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Human Health and Ecological Risk Assessment
for the Use of Wildlife Damage Management Methods
by USDA-APHIS-Wildlife Services

Chapter X

The Use of Zinc Phosphide in Wildlife Damage Management

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THE USE OF ZINC PHOSPHIDE IN WILDLIFE DAMAGE MANAGEMENT

EXECUTIVE SUMMARY

Zinc phosphide is a rodenticide used by the USDA-APHIS-Wildlife Services Program in rodent damage management projects. Zinc phosphide is used to control various rodents in terrestrial and aquatic environments. Several formulations are available for use including wheat and oat bait products, a concentrate used to mix with bait material attractive to the target pest, pellets and tracking powders. Application methods vary from hand applications at the entrance of burrows to aerial broadcast applications depending on the pest and use area. When rodents ingest zinc phosphide treated baits, the zinc phosphide reacts with acidic conditions in the stomach of vertebrates to generate phosphine gas. Phosphine distributes rapidly throughout the body resulting in hypoxia and eventual death of target mammals.

The USDA-Animal and Plant Health Inspection Service has evaluated the human health and ecological risk of zinc phosphide under various use patterns. The risk to human health, including workers that would mix and apply zinc phosphide, was found to be low based on conservative assumptions of exposure and toxicity. The low risk to workers was determined based on label requirements regarding the use of the appropriate personnel protective equipment designed to reduce exposure.

Label restrictions, use patterns, and available environmental fate and aquatic ecological toxicity data for zinc phosphide and its degradates suggest low risk to aquatic organisms and their habitats. Zinc phosphide risks to terrestrial invertebrates and plants are also low. However, potential risks have been identified to nontarget terrestrial vertebrates that consume bait (primary risk), or prey or scavenge on target animals (secondary risk) that have received a dose of zinc phosphide. Primary risks are greatest for those nontarget vertebrates attracted to the bait and ingest a sufficient quantity of zinc phosphide to result in sublethal or lethal effects. Many terrestrial vertebrate species are averse to the bait or treated food material, or ingestion results in emesis, which reduces exposure and risk. Secondary risks to nontarget terrestrial vertebrates are lower when compared to primary risks because zinc phosphide does not accumulate in target pests. Secondary risks to wildlife and domestic animals can mostly result from nontarget animals ingesting dosed animals with undigested zinc phosphide. Label restrictions reduce the probability of significant exposure and risk to humans and nontarget terrestrial vertebrates.

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1 INTRODUCTION

Zinc phosphide is a rodenticide used by the U.S. Department of Agriculture (USDA), Animal and Plant Health Inspection Services (APHIS), Wildlife Service (WS) Program to reduce wildlife hazards at airports (rodents attract predatory wildlife species to the air operating area that are dangerous to aircraft) and reduce damage to crops, property, and parks. This Human Health Risk Assessment (HHRA) and Ecological Risk Assessment (ERA) provides a qualitative evaluation of potential risks and hazards to human health and nontarget fish and wildlife as a result of exposure to zinc phosphide from its proposed use by WS.

The methods used in this HHRA to assess potential human health effects follow standard regulatory guidance and methodologies from the National Research Council (1983) and the U.S. Environmental Protection Agency (USEPA 2017), and generally conform to additional Federal agency standards such as USEPA Office of Pesticide Programs. The methods used in the ERA to assess potential ecological risk to nontarget fish and wildlife generally follow USEPA methodologies.

The risk assessment starts with problem formulation (identifying hazard), then evaluates potential exposure (identifying potentially exposed populations and determining potential exposure pathways for these populations), and toxicity (the dose-response assessment). Lastly, the combination of information from the exposure and toxicity assessment is integrated into the risk characterization, the determination of potential adverse human health and ecological risks. A discussion of the uncertainties associated with the risk assessment and cumulative effects is included in this risk assessment.

1.1 Use Pattern

Zinc phosphide is a toxicant used to control rodents (ground squirrels, prairie dogs, voles, commensal rodents, field rodents, gophers, nutria, and muskrats[1]) and jackrabbits. It has a strong, pungent, garlic-like odor that actually is attractive to rodents such as rats, but unattractive to other animals. Zinc phosphide comes in prepared baits on wheat and oats, the concentrate can be prepared on apples, carrots, or other baits attractive to the target animal, or be in tracking powder (the animal accumulates dust on its body and when cleans ingests zinc phosphide). Prebaiting with the same baits (i.e., oats, wheat, vegetables and fruits) to be used prior to bait application makes treatment more effective, especially for voles, muskrats, and jackrabbits. Toxicity is the result of zinc phosphide reacting with dilute acids in the stomach to form phosphine gas. The gas is absorbed through the respiratory passages and enters the bloodstream to block physiological processes in cells and alter hemoglobin (Witmer and Fagerstone 2003). Animals that ingest lethal amounts of bait usually succumb overnight with terminal symptoms of convulsions, paralysis, coma, and death from asphyxia. If death does not occur overnight, intoxication occurs which severely damages the liver killing the animal in a few days. Animals that are alive after 3 days completely recover. Zinc phosphide baits deteriorate very slowly into phosphine gas; therefore, dry bait is considered toxic indefinitely. Lecithin-mineral oil or other surfactants used to adhere zinc phosphide to grain baits, offer protection against moisture and may increase its stability. Under field conditions, zinc phosphide baits remain toxic for 2-3 months, and potentially for several more months. However, zinc phosphide mixed onto wet baits such as cubed vegetables and fruits break down in a few days. Once in the soil, zinc phosphide breaks down rapidly to phosphine, which is either released into the atmosphere or converted into phosphates and zinc complexes (Jacobs 1994). Translocation of phosphine has been demonstrated, but it is rapidly converted to harmless phosphates. A safety factor of

[1] Scientific names are given in the Risk Assessment Introduction Chapter I.

zinc phosphide is that it is a greenish black color unattractive to birds. It also causes an emetic response (regurgitation) in most animals except rodents.

WS took an annual estimated average of 230,546 rodents of 27 species and used an average 32,616 pounds[2] of bait in 18 states from FY11[3] to FY15 (Table 1). During this time, APHIS-WS applied or demonstrated nine formulations of zinc phosphide operationally and distributed or sold nine formulations of zinc phosphide through technical assistance (Table 2). WS worked on an average of 206 agreements annually from FY11 to FY15. The most common resources protected by WS were aircraft (zinc phosphide is frequently used at airports to reduce rodent populations that attract raptors, herons, and predatory mammals to air operating areas thereby protecting aircraft from wildlife strikes and reduce infrastructure damage from rodents, especially underground wiring), crops, and parks. The most common target species, 27 in all, included black-tailed prairie dogs, house mice, white-footed deermice, and meadow voles where WS personnel applied zinc phosphide baits.

Table 1. The annual average number of target rodents taken with zinc phosphide baits used by WS in WDM from FY11 through FY15. Take was estimated for WS projects not estimate take. The maximum take is based on daily intake less 10% wastage or the acreage treated (up to 10 pounds per acre can be used depending on target species).

ANNUAL AVERAGE ZINC PHOSPHIDE USE AND SPECIES TAKEN				
Species*	**Take**	**Lbs. Bait Used**	**How Estimated Numbers**	**States Where Used**
Burrowing Rodents				
Yellow-bellied Marmot	384	33.3	9.5 per pound	ID MT OR UT
Black-tailed Prairie Dog	143,504	23,917.3	15 per acre; max 6 per pound	CO KS NE TX
Gunnison's Prairie Dog	1,786	195.0	10 per acre; max 7 per pound	AZ CO NM
White-tailed Prairie Dog	399	57.0	10 per acre; max 7 per pound	CO UT
California Ground Squirrel	600	97.5	5 per acre; max 9 per pound	NV OR
Paiute Ground Squirrel	112	5.3	14 per acre; max 21 per pound	ID
Uinta Ground Squirrel	40	12.2	10 per acre; max 16 per pound	ID
Belding's Ground Squirrel	3,321	99.2	25 per acre; max 20 per pound	OR
Thirteen-lined Ground Squirrel	60	2.4	10 per acre; max 21 per pound	NE TX
Mexican Ground Squirrel	111	5.3	8 per acre; max 21 per pound	TX
Round-tailed Ground Squirrel	6,880	376.9	80 per acre; max 44 per pound	AZ
Attwater's Pocket Gopher	234	10.6	7 per acre; max 22 per pound	TX
Sagebrush Vole	572	10.6	9 per acre; max 174 per pound	OR WA
Gray-tailed Vole	4,000	1,000.0	N/A	OR
Long-tailed Vole	158	14.8	16 per acre	OR WA
Montane Vole	999	18.6	100 per acre; max 80 per lb	OR WA
Prairie Vole	7,015	1,861.5	10 per acre; max 142 per lb	CO IL KS NE
Meadow Vole	13,313	2,473.1	32 per acre; max 97 per pound	IL MI NE OH UT
Woodland Vole	4,450	2,237.8	10 per acre; max 174 per lb	KY TN
Ord's Kangaroo Rat	1,660	187.2	16 per acre; max 79 per pound	NE NM
TOTAL (20 sp.)	**189,598**	**32,615.6**		**17 States**
Terrestrial Rodents				
North American Deermouse	26	0.2	10 per acre; max 131 per lb	UT
White-footed Deermouse	18,658	2,435.0	7 per acre; max 131 per pound	IL MI
House Mouse*	21,281	701.9	10 per acre; max 148 per lb	MI TX
Brown Rat*	15	0.8	Max 20 per pound	NE
Black Rat*	937	15.9	Max 24 per pound	TX
TOTAL (5 sp.)	**40,917**	**3,153.8**		**5 States**
Aquatic Rodents				
Muskrat	1	0.2	N/A	ID
Nutria*	30	6.9	Max 9.5 per pound	LA TX
TOTAL (2 sp.)	**31**	**7.1**		**3 States**
GRAND TOTAL	**230,546**	**35,776.5**		**18 States**

N/A – Not applicable as take was estimated by applicator * Introduced species

[2] Zinc phosphide concentrate, measured in ounces of concentrate, was converted to the standard 1.9 pounds of treated bait made to estimate take.
[3] FY11 equals the federal Fiscal Year 2011, which is October 1, 2010-September 30, 2011 (the year is denoted by FY11, FY12, and so on).

Table 2. The annual average number of pounds of zinc phosphide treated baits and ounces of zinc phosphide concentrate and tracking powder applied, demonstrated, distributed, and sold by APHIS-WS in WDM from FY11 thru FY15.

ANNUAL AVERAGE ZINC PHOSPHIDE USE BY FORMULATION FO FY11 TO FY15			
Formulation	**EPA Registration No.**	**Applied/Demonstrated**	**Sold/Distributed**
Zinc Phosphide Formulated Baits (Lbs.)			
ZnPh (Gopha-rid)	12455-30	0	0
ZnPh AG (Rodent)	12455-17	192.3	19
ZnPh AG Oats (Rodent)	12455-102	1,337.3	9
ZnPh Bait (Prairie Dog)	13808-6	20,519.8	35
ZnPh Bait (Rodent) CA SLN	10965-50015	0	0
ZnPh Oats	4271-16	10	0
ZnPh Oats (Hacco Rodent)	61282-14	8.8	10
ZnPh Oats (Prairie Dog)	56228-14	2,608.9	1,452.4
ZnPh Pellets (Hopkins)	61282-49	0	0
ZnPh Pellets (Rodent)	12455-18	4.8	7.2
ZnPh Wheat	56228-3	10,537.5	16,195
TOTAL	**11 Registrations**	**35,219.4 lbs.**	**17,805.8**
Zinc Phosphide Concentrate (Oz.)			
ZnPh Concentrate	56228-6	300.7	68.6
ZnPh Tracking Powder	12455-16	0	9.6
TOTAL	**2 Registrations**	**300.7 oz.**	**78.2**

No nontarget species was documented to be taken with WS zinc phosphide use, either primarily or secondarily. It is possible that nontarget species could have been taken, but not found. Nontarget species would likely include nontarget rodents or rabbits that occupy the same habitat as the target species. WS did not take any T&E species.

2 PROBLEM FORMULATION

Zinc phosphide is a nonselective rodenticide, first registered as a pesticide in the U.S. in 1947 (USEPA 1998, 2016a). Registered with EPA for use against mammalian pest species including mice, ground squirrel, prairie dogs, voles, moles, rats, muskrats, nutria, and gophers, zinc phosphide is used on agricultural, non-agricultural, and residential sites (USEPA 2016a). WS uses zinc phosphide to reduce rodent damage such as prairie dogs, voles, rats, mice, ground squirrels, gophers, muskrat, nutria, marmots, and jackrabbits. Toxicity occurs when zinc phosphide reacts with water and hydrochloric acid (HCl) in the gastrointestinal tracts of poisoned animals to form the highly toxic phosphine gas (HSDB 2017). The following sections discuss the chemical description and product use, physical and chemical properties, environmental fate, and hazard identification for zinc phosphide.

2.1 Chemical Description and Product Use

Zinc phosphide (Zn_3P_2; CAS #1314-84-7) is an inorganic compound with a molecular weight of 258.12 and a molecular structure of Zn_3P_2 (Figure 1). Zinc phosphide comes in prepared baits on wheat and oats, or comes as a concentrate that is applied to apples, carrots or other baits attractive to the target animal. Use of zinc phosphide on various types of fruit, vegetable, or cereal substrates is more effective at targeting specific animals. The type of bait applied with the bait labeling directions and baiting strategies is designed to enhance target take while minimizing nontarget hazards. Pre-baiting with the same bait carrier is required, in some cases, or recommended prior to bait application to increase treatment efficacy; some target species are

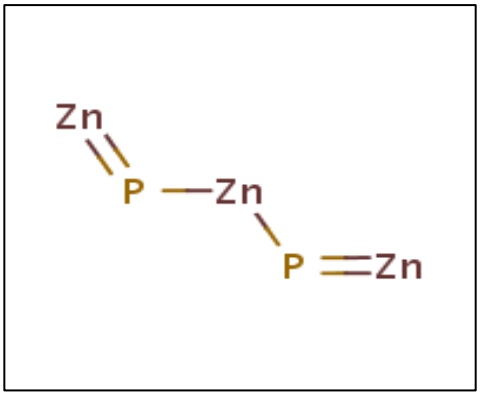

Figure 1. Chemical structure of zinc phosphide.

repelled to a certain degree by the pungent odor. When an animal ingests zinc phosphide and contacts stomach acids, phosphine gas is released which can cause death.

APHIS is the registrant of three zinc phosphide products (Zinc Phosphide on Wheat – EPA Reg. No. 56228-3 (rev. 6/25/13), Zinc Phosphide Concentrate – EPA Reg. No. 56228-6 (rev. 6/21/13), and Zinc Phosphide on Oats – EPA Reg. No. 56228-14 (rev. 6/25/13)). The wheat and oat formulations contain 1.82% and 2% zinc phosphide active ingredient (ai), respectively. The concentrate formulation contains 63.2% zinc phosphide ai; the concentrate is mixed with food materials attractive to the target species. WS used seven other zinc phosphide formulations from FY11 to FY15 (Table 2). All three APHIS labels and the additional seven labels are restricted-use and restricted to non-residential uses; only certified applicators or persons under their direct supervision may use these products. The labels list precautions to protect workers and others from direct exposure or through drift, and allowing only protected handlers to enter the treatment area during applications. Other limitations exclude applications on roads, near residential areas, over water, broadcast over crops (unless use directions specifically permit aerial application) when wind velocity exceeds 10 mph, in areas inhabited by livestock, and when precipitation is expected.

The zinc phosphide concentrate formulation (EPA Reg. No. 56228-6) is used on non-crop areas such as airport grasses, golf courses, highway medians, non-residential lawns, parks, rights-of-ways, turf, orchards, crop borders, nurseries, vineyards, floating rafts and waterway borders, in and around buildings, rangeland pasture, and reforestation areas. The label does not allow use in specific areas such as near roads or over water. The food and feed crop uses are limited to dormant crops, pastures, and rangelands, and artichoke fields in California to control voles. The label restricts bait containing the concentrate from use on bare ground, or by aerial application for sites such as airport grasses, golf courses, and parks, and hand baiting in pastures.

The zinc phosphide wheat formulation (EPA Reg. No. 56228-3) is used in dormant pome and stone fruit orchards, non-fruit bearing nursery stock and ornamental trees, conifer tree groves, rangelands and reforestation seeding areas, vineyards, airports, highway medians, turf farms, nonresidential lawns, golf course, park, alfalfa, and barley to control voles and mice.

The zinc phosphide formulation mixed with oats (EPA Reg. No. 56228-14) is used in the following areas: 1) rangeland and pasture to control prairie dogs; 2) along canal and ditch bank rights-of-ways and crop borders to control California ground squirrels; 3) on airport runways and grasses, highway medians, and other non-crop areas to control prairie dogs, voles and mice; 4) in fruit and nut tree orchards; 5) around agricultural and industrial structures; and 6) in alfalfa, barley, potatoes, sugar beets, wheat and dry beans to control voles and mice. The zinc phosphide wheat formulation has the same use restrictions.

Zinc phosphide has been used to reduce damage associated with many species of rodents. Zinc phosphide has been used to reduce damage from invasive nutria to wetlands and dikes. The use of zinc phosphide at airports to reduce rodents that attract raptors and other predators was found to reduce wildlife strikes to aircraft (Witmer 2011); inherently, a reduction in aircraft strikes reduces the chance of a catastrophic incident involving the loss of human lives.

Zinc phosphide labels require that only protected handlers of the product can be in the area of the application and that no other people can be in the area including the area of drift. All personnel that handle the product, including loaders and applicators, must wear long sleeved shirts, long pants, shoes, socks, and waterproof gloves. In addition, persons loading zinc phosphide pesticide products or toxic baits into aircraft or

mechanical ground equipment and persons loading or applying zinc phosphide with hand-pushed or hand-held equipment, such as a push-type or cyclone spreader, must wear a dust or mist filtering respirator (MSHA/NIOSH approval number prefix TC- 21C or a NIOSH-approved respirator with an R, P, or HE filter[4]) and protective eyewear. Any person who retrieves carcasses or unused bait following application of zinc phosphide must wear waterproof gloves.

Since zinc phosphide is highly toxic to wildlife, it cannot be applied directly to water, to intertidal areas below the mean high-water mark, or to areas where surface water is present or runoff is likely to occur. This includes water contaminated with zinc phosphide from cleaning equipment or disposing wastes. The use of this product may pose a hazard to federally listed threatened and endangered (T&E) species. The label identifies several species that must be considered prior to use. It is a federal offense to use any pesticide in a manner that results in the death of a T&E species. The label requires that the U.S. Fish and Wildlife Service (USFWS) be consulted on local species that could be impacted by use of zinc phosphide. WS conducts Section 7 consultations with USFWS to ensure that T&E species are not impacted.

Zinc phosphide cannot be stored or disposed of in an area where it could contaminate water, food, or feed. Improper disposal of excess pesticide, spilled bait, or rinsate is a violation of Federal law. If the zinc phosphide concentrate or baits are not used, the product is disposed of according to label instructions by contacting State Pesticide or Environmental Control Agency or the Hazardous Waste representative at the nearest EPA Regional Office for guidance. It must be stored in the original container and in a dry place inaccessible to children, pets and domestic animals. The container must be kept closed when stored and away from other chemicals. After use, the bags or containers are nonrefillable or reusable. All particles are shaken from bags, thoroughly rinsed off plastic bags, or removed from other containers into the application equipment at the treatment site. Empty bags and containers can be recycled, disposed of at a landfill, or incinerated, but cannot be reused or refilled. If the container cannot be recycled, then it is disposed of at a sanitary landfill or by incineration if allowed by State and local authorities.

Zinc phosphide cannot be applied on roads, near residential areas, in areas with livestock, when precipitation is expected, or over water. Airports were the latest addition where it can be applied because a study found that wildlife strikes at an airport were reduced following a reduction of rodents (prey) with zinc phosphide (Witmer 2001). It cannot be broadcast over crops unless the use directions specifically permit aerial application. When zinc phosphide is aerially applied, wind velocity cannot exceed 10 mph. Prebaiting with untreated oats at rates appropriate for treated bait typically improves acceptance of this zinc phosphide bait and usually 2 or 3 days before toxic baits are used. On the other hand, it should be noted that

2.2 Physical and Chemical Properties

Zinc phosphide is a gray to black powder with a phosphine odor (USEPA 1998). It has a high melting and boiling point (420°C and 1100°C, respectively). It has negligible vapor pressure and is stable in dry conditions. It reacts slowly with water, including atmospheric moisture, to form phosphine gas. Zinc phosphide is decomposed by acids to produce phosphine. The Hazardous Substances Data Bank (HSDB 2017) lists zinc phosphide as insoluble in water.

[4] This is on the current labels, but APHIS-WS in the process of updating this restriction to a NIOSH approved particulate respirator with any N (not resistant to oil), R (resistant to oil), or P (oil proof) filter with NIOSH approval number prefix TC-84A.

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2.3 Environmental Fate

The environmental fate describes the processes by which zinc phosphide moves and transforms in the environment. The environmental fate processes include: 1) mobility, persistence, and degradation in soil, 2) movement to air, and 3) migration potential to groundwater and surface water.

Zinc phosphide has low mobility in soil (USEPA 1998). Microbial degradation in soil is slow with hydrolysis the major route of degradation. The degradation rates increase as soil pH increases or decreases from neutrality. Zinc phosphide dissipation half-lives of one month or longer have been reported in dry soils with half-lives less than one week in moist soils (USEPA 1998).

Zinc phosphide exists solely in the particulate phase in air because it has negligible vapor pressure. However, wet or dry deposition removes particulates from the atmosphere or moist air may hydrolyze the particulates (HSDB 2017). Phosphine gas degrades quickly in the atmosphere by reaction with photochemically produced hydroxyl radicals with a reported atmospheric half-life of less than one day.

Zinc phosphide is insoluble in water and susceptible to hydrolysis and degradation to the zinc ion and phosphine gas. Degradation is pH dependent occurring more rapidly in acidic conditions (USEPA 1998). Zinc phosphide bait degradation under neutral pH conditions has shown stability for greater than a month while under acidic conditions anywhere from 78 to 100% of the rodenticide will be degraded to zinc ions and phosphine gas. Zinc phosphide in water under neutral conditions degrades in sediments to phosphine under anaerobic conditions or phosphate under aerobic conditions (Hilton and Robison 1972). Phosphine occurs as a volatile gas with very low water solubility and does not persist in water. The presence of zinc ions in water and sediment in various forms depends on site-specific water and sediment conditions. Site-specific conditions influence the partitioning of zinc between the water and sediment phases and determine bioavailability to fish as well as potential prey items. As a result of hydrolysis, bioconcentration is not an important fate pathway for zinc phosphide (HSDB 2017).

In addition to the environmental fate of zinc phosphide, the longevity of the bait in the field can be a factor in determining how long zinc phosphide persists in the environment. Longevity of zinc phosphide varies depending on the environmental factors where the baits are applied, and whether the baits are pellets or whole-grain. Zinc phosphide degradation was shown to increase under higher rainfall and humidity levels (Hilton and Robison 1972; Sterner and Ramey 1995, Twigg et al. 2001). Twigg et al. (2001) demonstrated a more rapid loss of zinc phosphide whole-wheat bait under seasonal wet conditions compared to dry conditions, but the available bait was considered lethal to rats for 8-14 days under either condition. Degradation of zinc phosphide is more rapid for whole-grain formulations compared to pelletized baits. Sterner and Ramey (1995) found that about 33% and 87% of zinc phosphide (2.0% ai) was lost from broadcast rolled-oat bait within 7 and 14 days in a semi-arid environment receiving 10 mm of rainfall and a soil pH of 6.0. Koehler et al. (1995) reported that pelletized baits contained greater than 80% of the original levels of zinc phosphide after 16 days exposure with 15 to 45 mm of rainfall. In tropical environments, bait has shown to be less effective and degrade within three weeks (Sugihara et al. 1995).

2.4 Hazard Identification

At sufficient doses, zinc phosphide can be a hazard to human health and can be lethal at high doses due to acute oral and inhalation toxicities. The following section summarizes available acute and chronic toxicity data

for mammals that are used to evaluate the hazards of zinc phosphide to human health and nontarget mammals.

2.4.1 Toxicokinetics

Animal studies show that zinc phosphide is absorbed from the respiratory tract and gastrointestinal tract. It can also be absorbed through abrasions on the skin. It has a toxic action on the heart, liver, and kidneys. Death results from heart and kidney failure within 24 hours. Zinc phosphide does not accumulate in bodily tissues, but is excreted in the urine either as a hypophosphite or as dissolved phosphine. Other metabolites include phosphoric acid and phosphate. Animals that experience a delay in death after receiving a large dose of zinc phosphide may become prostrate with deep slow respiration, finally terminating in convulsions. After repeated doses, kidney damage and hyaline degeneration of the myocardium are observed, as well as cloudy swelling in liver, hyaline degeneration and necrosis (usually located in the center of the lobules) (HSDB 2017, Gervais et al. 2010).

Serious illness and fatalities have resulted from zinc phosphide ingestion. Periodic medical examination of workers is desirable. Workers suffering from active hepatic or renal disease should avoid contact with zinc phosphide (HSDB 2017).

2.4.2 General Poisoning Effects

There have been no reports of human deaths in the United States from exposure to zinc phosphide, but non-severe poisonings include occupational exposures at four veterinary hospitals (two in Michigan, one in Iowa, and one in Washington) when veterinary staff breathed in phosphine gases emitted from zinc phosphide exposed dogs (Centers for Disease Control (CDC) 2012). The dogs were accidentally poisoned by consuming rodenticides containing zinc phosphide. Following exposure, staff members experienced acute adverse health effects (such as shortness of breath, difficulty breathing, headache, nausea, dizziness, and chest pain), but everyone recovered in the presence of fresh air. While no deaths have been reported in the U.S., Europe has reported at least 25 deaths from zinc phosphide exposure (HSDB 2017). Currently, only supportive and resuscitative measures are available for zinc phosphide because antidote or specific therapies do not exist. The amount of zinc phosphide toxic to humans varies. Ingestion of 4-5 g has produced death in human adults on at least two occasions, whereas two other victims have survived doses of 25 and 50 g (HSDB 2017). Phosphine gas, which forms when zinc phosphide reacts with gastric acid in the stomach, is toxic. Phosphine toxicity causes cardiac, respiratory, and metabolic damage leading to death (Proudfoot 2009, Marino et al. 2014).

2.4.3 Acute Toxicity

Zinc phosphide is listed in Toxicity Category I (the highest of four categories) for acute effects via oral or inhalation route of exposure, Toxicity Category III for dermal route, and Toxicity Category IV for eye irritation (USEPA 1998) (Table 3). Comparative acute toxicity between technical grade and formulated zinc phosphide products suggest that the technical active ingredient is the more hazardous material, followed by the concentrate, which is still highly toxic, followed by bait formulations.

2.4.4 Subchronic and Chronic Toxicities

A 90-day study with rats using technical zinc phosphide (97% ai) established a no observable effect level (NOEL) of 0.1 mg/kg/day and lowest observable effect level (LOEL) of 1.0 mg/kg/day based on increased mortality and kidney hydronephrosis (USEPA 1998). Kidney related effects such as hydronephrosis and pyelonephritis were detected by microscopic histopathology in males at 3.0 mg/kg/day and 1.0 mg/kg/day. Lesions were not observed at 0.1 mg/kg/day (USEPA 1998).

A chronic toxicity study was not performed because chronic exposure to zinc phosphide, or degradates, is expected to be negligible (USEPA 1998).

Table 3. Acute technical and formulation zinc phosphide toxicity data for mammals.

Test species	% ai	Test	Results	Toxicity Category
Zinc Phosphide Technical (USEPA 1998)				
Rat	89%	Oral LD_{50}	21 (13-35) mg/kg	I
Rabbit	94%	Dermal LD_{50}	2000-5000 mg/kg	III
Rabbit	94%	Eye irritation	Slightly conjunctional redness, chemises and discharge	IV
Rabbit	94%	Dermal irritation	Non irritating	N/A
Rabbit	97%	Acute neurotoxicity	NOEL =5 mg/kg, LEL=10 mg/kg (myelin debris and vacuoles in peripheral nerves of 2 female rats)	N/A
Zinc Phosphide Concentrated Formulation (EPA Reg. No. 56228-6) (Pocatello Supply Depot 2004a)				
Rat	63.2%	Oral LD_{50}	40.5-46.7 mg/kg	I
Zinc Phosphide Grain Bait Formulations (EPA Reg. No: 56228-3, 14) (Pocatello Supply Depot 2004b)				
Rat	1.82%	Oral LD_{50}	911 mg/kg	III
Rat	2.0%	Oral LD_{50}	888 mg/kg	III

NOEL – No observable effect level

2.4.5 Developmental and Reproductive Effects

A developmental toxicity study (USEPA 1998) was performed in mated female rats (25/group) by administering zinc phosphide in single daily doses at levels of 0, 1, 2 or 4 mg/kg on days 6 through 15 of gestation. Nine maternal animals from the 4.0 mg/kg group were dead between days 10 and 16 of gestation. A gross examination did not identify the apparent cause of death. Mean body weight and food intake reductions in females were significantly lower for gestation days 6 to10. The maternal NOEL was 2.0 mg/kg and the lowest effect level (LEL) was 4.0 mg/kg based on mortality. The developmental NOEL was determined to be at or above 4.0 mg/kg (the highest dose test). A two-generation reproductive toxicity study in rats was not performed because residues were expected to be negligible.

2.4.6 Neurotoxicity Effects

Two 13-week subchronic neurotoxicity studies (USEPA 1998) were conducted in rats (11/sex/group) dosed by gavage with zinc phosphide (97% ai and 95% ai) daily via oral gavage at levels of 0, 0.1, 0.5, or 2 mg/kg. Neuropathology evaluation of the first study showed systemic, behavioral, and neuropathological NOELs of 2 mg/kg/day (the highest dose tested). Neuropathological examination of the second study suggested no adverse changes in morphology. However, alterations of posture, rearing, touch, click and pinch were observed in the mid- or high-dosed rats. Based on the two subchronic studies, USEPA (1998) established the overall NOEL for subchronic neurotoxicity at 0.1 mg/kg/day (the lowest dose tested).

2.4.7 Carcinogenicity and Mutagenicity

The USEPA (1998) did not require carcinogenicity studies during registration because chronic exposure to zinc phosphide, or its byproducts, is expected to be negligible. The mutagenicity potential of Zn_3P_2 has been evaluated by in vitro and in vivo testing. Zn_3P_2 (97% ai) has been shown to be non-mutagenic in two out of three toxicology studies (USEPA 1998). Zn_3P_2 was negative in both Ames test (Ames *Salmonella*) and mouse micronucleus test (chromosome aberrations). However, it produced a positive result in a mouse lymphoma assay (USEPA 1998).

3 DOSE-RESPONSE ASSESSMENT

3.1 Human Health Dose-Response Assessment

A dose-response assessment evaluates the dose levels (toxicity criteria) for potential human health effects. USEPA (1998) developed an oral reference dose (RfD) of 0.0003 mg/kg-day from a 13-week oral toxicity study in rats (Bai et al., 1980). This subchronic oral toxicity study reported a lowest observable adverse effect level (LOAEL) of 50 mg/L (3.48 mg/kg/day) based on decreased food intake and body weight. USEPA (1998) derived the RfD by applying an uncertainty factor of 10,000 (10 for interspecies extrapolation, 10 for providing added protection to unusually sensitive individuals, 10 for expanding subchronic to chronic exposure, and 10 for the lack of a no observable adverse effect level (NOAEL)) (USEPA 2012a). USEPA (2016a) developed an acute dietary RfD of 0.02 mg/kg-day based on a NOAEL of 2 mg/kg/day with an uncertainty factor of 10 for interspecies variability and an uncertainty factor of 10 for intraspecies variability.

Table 4. Acute median lethality zinc phosphide toxicity studies for mammals and birds.

Test species	Test	Results	Reference
Mammals			
Brushtail Opossum *(Trichosurus vulpecula)*	LD_{50}	9.6 mg/kg	Ross and Henderson 2006
California Ground Squirrel	LD_{50}	33.1 mg/kg	USEPA 1998
Pocket Gopher sp.	LD_{50}	6.8 mg/kg	USEPA 1998
Meadow Vole	LD_{50}	18 mg/kg	USEPA 1998
North American Deermouse	LD_{50}	42 mg/kg	Schafer and Bowles 1985
Roof Rat	LD_{50}	2.9-40 mg/kg	USEPA 1998
Nutria	LD_{50}	5.5 mg/kg	USEPA 1998
Black-tailed Jackrabbit	LD_{50}	8.2 mg/kg	USEPA 1998
Domestic Dog	ALD	40 mg/kg *	Johnson and Fagerstone 1994
Kit fox	LD_{50}	93 mg/kg	Schitoskey 1975
Birds			
White-fronted Goose	LD_{50}	7.5 mg/kg	USEPA 2004
Canada Goose	LD_{50}	12.0 mg/kg	Glahn and Lamper 1983
Mallard	LD_{50}	35.7-67.4 mg/kg	USEPA 2014
Northern Bobwhite	LD_{50}	12.9 mg/kg	USEPA 2014
Gray Partridge	LD_{50}	27 mg/kg **	Janda and Bösseová 1970
Domestic Chicken	LD_{50}	25 mg/kg	Shivanandappa et al. 1979
Ring-necked Pheasant	LD_{50}	16.4 mg/kg	USEPA 2014
Mourning Dove	LD_{50}	34.3 mg/kg	USEPA 2004
Golden Eagle	LD_{50}	>20 mg/kg	Hudson et al. 1984
Horned Lark	LD_{50}	47.2 mg/kg	Hudson et al. 1984
Red-winged Blackbird	LD_{50}	23.7 mg/kg	USEPA 2004

*ALD – Acute Lethal Dose – estimated LD_{50} when unable to calculate **Reported LD_{05} of 16 mg/kg and LD_{95} of 46.5 mg/kg

3.2 Ecological Effects Analysis

This section of the risk assessment discusses available ecological effects data for terrestrial and aquatic biota. For certain nontarget organisms, the effects of zinc phosphide after degradation is discussed since the mode of action precludes significant effects from exposure to zinc phosphide, which requires acidic conditions to release phosphine gas.

3.2.1 Aquatic Effects Analysis

Aquatic Vertebrates and Invertebrates

Aquatic toxicity data for zinc phosphide is limited to three acute toxicity studies with fish and a freshwater invertebrate (APHIS 2002). These studies were submitted to USEPA to support registration of zinc phosphide and summarized by the Zinc Phosphide Consortium that was formed in 1991 to respond to requests for new studies. USEPA determined that the studies were not acceptable because they were not conducted using a flow-through system and zinc ions were measured instead of phosphine. The lack of standardized toxicity data is due to the low solubility of zinc phosphide in water and limitations in being able to get the material into solution using various solvents. Available data for fish report 96-hr median lethal concentration (LC_{50}) values of 0.7 mg/L for bluegill and 0.8 mg/L for the rainbow trout. One study using the freshwater cladoceran (*Daphnia magna*) reported a 96-hr LC_{50} of 0.23 mg/L of Zn^{++} based on the assumption that all the zinc phosphide disassociated during the exposure. Zinc phosphide is expected to degrade to a zinc ion, so effects data for zinc are also evaluated. The other by-product of zinc phosphide degradation, phosphine, is not expected to occur in aquatic systems due to its volatility and other fate properties. Available aquatic toxicity data for phosphine is limited to a 96-hr LC_{50} for rainbow trout (0.0097 mg/L) and a 24-hr EC_{50} value for *D. magna* (0.2 mg/L).

An extensive amount of literature exists regarding the toxicity of zinc to aquatic organisms. The following discussion relies on summaries of the literature regarding the impacts of zinc to aquatic organisms under various conditions. This is not a comprehensive analysis of all available research since the potential for significant zinc exposure to aquatic vertebrates and invertebrates is considered negligible based on the proposed use pattern.

Zinc toxicity to animals in aquatic environments varies depending on water quality parameters, as well as other variables such as organic matter, acid-volatile sulfide levels, suspended sediment, and other water characteristics that can affect bioavailability. Decreasing pH results in reduced zinc toxicity to fish such as steelhead (Cusimano et al. 1986). Reported 96-hr LC_{50} values ranged from 66 µg/L at a pH of 7.0 to 671 µg/L at a pH of 4.7. Water quality parameters can also affect the toxicity of zinc to prey items for aquatic vertebrates. For example, increasing water hardness and humic acid concentrations resulted in decreased acute and chronic effects to freshwater cladoceran (*D. magna*). In 50-day exposures, no observable effect concentrations (NOEC) ranged from 25 µg/L at a low humic acid concentration to 100 µg/L, at elevated humic acid concentrations (Paulauskis and Winner 1988). A similar decrease in toxicity with increasing hardness and decreasing pH has also been observed in rainbow trout (Bradley and Sprague +85). The variability in the toxicity of metals, including zinc, based on water quality parameters has been characterized using biotic ligand models in acute and chronic exposures (Deschamphelaere and Janssen 2004, Santore et al. 2002, Heijerick et al. 2005). These papers summarize much of the toxicity data for zinc demonstrating that acute median lethal effects typically range from 65 µg/L to greater than 10 mg/L for various fish species (Chapman 1978a, Cusimano et al. 1986). A summary of chronic effect data for various fish species shows that NOECs can

generally vary from 16 µg/L to 6210 µg/L in exposures lasting a few days to several months (Eisler 1993, Deschamphelaere and Janssen 2004). Chronic exposure studies using sockeye salmon demonstrated no sublethal impacts on adults or reproduction in a 21-month exposure to zinc at concentrations of 112 and 242 µg/L (Chapman 1978b).

Aquatic invertebrate acute toxicity is highly variable depending on the test species and water quality parameters. Available median lethality data ranges from about 30 µg/L to greater than 40 mg/L with freshwater aquatic crustaceans being the most sensitive taxa tested (Eisler 1993).

Aquatic Plants

Phytoplankton and aquatic macrophyte data shows zinc-related negative effects to growth in various aquatic plant species ranging from 19 µg/L for the diatom, to greater than 10 mg/L for a variety of algal species (Eisler 1993).

3.2.2 Terrestrial Effects Analysis

Mammals

A large number of acute lethality studies (>60) using zinc phosphide have been summarized for various mammal species (Johnson and Fagerstone 1994, USEPA 2004, Eason et al. 2013). Median lethality values range from 2.9 mg/kg for the roof rat to 93 mg/kg for the kit fox demonstrating that zinc phosphide is highly acutely toxic to mammals (Table 4).

Sublethal and chronic effects to nontarget mammals have been summarized in Sections 2.4.4 – 2.4.7 and would be expected to be similar for various wild mammal species. In addition to the previously discussed physiological effects, nontarget wild mammals that ingest sublethal doses of zinc phosphide through primary or secondary pathways could possibly display behavioral effects. Bell and Dimmick (1975) reported lethargy and food avoidance in gray and red foxes fed voles that had received lethal doses of zinc phosphide.

Birds

Acute avian toxicity to zinc phosphide is high based on various oral dosing studies using several test species (Table 4). Geese appear to be the most sensitive species to zinc phosphide toxicity while mallards and horned larks seem to be the least sensitive test species.

Eight-day dietary median lethality values ranged from 469 to 859 ppm for Northern bobwhites and 1285 to 2885 ppm for mallards (USEPA 2014). Sublethal impacts in various bird dosing studies include reduced weight, lethargy, and ataxia. Chronic avian reproduction studies were not available. Sublethal exposure to zinc phosphide *in vivo* and *in vitro* result in mutagenic impacts to chickens in a dose dependent manner at concentrations ranging from 0 to 0.5 g/mL (Muid et al. 2012).

Reptiles and Terrestrial Phase Amphibians

Some zinc phosphide toxicity data for reptiles has been reported for non-native lizards (Avery et al. 2011), but none for terrestrial phase amphibians. Black spiny-tailed iguanas were given seven doses of zinc phosphide (includes control group 13 mg/kg to 488 mg/kg. All animals dosed at 61 mg/kg or greater died

during the study while no animals in the 13 or 27 mg/kg dosing group died. Time to death ranged from 82 hours in the 61 mg/kg dosing group to 16 hours in the 488 mg/kg group. Witmer et al. (2010) orally dosed American alligators in two separate studies with zinc phosphide levels ranging from 3 to 240 mg/kg and determined a combined LD_{50} value of 28 mg/kg.

Terrestrial Invertebrates and Microorganisms

Standardized toxicity studies do not appear to be available for zinc phosphide effects to terrestrial invertebrates. Significant impacts to most terrestrial invertebrates are not expected due to the mode of action and method of application for zinc phosphide. Potential effects would be greatest for ground dwelling invertebrates. Deisch et al. (1989) studied the effect that zinc phosphide had on invertebrates. They determined that zinc phosphide bait reduced ant densities, but spider mites, crickets, wolf spiders, ground beetles, darkling beetles, and dung beetles were not affected. Wolf spiders and ground beetles showed increases after one year on zinc phosphide treated areas (Deisch 1986). Generally, direct long-term impacts from rodenticide treatments were minimal for the insect populations sampled (Deisch et al. 1989). Long-term effects were not directly related to rodenticides, but more to habitat changes (Deisch 1986) as vegetative cover and prey diversity increased without prairie dogs grazing and clipping the vegetation (Deisch et al. 1989). Eason et al. (2013) summarized results from an unpublished technical report that evaluated the acute and chronic toxicity of zinc phosphide to earthworms (*Eisena foetida*). The reported LOECs ranged from 32 mg/kg for cocoon production, 1000 mg/kg based on reproduction, and 3200 mg/kg for growth. No lethality was observed at the highest test concentration, which was greater than 3200 mg/kg.

Terrestrial Plants

The mechanism of action for zinc phosphide suggests negligible effects to terrestrial plants. The possibility exists that unconsumed baits not removed after treatment would degrade and allow zinc to occur in soils and be available for plant uptake. Zinc is a naturally occurring element in soils and is important for plant growth. However, excessive zinc in soils can result in phytotoxicity. Toxicity of zinc to terrestrial plants varies based on the test species and soil specific factors that can greatly influence bioavailability and plant uptake. Zinc can affect various metabolic functions in plants resulting in chlorosis, reduced growth and early senescence (Nagajyoti et al. 2010). Plant tissue levels resulting in greater than 10% effects to plant growth parameters are typically greater than 100 mg/kg (Reichmann 2002). The amount of zinc that would be available for plant uptake from soils under any scenario where zinc phosphide baits were left to degrade would not be a high enough concentration to cause adverse effects to terrestrial plants.

4 EXPOSURE ASSESSMENT

4.1 Human Health Exposure Assessment

Exposure assessments estimate the potential exposure of humans to zinc phosphide. The exposure assessment begins with the use and application method of zinc phosphide. An identified exposure pathway for zinc phosphide includes (1) a release from a source, (2) an exposure point where contact can occur, and (3) an exposure route such as ingestion, inhalation, or dermal contact by which contact can occur (USEPA 1989). Exposures for the identified human populations are qualitatively evaluated for each identified exposure pathway.

4.1.1 Potentially Exposed Human Populations and Complete Exposure Pathways

Based on the expected use pattern for zinc phosphide applications, workers who are mixing the APHIS-registered concentrated formulation and applying the rodenticide in the field are the most likely subgroup of the human population that could be exposed to zinc phosphide. Exposure during transportation is not anticipated because the container of the concentrated material is sealed. Among the three products that APHIS is the registrant, only the concentrated formulation requires mixing in the field. The other two formulations are ready to use grain baits. Zinc phosphide concentrate granular powder bait for bait preparation is mixed with a lecithin and mineral oil mixture and poured over grains, mixed with vegetable oil and poured over fruits or vegetables, or slowly blended into meat. The application methods for the baits mixed from the concentrate formulation include spot and hand baiting, burrow and burrow trail builder baiting, ground and aerial broadcast applications, and filling a bait station. The burrow builder application is a tractor drawn device that creates a tunnel through the soil and drops a measured quantity of zinc phosphide into the tunnel. Trail builder baiting involves the use of machinery to make applications in furrow. The application methods for the oat and wheat baits formulations include hand and trail-builder baiting, and ground and aerial broadcast applications. Strictly adhering to label directions, especially the use of proper personal protective equipment (PPE) minimizes worker exposure to zinc phosphide baits via inhalation and dermal contact routes. PPE for the concentrated formulation requires chemical-resistant gloves, protective eyewear, long sleeve shirt, long pants, socks, and shoes. In addition, workers must wear a dust/mist filtering respirator (MSHA/NIOSH) approved number prefix TC-21C or a NIOSH-approved respirator with an R (resistant to oil), P (oil proof), N (not resistant to oil), or high efficiency (HE) particulate filter[4]. All three formulations are restricted use, which means only certified applicators or persons under their direct supervision may use the product. Accidental exposure may occur during mixing, loading, and application activities. As a conservative approach, potential accidental exposure for workers was further quantified for dermal contact and inhalation routes during mixing. Comparing to loading and application activities, mixing represents the most likely exposure potential. The ingestion route was not quantified because the exposure is minimal.

A significant exposure pathway was not identified for direct contact with zinc phosphide by the general public because the use restrictions and use patterns of each formulation minimize or nullify this potential (see Section 2.1). Applications near residential areas are prohibited. The general public is unlikely to access areas such as golf courses and parks where zinc phosphide baits to control rodents may be applied; the potential exposure to zinc phosphide is low, though, since the general public is not allowed in the treated areas during application and unused baits following treatment are retrieved where possible (broadcast and underground bait applications are generally not retrieved or monitored on-site). Warning signs are posted when zinc phosphide is being applied in an area potentially accessible by the public. A significant exposure pathway was not identified for human dietary plant consumption exposed to zinc phosphide because the rodenticide will not be applied directly to most food crops or fruit trees. Use of zinc phosphide in potato, timothy hay, and wheat crops is allowed as no residues above the LOQ were found in these commodities (USEPA 2016a). However, residue data for alfalfa, barley, dry beans, and sugar beets was not as conclusive, but supported use regions in Washington, Oregon and Idaho (USEPA 2016a). Residue data in California supported use in alfalfa.

A significant exposure pathway was not identified for groundwater or surface water because zinc phosphide is expected to be insoluble in water and susceptible to hydrolysis and degradation to zinc ions and phosphine gas. In addition, label restrictions regarding applications near surface water further protect those sources that could be used for drinking water.

4.1.2 Exposure Evaluation

This section quantitatively evaluates worker exposures from an accidental direct contact pathway while mixing zinc phosphide baits using the concentrate formulation. Under the accidental exposure scenario, it assumes that chemical-resistant gloves are broken for dermal route and respirators do not function properly for inhalation route.

Chemical-specific data to assess potential exposure to occupational pesticide handlers were not available. The estimates of exposure to pesticide handlers are based on surrogate study data available in the Pesticide Handlers Exposure Database (PHED) (USEPA 2016b). The mixing and loading granules exposure scenario in the PHED was used to estimate potential exposure during mixing. Under this exposure scenario, the unit exposures are 8.4 ug/lb ai and 1.7 ug/lb ai for dermal contact under the single layer (long-sleeve shirt, long pants, shoes plus socks) and no gloves, and for inhalation under no respirators PPE levels, respectively (USEPA 2016b). Per the zinc phosphide concentrate formulation label instructions for preparation of 2% grain baits, a worker may be exposed to 2 lb of zinc phosphide during mixing to make 100 lb of bait under a typical scenario. Under an occasional scenario when a worker needs to make enough grain baits to treat 200 acres per day (0.2 lb a.i. per acre), the worker may be exposed to 40 lb zinc phosphide active ingredient during mixing. The occasional scenario represents a worst-case exposure scenario. The following equations were used to estimate the exposure dose of direct contact for workers:

Exposure Dose = Daily Dose Rate/Body Weight

Daily Dose Rate = Unit Exposure (mg/lb ai) x Daily Mixing Concentration (lb ai/day)

Estimates of exposure doses from zinc phosphide from dermal and inhalation routes are summarized in Table 5.

4.2 Ecological Exposure Assessment

4.2.1 Aquatic Exposure Assessment

The environmental fate and chemical properties of zinc phosphide suggests that any treated bait that could fall into the water would not result in detectable levels. Zinc phosphide is not soluble in water and would degrade to the zinc and phosphine gas or be degraded in the sediment to phosphine or phosphate. Phosphine gas is volatile and insoluble in water, but zinc ions would remain in aquatic environments either in solution or in sediment and could result in exposure to aquatic organisms. Zinc phosphide use for nutria control was used as a means to make a conservative estimate regarding potential aquatic residues. The methods of application for the other two formulations, the type of formulation (bait), and their use restrictions suggest detectable residues would not occur in aquatic habitats.

A highly conservative approach to determine the potential for zinc phosphide and zinc residues related to nutria applications in shallow ponds was used as a means to determine potential concentrations. The maximum amount of formulated zinc phosphide that can be added to the bait is approximately 1.4 ounces or 39.69 grams. The formulation contains 63.2% zinc phosphide so the percentage by weight conversion would result in 25.08 grams of zinc phosphide that would be mixed with 10 pounds of bait. The amount of zinc would be less since the assumption is that all of the material is zinc where a proportion would also be phosphide. Assuming that all bait applied to a large raft on a given day is deposited into a one-foot deep acre

pond results in a total loading of 0.020 mg/L zinc phosphide. Bait may be replenished if consumed by rodents up to an amount of 5 pounds each day over a 72-hour period. However, if rodents removed the baits, it would not be available to aquatic fish and invertebrates, and, therefore, was not considered in the exposure analysis. The occurrence of all 10 pounds of bait deposited into the water is not anticipated since rodents typically consume most of the bait, excess baits are removed following application, and the raft is constructed in a way to minimize bait falling into the water. Under a typical application, following label directions, zinc is not expected to be present in water based on the known chemistry and environmental fate properties of zinc phosphide. As previously mentioned zinc phosphide is considered insoluble in water so detectable levels of zinc phosphide would not be anticipated in the water column. However, for the sake of analysis, we will assume all 25.08 g or almost 1 ounce falls into the water prior to rodents consuming any, which could possibly happen under unexpected excessive winds or torrential rains. This unrealistic exposure scenario was used as a conservative estimate of exposure to account for some of the uncertainty regarding the potential for exposure and available effects data. Additionally, zinc phosphide will be present at the air and water interface in ponds since zinc phosphide is mixed with vegetable oil. However, this material would be susceptible to degradation and the amount of oil used in a 10 pound batch of treated bait is relatively low (1 oz.).

4.2.2 Terrestrial Exposure Assessment

The primary exposure pathway for terrestrial nontarget wildlife will occur primarily through ingestion of the bait. Dermal and inhalation exposure is not expected to be a significant exposure pathway based on formulation properties and anticipated application methods. Drinking water is not expected to be contaminated due to restrictions regarding zinc phosphide applications near water and the environmental fate of zinc phosphide in water. Hence, drinking water was determined not to be a significant pathway either. Dietary exposure may also occur for nontarget wildlife through scavenging or preying on target mammals that have received a dose of zinc phosphide. The qualitative risks from this type of exposure are discussed in the risk characterization section of this risk assessment.

Estimates of primary exposure to bait for nontarget wildlife were based on the range of application rates labelled for zinc phosphide. Several methods of application are allowed for zinc phosphide, but use-rates allowing broadcast application were used to estimate the exposure to nontarget wildlife from zinc phosphide. Broadcast applications were assumed to result in the highest potential nontarget wildlife exposure compared to other application methods for the three zinc phosphide labels. Estimated environmental concentrations were calculated based on the ai percentage in the zinc phosphide wheat formulation (1.82%) and the assumption that one bait kernel weighs approximately 31 mg and each kernel has an average of 0.62 mg of zinc phosphide on it. The number of kernels required to exceed toxicity for consumption by various sized mammals and birds was estimated and is discussed in the risk characterization section of this risk assessment (Table 6).

5 RISK CHARACTERIZATION

This section discusses the quantitative and qualitative risks associated with the use of zinc phosphide. Integration of the effects data with the exposure assumptions can be made to determine the risk to human health and nontarget fish and wildlife. Deterministic methods are used, where appropriate, to determine if expected environmental residues exceed toxicity data, which would suggest possible risk. To elaborate the potential risks, a qualitative discussion may be used relying on literature and other information.

5.1 Human Health

Risks associated with adverse human health were characterized quantitatively for the potential complete exposure pathways in this section. Under the WS uses, zinc phosphide applications to control rodent populations should pose minimal risks to human health.

To quantify the potential risks to workers from accidental exposure during mixing using zinc phosphide, estimated exposure doses were derived based on conservative assumptions (Table 5). The accidental risk estimates are based on the typical exposure scenario during a typical workday and an occasional scenario when 200 acres may be applied per day. The exposure estimates were then compared to the reference dose of 0.02 mg/kg/day for workers to calculate hazard quotient (HQ) values as shown in the below equation:

HQ = Exposure Dose/Reference Dose

The estimated HQs for the typical exposure scenario were 0.01 (dermal) and 0.002 (inhalation). The estimated HQs for an occasional exposure scenario were 0.2 (dermal) and 0.04 (inhalation) (Table 5). The estimated hazard quotients from accidental exposure for both scenarios are below the USEPA (2000) acceptable hazard quotient of one. The risk assessment results show that the estimated risks from accidental zinc phosphide exposure are low.

Table 5. Risk estimations for potential accidental exposure during mixing.

PARAMETER UNITS	EXPOSURE SCENARIO	MIXING		SOURCES
		DERMAL	INHALATION	
Dose = PDR/BW				
Dose mg/kg-day	Typical	2.1E-04	4.3E-05	Calculated
	Occasional	4.2E-03	8.5E-04	
BW - kg	(Adult BW used)	80	80	USEPA 2012b
PDR = UE * DMC mg/day				
PDR mg/day	Typical	0.0168	0.0034	Calculated
	Occasional	0.336	0.068	
UE mg/lb a.i	single layer, no gloves or respirator	0.0084	0.0017	UE for mixing and loading granules minimal PPE level (USEPA 2016b).
DMC lb ai/day (based on treating a max of 200 acres/day hand baiting)	Typical	2	2	Grain Baits/Mixed Baits = 2% ai ZnPh (per ZnPh grain and concentrate labels)
	Occasional	40	40	Mixing the amount for 200 acres per day with 0.2 lb a.i. per acre.
Reference Dose				
RfD - mg/kg/day		0.02	0.02	Acute oral RfD, USEPA 2016a
HQ = Dose/RfD				
HQ mg/kg-day	Typical	0.01	0.002	Calculated
	Occasional	0.2	0.04	

PDR = Potential Dose Rate BW = Body Weight UE = unit exposure DMC = daily mixing concentration
RfD = reference dose HQ = Hazard Quotient

5.2 Ecological Risks

5.2.1 Aquatic

The risk from the use of zinc phosphide will be negligible to all aquatic organisms. The exposure analysis used unrealistic, overly conservative, assumptions about zinc residues in static shallow water bodies where rafts containing treated bait could occur. Not all bait from an application would enter the water since the target animals consume most of the baits and the construction of large rafts prevents the bait from rolling off into an aquatic habitat. If all bait from an application was deposited into an isolated static pond the environmental fate of zinc phosphide and its degradates would not result in concentrations that would exceed acute or chronic thresholds for aquatic organisms. The estimated residue of 20 µg/L of zinc phosphide is an order of magnitude below the available effects data for zinc phosphide. Recognizing that this assumption is based only on a limited amount of lethality data there is some uncertainty regarding sublethal risks and other taxa. However, we do not expect these types of risks based on the proposed use pattern and environmental fate of zinc phosphide. Assuming all applied zinc phosphide disassociates in the water column and is bioavailable as zinc, the estimated residue is below zinc acute and chronic effects data for fish and aquatic invertebrate as discussed. Impacts to aquatic vertebrates through loss of prey items or ingestion of prey that may contain zinc residues is not expected based on the proposed use pattern of zinc phosphide, the very low potential for detectable residues, and the lack of zinc concentrations that could result in negative impacts to prey populations. Protection of isolated shallow water bodies such as the one considered in this assessment was assumed to also be protective of other aquatic habitats such as flowing waterways and larger static water bodies such as lakes and ponds.

Label restrictions for the various zinc phosphide formulations further reduce risks to aquatic biota as they are designed to reduce exposure. For example, the zinc phosphide labels have application restrictions prohibiting treatment of areas when precipitation is predicted. This type of restriction reduces the probability of runoff related to storm events. The same label also states that prohibited applications include applying directly to water, to areas where surface water is present, or to intertidal areas below the mean high-water mark. Additional restrictions state that applications should not occur when runoff is likely to occur and to avoid contaminating water from cleaning equipment or waste disposal near water sources.

5.2.2 Terrestrial

The lowest reported mammalian LD_{50} (roof rat LD_{50} = 2.9 mg/kg) and the lowest reported avian LD_{50} (bobwhite LD_{50} = 12.9 mg/kg) was used to calculate weight adjusted LD_{50} values for three different size classes of mammal and birds (Table 6). Those values were then compared to residues that would be expected in zinc phosphide bait using the wheat formulation (1.82% ai). The number of pellets that would have to be consumed to reach the LD_{50} level and 1/10 of the LD_{50} level were estimated based on the parameters that the average wheat kernel weighs 31 mg and that each kernel contains 0.62 mg of zinc phosphide (Table 6). The number of pellets needed to be consumed to exceed the LD_{50} is a minimal number considering it is only part of their daily ingestion. For example, a species that weighs 20g probably has a high intake level, about 10% of the body weight or 2 g, so 1 kernel is 1.5% of their daily intake whereas a species that weighs 2000 g has a lower intake, probably about 5% of their body weight, so birds need to eat about 3% of their daily intake to reach the Oral LD_{50} (Table 6). The number of baits needed to exceed weight adjusted acute median lethality values ranged from 1 to 21 for the differing weights of mammals and 1 to 92 for birds. The low number of pellets required to exceed acute lethality are within daily food consumption rates that would be expected for

mammals and birds in these size classes suggesting high acute risk to mammals and birds. These risk estimates do not account for any potential sublethal impacts that could occur from ingestion of bait pellets.

Table 6. Number of wheat kernels required to exceed acute median lethality values for zinc phosphide in mammals and birds of specific weight classes. Each wheat kernel weighs 31 mg and has 0.62 mg zinc/kernel.

Weight (g)	Example Species for Weight	Oral LD_{50} for Weight	# Kernels Needed (wt) to Exceed Oral LD_{50}	# Kernels Needed for 1/10 Oral LD_{50}
Mammals (based on 2.9 mg/kg)				
20 g	Pocket Mouse (*Chaetodipus* sp.)	0.13 mg	1 (0.03 g)	1
200 g	White-toothed Woodrat	1.28 mg	3 (0.1 g)	1
2000 g	Raccoon (small)	12.77 mg	21 (0. 7 g)	2
Birds (based on 12.9 mg/kg)				
20 g	Lincoln's Sparrow	0.57 mg	1 (0.03 g)	1
200 g	Northern Bobwhite	5.68 mg	10 (0.3 g)	1
2000 g	Cackling Goose	56.83	92 (2.9 g)	10

Field data collected on risks to nontarget mammals has shown that the risk of take in the field is less than laboratory estimates of risk. Uresk et al. (1987) showed a decline in field mice populations after treatment of fields with zinc phosphide to control prairie dogs. The difference was not statistically significant due to variability observed in the field mice populations. Johnson and Fagerstone (1994) reviewed published as well as unpublished reports and found that nontarget mammal impacts occurred, but risks were minimal with low numbers taken in local areas. Several rodent species are associated with target mammals such as prairie dogs and ground squirrels so nontarget rodent mortality can occur.

Several field studies have been conducted to evaluate the potential impacts of zinc phosphide on various avian species. Fellows et al. (1988) reported low risk to the spotted dove and northern cardinal in a macadamia orchard treated with oat bait containing zinc phosphide. No zinc phosphide related mortality was observed in a study area and no sublethal effects related to zinc phosphide were noted in mist-netted birds. A tracer in the bait was used to determine if birds had been exposed and in the case of the northern cardinals, >70% of the captured birds showed that they had consumed some bait. Uresk et al. (1987) showed no statistical difference in the number of ground feeding bird species when comparing zinc phosphide treated fields to controls. Hegdal and Gatz (1977) conducted carcass searches over a two-week period after zinc phosphide bait (2%) applications to control voles in Michigan orchards. One blue jay carcass and 1 of 5 radio-equipped pheasants were recovered during the study. In another study using radio-equipped pheasants, Ramey et al. (1998) evaluated risk to birds in zinc phosphide treated alfalfa fields. No mortality was observed related to the treatment. However, some of the pen-reared pheasants moved into other habitats and several were captured by predators. Ramey and Sterner (1995) reported no mortality of sublethal signs of intoxication for California quail within outdoor enclosures planted in alfalfa and treated with 2.0% zinc phosphide. In the same study, pheasant mortality was 62% with greater than 90% of the deaths occurring in less than 24 hours. Surviving birds exhibited sublethal effects consistent with zinc phosphide poisoning (lethargy, hypotaxia, and ataxia). The difference in response between the two bird species was attributed to bait aversion by the California quail. Four bird mortalities were observed in zinc phosphide treatments to control the house mice (Brown et al. 2002). No bird mortalities were found following zinc phosphide treatments on rangeland for Richardson's ground squirrels (Matschke et al. 1983) and black-tailed prairie dogs (Apa et al. 1991). Johnson and Fagerstone (1994) provided a review of several published and unpublished field studies that were conducted to determine the impacts of zinc phosphide to avian species in various habitats (rangeland, orchards, agricultural fields). The number of avian mortalities observed in these studies was low to nonexistent. Passerine birds occupy many of the study areas and are less sensitive to zinc phosphide

compared to other birds such as waterfowl. In addition, most of these treatments occurred during times when other feed was available for foraging, thereby reducing the potential for exposure. While these types of studies suggest avian risk is less than predicted using deterministic methods, uncertainty exists regarding some results. Carcass searches can be used as a tool for evaluating mortality events, but search efficiency varies widely and underestimates the actual number of mortalities (Vyas 1999). Scavenging may also occur and nontarget animals may move offsite after receiving a lethal dose of zinc phosphide and, therefore, would not be found in mortality searches. In addition, variability in population numbers makes it difficult to determine statistically significant difference between populations in treated and untreated fields when those types of comparisons are assessed.

The differences in avian risk between field evaluations and laboratory estimates relate to several factors. Bait attractiveness can vary among species based on the type and color of zinc phosphide baits used in a control program. Bait size, composition, and color can affect whether birds may consume treated bait pellets (Jacob and Leukers 2008). In cases where birds are bait-averse because of these characteristics, exposure and risks to zinc phosphide are reduced. Exposure and risks are further reduced if availability is reduced by the target species removing baits quickly because baits that remain in place over a longer period are also available for birds to forage. Birds are able to regurgitate bait pellets as a result of the emetic action reducing exposure and the probability of mortality whereas rodents cannot. However, in cases where the bait is available and attractive to birds, mortality events and sublethal effects can occur. Bildfell et al. (2013) summarizes several epizootic mortality events for various species of geese that occurred in Oregon between 2004 and 2011. The events range in size from 5 to greater than 300 birds where toxicosis was determined to be a result of zinc phosphide poisoning. Geese appear to be particularly sensitive with other poisoning incidents occurring in other parts of the United States (Johnson and Fagerstone 1994). Zinc phosphide poisoning in wild turkeys has occurred multiple times in Pennsylvania (Poppenga et al. 2005). In addition, the available EPA incident database reported 25 incident reports for zinc phosphide with 22 of the reports related to birds (USEPA 2004).

Several label restrictions for each of the formulations are designed to reduce exposure to domestic animals and nontarget wildlife. Prohibiting access to treated areas reduces risk to livestock. For several uses of zinc phosphide, applications are made by hand and focus just around mounds or burrows, or other active areas for the target mammal, which inherently minimizes potential exposure. In those cases, consumption of the bait by the target pest is more likely and the lack of broadcast treatments further reduces exposure to nontarget wildlife. Applicators do not treat an area if nontarget animals are found feeding during the prebait period. Tamper-resistant bait stations in areas accessible by children, pets, or nontarget wildlife also reduces risk. These types of restrictions reduce the potential for exposure and risk to nontarget organisms such as domestic animals and nontarget wildlife.

Secondary poisoning risks to predators and scavengers from zinc phosphide exposure are low especially when compared to other rodenticides. Zinc phosphide and phosphine does not accumulate in the tissue of target animals, and therefore, secondary risk is related to only the consumption of undigested bait in target animals. USEPA (2004) summarized incident reports for various rodenticides and no incidents involved raptors or corvids. These types of birds would be more likely to scavenge or prey on mammals that have received a dose of zinc phosphide. While the total number of incidents most likely underestimates the actual number that may have occurred, the proportion of incidents were almost exclusively granivorous birds and mammals that consumed treated baits directly, primary exposures. The lack of secondary risk to nontarget vertebrates has also been summarized in other studies. Schitoskey (1975) observed no mortality to kit fox fed kangaroo rats that received well above a lethal dose of zinc phosphide. Matschke et al. (1992) observed no poisoning symptoms or lethality to domestic ferrets that were fed various tissues of zinc phosphide-killed

black-tailed prairie dogs. Two tissue groups were evaluated to determine potential secondary poisoning effects: stomach, liver and intestine in one group and the remaining carcass in the second group. Hill and Carpenter (1982) did not observe lethality in Siberian ferrets or Steppe polecats (*Mustela eversmanii*) fed rats given lethal doses of zinc phosphide. Acute effects related to emesis were reported for ferrets, which reduces the probability of lethality by reducing the formation of phosphine. Sublethal effects related to multiple blood chemistry parameters suggested alterations in kidney and liver function. These and other studies have been summarized in various publications documenting a low potential for secondary poisonings related to zinc phosphide (Johnston and Fagerstone 1994, Joermann 1998, USEPA 2004). This includes birds such as corvids, raptors and various owl species as well as mammals that may prey or scavenge zinc phosphide dosed animals. Many of these studies evaluated lethality as the primary endpoint. However, uncertainty exists regarding potential sublethal impacts, which have been observed in other studies. Available studies using reptiles suggest a low secondary poisoning risk from zinc phosphide based on a study conducted using gophersnakes. Test animals were fed zinc phosphide dosed mice and were observed for any mortality or other clinical signs. No mortalities were observed, but emesis was observed in 2/3 of the test subjects (Brock 1965).

Secondary poisoning risks to domestic cats and dogs are also low. Sterner (1996) established a low risk potential for secondary poisoning of cats and dogs based on expected zinc phosphide residues in treated voles. Estimated zinc phosphide residues in voles were compared to the available acute toxicity data for cats and dogs to determine the number of voles that would have to be consumed to reach a lethal dose. The estimates showed that an unrealistic number of voles would have to be consumed to reach a lethal dose, but sublethal impacts to domestic animals was not discussed. The probability of lethal or sublethal impacts from secondary poisoning to wildlife and domestic animals is related to the amount of undigested bait in targeted animals at the time of death, which parts of the carcass are ingested, and environmental factors affecting the decomposition of the carcass.

Available data regarding effects to terrestrial plants and soil invertebrates suggest that risk from zinc phosphide, and associated degradates, is very low. Toxicity data for zinc phosphide and zinc when compared to the application rates of zinc phosphide show that soil levels would be well below any potential effects. Residues would actually be much less since bait would be removed by the target animals and in certain use patterns applications are focused in small areas where the animals are more active. Any potential impacts would be localized to areas immediately under a pellet that was not removed and allowed to degrade.

6 UNCERTAINTIES AND CUMULATIVE IMPACTS

The uncertainties associated with this risk evaluation arise primarily from a lack of toxicity information on the various formulations. Safety data sheets provide some acute mammalian toxicity values that can be used to make general comparisons between the formulations and the active ingredient. That information shows comparable or reduced hazard when comparing effects to the technical active ingredient. Other uncertainties related to chronic and sublethal effects data for some fish and wildlife, as well as surrogacy of test organisms are typical for most pesticides. However, a considerable amount of field data related to nontarget zinc phosphide poisoning of wildlife was found. This information provides a weight of evidence approach to evaluate the risk of zinc phosphide to nontarget organisms. The conservative assumptions regarding the potential for exposure to human health and the environment address the uncertainties to some extent. A lack of risk using these conservative assumptions supports the reasonable certainty that impacts to human health and the environment will be negligible.

Zinc phosphide use patterns vary for different sites and application methods. To account for variations in use patterns, the risk assessment used conservative assumptions regarding human exposure to ensure that risks are not underestimated for workers. Two exposure scenarios representing burrow builders and hand baiting were used to estimate the lower bound and upper bound of risk. The maximum application rate of 0.4 lb ai per acre per year was used for the upper bound risk calculation. The oral RfD was used to estimate dermal risk, which is also highly conservative since dermal toxicity, is 100 fold less than oral LD_{50} values.

Another area of potential uncertainty in this risk assessment is the potential for cumulative impacts to human health and the environment from the proposed use of zinc phosphide in the WS program. Areas where cumulative impacts could occur are: 1) repeated worker and environmental exposures to zinc phosphide from site applications and other uses; 2) co-exposure to other chemicals with a similar mode of action; and 3) exposures to other chemicals affecting the toxicity of zinc phosphide.

Repeated exposures that could lead to significant risk from zinc phosphide from program applications are not expected because workers are required to wear PPE. An accidental exposure may occur from the improper use of PPE, but these are not anticipated to lead to substantial risk or accumulation in the body (See Section 4.1). Label restrictions against applications near residential areas will reduce the potential for cumulative impacts related to exposures to zinc phosphide and other stressors.

Program applications of zinc phosphide may occur in areas where other pesticide applications have taken place. All zinc phosphide formulations are restricted-use, therefore applications will only occur by certified applicators or persons under their direct supervision. Other chemicals with a similar mode of action are aluminum and magnesium phosphide fumigants. Similar to zinc phosphide, these fumigants degrade to form phosphine. WS uses aluminum phosphide to control burrowing rodents as well, so the potential exists for WS personnel to be exposed to both zinc and aluminum phosphide. However, the proper use of required PPE for both products reduces exposure to WS personnel and the aggregate risks would be minimal. Aluminum and magnesium phosphide are also restricted-use chemicals and would not be applied near residential areas where public exposure could occur. Exposure to other pesticides could occur during program application in orchards, agricultural fields, and rangeland. It is anticipated that these areas could receive pesticide applications for other pests at certain times. Mixture related effects to human health and the environment from zinc phosphide in combination with other pesticides, and other chemicals, is not well understood. The cumulative impacts for human health is expected to be minor since use of PPE for WS zinc phosphide applications would also provide protection against exposure to other chemicals. Impacts to fish and wildlife are more difficult to assess.

Cumulative impacts related to zinc phosphide in combination with other natural and anthropogenic stressors to aquatic systems would be low since zinc phosphide poses little risk to aquatic organisms. In addition, label restrictions further reduce the potential for exposure to aquatic systems. Cumulative impacts to terrestrial nontarget organisms from various stressors, including zinc phosphide, could occur for terrestrial vertebrates that are at risk from zinc phosphide poisoning while other natural and anthropogenic stressors are present. For example, other types of pesticide applications may result in loss of prey or habitat for certain nontarget animals, and if those species are sensitive to zinc phosphide as well, cumulative impacts could occur from the combination of treatments. These types of cumulative impacts are difficult to quantify and need to be evaluated on a site-specific basis when considering the use of zinc phosphide. Consideration of the nontarget terrestrial fauna and their life histories in a particular treatment area, as well as temporal and spatial variability in the various stressors, would need to be evaluated.

Uneaten zinc phosphide baits are expected to degrade and could result in cumulative loading of zinc and phosphorus into soil. The potential loading would be incrementally minor when compared to background levels and other sources of zinc and phosphide. Over time, phosphide in the bait could be metabolized to form phosphine, but the amount produced at any one time would be minor. Phosphine produced from degradation of zinc phosphide would enter the atmosphere and degrade rapidly. The contribution of atmospheric phosphine from bait that is left in fields and allowed to degrade would be negligible. Phosphine can occur naturally from sources such as anaerobic degradation in soil, sediments, and decomposing plant matter and sludge (Glindemann et al. 1996, Eismann et al. 1997). Phosphide could also degrade to a phosphate that would be available for plant uptake, but based on the minor amounts that are applied and available during bait degradation, the contribution from any WS applications would be negligible.

Similar concerns regarding cumulative impacts to human health and the environment from additional zinc in the environment would be negligible. Zinc occurs naturally in soil, sediments, and plants and any zinc contributions from the degradation of zinc phosphide bait left in treated fields would be negligible. Zinc also has a wide variety of anthropogenic sources that can result in environmental loading to air, soil, sediments, and water. The natural and anthropogenic sources of zinc vary across the United States. However, the potential for cumulative impacts from WS use will not result in degradation of air, soil, or water quality, and therefore, would not impact human health and the environment.

7 SUMMARY

WS uses zinc phosphide to manage several rodent species that damage a variety of resources. The WS use pattern, application rates, mostly on private lands away from the public, reduces the risk to minimal for the public. The risk to WS applicators is low because they receive training in the product's use, are certified by the State to use restricted-use pesticides, and follow label instructions, including the appropriate personal protective equipment. The release of zinc phosphide into the environment is expected to have minimal or low impacts to nontarget species, the public, and the environment. WS does not know of any accidental exposures to the public or in the environment and believes this will continue for the foreseeable future.

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9.1 APHIS WS Methods Risk Assessment Committee

Writers for "Use of Zinc Phosphide in Wildlife Damage Management Risk Assessment":

Writer/Editor: Thomas C. Hall
Position: USDA-APHIS-WS, Operational Support Staff, Staff Wildlife Biologist, Fort Collins, CO
Education: BS Biology (Natural History) and BA Psychology – Fort Lewis College; MS Wildlife Ecology – Oklahoma State University
Experience: Special expertise in wildlife biology, identification, ecology, and damage management. Thirty-one years of service in APHIS Wildlife Services including operations and research in CO for research and OR, GU, CA, OK, and NV for operations conducting a wide variety of programs including bird damage research and management, livestock protection (predators and birds), invasive species management, wildlife hazard management at airports, property and natural resource protection including waterfowl, brown tree snake, feral swine, rodent, and beaver damage management. Applied and supervised the use of zinc phosphide.

Primary Writer: Fan Wang-Cahill
Position: USDA-APHIS-Policy and Program Development (PPD), Environmental and Risk Analysis Services (ERAS), Environmental Health Specialist
Riverdale, MD
Education: B.S. Biology and M.S. Hydrobiology - Jinan University, Guangzhou, China; Ph.D. Botany (Ultrastructure/Cell Biology) – Miami University
Experience: Joined APHIS in 2012, preparing human health risk assessments and providing assistance on environmental compliance. Prior experience before joining APHIS includes 18 years environmental consulting experience specializing in human health risk assessments for environmental contaminants at Superfund, Resource Conservation and Recovery Act (RCRA), and state-regulated contaminated facilities.

Primary Writer: Jim Warren
Position: USDA-APHIS-Policy and Program Development (PPD), Environmental and Risk Analysis Services (ERAS), Environmental Toxicologist, Little Rock, AR
Education: B.S. Forest Ecology and M.S. Entomology – University of Missouri; Ph.D. Environmental Toxicology – Clemson University
Experience: Eight years of experience working for APHIS preparing ecological risk assessments and providing assistance on environmental compliance. Prior experience before joining APHIS includes other government and private sector work regarding ecological risk assessments related to various environmental regulations.

Editors/Contributors for "Use of Zinc Phosphide in Wildlife Damage Management Risk Assessment":

Editor: Nikeeya Ali
Position: USDA-APHIS-WS, Operational Support Staff, Fort Collins, Colorado, 2017 Sumer Intern
Education: BS in Communications, South Carolina State University
Experience: Three years as a staff writer for The Collegian Newspaper, one year as Editor-in-Chief. Skilled in production, video editing. Previously edited two Risk Assessments for USDA-APHIS-WS.

Editor/Contributor: Andrea Lemay
Position: USDA-APHIS-Policy and Program Development (PPD), Environmental and Risk Analysis Services (ERAS), Biological Scientist, Raleigh, NC
Education: BS Plant and Soil Science (Biotechnology) - University of Massachusetts; MS Plant Pathology -North Carolina State University
Experience: Thirteen years of service in APHIS conducting risk analysis. Four years of experience in preparing environmental analyses in compliance with the National Environmental Policy Act.

Editor/Contributor: Jeanette O'Hare
Position: USDA-APHIS-Wildlife Services (WS), National Wildlife Research Center (NWRC), Registration manger, Fort Collins, CO
Education: B.S. Biology – College of Saint Mary; M.A. Biology – University of Nebraska - Omaha
Experience: 13 years of experience working for WS NWRC providing regulatory compliance support for the development of wildlife damage management tools. Prior experience before joining APHIS includes assessing the environmental fate of pesticides and providing the agency guidance on water quality issues at the state government level, and laboratory experience in the fields of pharmacology and toxicology, and immunology.

Editor/Contributor: Emily Ruell
Position: USDA-APHIS-WS, NWRC, Registration Specialist, Fort Collins, CO
Education: B.S. Zoology and Biological Aspects of Conservation – University of Wisconsin - Madison; M.S. Ecology – Colorado State University (CSU); M.A. Political Science – CSU
Experience: Three years of experience with WS NWRC preparing and reviewing vertebrate pesticide registration data submissions and other registration materials, and providing pesticide regulatory guidance to WS, WS NWRC, and collaborators. Prior experience before joining APHIS includes seven years of conducting field and laboratory wildlife research at CSU, and environmental policy research for the U.S. Geological Survey.

Editor/Contributor: Ryan Wimberly
Position: USDA-APHIS-WS, Operational Support Staff, Staff Wildlife Biologist, Madison, TN
Education: BS Wildlife Management and Ecology – Northwest Missouri State University
Experience: Special expertise in wildlife biology, ecology, and damage management. Seventeen years of service with APHIS Wildlife Services, including operations and research, conducting a wide variety of programs, including bird damage research and management, livestock protection, invasive species management, wildlife hazard management at airports, property, and natural resource protection. Expert in preparing environmental documents for WS programs to comply with the National Environmental Policy Act and the Endangered Species Act.

Data Contributor: Joey Millison
Position: USDA-APHIS-WS Information and Technology (IT), Junior Applications Developer
Education: Information and Technology coursework from various sources
Experience: Eleven years of experience in APHIS, WS Management Information System (MIS) Group. Retrieves WS field data from the MIS for writers, reviewers, and editors.

9.2 Internal Reviewers

USDA APHIS Wildlife Services

Reviewer: Jon Grant
Position: USDA-APHIS-WS, District Supervisor/ Wildlife Biologist, Albuquerque, NM
Education: BS Wildlife - Univ. Wisconsin, Stevens Point
Experience: Expertise in wildlife biology and wildlife damage management operations and research. Twenty-four years of service in APHIS Wildlife Services operational programs in WI and NM. Experience in a wide variety of damage management to include feral swine, livestock protection, bird damage, wildlife hazard management at airports. Have extensive experience applying and supervising the use of zinc phosphide.

Reviewer: Jeff Jones (retired)
Position: USDA-APHIS-WS, Staff Wildlife Biologist, Riverdale, MD
Education: BS Wildlife and Fisheries Sciences, Texas A&M University
Experience: Special expertise in wildlife biology, ecology, and damage management including overseeing the WS Pesticide Program. Thirty years of Federal time and six years of State program service in TX, AR, CA, OR and MD with experience in a wide variety of programs (livestock, aquaculture, property, public safety and natural resource protection) including predator, bird, beaver, feral swine, and rodent damage management activities including supervising and monitoring the use of and applying zinc phosphide in the course of duties.

Reviewer: Shane T. Koyle
Position: USDA-APHIS-Wildlife Services, District Supervisor/Wildlife Biologist, Pueblo, CO
Education: B.S. Wildlife Science- Utah State University
Experience: Twelve years of experience with APHIS Wildlife Services identify and managing animal damage. Qualified supervisor of Colorado restricted use pesticide license, with endorsement in outdoor vertebrate pest. Expert in surveying for non-targets and endangered species, pre-baiting, applying zinc phosphide, and monitoring treatment sites. Trainer in proper use and safe handle of zinc phosphide.

Reviewer: Chad Richardson
Position: USDA-APHIS-WS, Operational Support Staff, Staff Wildlife Biologist, Ogallala, NE
Education: BS Fisheries and Wildlife Biology – Kansas State University
Experience: Expertise in wildlife biology and wildlife damage management operations and research. Twenty-two years of service in APHIS Wildlife Services operational programs in Kansas and Nebraska. Experience in a wide variety of damage management to include feral swine, livestock protection, bird damage, wildlife hazard management at airports. Have extensive experience applying and supervising the use of zinc phosphide.

Reviewer: Gary Witmer
Position: USDA/APHIS/WS/NWRC Supervisory Research Wildlife Biologist, Fort Collins CO
Education: B.S. and M.S. General Biology – University of Michigan; M.S. Wildlife Ecology – Purdue University; Ph.D. Wildlife Science with minors in Statistics and Forest Management – Oregon State University
Experience: Twenty-five years as a USDA research wildlife biologist with a focus on rodent population and damage management. Three years on the wildlife faculty of Penn State University. Four years as a terrestrial ecologist with Argonne National Laboratory.

9.3 Peer Review

The Office of Management and Budget requires agencies to have peer review guidelines for scientific documents. The APHIS guidelines were followed to have "Use of Zinc Phosphide in Wildlife Damage Management" peer reviewed. WS worked with the Association of Fish and Wildlife Agencies to have experts review the documents.

9.3.1 Peer Reviewer Agencies Selected by the Association of Fish and Wildlife Agencies

California Department of Fish and Wildlife
Colorado Parks and Wildlife
Nevada Department of Wildlife
Texas Parks and Wildlife Department

9.3.2 Comments

Comments regarding concerns with the risk assessment and a response:

1. **Comments:** My experience with non-target losses support the conclusions that there was a high acute risk to birds and mammals and that lack of palatability may be what limits non-target exposure.
   Response: We agree that zinc phosphide has high acute risks, especially as outlined in Sections 3, 4, and 5, and that low palatability as well as color reduce nontarget hazards.

2. **Comments:** We have had nontarget losses that included several incidents with Canada geese and one with wild turkeys. These cases may have been prevented by using bait stations or closer monitoring of prebaiting.
   Response: WS is aware of nontarget take with zinc phosphide, especially birds as discussed in Section 5.2.2. WS typically prebaits fields when using zinc phosphide to increase palatability and bait uptake by the target species. Bait stations are used primarily for commensal rodents, species living in close association with people, for aquatic rodents (e.g., nutria), or where required per the label. WS did not record a nontarget species take using zinc phosphide for FY11-FY15, but realize that it likely has occurred, especially with other rodents in the area. WS attempts to minimize nontarget take by conservation steps such as monitoring fields for nontarget species prior to broadcasting baits, using bait stations as appropriate, and following label specifications.

Comments received not requiring a response.

1. **Comment:** I have reviewed the risk assessment and suggest use as described.

2. **Comment:** The risk assessment does good job of covering ecological risks.

3. **Comment:** I have no comments but say the risk assessment appears complete and the protocols/reviews seem appropriate.