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

Chapter IX

The Use of Aluminum Phosphide in Wildlife Damage Management

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EXECUTIVE SUMMARY

Aluminum phosphide is a fumigant used by the USDA-APHIS-Wildlife Services (WS) Program to control various burrowing rodents. Formulations for burrow treatment are in 3-g tablets or 600-mg pellets containing about 56% active ingredient. WS applicators place the tablets or pellets into the target species’ burrow and seal the burrow entrance with soil. Aluminum phosphide reacts with moisture in burrows to release phosphine gas, which is the toxin in the fumigant.

APHIS evaluated the human health and ecological risk of aluminum phosphide under the WS use pattern for rodent control. Although phosphine gas is toxic to humans, the risk to human health is low because inhalation exposure is slight for the underground applications. WS applicators wear proper personal protective equipment (PPE) according to the pesticide label requirements, which reduces their exposure to aluminum phosphide and phosphine gas, lowering the risk to their health. Label restrictions protect the public from exposure to the fumigant when used accordingly.

Label restrictions, use patterns, environmental fate and aquatic toxicity data for aluminum phosphide suggest low risk to aquatic species and their habitats. Aluminum phosphide risks to terrestrial plants is also low, however there is a risk to terrestrial vertebrates and invertebrates that are in burrows and exposed to phosphine gas during treatment. WS personnel will reduce the exposure and risk to nontarget species by checking burrows and confirming the presence of target animals before applying aluminum phosphide. There is no secondary risk to nontarget species because the gas rapidly dissipates and aluminum phosphide and phosphine do not accumulate in target animals. Restrictions are present on all labels to lower the chance of exposure and risks to nontarget terrestrial species.
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1 INTRODUCTION

This human health and ecological risk assessment is a qualitative evaluation of the risks and hazards to human health and nontarget species from the use of aluminum phosphide (AlP) by the U.S. Department of Agriculture (USDA), Animal and Plant Health Inspection Service (APHIS), Wildlife Services (WS) Program. The methods used to assess human health effects conform to the guidance of other Federal agencies, such as the U.S. Environmental Protection Agency (USEPA) Office of Pesticide Programs and the National Research Council (NRC), and follow regulatory methods (NRC 1983, USEPA 2016) that these agencies have outlined. The methods used to assess the ecological risk to nontarget species generally follow USEPA (2016) ecological risk assessment methods.

This assessment starts with the problem formulation (identifying the hazard) and then evaluates toxicity (the dose-response assessment) and exposure (identifying exposed populations and determining exposure pathways for these populations). Lastly, the integration of the toxicity and exposure analysis provides a characterization of risks (a determination whether adverse human health or ecological risks are present and the significance). Uncertainties and cumulative effects are part of this assessment.

1.1 Use Pattern

For FY11-FY15, WS killed an estimated average annual 54,095 target rodents representing 19 species (Table 1) with aluminum phosphide. Of the targeted animals, black-tailed and Gunnison’s prairie dogs1 were the species with most taken. WS personnel record burrows taken in the MIS, so these were converted to numbers using values noted in Table 1. Aluminum phosphide was used in nine states (Table 2).

Table 1. The annual average number of target burrowing rodents killed with aluminum phosphide by WS in WDM activities for FY11 to FY15 throughout the United States. No known nontarget take occurred in this time.

<table>
<thead>
<tr>
<th>Species</th>
<th>Target</th>
<th>AIP Tablets2 Used</th>
<th>Est. #/burrow</th>
<th>% of Take</th>
</tr>
</thead>
<tbody>
<tr>
<td>Black-tailed Prairie Dog</td>
<td>28,479</td>
<td>104,543</td>
<td>1</td>
<td>52.6%</td>
</tr>
<tr>
<td>Gunnison’s Prairie Dog</td>
<td>16,161</td>
<td>95,242</td>
<td>0.67</td>
<td>29.9%</td>
</tr>
<tr>
<td>White-tailed Prairie Dog</td>
<td>1,772</td>
<td>8,688</td>
<td>0.67</td>
<td>3.3%</td>
</tr>
<tr>
<td>California Ground Squirrel</td>
<td>385</td>
<td>140</td>
<td>3</td>
<td>0.7%</td>
</tr>
<tr>
<td>Rock Squirrel</td>
<td>90</td>
<td>60</td>
<td>3</td>
<td>0.2%</td>
</tr>
<tr>
<td>Paiute Ground Squirrel</td>
<td>720</td>
<td>464</td>
<td>3</td>
<td>1.3%</td>
</tr>
<tr>
<td>Uinta Ground Squirrel</td>
<td>44</td>
<td>59</td>
<td>3</td>
<td>0.1%</td>
</tr>
<tr>
<td>Belding’s Ground Squirrel</td>
<td>60</td>
<td>117</td>
<td>3</td>
<td>0.1%</td>
</tr>
<tr>
<td>Columbian Ground Squirrel</td>
<td>1,244</td>
<td>1,669</td>
<td>3</td>
<td>2.3%</td>
</tr>
<tr>
<td>Thirteen-Lined Ground Squirrel</td>
<td>828</td>
<td>552</td>
<td>3</td>
<td>1.5%</td>
</tr>
<tr>
<td>Mexican Ground Squirrel</td>
<td>1,216</td>
<td>1,620</td>
<td>3</td>
<td>2.2%</td>
</tr>
<tr>
<td>Yellow-Faced Pocket Gopher</td>
<td>2,564</td>
<td>5,191</td>
<td>2</td>
<td>4.8%</td>
</tr>
<tr>
<td>Botta’s Pocket Gopher</td>
<td>452</td>
<td>904</td>
<td>2</td>
<td>0.8%</td>
</tr>
<tr>
<td>Camas Pocket Gopher</td>
<td>18</td>
<td>28</td>
<td>2</td>
<td>0.03%</td>
</tr>
<tr>
<td>Other Rodent (4T – 4 spp.)</td>
<td>15</td>
<td>21</td>
<td>2</td>
<td>0.03%</td>
</tr>
<tr>
<td>Townsend’s Mole</td>
<td>17</td>
<td>26</td>
<td>2</td>
<td>0.03%</td>
</tr>
</tbody>
</table>


*Includes yellow-bellied marmot (2/burrow), plains pocket gopher (2), meadow vole (4), and sagebrush vole (4)

1 - The burrows taken were multiplied by the estimated number of rodents in each burrow. Depending on time of year, you could have 0 (often burrowing rodents use more than one burrow system so some have none, especially the prairie dogs) to 9 for ground squirrels (several species have the potential for 1 adult and 8 young until July when they disperse). Males are often in their own burrows.

2 - If pellets were used, they were converted to an equivalent number of tablets (1 tablet equals 5 pellets)

1 Scientific names are given in the Risk Assessment Introduction Chapter
2 MIS - Computer-based Management Information System used for tracking WDM activities. Throughout the text, data for a year (i.e. FY11 to FY15) will be given and is from the MIS. MIS reports will not be referenced in the text or Literature Cited Section because MIS reports are not kept on file. A database is kept that allows queries to be made to retrieve the information needed.
Table 2. The annual average burrowing rodent take targeted with aluminum phosphide by WS in WDM throughout the United States for FY11-FY15.

<table>
<thead>
<tr>
<th>STATE</th>
<th>Target Take</th>
<th>AIP Tablets Used</th>
<th>% of AIP Use</th>
</tr>
</thead>
<tbody>
<tr>
<td>Colorado</td>
<td>30,640</td>
<td>119,674</td>
<td>54.5%</td>
</tr>
<tr>
<td>Idaho</td>
<td>720</td>
<td>465</td>
<td>0.2%</td>
</tr>
<tr>
<td>Nebraska</td>
<td>2,359</td>
<td>4,813</td>
<td>2.2%</td>
</tr>
<tr>
<td>New Mexico</td>
<td>13,435</td>
<td>80,509</td>
<td>36.7%</td>
</tr>
<tr>
<td>Oklahoma</td>
<td>120</td>
<td>600</td>
<td>0.3%</td>
</tr>
<tr>
<td>Oregon</td>
<td>484</td>
<td>584</td>
<td>0.3%</td>
</tr>
<tr>
<td>Texas</td>
<td>4,395</td>
<td>9,139</td>
<td>4.2%</td>
</tr>
<tr>
<td>Utah</td>
<td>698</td>
<td>2,141</td>
<td>1.0%</td>
</tr>
<tr>
<td>Washington</td>
<td>1,244</td>
<td>1,669</td>
<td>0.8%</td>
</tr>
<tr>
<td><strong>Total (9 States)</strong></td>
<td><strong>54,095</strong></td>
<td><strong>219,594</strong></td>
<td></td>
</tr>
</tbody>
</table>

1 - Numbers of animals were estimated for the burrows taken.
2 - If pellets were used, they were converted to an equivalent number of tablets (1 tablet equals 5 pellets).

2 PROBLEM FORMULATION

The first registration for AlP as a pesticide in the United States was in 1958 (USEPA 1998b). AlP reacts with moisture in the atmosphere and soil to form phosphine gas, a toxin (USEPA 1998a). As an indoor fumigant, AlP controls insect pests in raw and processed food commodities, and non-food commodities in sealed containers or structures. As a burrow fumigant, AlP controls rodents and moles in non-residential and agricultural areas (USEPA 1998b). WS uses AlP to reduce rodent damage from prairie dogs, voles, rats, mice, ground squirrels, gophers, woodchucks, and chipmunks. WS conducted an annual average of 448 work tasks3 per year during FY11-FY15 with aluminum phosphide.

2.1 Chemical Description and Product Use

Aluminum phosphide (AlP; CAS # 20859-73-8) is an inorganic compound with a molecular weight of 57.96 and a molecular structure shown in Figure 1 (USEPA 1998a). AlP is available in tablets, pellets, impregnated materials and dusts (USEPA 1998a). AlP is a fumigant used to control insects and rodents (USEPA 1998a). AlP reacts with moisture to produce hydrogen phosphide, or phosphine gas (CAS# 7803-51-2), which is the toxin in the pesticide (USEPA 1998a).

WS uses AlP as a burrow fumigant for rodents such as prairie dogs, except the threatened Utah prairie dog (Cynomys parvidens), ground squirrels, pocket gophers, native mice (voles, deer mice), marmots, and moles per label directions. AlP comes in 3-g tablets or 600-mg pellets containing between 55% to 60% active ingredient (a.i.) (Witmer and Fagerstone 2003). WS applicators place one to four 3-g tablets or five to twenty 600-mg pellets in burrows and immediately seal the burrow entrances with soil (Snider 1983, USEPA 1998b). Applicators may plug the entrance to the burrow with crumpled newspaper or another material to prevent soil from covering the tablets or pellets (Virchow et al. 2002). Applicators may drop AlP tablets or pellets through a four to five foot plastic pipe with a 1.5 to 1.75 inch diameter inserted deep into the burrow entrance (Virchow et al. 2002). AlP reacts with moisture in burrows to release phosphine gas. Phosphine reaches peak concentrations in 48 – 60 hours (Virchow et al. 2002). Several product labels specify a maximum application of 12 grams of AlP (about 4 grams of

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3 A Work Task is defined as a visit to a property, or a portion of it, where a WS employee conducts field work. However, duration is not taken into account and, thus, a Work Task could be 10 minutes to 10 hours in duration.
phosphine) per burrow, which produces around 1,000 ppm phosphine near the pellets or tablets (Snider 1983). Burrow residents inhale phosphine which then enters the bloodstream and interferes with cell physiology and alters hemoglobin (Witmer and Fagerstone 2003).

Only certified pesticide applicators or persons under their direct supervision may use AlP (USEPA 1998b, Witmer and Fagerstone 2003). WS uses several AlP products, including DEGESCH Phostoxin® (USEPA Reg. No. 72959-4), Fumitoxin® (USEPA Reg. No. 72959-1), Weevil-Cide® (USEPA Reg. No. 70506-13), Gastoxin® (USEPA Reg. No. 70506-14), and DREX-PH3™ (USEPA Reg. No. 19713-571) aluminum phosphide tablets (Table 3).

Table 3. Aluminum phosphide formulations used by WS to control burrowing rodents. Manufacturer websites can be visited for labels and material safety data sheets.

<table>
<thead>
<tr>
<th>Product</th>
<th>Registration date</th>
<th>USEPA registration number</th>
<th>Active/Inert ingredients</th>
<th>Animals labelled to be treated</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fumitoxin®</td>
<td>10/10/1958</td>
<td>Tablet: 72959-1, Pellet: 72959-2</td>
<td>ai: AlP 55% Inert: 45%</td>
<td>Burrowing Rodents: prairie dogs except Utah prairie dog, woodchucks, yellow-bellied marmots, ground squirrels, pocket gophers, chipmunks, brown rats, black rats, mice, voles, and moles</td>
</tr>
<tr>
<td>Mfg: D&amp;D Holdings</td>
<td>Last update: 9/30/2013</td>
<td>Pellet: 72959-5</td>
<td>ai: AlP 55% Inert: 45%</td>
<td></td>
</tr>
<tr>
<td>Mfg: D&amp;D Holdings</td>
<td>Last update: 9/30/2013</td>
<td>Pellet: 72959-5</td>
<td>ai: AlP 57% Inert: 43%</td>
<td></td>
</tr>
<tr>
<td>Gastoxin®</td>
<td>8/14/1980</td>
<td>Tablet: 43743-1, Pellet: 43743-2</td>
<td>ai: AlP 60% Inert: 40%</td>
<td></td>
</tr>
<tr>
<td>GASTOXIN®</td>
<td>3/10/1981</td>
<td>Tablet: 19713-571, Pellet: 19713-569</td>
<td>ai: AlP 60% Inert: 40%</td>
<td></td>
</tr>
<tr>
<td>Weevil-Cide®</td>
<td>12/13/2005</td>
<td>Tablet: 70506-13, Pellet: 70506-14</td>
<td>ai: AlP 60% Inert: 40%</td>
<td></td>
</tr>
<tr>
<td>Mfg: United Phosphorous, Inc.</td>
<td>Last update: 3/20/2016</td>
<td>Pellet: 70506-14</td>
<td>ai: AlP 60% Inert: 40%</td>
<td></td>
</tr>
</tbody>
</table>

2.2 Physical and Chemical Properties

AlP is a grayish green or black to yellow granular or powdered solid with a sulfurous/garlic odor. It has a melting point above 1000°C, vapor pressure of 40 mm Hg at 129.4°C, and a Henry’s Law Constant of 0.1 atm m³/mol (USEPA 1998b). AlP is stable when dry, but readily reacts with water, including moisture in the air, to release phosphine gas (USEPA 1998b).

Phosphine is a colorless gas, with a melting point and boiling point of -133.5°C and -87.4°C, respectively (World Health Organization (WHO) 1988). Its vapor density (air = 1) is 1.17 (WHO 1988).

2.3 Environmental Fate

The environmental fate describes how AlP moves and transforms in the environment. This includes its: 1) mobility, persistence, and degradation in soil, 2) movement to air, and 3) migration potential to ground and surface water. AlP is non-persistent under most environmental conditions. It reacts with moisture to form phosphine gas. Phosphine is non-persistent and non-mobile in the soil environment and is unlikely to be found in groundwater and surface waters (USEPA 1998b).

Under normal environmental conditions, phosphine exists as a gas. Phosphine reacts with photochemically-produced hydroxyl radicals in the atmosphere, degrading in 5 hours (half-life) (WHO 1988, USEPA 1998b). The reaction products are non-volatile oxycacids of phosphorous and inorganic phosphate, both of which contribute nutrients to soil and surface water (WHO 1988, USEPA 1998b). Without light, the half-life can be as long as 28 hours (Gurusinghe 2014). Phosphine has a high vapor pressure and high Henry’s Law Constant, thus at the soil surface it undergoes rapid dissipation into the atmosphere (Gurusinghe 2014). Studies suggest phosphine below the soil surface is quickly adsorbed and degraded (Berck and Gunther
1970, Hilton and Robison 1972a, Eismann et al. 1997a, USEPA 1999). Phosphine (at 1,000 ppm of phosphorus) reaches undetectable levels quicker in dry soils than saturated soils. In air-dried soils, phosphine disappeared within 18 days, whereas in saturated soils it disappeared in 40 days (Hilton and Robison 1972b, Gurusinghe 2014). The USEPA does not require environmental fate studies for AlP because of its physical properties and use patterns (USEPA 1998b).

2.4 Hazard Identification

AlP can be a hazard to human health and lethal at high doses because of its acute inhalation toxicity. This section summarizes human incidents and acute and chronic toxicity data for mammals.

2.4.1 Human Incidents

Human toxicity occurs from the ingestion of AlP or from the inhalation of phosphine (Gurjar et al. 2011). Ingested AlP reacts with water and stomach acids to produce phosphine gas which passes into the bloodstream (Gurjar et al. 2011). Inhaled phosphine passes through the lung epithelium (Gurjar et al. 2011). This can lead to systemic effects involving the heart, lung, kidney, and liver with symptoms of irregular heartbeat (cardiac arrhythmia), acidosis, and excess fluid in the lungs (pulmonary edema) (Gurjar et al. 2011). The reaction of phosphine with hemoglobin causes a denaturation of oxyhemoglobin and interferes with several enzymes important for metabolism and respiration, and may also affect the integrity of cell membranes (WHO 1988). Excretion of phosphine is through urine in the form of hypophosphite, and exhalation from the lungs (Gurjar et al. 2011). Symptoms of mild to moderate acute AlP toxicity include nausea, abdominal pain, tightness in chest, restlessness, agitation and chills (Degesch America Inc. 1988, USEPA 1998b). Symptoms of more severe toxicity include diarrhea, cyanosis, difficulty breathing, pulmonary edema, respiratory failure, tachycardia (rapid pulse) hypotension (low blood pressure), and dizziness (Gurjar et al. 2011). Toxicity can cause death (Gurjar et al. 2011).

USEPA (2013) reviewed human incidents for AlP and phosphine. The fatality of two children after an applicator (not WS) applied Fumitoxin® pellets within 7 feet of a residence as opposed to the minimum of 15 feet triggered a revision of the label to prevent accidental poisonings. The revision increases the buffer distance from 15 to 100 feet for applications around buildings occupied by a human or animal and prohibits use in residential areas.

2.4.2 Acute Toxicity

AlP has a high acute inhalation toxicity (LC₅₀ > 0.014 mg/L) placing it in Toxicity Category I (USEPA 1998b). The Phostoxin®, Fumitoxin®, Gastoxin®, DetiaPhos™ and DREX-PH₃™ formulations of AlP have an acute median oral toxicity of 11.5 mg/kg of body weight as discussed on material safety data sheets for the various formulations. The Phostoxin® and Fumitoxin® material safety datasheet give a dermal toxicity LD₅₀ greater than 5,000 mg/kg for a 1-hour exposure to AlP, which is very low.

2.4.3 Subchronic Toxicity

Researchers exposed male and female rats to phosphine in three inhalation exposure tests (USEPA 1998b). In test 1, they exposed rats to 0, 0.3, 1.0 or 3.0-ppm phosphine for 6 hours a day, five days per week for 13 weeks. In test 2, they exposed rats to 0 or 10 ppm of phosphine on day 48 of the study. Test 2 stopped after 3 days of exposure because four of the 10 females died. In test 3, they exposed rats to 0 or 5 ppm of
phosphine for 15 days, starting on day 75 of the study. This subchronic toxicity study set a no observable
effect level (NOEL) of 3 ppm (USEPA 1998b). In the 13-week exposure to 3 ppm or less of phosphine (test
1), rats decreased their food consumption which caused their weight to drop but no rats died (USEPA
1998b). In rats exposed to 10 ppm for 3 days (test 2), several of the females died while no males died. The
kidneys in both sexes had coagulative necrosis in the tubules. In the 5 ppm for 15-day exposure (test 3),
males had an increase in alkaline phosphatase activity and blood urea nitrogen but these increases were
not significant. At this exposure level, there were no effects on survival, food consumption, body weight, or
eye and blood parameters in either sex (USEPA 1998b).

2.4.4 Chronic Toxicity

Male and female rats fed phosphine-treated food for two years did not show any changes in normal food
consumption, growth, hematology, survival and morbidity, or show signs of carcinogenicity (USEPA 1998b).
Just prior to feeding, phosphine concentrations in the food ranged from 0.2 to 7.5 ppm. The precise amount
of phosphine ingested by the rats is unknown because some of the gas dissipates from the food prior to
consumption.

In another study, rats kept in enclosed chambers did not show any abnormal change in body weight, food
consumption, blood and urinary analysis, and other pathology and histopathology tests after a one-year
inhalation exposure of up to 3 ppm phosphine gas (USEPA 1998b).

2.4.5 Reproductive or Developmental Effects

The maternal NOEL is 5 ppm for phosphine and the maternal lowest observable effect level (LOEL) is 7.5
ppm for phosphine, based on a developmental study on mated female rats (USEPA 1998b). In this study,
inhalation exposure of rats to 7.5-ppm phosphine gas six hours a day for 15 gestation days caused an
increase in resorption of litters. The resorption occurred by day 10, at which point the study stopped due to
the high rat mortality. Rats exposed to concentrations of phosphine below 7.5 ppm had body weight, food
consumption, corpora lutea, fetus survival, resorptions, external malformations and visceral and skeletal
defects of pups similar to the untreated rats (USEPA 1998b).

The USEPA (1998b) waived reproductive toxicity studies because residues of phosphine in food or drinking
water are not expected. The available evidence for developmental and reproductive effects in animals
suggests that such effects are not likely in humans.

2.4.6 Carcinogenicity and Mutagenicity

There is no evidence that phosphine gas causes carcinogenicity in rats (USEPA 1998b). Several studies
suggest phosphine is not likely to cause mutations or increase the mutation rate in bacteria but may cause
breakage of the chromosome in vitro (USEPA 1998b).

Exposure of human lymphocyte cells in culture to phosphine caused chromosomal aberrations (Garry et al.
1989). People exposed to phosphine in their job had a higher occurrence of chromosomal aberrations (e.g.,
deletions, gaps, breaks), in comparison to people exposed less or not at all, indicating a dose-related
increase in aberrations (Garry et al. 1989).
2.4.7 Neurotoxicity Effects

Inhalation exposure of male and female rats to 20, 30, and 40-ppm phosphine for four hours caused a decrease in body temperature and motor activity compared to the control (USEPA 1998b). Other neurotoxicity observations in the study were suspect as a result of study design, lack of positive control data, and deficiency in reporting the study’s findings (USEPA 1998b).

In a subchronic inhalation neurotoxicity study, no rats died after exposure to 3-ppm or less of phosphine for six hours per day, five days per week over a 90-day period. A tentative NOEL for subchronic systemic/neurobehavioral finding was 3 ppm for male and female rats because by week 13, male rats had effects in sleep patterns, body temperature, and motor activities (USEPA 1998b). A LOEL was not determined in this study (USEPA 1998b).

Several labels specify a maximum application of 12 grams of AlP (about 4 grams of phosphine) per burrow, which produces around 1,000 ppm phosphine near the pellets or tablets (Snider 1983). Phosphine gas diffuses from the source to lesser concentrations (Snider 1983).

3 DOSE-RESPONSE ASSESSMENT

3.1 Human Health and Safety

A dose-response assessment evaluates the dose levels (toxicity criteria) for potential human health effects. The chronic reference dose (RfD) is “the amount of pesticide that could be consumed daily without causing adverse effects” (USEPA 1998a). The chronic RfD for AlP is 0.0113 mg/kg/day. This value is based on the NOEL of 1.13 mg/kg/day from the 90-day inhalation study in rats described in the Subchronic Toxicity section (USEPA 1998a, b).

The acute reference dose of 0.018 mg/kg/day for AlP is based on no detectable effects after 15 days of exposure in the 90-day inhalation study in rats described above (USEPA 1998a, b). The dietary risk for AlP is based on residues in food since phosphine residues are not likely to occur in drinking water (USEPA 1998a).

The Food Quality Protection Act of 1996 requires a 10-fold safety factor for the establishment of tolerances to protect infants and children; however, change to this requirement may occur if data demonstrate it is appropriate to reduce or remove the safety factor (USEPA 1998b). In the prenatal developmental inhalation toxicity study in rats described in the Reproductive and Developmental Effects section, the data “provides no indication of increased susceptibility of rats to in utero or postnatal exposure” to AlP (USEPA 1998a). Additionally, the restrictions for residential use and the expected lack of phosphine residue in drinking water indicated a lack of exposure to infants and children (USEPA 1998a). Given these factors, the USEPA (1998a) determined that the additional 10-fold safety factor is not necessary.

3.2 Ecological Effects Analysis

This section of the risk assessment discusses available ecological effects data for terrestrial and aquatic species. Because of the physical properties and use patterns of AlP, the USEPA (1998a) determined that no environmental fate studies were required (USEPA 1998b).
AIP is non-persistent under most environmental conditions and degrades rapidly in the presence of water to aluminum hydroxide and phosphine. The instability of AIP at atmospheric moisture contents indicates it is non-mobile in soil. Phosphine degrades in days. Phosphine near the soil surface diffuses into the atmosphere where it degrades through reaction with hydroxyl radicals. Phosphine binds to soil, inhibiting movement, and oxidizes to phosphates (USEPA 1998b). Because of these attributes, AIP and phosphine are at low risk for contaminating surface waters (USEPA 1998b) and causing harm to aquatic species.

WS uses AIP as a burrow fumigant for the control of rodents, potentially exposing nontarget animals including some endangered or threatened species that may be present in fumigated burrows. Phosphine gas is highly toxic and all animals in a fumigated burrow will die (USEPA 1998b).

The United States Fish and Wildlife Service (USFWS) determined that AIP used as a burrow fumigant may jeopardize the following federally-listed endangered species: the black-footed ferret (*Mustela nigripes*), the Utah prairie dog, the San Joaquin kit fox (*Vulpes macrotis mutica*), the desert tortoise (*Gopherus agassizii*), the blunt-nosed leopard lizard (*Gambelia sila*), and the eastern indigo snake (*Drymarchon couperi*) (USEPA 1998b). AIP labels contain use restrictions designed to prevent the take of endangered species. For example, the labels specify where in the states and counties AIP may be applied as well as requirements for conducting surveys for the presence of species of concern.

### 3.2.1 Aquatic Effects Analysis

AIP is highly toxic to fish, with a reported 96-hour LC50 of 4.1 µg/L in rainbow trout (Gurusinghe 2014) and 0.178 mg/m³ for bluegill (WHO 1988, Gurusinghe 2014). The 96-hr LC50 for phosphine toxicity in rainbow trout was 0.0097 mg/L. The 24-hr EC50 value for *Daphnia magna* was 0.2 mg/L. In frogs, the LC50 for 30-minute phosphine exposure was 0.56 mg/L and for a 15-minute phosphine exposure was 0.84 mg/L (WHO 1988). No data was available regarding the specific toxicity of AIP or phosphine to additional fish or aquatic species, but due to the mechanism of action, it is likely that it will be very highly toxic to other aquatic species. However, such exposure is unlikely as AIP will rapidly react to form phosphine gas, which is somewhat soluble in water, but volatilizes into the atmosphere based on the high Henry’s Law Constant (USEPA 1998b).

### 3.2.2 Terrestrial Effects Analysis

**Mammals**

AIP and phosphine is used as a mammal toxicant with labeled uses to control target animals inside burrows. The effect of AIP to wild and domestic mammals is similar to the types of impacts discussed in the hazard identification section of this risk assessment (Section 2.4). Toxicity studies on the effects of aluminum phosphide on nontarget mammals are limited. The lethal inhalation concentration values for the mouse and the cat are 380 mg/m³/2hr and 70 mg/m³/2hr, respectively (Witmer and Fagerstone 2003).

An assumption is that phosphine will kill all animals residing in treated burrows (Odenkirchen 2010), including nontarget animals that use burrows such as rodents, rabbits, raccoons, foxes, weasels, skunks, burrowing owls, reptiles, and amphibians, (Witmer and Fagerstone 2003). WHO (1998) summarized secondary poisoning studies where animals killed by phosphine (poisoned with zinc phosphide, not AIP, but ultimately died from phosphine gas) were fed to fox, rats, cats, and mice with no toxic effects observed. In one of the studies, kit fox fed one kangaroo rat a day for three days that was dosed with 480 mg of zinc...
phosphide (a dose that is around three times the LD50 for kit fox) showed no ill effects other than regurgitation and one fox refused to eat the rat for two days (Schitoskey 1975). Studies indicate bioaccumulation does not occur (WHO 1988, Witmer and Fagerstone 2003).

**Birds**

Information on the oral and inhalation median lethal doses for AlP and phosphine in birds is lacking. Exposure of three tom turkeys and six hens to 211 mg/m^3 for 74 minutes and 224 mg/m^3 for 59 minutes, respectively, caused labored breathing, apathy, and restlessness. The birds died in less than 2 hours (Gurusinghe 2014, Klimmer 1969 cited in WHO 1998). Exposure of other bird species to similar levels of phosphine is likely to cause similar results given the mode of action. However, exposure to these concentrations and durations is unlikely, as phosphine dissipates quickly in open air and birds would not be expected to nest or forage in burrows (Gurusinghe 2014).

**Reptiles**

Data on AlP toxicity for reptiles is lacking. However, studies on the use of AlP as a fumigant for brown tree snake control in Guam suggest that it may be effective for the control of snakes but did not include specific dosages (Savarie and Bruggers 1999, Witmer and Fagerstone 2003). The USFWS determined that use of AlP as a burrow fumigant may jeopardize the blunt-nosed leopard lizard, the eastern indigo snake, the desert tortoise, and other threatened and endangered reptile species (USEPA 1998b, Odenkirchen 2010).

**Terrestrial Invertebrates and Microorganisms**

Insects are a principal target of AlP. Susceptibility varies at different life stages, with larvae in diapause exhibiting tolerance, and some species are resistant (WHO 1988). The common fruit fly (*Drosophila melanogaster*) exhibits adverse effects to 1.4 mg/m^3 (1 ppm) of phosphine (Noack and Reichmuth 1981 cited in WHO 1988), a threshold similar to the acute inhalation effects in mammals (WHO 1988). Mites appear to be less sensitive than insects (WHO 1988). As with insects, microorganisms vary in their sensitivity to phosphine with many species surviving levels that are fatal to arthropods (WHO 1988). The Mediterranean white snail (*Cernuella virgata*) and the small conical snail (*C. acuta*) exhibit tolerance to phosphine, with an LD100 of 4.4 g/m^3 and 5.4 g/m^3, respectively, for 10 days (Cassells et al. 1994).

**Terrestrial Plants**

Data on the effect of phosphine on terrestrial plants is limited. Phosphine levels between 3 mg/m^3 and 8 mg/m^3 in the air had harmful effects on growing lettuce (*Lactuca sativa capitata*) (Noack and Reichmuth 1982 cited in WHO 1988). WHO (1988) summarized several studies that indicated exposure of a variety of seed to phosphine had minimal or no effect on seed germination. For example, exposure of watercress seeds to air concentrations of phosphine between 20 and 1400 mg/m^3 for three days did not affect germination (Noack and Reichmuth 1982 cited in WHO 1988).
4 EXPOSURE ASSESSMENT

4.1 Human Health and Safety

This qualitative exposure assessment estimates the potential exposure of humans to AIP and phosphine. It begins with the use and application method of AIP. An identified exposure pathway for AIP includes (1) a release from a source, (2) an exposure point where contact can occur, and (3) an exposure route such as ingestion or inhalation by which contact can occur (USEPA 20). Each AIP pellet weighs 0.6 g and releases 0.2 g of phosphine. Each AIP tablet weighs 3 g and releases 1 g of phosphine. For outdoor fumigation of rodent burrows (agricultural and non-crop land areas), USEPA (1998b) recommends dosages of 5 to 20 pellets/burrow and 1 to 4 tablets/burrow.

The most at risk group for exposure to AIP or phosphine is WS applicators. AIP comes in prepared pellets or tablets, which require no mixing by applicators and, therefore, reduces potential dermal exposure to AIP. Dermal contact with AIP pellets or tablets is unlikely because the label requires applicators to wear dry cotton gloves, which prevents hands from contacting the formulation and prevents hand moisture from reacting with AIP.

Acute inhalation exposure is the primary concern for applicators. USEPA (1998b) used an 8-hour time-weighted-average (TWA) level of 0.3 ppm as the short-term exposure standard for applicators. This includes the time it takes to open the first container, apply the appropriate first dosage of fumigant, and close the burrow opening. The Occupational Safety and Health Administration developed a permissible exposure limit (PEL) of 0.3 ppm (0.0004 mg/l) for phosphine. The maximum concentration after application must not exceed 0.3 ppm for applicator exposure (USEPA 1998b). The current labels for AIP (Table 3) restrict exposure to phosphine to not exceed the 8-hour TWA of 0.3 ppm (0.0004 mg/l) or the 15 minute short-term exposure limit (STEL) of 1.0 ppm phosphine for applicators during application (USEPA 1998b).

The current Phostoxin® label states the tablets and pellets release phosphine gas slowly when encountering moisture from the air, depending upon the temperature and humidity. In general, this release is slow enough to permit applicators to deposit the fumigant in the desired areas and then leave the premises without significant human exposure to the gas. The label requires monitoring to determine the fumigator’s exposure and to ensure their safety. The current labels also require a written fumigation management plan prepared for all fumigants prior to actual treatment to ensure a safe, legal and effective fumigation. This fumigation management plan must describe the steps used to comply with requirements of pesticide product labels, such as (1) characterizations of the area, (2) personnel qualifications, (3) appropriate monitoring and notification requirements, (4) sealing procedures, (5) application procedures and fumigation period, and (6) post-application operations.

Exposure of WS applicators to high levels of phosphine gas during or after applications to burrows is unlikely. Applications occur outdoors where phosphine gas that escapes from the burrow dissipates into the atmosphere. An industrial hygiene study looked at phosphine gas levels during applications of AIP to rodent burrows through hand and mechanical applications. The hand application method resulted in detectable phosphine gas levels below the 8-hour TWA of 0.3 ppm according to air monitoring results in the applicator-breathing zone (USEPA 1998b, Baker 1992). The mechanical application method did not expose applicators to detectable levels of phosphine gas (Baker 1992). In another study, however, applicators who used AIP tablets (Phostoxin®, Degesch America, 3-g tablet released 1 g hydrogen phosphide gas) to control Richardson’s ground squirrel in grasslands experienced discomfort and nausea after hand-applying tablets.
despite using the safety measures recommended at the time of the study (2007 to 2009) (Proulx et al. 2011). This study did not specify the safety measures used or environmental conditions. The respirator protections required by the current labels, including an approved full face gas mask-phosphine canister combination or SCBA or equivalent, when concentration of phosphine in the air is unknown or the concentration from monitoring exceeds 0.3 ppm will minimize unexpected exposures. Following label directions during the burrow applications will minimize exposure for WS worker to AlP.

Once the pellets/tablets are placed in the burrow, the applicator plugs the entrance to the burrow with crumpled newspaper or another material to prevent soil from covering the tablets or pellets (Virchow et al. 2002) and immediately seals the burrow entrances with soil (Snider 1983, USEPA 1998b); some rodents have multiple burrow openings and all entrances are sealed after one or all entrances are treated. The sealing of the burrow entrance traps the phosphine gas within the burrow reducing the potential for exposure of the applicator. After placement, applicators move away from the treated burrows, reducing exposure to phosphine gas that may escape from the burrow entrance. Lethal exposure is unlikely during proper use; however, an accidental event could expose applicators to phosphine. In the event of an accidental spill or leak, instructions on the product label explain the clean-up steps including the use of proper PPE to protect those cleaning up the spill or leak. It is important not to use water at any time to clean up a spill as noted on labels and material safety data sheets for the labels in Table 3.

WS Directive on Pesticide Use (WS Directive 2.401, 12/08/2009)\(^4\) ensures that WS personnel are trained regarding proper and safe pesticide use and adherence to all label requirements. For burrowing rodent applications, the label prohibits use within 100 feet of any building occupied by humans, domestic animals, or may where they may reside, in single or multi-family residential properties and nursing homes, schools except athletic fields, daycare facilities and hospitals. WS uses these products outdoors and applies the pellets or tablets directly to underground burrow systems not connected to an occupied building. A recent paper assessed the minimum distance from buildings necessary to fumigate burrows given the length of North American rodent species’ burrows; the length, depth and number of burrow openings was described for each species (Eisemann et al. 2017).

The label restrictions and WS use patterns result in a low exposure potential to the public. The public would have limited access to AlP, as it is a restricted use pesticide and the label prohibits its use on residential properties. Applicators post warning signs at all entry points to a treatment area that is accessible to the public, such as athletic fields or parks. The signs, which remain in place for a minimum of two days after treatment, warn of the dangers of AlP, indicate reentry period, and provide emergency contact information as discussed on labels.

The use pattern, label restrictions, and fate of AlP and phosphine in the environment indicate no exposure pathway for dietary consumption. The risk of phosphine contamination of drinking water is negligible, thus the exposure to phosphine through drinking water is not likely (USEPA 1998b). Human consumption of animals treated with AlP during WS program activities is unlikely, especially considering the fact that animals die in their burrows underground. A USEPA (1998b) analysis indicated that phosphine residues are unlikely in food because the gas dissipates into the atmosphere, especially during preparation and cooking of food. WS uses AlP only as a burrow fumigant to control rodents and moles, not for the treatment of food commodities. The USEPA (1998b) concluded that aggregate acute dietary risk is not a concern.

\(^4\) All WS Policy Directives referenced in this document can be found @ http://www.aphis.usda.gov/wps/portal/aphis/ourfocus/wildlifedamage under Wildlife Damage – WS Program Directives.
4.2 Environmental

4.2.1 Aquatic Exposure Assessment

Aquatic exposure to AIP is unlikely based on its environmental fate and chemistry characteristics and use pattern. AIP reacts with water, forming phosphine gas which is volatile and has low solubility in water (Gurusinghe 2014). Phosphine degrades in days to phosphates (WHO 1988) and is at low risk for contaminating ground or surface water (Gurusinghe 2014). It is unlikely that AIP or phosphine will be in surface waters making the likelihood of aquatic exposure low (USEPA 1998b).

4.2.2 Terrestrial Exposure Assessment

The primary exposure pathway to terrestrial species is the inhalation route. There are no expected exposure pathways above ground as the label requires animal burrows to be sealed (Odenkirchen 2010). Because of the high toxicity of phosphine gas, an assumption is that phosphine will kill all animals residing in treated burrows, including nontarget animals (USEPA 1998b, Odenkirchen 2010). Product labels instruct applicators to verify that burrows are occupied by target animals before treatment with AIP (Witmer and Fagerstone 2003).

Exposure of plant roots growing in and around treated burrows to phosphine is possible; however, this potential damage was not quantifiable (Odenkirchen 2010). Phosphine trapped beneath the soil surface will bind to soil, inhibiting movement, and be oxidized to phosphates (USEPA 1998b). Phosphates occur naturally in the environment and are nutrients to plants and other organisms (WHO 1988). Phosphine disappeared from air-dried soils within 18 days, but it took 40 days for it to disappear completely from 100% saturated soils (Gurusinghe 2014).

5 RISK CHARACTERIZATION

This section discusses the qualitative risks associated with the WS use of AIP. The determination of risks to human health and nontarget species uses the effects data with the exposure assumptions.

5.1 Human Health and Safety

Although phosphine has high acute inhalation toxicity in humans, the WS use pattern, label restrictions, and environmental fate indicate minimal risk to human health from AIP and phosphine. Potential exposure of the public is at most negligible as AIP is a restricted use pesticide. Dietary exposure to the public is expected to be nil since WS does not apply AIP to food commodities. WS personnel do not use AIP where it would contaminate drinking water.

WS applicators are the most likely to be exposed to phosphine through inhalation. Following label instructions including the use of PPE and other requirements will minimize exposure and risk to applicators.
5.2 Environmental

5.2.1 Aquatic

AIP does not pose a significant ecological risk to aquatic resources given its chemical characteristics and WS use pattern (USEPA 1998b). AIP reacts with water, forming phosphine gas which quickly dissipates (Gurusinghe 2014). Phosphine degrades in days to phosphates and is at low risk for contaminating ground or surface water (USEPA 1998a, Gurusinghe 2014). While phosphine has been found to be toxic to some fish species (WHO 1988), it is highly unlikely that AIP or phosphine will be found in surface waters making the probability of risk negligible (USEPA 1998b).

5.2.2 Terrestrial

Exposure and risk to non-target animals will be greatest for those animals in a burrow that WS treats. Because of the high degree of toxicity of phosphine gas, all animals in a treated burrow will likely be killed (USEPA 1998b). The method of application and environmental fate of AIP keeps impacts localized to any sensitive terrestrial vertebrates and invertebrates inside treated burrows. WS applicators minimize the potential for exposure and risk by monitoring burrows and verifying occupation by target animals before applying AIP. Risk of secondary exposure to animals that consume animal carcasses killed by fumigation is unlikely since the phosphine gas quickly dissipates from the body, which eliminates secondary toxicity concerns (Snider 1983, Witmer and Fagerstone 2003, Baldwin 2012).

WS recorded no non-target animals taken between FY11 and FY15, but some likely occurred. In one study, the treatment of 97 woodchuck burrows with gas cartridges (carbon monoxide fumigation) took 4 non-target animals - one juvenile eastern cottontail rabbit in a burrow and three deer mice in two other burrows (Dolbeer et al. 1991). Without excavating the burrows, WS would not likely know the non-target species taken. Following the Dolbeer et al. (1991) study, it would be expected that if four non-targets were taken for every hundred burrows treated, WS could have potentially taken about 2,333 non-target animals in the 58,330 rodent burrows treated. Considering the species found in the woodchuck burrows, this would be minimal. It should be noted that WS personnel inspect burrow entrances for potential non-targets and does not treat them if present; WS personnel also does not treat in areas where threatened or endangered species could be exposed. Thus, this level of non-target take would be minimal.

AIP and phosphine “degrade to mineral constituents in the soil, so no biologically significant effects are expected for terrestrial plants above ground” (Odenkirchen 2010). In addition, the use pattern for AIP prevents exposure of above ground parts of terrestrial plants. However, exposure of roots inside the burrows is possible. AIP will have a negligible effect to roots since it rapidly reacts with water to produce phosphine gas (Odenkirchen 2010). Data on the effects of phosphine gas on plant roots is limited. As summarized in section 3.2.2.5, observations of harmful effects on growing lettuce occurred at phosphine gas concentrations between 3 and 8 mg/m³. Phosphine concentrations inside burrows can reach 1,000 ppm near the pellets or tablets (Snider 1983), indicating phosphine gas may reach levels that are damaging to roots (Odenkirchen 2010). Any effects that could occur to plants would be localized areas adjacent, or in treated burrows.

6 UNCERTAINTIES AND CUMULATIVE IMPACTS

The predominant uncertainty associated with this assessment is the lack of definitive toxicity estimates in terrestrial organisms. The assumption that all vertebrates and invertebrates in a treated burrow would die
may overestimate acute lethal risk within burrows. Recommended dosage often generates over 1,000 ppm phosphine near the pellets or tablets, but the gas concentration decreases as the gas diffuses out from the source (Snider 1983). Phosphine gas concentrations in burrows may be lower than expected to achieve complete mortality of the inhabitants.

Areas where cumulative impacts could occur are: 1) repeated worker and environmental exposures to AlP from program activities and other sources, 2) co-exposures to other chemicals with a similar mode of action, and 3) exposures to other chemicals affecting the toxicity of AlP.

WS applicators wear PPE that reduce the risk to negligible for repeated exposures to AlP and phosphine. A failure in the function of PPE or misuse of the product could expose applicators to AlP, but this exposure is still unlikely to cause substantial risk or accumulation in the body (See Section 4.1).

The use of phosphine gas to control pests in and around stored food can cause phosphine residue in food. WHO (1988) summarized several studies on phosphine residue concentration in treated food items (a variety of grain and bean commodities treated at several phosphine concentrations) as well as the toxicity of residues in food to rodents. Residue levels in food dropped to negligible levels within several days and residues did not have significant toxicological effects on rodents. The phosphine residue in food is unlikely to contribute cumulatively in applicators who apply burrow fumigants because the gas quickly dissipates in the air, particularly during food preparation and cooking and worker exposure to phosphine during burrow fumigation is negligible.

WS use of AlP may occur in areas where other pesticide applications may occur. Other chemicals with a similar mode of action include the fumigants magnesium and zinc phosphide. Similar to AlP they degrade to phosphine. WS uses zinc phosphide in its program to control burrowing rodents so there is the potential for exposure to both aluminum and zinc phosphide. WS does not use multiple fumigant types in the same burrow at the same time. The proper use of required PPE for both products reduces exposure and the aggregate risks would be minimal for applicators. Zinc and magnesium phosphide are also restricted use chemicals with buffer restrictions in residential and public areas where public exposure could occur.

Cumulative impacts to ecological resources from the use of phosphide compounds (aluminum and zinc) in the WS program are also unlikely because AlP degrades rapidly in the environment, phosphine has a low risk for contaminating ground or surface waters, and it is not persistent or mobile under most environmental conditions.

Phosphine in the environment comes from other sources such as aerobic degradation in soil, sediments, decomposing plant matter and sludge, but is somewhat rare (Glindemann et al. 1996, Eismann et al. 1997b). The contribution of phosphine from WS applications would be negligible, particularly in comparison to its use in non-vertebrate applications and other sources of phosphine in the environment. For example, in California, vertebrate pest control accounted for 6% of the aluminum phosphide chemical used in agricultural and structural applications (California Department of Pesticide Regulation 2017). This percentage would be much less if restricted to just vertebrate applications by the WS program (aluminum phosphide was not used by WS-California during this time).
7 LITERATURE CITED


_____USEPA. 1998a. R.E.D. Facts Aluminum and Magnesium Phosphide. USEPA.

_____USEPA. 1998b. Reregistration Eligibility Decision (RED) Aluminum and Magnesium Phosphide. USEPA.


_____USEPA. 2013. Aluminum Phosphide, Magnesium Phosphide and Phosphine: Review of Human Incidents. USEPA.


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