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SHORT COMMUNICATIONS

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Zinc Phosphide Intoxication of Wild Turkeys (*Meleagris gallopavo*)

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ABSTRACT: Zinc phosphide (Zn_3P_2) is a rodenticide used to control a variety of small mammal species. It is available over-the-counter or as a restricted-use pesticide depending on how it is to be applied. The toxicity of Zn_3P_2 is dependent on the species exposed, whether the animal is able to vomit or not, and whether it is ingested on a full or empty stomach. Nontarget species can be exposed through inadvertent or intentional product misapplication. In this article we describe four mortality events in which wild turkeys (*Meleagris gallopavo*) were believed to have been intoxicated following the ingestion of baits containing Zn_3P_2.

Key words: Intoxication, *Meleagris gallopavo,* poisoning, wild turkeys, zinc phosphide.

Zinc phosphide (Zn_3P_2) is a dark-grey, crystalline compound used as a rodenticide to control rats, mice, voles, ground squirrels, prairie dogs, nutria, muskrats, feral rabbits, and gophers (Casteel and Bailey, 1986; Clarkson, 2001; Meister, 2001). It is used on crop and noncrop areas including nut and fruit orchards, Christmas tree farms, lawns, golf courses, nurseries, highway medians, and wetlands (US EPA, 2004). Approved bait formulations are primarily grain-based, but a tracking powder, a concentrate used to formulate grain-based baits, and meat- and fruit-based baits are available. Concentrations of Zn_3P_2 range from 1.82–2.00% for most prepared baits to 10% for the tracking powder. The concentrate is 63.2% Zn_3P_2. Baits containing Zn_3P_2 are available over-the-counter (OTC) or as a restricted-use pesticide (RUP) depending on whether the formulation is designed to be placed underground in burrows (OTC) or to be used above ground (RUP).

Zn_3P_2 is toxic to wild birds, nontarget mammalian species, and freshwater fish (Casteel and Bailey, 1986; Extoxnet, 1996; Knight, 2001). However, toxicity varies among species (Albretsen, 2004). Lethal, oral doses of Zn_3P_2 for most domestic mammalian species are reported to be between 20 mg/kg and 40 mg/kg body weight (Casteel and Bailey, 1986; Albretsen, 2004). The rodent, oral, median lethal dose (LD_{50}) of technical-grade Zn_3P_2 is reported to be 45.7 mg/kg (Clarkson, 2001). The minimum lethal, oral dose for chickens given technical-grade Zn_3P_2 combined with starch in gelatin capsules is 10 mg/kg body weight (Robertson et al., 1945), whereas the oral LD_{50} for partridges and pheasants (species not reported) given Zn_3P_2 applied to wheat is 26.7 mg/kg body weight (Janda and Bosseova, 1970). White-fronted geese (*Anser albifrons*) are the most sensitive avian species thus far tested with an oral LD_{50} of 7.5 mg/kg technical-grade Zn_3P_2 when applied to hulled oats (Glahn and Lamper, 1983).

Zn_3P_2 has a disagreeable odor resembling acetylene or rotten fish (Casteel and Bailey, 1986). It is hydrolyzed in the acidic environment of the stomach, liberating phosphine gas and free radicals. Zn_3P_2 is more toxic when ingested with food because it lowers the stomach pH (Stowe et al., 1978).

There are several case reports of wildlife and domestic animal intoxication following ingestion of Zn_3P_2 (Mohr, 1959; Stowe et al., 1978; Glahn and Lamper, 1983; Drolet

et al., 1996). However, there have been no published reports of intoxication in wild turkeys (*Meleagris gallopavo*). We report here a series of cases submitted to the Pennsylvania Animal Diagnostic Laboratory System (Kennett Square, Pennsylvania, USA) in which the deaths of wild turkeys were attributable to ingestion of Zn_3P_2 used for rodent control in orchards and tree nurseries.

The first mortality event occurred in February 1998. Nine turkeys were found dead along an old railroad bed near two orchards in southeastern Pennsylvania (Montgomery County, 40°07′N, 75°20′W) by a wildlife conservation officer (WCO) with the Pennsylvania Game Commission (PGC). Several of the carcasses showed evidence of predation or scavenging. The carcasses were submitted for necropsy. Gross-postmortem examination revealed four birds with congested lungs, two with hepatic and peritoneal hemorrhages, and two with petechial epicardial hemorrhages. A variety of seeds was found in the gizzards. The most numerous were unidentified species of smartweed (*Polygonum* sp.) and wild grape (*Vitis* sp.) that were determined to be insignificant as a cause of death. Microscopic examination of lung specimens showed severe congestion, interstitial edema, and mild, interstitial inflammation that was predominantly heterophilic. Aerobic bacterial cultures of lung, liver, and pericardial fluid specimens were negative. A metal screen of kidney samples for arsenic, cadmium, calcium, copper, iron, lead, magnesium, manganese, molybdenum, total phosphorus, selenium, zinc, and thallium performed by inductively coupled plasma mass spectrometry was negative. A pooled sample of crop contents was negative for cyanide or other compounds detectable with routine screening by gas chromatography-mass spectroscopy (GC-MS) such as organophosphate insecticides. However, phosphine was detected by GC-MS, but not quantified, in the pooled sample. The owner of one of the orchards admitted to using a zinc-phosphide bait for rodent control. Although allowed by the label, the bait was placed directly on the ground, which was accessible by the turkeys.

The second mortality event occurred in December 1999. Eleven, frozen, wild turkey carcasses were found by a WCO with the PGC adjacent to a woodlot and peach orchard in northwestern Pennsylvania (Erie County, 42°07′N, 80°05′W). The dead turkeys were part of a flock of approximately 33 birds being fed by a homeowner adjacent to the orchard. Seven carcasses were submitted for necropsy. Three of the seven carcasses were severely damaged by predators or scavengers and were not suitable for necropsy. The other four carcasses were in reasonably good condition, and all four birds had crops filled with grain and grass. Dark, hemorrhagic lungs were noted in all birds; there were no other consistent lesions noted. Postmortem autolysis and freeze-thaw artifacts precluded an accurate histologic evaluation. A variety of tissues, including brain and crop contents, were collected for toxicologic testing. Historically, several pesticides had been used on the farm where the birds were found, including diphacinone, methomyl, diazinon, guthion, dimethoate, carbaryl, carbofuran, permethrin, methyl parathion, chlorothalonil, metribuzin, and Zn_3P_2. Liver tissue was negative for anticoagulant rodenticides (brodifacoum, bromodiolone, chlorophacinone, coumafuryl, difenacoum, difethiolone, diphacinone, pindone, valone, or warfarin). General organic chemical screens by GC-MS and liquid chromatography–mass spectroscopy were negative for organophosphate or carbamate insecticides. Because of a history of the use of Zn_3P_2 and consistent, gross lesions of pulmonary edema, crop contents were tested for phosphine by GC-MS and found to be positive. The phosphine was not quantified.

The third mortality event occurred in January 2001 and involved the deaths of six wild turkeys in south central Pennsylvania (Adams County, 39°49′N, 77°13′W).

One turkey was radio-tagged as part of a population study being conducted by the PGC. Investigation of its lack of movement led to the discovery of the six turkeys at a roost site approximately 50 m from a fruit orchard. One carcass and four crops were submitted to the laboratory for examination. All five crops contained fruit pulp and skin that appeared to be derived from apples. In addition, all crops contained blue-green material with a doughy consistency along with unidentified seeds, grasses, and leaves. There was a suspicion that the birds might have been exposed to Zn_3P_2 because the rodenticide was used in the proximity of the birds. Crop contents were tested for zinc. Zinc concentrations in the five crops, determined by atomic absorption spectroscopy, were 40 parts per million (ppm), 438 ppm, 975 ppm, 1,513 ppm, and 1,850 ppm (wet weight). The anticoagulant rodenticide, chlorophacinone, was also detected by high-performance liquid chromatography but not quantified. Zinc concentrations in two control-turkey crops were determined to be 16 and 7 ppm. A diagnosis of Zn_3P_2 intoxication was made based on a history of the use of Zn_3P_2 in the area, apparent sudden deaths of several birds, high concentrations of zinc in four of five crop contents, and no evidence of a coagulopathy, consistent with chlorophacinone intoxication, on postmortem examination.

Mortality event number four occurred in February 2002 and involved ten birds. All were found dead on the ground within 42 m^2 in western Pennsylvania (Allegheny County, 40°25N, 79°47W) by a local homeowner who notified a WCO with the PGC. Two carcasses were heavily scavenged; eight were submitted for necropsy. Although there was evidence of some scavenging and autolysis, the carcasses were in relatively good postmortem condition. Grossly, all birds were noted to have dark red to black lungs; several lungs were saturated with serosanguineous fluid. Fecal examination for intestinal parasites was negative as were aerobic cultures of liver and pericardial samples for bacterial pathogens. Analysis of crop contents was positive for phosphine using a colorimetric procedure. A Zn_3P_2-laced, cracked-corn bait had been used according to label directions for mouse control by a nearby nursery. The bait had been broadcast between rows of fruit trees. The same procedure had been used for a number of previous years without apparent problem. However, it was believed that the lack of snow cover made the bait more readily accessible by the birds.

Zn_3P_2 has been used as a rodenticide for many years and was particularly popular during World War II, when the supply of red squill (*Urginea maritima*), containing the active ingredient scilliroside, was limited (Casteel and Bailey, 1986). For many rodenticide applications, the anticoagulants have replaced Zn_3P_2. However, Zn_3P_2 is still readily available and has a range of both crop and noncrop uses for a variety of small-mammal pests (Casteel and Bailey, 1986; Clarkson, 2001; Meister, 2001). Zinc phosphide is directly irritating to the gastric and intestinal mucosa and causes rapid emesis (Casteel and Bailey, 1986). The ability of many nontarget mammalian and avian species to vomit, compared with the inability of many target mammalian species to vomit, confers some degree of selectivity of Zn_3P_2 as a rodenticide. However, it is a highly toxic compound to most species (Casteel and Bailey, 1986; Extoxnet, 1996; Knight, 2001). The inherent toxicity of Zn_3P_2 combined with baits that contain relatively high concentrations makes its use potentially hazardous for nontarget species. For this reason, above-ground application is restricted to licensed individuals.

Populations of wild turkeys have increased dramatically in recent years in many areas of the country where they had been largely extirpated (Dickson, 1992). Given the increasing numbers of wild turkeys, it is likely that there will be a higher prevalence of intoxication from commonly used insecticides and rodenticides. All four mortality events occurred during winter when other

food items may have been scarce and baits containing Zn_3P_2 more attractive. It is believed that birds will generally avoid baits containing Zn_3P_2 if other food is available (Glahn and Lamper, 1983). Assuming that wild turkeys are as sensitive to Zn_3P_2 as pheasants (LD_{50} of ~26 mg/kg body weight; Janda and Bosseova, 1970), a typical, adult, female, wild turkey weighing 5 kg would only have to ingest approximately 6 g of a 2% bait to reach a potentially fatal dose.

As is the case with the most wildlife-mortality events, animals are most often found dead with no prior observation of clinical signs. In addition, the quality of samples available for postmortem examination is generally poor, which precludes good histopathologic, microbiologic, and virologic evaluations. In many cases, only gross-postmortem examinations and toxicologic analyses can be performed. Lesions associated with Zn_3P_2 intoxication are nonspecific and primarily involve generalized organ congestion. In the cases described here, notable pulmonary congestion was the only clue to possible intoxication by this rodenticide. In some cases, perceptive diagnosticians may note a distinctive odor to the gastrointestinal contents, which has been described as smelling like acetylene, garlic, or rotten fish (Casteel and Bailey, 1986).

There are several analytical approaches to detecting Zn_3P_2 including colorimetric, gas chromatography, and GC-MS methods (Guale et al., 1994; Drolet et al., 1996; Sterner, 1996). All involve the detection of phosphine gas and not intact Zn_3P_2. Crop or stomach content samples are preferred to confirm exposure because other samples are unlikely to contain detectable concentrations (Robertson et al., 1945; Matschke et al., 1992; Guale et al., 1994). Phosphine gas dissipates rapidly in air, and ideally, samples of gastrointestinal contents should be packed in air-tight containers and stored and shipped frozen to prevent phosphine loss (Guale et al., 1994). Alternatively, a frozen, whole carcass can be submitted. Phosphine recovery from gastrointestinal tracts declines as the postmortem interval increases (Robertson et al., 1945). However, in the described cases, no particular precautions were taken to preserve gastrointestinal contents samples, so less-than-ideal sample handling should not preclude an attempt to detect phosphine. Although phosphine was not quantified in the three cases in which it was detected, its presence, along with other case information, was deemed sufficient to support a diagnosis of Zn_3P_2 intoxication. The diagnosis in event number three is the most tenuous because testing for phosphine in gastrointestinal contents was not performed. An indirect indication of ingestion of Zn_3P_2 involves the measurement of elevated concentrations of zinc in gastrointestinal contents and tissues (Casteel and Bailey, 1986). Detection of zinc at 1,513 ppm and 1,850 ppm (wet weight) in two of five submitted crop contents, along with the known use of Zn_3P_2 within 50 m of the dead birds, is highly suggestive of intoxication. Zinc concentrations in a variety of seeds and grasses are generally well below 100-ppm wet weight (NRC, 1982).

Like Zn_3P_2, aluminum phosphide (AlP) degrades readily to release phosphine gas (H_3P). Aluminum phosphide is used primarily as a fumigant of enclosed grain storage and transport areas and, less commonly, in rodenticide baits and for fumigation of rodent burrows. Although wildlife species are less likely to be exposed to AlP, detection of phosphine in gastrointestinal contents does not differentiate between the two, and a diagnosis should be based on exposure history and/or elevated concentrations of zinc in gastrointestinal contents and tissues.

Compared with other rodenticides, such as strychnine and compound 1080, Zn_3P_2 presents less risk of a lethal, secondary intoxication to scavengers, such as foxes, dogs, cats, and raptors, following their ingestion of a target species killed by Zn_3P_2 (Bell, 1975; Hill and Carpenter, 1982; Matschke et al., 1992; Sterner and Mauldin, 1995; Sterner, 1996). In one study, it was estimated that cats and dogs of various

body weights would have to ingest between five and 847 intoxicated voles in a short period of time to approach a lethal dose (Sterner, 1996).

The mechanism of toxic action of Zn_3P_2 is not clear. It is likely that phosphine gas, released in acidic portions of the gastrointestinal tract and absorbed, causes oxidative tissue damage (Hsu et al., 2002). Damage to heart and lungs is likely to be responsible for early deaths. Onset of clinical signs following ingestion is variable but is most often within 4 hr (Casteel and Bailey, 1986). Partridges and pheasants developed clinical signs between 2 hr and 6 hr after ingestion (Janda and Bosseova, 1970). Clinical signs are nonspecific; experimentally poisoned poultry exhibited depression, ruffled feathers, anorexia, and diarrhea (Roberston et al., 1945). Partridges and pheasants exhibited listlessness, anorexia, tachypnea, stiffness, and muscle spasms (Janda and Bosseova, 1970). Likewise, postmortem lesions are nonspecific. Gross lesions are limited to generalized organ congestion and pericardial, pleural, and peritoneal effusions. Histologic changes in intoxicated poultry include venous congestion, cloudy swelling in the epithelial cells of the small intestine, and fatty degeneration in the liver. (Robertson et al., 1945).

Direct ingestion of Zn_3P_2 baits by many wildlife species can cause intoxication and should be considered in the differential diagnosis of any wildlife-mortality event in regions in which it is used. The risk of nontarget-animal intoxication from ingestion of bait can be minimized by following label directions. Secondary intoxication of scavenger species is unlikely.

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