phosphide have been conducted on many species of birds. Approximately 18–25 wheat kernels treated with 2.5 percent zinc phosphide were enough to kill a ring-necked pheasant (*Phasianus colchicus*), while just 6–9 such kernels sufficed to kill a gray partridge (*Perdix perdix*) (Janda and Bosseova 1970). Further studies showed that this bait remained lethal to pheasants and partridges up to 4 weeks after application. Only 1 of 15 red-winged blackbirds (*Agelaius phoeniceus*) survived after being fed a 1:1 mix of cracked corn treated with 2 percent zinc phosphide and untreated cracked corn for 48 hours (Schafer et al. 1970 unpubl.). Caged spotted doves (*Streptopelia chinensis*) consumed as many as 35 kernels of oat groats treated with 2 percent zinc phosphide without dying (Hilton et al. 1972, Pank et al. 1972 unpubl.). These authors attributed lack of mortality to the emetic properties of zinc phosphide, which caused the doves to regurgitate approximately 1 hour after eating the bait. The fact that the doves ate 10 times as much untreated oat groats as treated groats is evidence that birds are repelled by zinc phosphide. When spotted doves were exposed to zinc-phosphide-treated oats after becoming accustomed to untreated oats, 40- to 60-percent mortality occurred (Pank et al. 1975 unpubl.).

Hines and Dimmick (1970) provided oat bait treated with 1.5 percent zinc phosphide to a group of 25 penned northern bobwhite quail for 10 consecutive days. Another group of 25 quail was provided the same formulation for 5 consecutive days. Two of the quail provided the bait for 10 days were killed: 1 on the first day (after consuming 18 kernels), and 1 on the fifth day. None of the birds provided bait for 5 days died during the study. When not fasted prior to the test, crowned guinea fowl (*Numida meleagris*) and laughing doves (*Streptopelia senegalensis*) fed zinc phosphide bait dyed green or black or left undyed refused to eat the bait (Siegfried 1968). However, four laughing doves that fasted for 30 hours prior to receiving zinc-phosphide-treated wheat readily consumed the bait. Although they regurgitated approximately 20 minutes later, all four died about 2 hours after eating.

Glahn and Lamper (1983) placed groups of either two captive Canada geese (*Branta canadensis*) or two white-fronted geese in alfalfa or fescue hay enclosures containing oat groats treated with 1 percent zinc phosphide at rates of 6.7, 20.1, and 67 kg/ha. After 4 days, one Canada goose in each of the fescue enclosures treated at 6.7 and 20.1 kg/ha had died, while both Canada geese in the fescue enclosure treated at 67 kg/ha had died. None of the Canada or white-fronted geese in the alfalfa enclosures treated with zinc phosphide at the same rates died. Birds in the fescue hay enclosure consumed more bait than birds in the alfalfa enclosure, presumably because alfalfa provided an alternative feed. Glahn and Lamper concluded that zinc phosphide applications at recommended rates will have minimal impact when alternative food sources are available.

The LD₅₀ of grain treated with 1 percent zinc phosphide was 260 kernels for snow geese (*Anser caerulescens*) and 310 kernels for white-fronted geese (California Department of Fish and Game 1962 unpubl.). In studies with captive snow geese, all birds survived treatment with 100 kernels of oat groats treated with 1 percent zinc phosphide. All of 3 adults dosed with 200 kernels died, while 1 of 3 immature birds dosed with 200 kernels died. All birds, including immatures, given 300 or more treated kernels died (Keith and O'Neill 1964 unpubl.). According to Keith and O'Neill, a 5-lb goose could eat about 6,400 kernels per feeding. Therefore, it is possible for geese to consume several lethal doses in just one feeding.

Results from these studies confirm that waterfowl and gallinaceous birds appear to be the most sensitive species to zinc phosphide poisoning. However, many birds appear capable of distinguishing treated from untreated bait, and they prefer untreated grain when given a choice. Several factors, including prior exposure to untreated bait, nutritional condition of the bird when provided treated baits, availability of alternate food sources, and likelihood of regurgitating treated baits, exert a large influence on the magnitude of effects.

Secondary Hazards

Secondary toxicity studies with zinc phosphide have been conducted using several species, including coyotes (*Canis latrans*) (Evans et al. 1970) and domestic dogs (*C. familiaris*) (Evans et al. 1965 unpubl., 1966b unpubl.); kit fox (Schitoskey 1975), red fox (*Vulpes fulva*), and gray fox (*Urocyon cinereoargenteus*) (Bell 1972, Bell and Dimmick 1975); domestic cats (*Felis domesticus*) (Doty 1945, Chitty and Southern 1954, Evans et al. 1965 unpubl., 1966b unpubl., Tkadlec and Rychnovsky 1990); mink (*Mustela vison*) (Evans et al. 1965 unpubl., 1966b unpubl., Tietjen 1976), least weasels (*M. nivalis*) (Tkadlec and Rychnovsky 1990), Siberian ferrets (*M. eversmanni*) (Hill and Carpenter 1982, 1983), and domestic ferrets (*M. putorius furo*) (Matschke and Andrews 1990 unpubl.); mongooses (*Herpestes auropunctatus*) (Doty 1945, Pank 1972 unpubl.); rats (U.S. Department of the Interior 1970 unpubl.); great-horned owls (*Bubo virginianus*) (Evans et al. 1970, Bell 1972) and spotted eagle owls (*B. africanus*) (Siegfried 1968); bald eagles (*Haliaeetus leucocephalus*) (Evans et al. 1965 unpubl., 1966b unpubl.) and golden eagles (*Aquila chrysaetos*) (Evans 1967 unpubl., Evans et al. 1970); black vultures (*Coragyps atratus*) (Evans et al. 1966b unpubl.); kestrels (*Falco tinnunculus*) (Tkadlec and Rychnovsky 1990); snapping turtles (*Chelydra serpentina*) (Evans et al. 1965 unpubl., 1966b unpubl.); alligators (*Alligator mississippiensis*) (Evans 1970); and gopher snakes (*Pituophis catenifer*) (Brock 1965). Results of these studies indicate that zinc phosphide poses little secondary risk to nontarget wildlife.

Five mink fed for 30 consecutive days 200 g/day of ground prairie dogs killed with oats treated with 2 percent zinc phosphide showed no acute or subacute effects during

the 30-day test or during a 15-day posttreatment observation period (Tietjen 1976). The theoretical maximum daily dose received by each mink, assuming no detoxification by the prairie dogs, was 13.6 mg/kg.

Four Siberian ferrets were fed rats killed with 2 percent bait or with single doses of 40, 80, or 160 mg zinc phosphide at the rate of 1 rat every other day for 10 days (Hill and Carpenter 1982, 1983). The researchers estimated the maximum dosage per feeding was 40–49, 41–45, 75–115, and 155–214 mg/kg for the 2 percent bait and the 40-, 80-, and 160-mg doses, respectively. The only effect noted was regurgitation by one ferret in each group (with the exception of the group given rats killed with the 80-mg dose). After their first experience, the ferrets avoided eating the rats' GI tracts. Subsequent blood chemistry analyses indicated that exposure to zinc phosphide may have resulted in cellular heme degradation, renal damage, and liver disorders. These researchers concluded that secondary exposure of ferrets to zinc phosphide will probably not cause direct mortality if alternative food sources are available. However, effects of long-term exposure on the respiratory system, liver, and kidneys were cited as a potential concern. Sublethal exposure also causes liver damage and hemorrhagic congestion in the lungs of rats (Johnson and Voss 1952).

In another secondary-hazard study, 1 group of 10 domestic ferrets was fed the stomachs, livers, and intestines from prairie dogs killed with a bait treated with 2 percent zinc phosphide. Another group of 10 ferrets was provided the remainder of treated carcasses without the GI tracts (Matschke and Andrews 1990 unpubl.). After a 3-day period, no mortality and no signs of poisoning or emesis occurred in the ferrets, prompting the investigators to conclude that the risk of ferret secondary poisoning from zinc phosphide is low. They attributed the lack of effect to the following factors: (1) 90 percent of the zinc phosphide ingested by the prairie dogs was eliminated by them; (2) 99 percent of the zinc phosphide residues occurred in the prairie dog GI tracts, with none occurring in the muscles; (3) ferrets tended to avoid the GI tracts containing zinc phosphide; and (4) the amount of zinc phosphide required to kill prairie dogs was not enough to kill ferrets that consumed prairie dog tissue.

Common voles (*Microtus arvalis*) killed with baits containing 5 percent zinc phosphide (a concentration much higher than currently labelled uses in the United States) were fed to two domestic cats, two kestrels, and two weasels (Tkadlec and Rychnovsky 1990). One of the cats died after consuming five vole carcasses, even though it regurgitated the voles prior to death. That cat's estimated dose was 37 mg of zinc phosphide per kg body weight. The other cat consumed nine voles, regurgitated them by the next day, and did not show any signs of intoxication. Additional voles were fed to this cat, who soon quit consuming the GI tracts and never displayed any symptoms of zinc phosphide poisoning. The two kestrels were each given one vole per day for 3 consecutive days. No mortality or signs of

toxicity occurred; however, the kestrels did not consume the GI tracts of any of the voles provided. Ninety-eight percent of the zinc phosphide consumed by the voles was restricted to their GI tracts, so the kestrels were not exposed to much zinc phosphide. The two weasels were also provided one vole each per day for 3 days; the weasels refused to eat the GI tracts, and no deaths or signs of intoxication occurred.

To evaluate potential effects of ground squirrel control with zinc phosphide on the endangered San Joaquin kit fox (*Vulpes macrotis mutica*), Schitoskey (1975) fed three closely related desert kit foxes (*V. m. arsipus*) kangaroo rats killed with 480 mg zinc phosphide each, at the rate of one kangaroo rat a day for 3 days. Two of the three foxes ate the rat every day, regurgitated, and then reconsumed the vomitus. The third fox refused to eat the rat until the third day, when it also consumed the rat, vomited, and ate the vomitus. None of the foxes displayed any signs of intoxication, despite the fact that the first two consumed the equivalent of nine LD₅₀ doses during the trial.

Secondary hazards posed by zinc-phosphide-killed voles to red fox, gray fox, and great-horned owls have been investigated (Bell 1972, Bell and Dimmick 1975). Two individuals of each species were fed zinc-phosphide-killed voles for 3 days, resulting in a calculated dose of 10.64 mg/kg for the red foxes, 8.60 mg/kg for the gray foxes, and 22.31 mg/kg for the great-horned owls. The zinc phosphide consumed by the foxes was probably well below the LD₅₀ dose, as the LD₅₀'s for dogs and kit foxes are 40 and 93 mg/kg, respectively. Although none of the predators was killed, one of the red foxes became ill, and a mucous discharge appeared in the feces of one of the gray foxes. The owls also developed significant behavior irregularities (e.g., roosting under rather than on top of shelters, running rather than flying when disturbed). Therefore, secondary exposure to these species could potentially reduce their ability to survive in the wild due to altered behavior.

Golden eagles, great-horned owls, and coyotes provided multiple feedings of black-tailed jackrabbits (*Lepus californicus*) killed with carrot bait treated with 0.75 percent zinc phosphide displayed no visible signs of secondary intoxication (Evans et al. 1970). The eagles did not consume the GI tracts of noneviscerated rabbits (Evans 1967 unpubl.). Three spotted eagle owls provided gerbils (*Tatera afra*) killed with bait treated with 2 percent zinc phosphide for 5, 10, and 40 consecutive days, respectively, avoided eating most of the GI tracts, and no mortalities occurred during the trial (Siegfried 1968).

Norway rats (*Rattus norvegicus*) and Polynesian rats (*R. exulans*) that had been killed with zinc phosphide were fed to four mongooses at the rate of one Norway rat or two Polynesian rats per day for 10 days. The mongooses rarely ate the GI tracts of the rats and gained more weight than three control animals during the test (Pank 1972 unpubl.). Based on these results, Pank believed that use of zinc phosphide for rat control in Hawaiian sugarcane would have little effect on mongooses. In another study to determine secondary effects on mongooses, rats killed with bait treated

with 1 percent zinc phosphide were fed to two mongooses over a 5-week period (Doty 1945). One mongoose consumed five rats while the other consumed seven. No visible effects were detected.

Doty (1945) also fed to each of three cats a rat killed with the same zinc phosphide bait. Two of the cats consumed the whole carcass. No emesis occurred, and no signs of toxicity were noted. In another secondary poisoning study with cats, rats killed after eating zinc phosphide at the rate of 80 mg/kg body weight were fed to six cats. Five cats ate the rats, and, although all five later regurgitated the rats, two died. The two cats that died consumed estimated zinc phosphide doses of 44 and 96 mg/kg, while the three surviving cats consumed doses of 33, 37, and 37 mg/kg (Chitty and Southern 1954). Taylor (1973 unpubl.) fed rats killed with a single acute dose of zinc phosphide (100 mg/kg) to four cats. Each cat received the equivalent of three rats, minus the skins and skeletons. No signs of secondary intoxication were observed; however, the maximum dose received by each cat was less than half the LD₅₀ value for this species.

Of five cats fed a mixture of meat from unpoisoned nutria and stomach contents from poisoned nutria, one died, two regurgitated, and two others displayed no symptoms of intoxication (Evans et al. 1966b unpubl.).

The U.S. Department of the Interior (1970 unpubl.) conducted the only secondary toxicity trial with rodents. River shrimp (*Atya bisulcata*) killed in water containing 250 p/m zinc phosphide were given to four Polynesian rats. In a 24-hour period, three rats each consumed 22 shrimp, and the remaining rat consumed 17. One of the rats died.

To evaluate potential for secondary poisoning from zinc phosphide used for nutria control, Evans et al. (1966b unpubl.) presented nutria killed by zinc phosphide to mink, dogs, cats, bald eagles, black vultures, and snapping turtles. The mink refused to eat zinc-phosphide-killed nutria that had intact GI tracts. These researchers conducted further tests with nutria livers and ground nutria (minus the stomachs and intestines) and observed no effects.

In dogs, the effects of zinc phosphide were highly variable. One dog died after consuming the stomach contents of a zinc-phosphide-killed nutria, while two other dogs that ate nutria stomachs regurgitated and survived. Two additional dogs provided whole carcasses showed no effects; however, they did not consume the GI tracts. Another dog fed livers from nutria and two dogs fed ground nutria (without stomachs and intestines) showed no visible reactions. For 5 months, two dogs consumed muscle tissue, livers, lungs, stomachs, and intestines of nutria killed with zinc phosphide without showing any ill effects (Evans 1965 unpubl.).

Over a 29-day period, an adult bald eagle that ate 13 nutria killed with 275 g of zinc phosphide displayed signs of intoxication once when it regurgitated and became listless for a 4-hour period (Evans et al. 1966b unpubl.). This one effect was attributed to the presence of zinc phosphide on the hairs of one of the nutria provided to the eagle. Two

additional immature bald eagles were each provided 28 nutria killed with zinc phosphide for a period of 28 and 35 days, respectively. The eagles regurgitated whenever they consumed stomach contents but displayed no further effects. Three black vultures fed for 10 to 11 days on nutria killed with zinc phosphide (Evans et al. 1966b unpubl.). The vultures avoided eating the GI tracts and never showed signs of poisoning.

In another experiment, 12 raptors, primarily yellow-billed kites (*Milvus aegyptius parasiticus*) were monitored for 6 months in an area treated with zinc phosphide for gerbil control in South Africa. The birds were apparently not affected, even though they were observed to consume dead and dying gerbils on a daily basis (W. R. Siegfried 1973, pers. comm. to L. Pank). As can be seen, zinc phosphide poses relatively little risk to raptors.

Evans et al. (1966b unpubl.) provided three snapping turtles for 21 days with meat, stomachs, and intestines of nutria killed with zinc phosphide, resulting in a dose to the turtles of 3,700 mg zinc phosphide. The turtles displayed no overt reactions during the trial. Evans (1970) also fed nutria to alligators and concluded that alligators in zinc-phosphide-treated areas would not be affected based on the large amount of the compound required to kill them. Doses ranging from 41.7 to 100 mg/kg elicited no reaction in six alligators; however, higher doses (150–250 mg/kg) killed all four alligators tested at those levels. In another study to evaluate potential for secondary zinc phosphide poisoning of reptiles, 11 zinc-phosphide-killed mice were fed to 8 gopher snakes (Brock 1965). The snakes regurgitated six of the mice but consumed the other five mice without any apparent effects.

Data collected by the Denver Wildlife Research Center indicate that 90 percent of zinc phosphide consumed by Richardson's ground squirrels (*Spermophilus richardsonii*) is metabolized by the time death occurs (Matschke et al. 1978 unpubl.). Matschke and Andrews (1990 unpubl.) analyzed carcasses of black-tailed prairie dogs (*Cynomys ludovicianus*) killed with an oat bait containing 2 percent zinc phosphide, and only 8.9 percent of the zinc phosphide consumed by the prairie dogs was in the carcasses. Virtually all (99.9 percent) of the recovered zinc phosphide occurred in the GI tracts. The remaining zinc phosphide was recovered from the kidneys, gall bladders/livers, and spleens. No zinc phosphide was recovered from the lungs, hearts, or muscle tissues.

In common voles, 98 percent of zinc phosphide consumed remained in the GI tracts, with 90 percent recovered from the stomachs alone (Tkadlec and Rychnovsky 1990). Based on this finding, those authors concluded that the stomachs of poisoned rodents can be considered the only source of secondary exposure to predators. In contrast to the low levels retained by prairie dogs and ground squirrels, however, the voles retained an average of 58 percent of the zinc phosphide upon death. In a similar study, Sterner and Mauldin (1991 unpubl.) measured zinc phosphide residues in the whole carcasses of meadow voles killed with an oat

Field Studies

bait treated with 2 percent zinc phosphide. The residues averaged approximately one-third of the estimated amount ingested. Based on a worst-case scenario using the maximum zinc phosphide ingested by the voles in this study (8.2 mg) and 100-percent retention of residues, Sterner and Mauldin estimated that a small (1-kg) cat or dog would have to ingest approximately five voles in less than 24 hours to consume a lethal dose, a very unlikely field scenario.

In summary, because zinc phosphide does not accumulate in muscle tissue of poisoned animals, no true secondary poisoning occurs. Death caused by eating animals poisoned with zinc phosphide results from primary ingestion of zinc phosphide remaining in the GI tracts. Although low numbers of mammals (particularly cats) and birds have been killed after consuming poisoned animals in the laboratory, the potential risk appears to be very low. The low risk of secondary intoxication with zinc phosphide shown by these studies has been attributed to several factors: (1) Mammalian predators appear less susceptible to zinc phosphide than do other species (Johnson and Voss 1952). (2) The strong emetic action of zinc phosphide reduces secondary risks (Hegdal et al. 1981): mammals, birds, and reptiles have all been shown to eliminate food containing zinc phosphide through emesis. (3) Zinc phosphide decomposes rapidly in the GI tracts of poisoned animals. (4) Most animals, when given a choice, refuse to eat the GI tracts of poisoned animals.

Field studies have been conducted in the United States to evaluate primary effects of zinc phosphide on nontarget wildlife for the following uses: Prairie dogs on rangeland (five studies), Richardson's ground squirrels on rangeland (two studies), jackrabbits (*Lepus* spp.) on rangeland (one study), California ground squirrels (*Spermophilus beecheyi*) on ditchbanks (two studies), rats on ditchbanks (one study), voles in apple orchards (one study), rats in or adjacent to macadamia nut orchards (two studies), and rats in or adjacent to sugarcane (two studies). Information on nontarget hazards has also been gathered from studies regarding the following uses: Voles in alfalfa (one study), rodents in sugarbushes (one study), muskrats in wetlands (one study), nutria in wetlands (one study), and rats in ricefields (one study in Thailand). However, these studies were not specifically conducted to evaluate nontarget effects. Results from both types of studies are presented below.

Rangeland

Several studies have evaluated effects on nontarget wildlife from zinc phosphide applications for controlling prairie dogs and ground squirrels on rangeland. One study (U.S. Fish and Wildlife Service 1979 unpubl.) monitored effects on nontarget wildlife of an experimental Gunnison prairie dog (*Cynomys gunnisoni*) control effort on pine forests near Flagstaff, AZ. Eight colonies were treated over a 2-year period using oats treated with 2 percent zinc phosphide. Researchers applied the treated oats at the rate of 4 g per burrow and assessed impacts to nontarget wildlife by systematically searching treated colonies and nearby areas for carcasses. In 22 person-hours of searching the first year of the study, two pocket gophers (*Thomomys* spp.) and one northern grasshopper mouse (*Onychomys leucogaster*) were found. The researchers considered one of the pocket gopher mortalities to be treatment related based on the presence of oats and the smell of phosphine in the animal's GI tract. The other carcasses were not suitable for analysis. Thirty person-hours of carcass-searching the second year of the study revealed one cottontail rabbit (*Sylvilagus* spp.) mortality attributed to zinc phosphide poisoning. The researchers concluded that some mortality of nontarget rodents associated with prairie dog colonies will occur and cannot be avoided. In the study area, they observed other nontarget species that were apparently not affected, including 13 species of birds, 7 species of mammals, several species of lizards, and 2 species of snakes.

In a similar study, oats treated with 2 percent zinc phosphide were applied to eight colonies of black-tailed prairie dogs on the Pine Ridge Indian Reservation in South Dakota (Tietjen and Matschke 1981 unpubl., U.S. Fish and Wildlife Service 1981 unpubl.). The researchers checked the eight treated areas as well as four control areas for nontarget mortality following treatment. No dead nontarget wildlife were found, although 13 nontarget bird species and 3 species of nontarget mammals were observed in or near treated colonies.

During the development process to register zinc phosphide for prairie dog control, oats treated with 2 percent zinc phosphide were applied at the rates of 4 or 14 g per burrow to 15 colonies of black-tailed prairie dogs in Montana, Colorado, and Nebraska (Tietjen 1976). Although large numbers of birds, primarily horned larks (*Eremophila alpestris*) and mourning doves (*Zenaida macroura*), appeared in and around treated areas, extensive surveys of these areas, adjacent rangelands, and at stock-watering tanks in the general vicinity of treated areas showed no evidence that avian mortality occurred. Based on this, Tietjen concluded that applying oat baits treated with 2 percent zinc phosphide for prairie dog control presented no significant hazards to nontarget wildlife.

Record and Swick (1983 unpubl.) compared effects of zinc phosphide, Compound 1080, and strychnine baits used for control of black-tailed prairie dogs on rangeland in Butte County, SD. The researchers applied zinc phosphide to 4 ha in late September and early October as a 2 percent bait on oats at the rate of 4 g per burrow entrance. The investigators evaluated effects on wildlife by conducting extensive surveys to determine presence of nontarget birds and mammals before and after treatment. Nontarget species considered common in the study area were killdeer (*Charadrius vociferus*), vesper sparrow (*Pooecetes gramineus*), horned lark, and western meadowlark (*Sturnella neglecta*), while less common species included burrowing owl (*Speotyto cunicularia*), ferruginous hawk (*Buteo regalis*), American kestrel (*Falco sparverius*), white-tailed jackrabbit (*Lepus townsendii*), cottontail, rattlesnake (*Crotalus* spp.), and red fox. Although four treatment-related vesper sparrow carcasses were found on the plots treated with zinc phosphide, no significant changes or decreases in nontarget species abundance occurred.

In another study to evaluate effects of zinc phosphide applications for prairie dog control in South Dakota, steam-rolled oats treated with 2 percent zinc phosphide were applied to three prairie dog colonies in September 1983, and an additional three colonies were used as controls (Apa 1985, Deisch 1986, Uresk et al. 1988, Deisch et al. 1990). A grid of Sherman live traps on each site sampled small mammals before and after treatment, and fecal pellet groups indicated cottontail and white-tailed jackrabbit abundance. The investigators monitored avian populations by counting the number of birds seen or heard along a transect located in each colony. Zinc phosphide reduced deer mouse (*Peromyscus maniculatus*) density by 79 percent; however, the difference between treated and control areas was not statistically significant due to high variability in the data. Pellet count data indicated that zinc phosphide had no impact on cottontail and white-tailed jackrabbit abundance. Although no significant differences in the densities of horned lark or other ground-feeding birds occurred, avian census data were highly variable, making it difficult to detect statistically significant changes.

Matschke et al. (1978 unpubl.) evaluated hazards to nontarget wildlife from a Richardson's ground squirrel control operation with zinc phosphide near Kremmling, CO, on three plots treated in late spring and three plots treated in late summer. The researchers monitored three control areas during the same time periods. A steam-rolled oat bait containing 2 percent zinc phosphide and Monastral green (an avian repellent) was applied to each burrow at the rate of 4 g per burrow entrance. Nontarget bird species in the study area included mourning dove, Brewer's blackbird (*Euphagus cyanocephalus*), western meadowlark, common raven (*Corvus corax*), and vesper sparrow. The researchers systematically searched treated and surrounding buffer areas as well as control areas for nontarget wildlife carcasses beginning 1 day prior to and ending 4 days after treatment. A blackbird roosting area near one of the plots was also searched.

Following the spring application, 73.5 person-hours of searching revealed one dead deer mouse. Zinc phosphide residues in the liver of the mouse were <0.01 p/m, but the stomach was not analyzed. During 83 person-hours of searching associated with the late summer application, the researchers found carcasses of two white-tailed jackrabbits, one deer mouse, and one common snipe (*Gallinago gallinago*) on or near treated areas and one vesper sparrow carcass on a control area. All of the nontarget carcasses found, including the vesper sparrow and the snipe (which is not granivorous), tested positive for zinc, indicating that high background levels of zinc or mishandling of the carcasses during analyses may have occurred. Least chipmunks (*Eutamias minimus*) were present on one plot prior to treatment but were not observed after treatment, so potential effects on chipmunks were unknown. The Matschke team concluded that bird populations were not seriously affected because none of the five species observed in the study area was found dead on treated plots.

In another study to monitor effects of Richardson's ground squirrel control with zinc phosphide on nontarget wildlife, a bait treated with 2 percent zinc phosphide was broadcast using a cyclone spreader on sagebrush (*Artemisia* spp.) rangeland in Montana (Matschke et al. 1980 unpubl., 1983). The bait was applied in 6.1-m-wide swaths to three 1-ha plots at the rate of 5.1 kg/ha. Nontarget wildlife observed in or near the treated plots included cottontails, long-tailed weasels (*Mustela frenata*), vesper sparrows, and sage grouse (*Centrocercus urophasianus*). The Matschke team evaluated effects on nontarget wildlife by systematically searching for carcasses the first 4 days following treatment. No nontarget wildlife carcasses were found following 22.3 person-hours of searching.

Evans et al. (1970) conducted several field trials to evaluate a carrot bait treated with 0.75 percent zinc phosphide for jackrabbit control on rangeland. Although jackrabbit carcasses were located, some as far as 4 km from treated areas, no nontarget animal carcasses were found during any of the field trials.

Several rangeland field studies have shown that the potential exists for predators to be exposed to zinc phosphide from eating carcasses, especially during prairie dog and ground squirrel control operations. Following application of a grain bait treated with 2 percent zinc phosphide for control of black-tailed prairie dogs in Montana, 12 percent of the estimated number of prairie dogs killed died above ground (Knowles 1986). Densities of dead prairie dogs on the ground ranged from 0.2 to 1.7 per ha. Avian or mammalian predators scavenged at least 7 of 67 prairie dog carcasses found above ground 24–48 hours after baiting. Tietjen (1976) monitored 25 carcasses of prairie dogs killed by zinc phosphide bait and found all missing by the following morning. Evidence indicated most had been scavenged by coyotes and badgers (*Taxidea taxus*). Despite evidence that badgers were consuming large numbers of prairie dogs killed by zinc phosphide, field crews found no badger or other carnivore carcasses during thorough ground searches following baiting. In a similar study, 13 Gunnison prairie dogs killed by bait treated with 2 percent zinc phosphide found above ground were purposely left in place and monitored (U.S. Fish and Wildlife Service 1979 unpubl.). Most were gone within 1 to 2 days of discovery. Investigators presumed the carcasses were taken by badgers, coyotes, or other mammalian or avian predators.

An estimated 15–22 percent of all Richardson's ground squirrels killed by zinc phosphide baiting during two studies died above ground (Matschke et al. 1978 unpubl.). The actual numbers of carcasses found above ground during the 2 studies were 77 and 162, and the mean number of exposed carcasses ranged from 0.6 to 0.9 per ha. Zinc phosphide residues in the stomachs of 64 Richardson's ground squirrels killed by bait treated with 2 percent zinc phosphide during the 2 studies ranged from 0.01 to 379 p/m, with means for the 2 studies of 84 and 86 p/m. Residues in the livers of 12 ground squirrels ranged from <0.01 to 2.3 p/m. Although badgers fed on the zinc-phosphide-killed Richardson's ground squirrels, no dead badgers were found. Examination of the remains of ground squirrel carcasses indicated that badgers were eviscerating the carcasses prior to consuming them.

In another study, zinc phosphide residues ranged from 0.12 to 599 p/m in stomachs of 14 Richardson's ground squirrels found above ground following a broadcast application of zinc phosphide bait (Matschke et al. 1983). Investigators saw no predators besides long-tailed weasels on the plots and no evidence of secondary mortality. Griffith (1972 unpubl.) observed magpies (*Pica pica*), crows (*Corvus brachyrhynchos*), ravens, and vultures feeding on jackrabbits killed with zinc phosphide but did not detect any effects on these species.

In summary, eight studies have been conducted to evaluate effects on wildlife from rangeland uses of zinc phosphide. Five examined effects of prairie dog control with treated oats, two examined Richardson's ground squirrel control with treated oats, and one examined effects resulting from jackrabbit control using treated carrots. In all these

studies, only one case of avian mortality was documented from zinc phosphide use on rangeland (four vesper sparrows were killed in South Dakota). Low avian mortality associated with rangeland applications can be attributed to the fact that passerines comprise the majority of rangeland birds, and toxicity data indicate that most passerines are less sensitive to zinc phosphide than other groups (e.g., waterfowl and gallinaceous birds). Also, avian density on rangeland is generally lower than on other habitat types, further reducing the opportunity for exposure. Finally, rangeland applications occur primarily in the summer and early fall, when alternative food supplies are generally abundant.

Because many species of rodents are associated with prairie dog and ground squirrel colonies, several instances of mortality to other rodents from zinc phosphide applications have been documented. Most mortality to nontarget rodents, however, has been localized and involved only a few individuals. No cases of secondary poisoning to scavengers from eating poisoned animals have been documented, primarily because scavengers avoid consuming the GI tracts of poisoned animals.

Ditchbanks

Field studies have also been conducted to assess hazards associated with zinc phosphide control of California ground squirrels and rats along ditchbanks in California. While conducting searches on 3.2 km of transects located on ditchbanks and adjacent areas during California ground squirrel efficacy trials, investigators found carcasses of two black-tailed jackrabbits, one pocket mouse (*Perognathus* spp.), and one deer mouse. All of the deaths were considered treatment related (U.S. Fish and Wildlife Service 1976a unpubl.).

To assess effects on birds, the researchers applied rolled oat groats treated with 2 percent zinc phosphide and labelled with Tracerite, an ultraviolet fluorescent marker, to two areas by broadcast baiting and placed bait stations on three additional areas (U.S. Fish and Wildlife Service 1976b unpubl.). The two bird species using ditchbanks most frequently were mourning doves and Brewer's blackbirds. These researchers conducted strip censuses four times prior to and four times after application and detected no significant decreases in numbers of mourning doves, Brewer's blackbirds, or total birds in the areas treated by broadcasting with zinc phosphide bait. The only significant decrease noted in the three sites treated with bait stations was a decrease in mourning doves on one site, which was attributed to an increase in human activity on this area after treatment. Systematic carcass-searching in each of the five treated areas did not produce any wildlife carcasses, although the researchers postulated that birds consuming lethal doses could have flown considerable distances from the treated area before dying. An attempt was made to examine feces for presence of Tracerite, but only 10 samples were collected on all study areas and none contained it. Additionally, five mourning doves, two Brewer's blackbirds,

and one house sparrow (*Passer domesticus*) were shot and analyzed for zinc phosphide residues. No significant levels of zinc occurred in the GI tracts or livers, and no treated grain was found in their crops. The investigators concluded that zinc phosphide bait has no significant effect on local bird populations inhabiting ditchbanks.

Collins (1966) monitored effects on wildlife of an aerial application of oat groats treated with 2 percent zinc phosphide at 9 kg/ha for rat control along ditches and canals in California. Daily searches of routes totalling 20.6 km took place in the 194-ha treated area for 6 days following application. A total of seven ring-necked pheasants, two mallards, two bitterns (*Botaurus lentiginosus*), one squirrel, one mouse, and one rabbit were found dead. Oat groats were found in the crop of one of the ring-necked pheasant carcasses; the other pheasant carcasses were too decayed for analysis. Collins did not report the status of the other wildlife carcasses.

Results of studies conducted on ditchbanks to evaluate effects on wildlife from control of California ground squirrels and rats with zinc phosphide indicate that some small mammals, rabbits, and birds can be killed by the treatment. However, the applications do not appear to result in substantial mortality to nontarget wildlife.

Apple Orchards

An intensive field investigation to monitor effects of zinc phosphide applications in apple orchards on nontarget wildlife, especially ring-necked pheasants and cottontails, was conducted in Michigan in 1975 (Hegdal and Gatz 1977 unpubl.). In all, 385 ha were treated for vole control using both aerial and ground broadcast methods. Cracked corn bait treated with 2 percent zinc phosphide was applied at either 5.6 or 11.2 kg/ha in late October and early November. Hegdal and Gatz determined effects on wildlife by (1) snap-trapping small mammals; (2) searching treated areas for carcasses; (3) collecting cottontails, ring-necked pheasants, northern bobwhite, and mourning doves after the treatment for residue analysis; and (4) monitoring survival of radiomarked cottontails and ring-necked pheasants.

Trapping results indicated that populations of deer mice, house mice, meadow jumping mice, and thirteen-lined ground squirrels (*Spermophilus tridecemlineatus*) decreased following application; however, deer mice populations also decreased on control sites. Investigators searched a total of 272 ha, including entire orchards as well as some of the surrounding cover, for carcasses between 1 and 14 days after application. They conducted an additional carcass search on one site 159 days after treatment. Five deer mice, eight cottontails, and one blue jay (*Cyanocitta cristata*) were found dead. A sick deer mouse was also found and collected. All six of the deer mice contained zinc phosphide residues. One of these was found near a bait spill during the last search, indicating that the bait remained toxic for over 5 months; at that time, zinc phosphide residues in the bait were only 0.11–0.18 percent. Four of the eight cottontail carcasses had tissue suitable for zinc phosphide

analysis, and all four contained zinc phosphide residues. The blue jay also contained zinc phosphide residues. Five cottontails, nine ring-necked pheasants, four northern bobwhites, and four mourning doves were collected by trapping or shooting in or near treated areas following applications. Three of the cottontails and two of the northern bobwhites contained residues of zinc phosphide, indicating sublethal exposure.

Of eight cottontails radiomarked during the study, three were lost prior to treatment. The researchers recovered a radio 10 days after treatment but found no carcass associated with it. They found one dead rabbit 8 days after treatment, but the carcass did not contain zinc phosphide residues. One of the remaining three rabbits was never tracked in a treated area and did not contain residues of zinc phosphide upon collection 15 days after treatment. The other two were frequently tracked in treated areas. The researchers trapped these two individuals 13 and 15 days after treatment, and one contained residues of zinc phosphide. Of 25 pheasants radiomarked in the study area, 12 were lost prior to treatment, 3 were killed by hunters, and 1 was killed by a predator. Only five of the remaining nine were ever tracked in treated areas; one of these five was apparently killed by zinc phosphide. The remaining eight were collected 8–20 days posttreatment and did not contain residues of zinc phosphide. Several Michigan apple growers interviewed by Hegdal and Gatz reported finding dead cottontails and ring-necked pheasants in or near orchards treated with zinc phosphide.

Hegdal and Colvin (1988) evaluated effects on eastern screech owls (*Otus asio*) of rodenticides used for vole control in apple orchards. These investigators noted no relationship between increased exposure to zinc phosphide and mortality of owls.

Based on results of these studies, zinc phosphide applications for vole control in apple orchards will likely result in some mortality to cottontails, gallinaceous birds, and granivorous passerines, but populations of these species will probably not be significantly reduced. There is no evidence to indicate that secondary exposure to raptors or other predators/scavengers is a problem with zinc phosphide use in apple orchards.

Sugarcane

An operational zinc phosphide bait application to sugarcane in Hawaii for rat control was monitored to assess nontarget effects (Pank et al. 1972 unpubl., Hilton et al. 1972). Oat groats treated with 2 percent zinc phosphide were aerially applied at the rate of 5.6 kg/acre. Investigators used marking and resighting birds, call counts, and road surveys to index bird populations prior to and after treatment. They used radiotelemetry to monitor five radiomarked ring-necked pheasants and four golden plovers (*Pluvialis dominica*). Of the 24 birds (representing 7 species) examined for the presence of zinc phosphide bait labelled with Tracerite, 6 had consumed treated bait, showing sublethal exposure. The researchers detected only 1 change in any

of the population indices following application; pheasant crow counts declined from 24 to 10 following baiting. However, rainy weather may have been responsible for the decrease. Using mist nets, the researchers captured 23 birds and marked them prior to application; the researchers captured 18 after application with the same effort. They did not make population estimates due to the small sample sizes. None of the radiomarked birds died from eating the bait, and no carcasses were found during 50 person-hours of searching in the sugarcane and adjacent areas. The investigators concluded that, although birds will consume zinc phosphide bait, mortality resulting from the applications is minimal.

Pank et al. (1975 unpubl.) evaluated hazards associated with zinc phosphide applications in noncrop areas adjacent to sugarcane. The primary objective of the study was to monitor effects on aquatic life inhabiting an intermittent stream in the treated area. Oat groats treated with 1.88 percent zinc phosphide were aerially applied at the rate of 5.6 kg/ha over an 18-m swath on a gulch adjacent to a sugarcane field. The resultant stream contamination did not significantly affect fish or crayfish (*Procambarus clarkii*), but river shrimp populations declined significantly. Pank's team noted no avian mortality or gross changes in numbers of birds but made no formal efforts to evaluate zinc phosphide effects on birds.

Results of these studies indicate that birds associated with sugarcane will consume zinc-phosphide-treated grain, but resultant mortality is low. Applications of zinc phosphide to wetlands may result in mortality of some aquatic invertebrates, but risk associated with zinc phosphide applications appears low.

Macadamia Nut Orchards

Fellows et al. (1978 unpubl.) evaluated the potential for avian exposure to zinc phosphide baits in Hawaiian macadamia nut orchards. Untreated baits were applied by broadcasting or by placing in trees. One day later, diurnal birds had consumed bait at 11.7 percent of the bait spots placed in trees and at 16.7 percent of the broadcast bait spots monitored.

Fellows et al. (1988) conducted a study to assess acute hazards to birds from zinc phosphide applications to macadamia nut orchards in Hawaii. The researchers monitored three 14.2- to 18.2-ha orchard blocks with paired controls. Oat groat bait treated with 1.88 percent zinc phosphide was applied using a truck-mounted blower at the maximum label rate of 5.62 kg/ha. The bait contained Tracerite to label birds that consumed bait. The avian community consisted primarily of northern cardinals (*Cardinalis cardinalis*), spotted doves (*Streptopelia chinensis*), zebra doves (*Geopelia striata*), common mynahs (*Acridotheres tristis*), which are all granivorous, and Japanese white-eyes (*Zosterops japonicus*), which are primarily insectivorous. Northern cardinals and spotted doves comprised 98 percent of all seed-eating birds observed during the study.

Fellows et al. (1988) conducted line-transect surveys to estimate avian density and estimated survival based on the number of birds marked and subsequently recaptured in Kniffin traps and mist nets. The investigators searched approximately 25 percent of the orchard and the adjacent windbreaks for carcasses beginning 5 days prior to and ending 5 days after application. To estimate the percentage of birds consuming bait, they collected fecal samples from captured birds and examined them for presence of Tracerite. The researchers also weighed birds captured before and after application to determine if bait consumption caused weight loss.

Line-transect data indicated that the treatment had no effect on population size of seed-eating birds in the study area. The researchers tagged and released 261 birds during the study and noted no treatment effects based on the 9.23 percent recaptured. The Fellows team found one dead nestling spotted dove and one dead Japanese white-eye during preapplication carcass searches and three dead adult spotted doves in the postapplication searches. One of the spotted doves contained Tracerite, but the other two did not have sufficient tissue to analyze. Of all birds caught in mist nets following application, 56 percent had Tracerite in their feces, whereas 70 percent of northern cardinals contained Tracerite. None of the exposed birds appeared sick, and none sustained significant weight loss. Even though bait was present throughout the study period, the percentage of birds labelled with Tracerite declined after 3 days, which indicated that the birds developed sublethal aversion to the treated grain. The investigators concluded that zinc phosphide bait should pose minimal hazards to birds in macadamia nut orchards, but they cautioned against applying zinc phosphide to macadamia nut orchards where indigenous or endangered seed-eating birds are present.

Pank et al. (1978) evaluated hazards from zinc phosphide baiting in noncrop borders adjacent to macadamia nut orchards in Hawaii. They selected six orchard blocks for study, each approximately 5.3 ha and bordered on at least three sides by uncultivated land. They applied oat groat bait treated with 1.88 percent zinc phosphide and Tracerite to the noncrop borders at 5.6 kg/ha over a 9.1-m swath. The researchers conducted carcass searches, trapping, and avian surveys to assess effects on birds and found no significant effect on numbers of birds observed or caught in traps. One dead cardinal (*Richmondia cardinalis*), positive for Tracerite, was found during the carcass searching; however, evidence indicated that consumption of anticoagulant rodenticides present in the area may have contributed to its death.

Data from studies conducted in macadamia nut orchards show that granivorous birds readily accept zinc-phosphide-treated grain baits. Although some treatment-related avian mortality could potentially occur with this use, avian populations should not decrease as a result of the treatment.

Ricefields

Tongtavee et al. (1987) monitored applications of a rice bait treated with 0.8 percent zinc phosphide for rodent control near a village in Thailand. The zinc phosphide bait was applied to ricefields in small heaps 10 m apart, and the same areas were retreated 14 days later. The investigators censused all domestic animals in the village before and after treatment and surveyed 6-km transects in the treated fields to estimate diurnal bird abundance and species composition. They surveyed transects before treatment and three times after treatment within 35 days of the first application. The investigators monitored barn owl (*Tyto alba*) roosts in the associated village and searched ricefields and adjoining areas for carcasses the first 4 days after each application. No wildlife carcasses were found on the plots treated with zinc phosphide, and no apparent changes occurred in avian abundance or species richness. Three dogs (3.9 percent of population), nine chickens (3.8 percent of population), and three ducks (2.5 percent of population) living in the village died as a result of the zinc phosphide applications. Tongtavee et al. concluded that neither wildlife nor domestic animal populations were significantly affected by the treatment.

Alfalfa

A live-trapping study conducted in Nebraska showed little risk to small mammals from applications of bait treated with zinc phosphide to alfalfa (Rutgers University 1985). The study was conducted to obtain registration of zinc phosphide for vole control in alfalfa at bait-application rates of 5–10 lb/acre. Over 100 trap-nights, captures of deer mice increased from an average of 5.6 per night before application to 6.6 afterwards, while captures of all small mammals increased on average from 6.8 to 8.6. Calculations were made based on captures of 52 deer mice, 6 *Microtus* spp., 4 *Sorex* spp., 2 *Onychomys* spp., 1 *Reithrodontomys* sp., and 1 *Spermophilus* sp.

Sugarbushes

May et al. (1991 unpubl.) monitored effects of zinc phosphide applications to two 0.81-ha plots in Vermont sugarbushes. The researchers applied a total of 5.67 kg of zinc-phosphide-treated grain to the two sites from late summer to early fall. They placed baits in bait boxes wired to trees for squirrel control and in polyvinyl chloride pipe attached to the base of trees for control of chipmunks, mice, and voles. Squirrel populations decreased, but results of trapping before and after treatment indicated no short-term impacts to the other rodent communities. No nontarget deaths were observed.

Wetlands

Scientists with the U.S. Fish and Wildlife Service (1967a–c unpubl.) studied the effects of muskrat baiting operations in several locations in Arkansas, Louisiana, and South Carolina. Apples, carrots, or potatoes treated with 0.75 percent zinc phosphide were placed along levees, ditchbanks, and in one case along a pond in a golf course. The researchers found no nontarget wildlife mortalities during examinations of any of the treated areas, but formal searches for nontarget mortalities were not conducted. Nontarget species known to occur in the study areas included fish, ducks, rabbits, raccoons (*Procyon lotor*), mink, skunks (*Mephitis mephitis*), opossums (*Didelphis virginianus*), and red foxes. Use of zinc phosphide for nutria control has resulted in muskrat mortality in the Southeastern United States (Evans et al. 1966a unpubl.); in that case, muskrats were considered a nontarget species.

While evaluating effects of nutria control with zinc phosphide on wildlife, Evans et al. (1965 unpubl.) observed dogs, opossums, raccoons, turtles, and blue crabs feeding on zinc-phosphide-killed nutria, apparently without ill effects. In Louisiana, 25 carcasses of nutria killed with a carrot bait treated with 0.75 percent zinc phosphide were placed near a roost containing 30–40 black vultures. The vultures consumed all carcasses within 12 hours, but no vultures became sick or died at the roost.

Available data from evaluations of muskrat and nutria control indicate that the potential for exposure to nontarget wildlife is low. The most likely source of exposure is to animals that scavenge carcasses of the target species, but no mortalities from this source of exposure have been documented.

Incident Reports

Although field studies to determine effects of zinc phosphide on nontarget wildlife have generally found no significant effects, under certain circumstances operational zinc phosphide applications have killed nontarget wildlife. Most of these incidents have involved misuse of zinc phosphide or application rates and concentrations that were much higher than currently labelled rates.

Several of the incidents involved geese, which are very sensitive to zinc phosphide. One of the more notable cases occurred on the Tule Lake National Wildlife Refuge in California, where 325 white-fronted geese, 105 Canada geese, and 25 snow geese were apparently killed by oat groats treated with 1 percent zinc phosphide. The bait was aerially applied to potatoes and barley at 6.7–9 kg/ha for *Microtus* control (Keith and O'Neill 1964 unpubl., Keith and Perry 1964). The geese consumed the bait on a barley field that was burned 3 months later, exposing large amounts of treated bait. The barley was burned despite a previous agreement not to conduct any burning in the treated areas. In 1958, 3,676 geese were killed in Oregon following large-scale applications of bait treated with 1 percent zinc phosphide to dormant alfalfa fields at rates of 22 kg/ha or higher (Oregon State College 1959). This rate is much higher than currently labelled rates for zinc phosphide uses. A kill of 30 Canada geese occurred on a Michigan golf course treated with zinc phosphide (U.S. Environmental Protection Agency 1988 unpubl.).

The U.S. Environmental Protection Agency (EPA) (1988 unpubl.) has documented other zinc-phosphide-suspected poisonings of wildlife in Michigan from 1982 to 1988: 10 gray squirrels (*Sciurus carolinensis*) in 1 incident and 24 wild turkeys (*Meleagris gallopavo*) in 7 different instances. According to the author of the memorandum describing these incidents, most of the wildlife poisonings in Michigan resulted from misuse of zinc phosphide.

In the former Czechoslovakia, approximately 3,000 birds of the family Corvidae were killed in 1988 following operational vole control on winter wheat fields using wheat bait treated with 2.5 percent zinc phosphide (Tkadlec and Rychnovsky 1990). The winter wheat was very sparse, and no snow was present to cover the bait. Baits containing more than 2.0 percent zinc phosphide are not labelled for use in the United States.

In their review of zinc phosphide hazards, Rudd and Genelly (1956) reported that several cottontails in California died as a result of zinc phosphide applications at the rate of 2.2 kg/ha and that 500–1,000 geese, as well as a considerable number of ducks, partridges, and hares, died in The Netherlands from such applications. Further details were not reported.

Ingram (1945) documented the death of a colt that consumed an unknown amount of zinc-phosphide-treated grain in England. In another case, consumption of a zinc phosphide bait resulted in the deaths of six of nine horses (Schoof 1970). The source of the bait was not reported. Chitty and Southern (1954) reported that zinc phosphide poisoning caused the deaths of four cows and a pig but did not provide further details.

Although most studies indicate little potential for poisoning to occur through consumption of animals killed by zinc phosphide, a few incidents have been reported. In Michigan, two red foxes reportedly died from eating mice killed with grain baits treated with zinc phosphide (U.S. Environmental Protection Agency 1988 unpubl.), but no details were provided to verify this claim. Storer and Jameson (1965) reported that dogs have been killed from eating ground squirrels poisoned by zinc phosphide; no further details were provided. In Illinois, two cats were found dead adjacent to a no-till corn field illegally treated with 2 percent zinc phosphide bait at 4.9 kg/ha. Zinc phosphide was detected in the stomachs of both individuals, and the deaths were attributed to zinc toxicosis. Rodent fur along with cracked corn was found in the stomach of one of the cats (U.S. Environmental Protection Agency 1988 unpubl.). Another cat mortality believed to be the result of zinc phosphide ingestion was reported by White and Vonesch (1970), but no details were provided. Many cases of secondary poisoning have involved cats and dogs, possibly because these species are known to consume stomach contents of poisoned animals, whereas wild carnivores tend to avoid consuming the GI tracts. In an unusual case of secondary zinc phosphide poisoning, 10 chickens reportedly died after eating remains of rats killed with zinc phosphide in India (Christopher et al. 1982).

Summary

At least 61 acute oral toxicity studies, representing 28 species of mammals and 16 species of birds, have been conducted on zinc phosphide. LC_{50} 's have been determined for northern bobwhite quail, coturnix quail, and mallards. Toxicity studies using formulated zinc phosphide baits have also been conducted on Canada geese, white-fronted geese, snow geese, ring-necked pheasants, gray partridge, northern bobwhite, crowned guinea fowl, spotted doves, laughing doves, and red-winged blackbirds. Based on these toxicity data, zinc phosphide is highly to extremely toxic to both mammals and birds.

Zinc phosphide is several times more toxic to rodents than to carnivores. Of the bird species tested, waterfowl and gallinaceous birds appear the most sensitive. Geese appear to be the most susceptible of all wild birds to primary zinc phosphide poisoning, as shown by the low LD_{50} 's for these species. Some passerines, such as red-winged blackbirds, also appear relatively sensitive. Many avian species can distinguish treated from untreated bait and prefer untreated grain when given a choice. Several factors, including prior exposure to untreated bait, nutritional condition of the bird when provided treated baits, availability of alternate food sources, and ability to regurgitate treated baits significantly influence the magnitude of hazard to birds posed by zinc phosphide grain baits.

Many secondary toxicity studies have also been conducted on a variety of species, including coyotes, kit fox, red fox, gray fox, domestic dogs and cats, mink, least weasels, Siberian ferrets, domestic ferrets, mongooses, rats, great-horned owls, spotted eagle owls, bald eagles, golden eagles, black vultures, kestrels, snapping turtles, alligators, and gopher snakes. Results from these studies generally indicate low risk of secondary toxicity. Because zinc phosphide does not accumulate in muscle tissue of poisoned animals, no true secondary poisoning occurs. Death caused by eating animals poisoned with zinc phosphide results from primary ingestion of zinc phosphide remaining in the GI tracts. The low risk of secondary intoxication with zinc phosphide shown by these studies can be attributed to several factors: (1) Mammalian predators appear less susceptible to zinc phosphide than do other species; (2) the strong emetic action of zinc phosphide reduces secondary risks—mammals, birds, and reptiles have all been shown to eliminate food containing zinc phosphide through emesis; (3) zinc phosphide decomposes rapidly in the GI tracts of poisoned animals; and (4) most animals, when given a choice, refuse to eat the GI tracts of poisoned animals.

Field studies have been conducted in the United States to evaluate primary effects of zinc phosphide on nontarget wildlife for the following uses: prairie dogs on rangeland (five studies), Richardson's ground squirrels on rangeland (two studies), jackrabbits on rangeland (one study), California ground squirrels on ditchbanks (two studies), rats on ditchbanks (one study), voles in apple orchards (one study), rats in or adjacent to macadamia nut orchards (two studies), and rats in or adjacent to sugarcane (two studies). Information on nontarget hazards has also been gathered from

studies on the following uses: voles in alfalfa (one study), rodents in sugarbushes (one study), muskrats in wetlands (one study), nutria in wetlands (one study), and rats in ricefields (one study). However, these studies were not specifically conducted to evaluate effects on nontarget animals.

Of the eight studies conducted to evaluate effects on wildlife from rangeland uses of zinc phosphide, only one case of avian mortality was documented: four vesper sparrows were killed in South Dakota following zinc phosphide applications to control prairie dogs. Low avian mortality associated with rangeland applications can be attributed to the fact that passerines comprise the majority of rangeland birds, and toxicity data indicate that most passerines are less sensitive to zinc phosphide than other groups (e.g., waterfowl and gallinaceous birds). Also, avian density on rangeland is generally lower than on other habitat types, further reducing the opportunity for exposure. Finally, rangeland applications occur primarily in the summer and early fall, when alternative food supplies are generally abundant.

Because many species of rodents are associated with prairie dog and ground squirrel colonies, several instances of mortality to these species from zinc phosphide applications have been documented. Most mortality to nontarget rodents, however, has been localized and involved only a few individuals. The potential for zinc phosphide exposure to scavengers from eating poisoned animals was shown in several studies conducted on rangeland, but no cases of secondary exposure have been documented, primarily because scavengers avoided the GI tracts of poisoned animals.

Results from studies conducted on ditchbanks to evaluate effects on wildlife from California ground squirrel and rat control with zinc phosphide indicate that some small mammals, rabbits, and birds will be killed by the treatment. However, the applications do not appear to result in substantial wildlife mortality and do not affect populations.

Zinc phosphide applications for vole control in apple orchards will likely result in some mortality to cottontail rabbits, gallinaceous birds, and granivorous passerines, but populations of these species will probably not be significantly reduced. There is no evidence to indicate that secondary exposure to raptors or other predators/scavengers is a problem with zinc phosphide use in apple orchards.

Data from studies conducted in macadamia nut orchards show that granivorous birds will readily accept zinc-phosphide-treated grain baits, but none of the studies found that avian populations were reduced by the treatment.

Birds associated with sugarcane will consume zinc-phosphide-treated grain, but resultant mortality appears to be minimal or does not occur. Applications of zinc phosphide to wetlands adjacent to sugarcane will result in mortality of some aquatic invertebrates.

Available data from evaluations of muskrat and nutria control in wetlands indicate that the potential for exposure to nontarget wildlife is low. The most likely source of exposure

Management Implications

is to animals that scavenge carcasses of the target species, but no mortalities from this source of exposure have been documented.

Although field studies to determine effects of zinc phosphide on nontarget wildlife have generally found no significant effects, under certain circumstances operational zinc phosphide applications have resulted in incidents of mortality of nontarget wildlife. Most of these incidents have involved misuse of zinc phosphide or application rates and concentrations that were much higher than currently labelled rates.

Zinc phosphide is one of the safest rodenticides available and should continue to be used as a field rodenticide. Field studies have generally shown few risks to nontarget species when zinc phosphide baits are properly applied. Few other alternative rodenticides are currently available for field use. Strychnine is registered for underground use in controlling pocket gophers but is currently under a court injunction prohibiting aboveground use because of potential primary hazards to seed-eating birds and some secondary hazards to raptors. All rodenticide uses of Compound 1080 have been cancelled by EPA because registrants did not submit required data. EPA is currently concerned about field use of the anticoagulants and of cholecalciferol because of potential primary and secondary hazards; reregistration data have not yet been supplied for either of these compounds to support field use patterns.

On rangelands, zinc phosphide is the preferred control technique because of its lack of secondary hazards. It is the only grain bait registered for prairie dog control and is preferred over burrow fumigants because fumigants kill everything in the burrow, including nontarget species.

Zinc phosphide is currently being reregistered by EPA under the Federal Insecticide, Fungicide and Rodenticide Act (as amended in 1988). The reregistration data requirements include studies on product chemistry, wildlife toxicity, human health hazards, environmental fate, and residue chemistry. Funding for completion of these studies is being generated by the Zinc Phosphide Consortium of registrants. This group has placed a \$2/lb surcharge on all technical zinc phosphide sold in the United States. Required studies are scheduled for completion within the next 3 years.

Acknowledgments

We gratefully acknowledge the efforts of E. W. Schafer, Jr., and S. D. Palmateer in reviewing this manuscript.

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