MSD MANUAL
Veterinary Manual

*PROFESSIONAL VERSION*

# Phosphide (Zinc, Magnesium, Aluminum) Poisoning in Animals

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Zinc phosphide was discovered as a chemical in 1740, and its use as a rodenticide was documented as early as 1911. Although zinc phosphide is most commonly and readily available in pelleted form, typically gray in color, and stored in containers designed to facilitate subterranean placement for the control of gophers and moles, both aluminum phosphide and magnesium phosphide remain available as well.

Formulations include pastes, powders, grains, and pellets, and they range in concentration from 0.5% to 10%. Phosphides remain stable for up to 2 years; a persistently wet environment, however, could decrease stability to 3 weeks to several months.

As with other products, nontarget species typically are exposed by direct ingestion of the bait product itself, although relay toxicosis after ingestion of prey or carrion remains theoretically possible, given phosphides' very narrow margin of safety. **Lethal doses in dogs have been suggested at 20–40 mg/kg, and one-tenth of the lowest lethal dose is widely accepted as a toxic dose requiring decontamination, monitoring, and care.**

Phosphides react in the liquid gastric environment to produce **phosphine gas**. Zinc phosphide requires an acidic pH < 4 to hydrolyze to the toxic phosphine gas, whereas magnesium and aluminum phosphides may do so at a neutral pH. The gas itself is **severely corrosive, very rapidly resulting in spontaneous vomiting with or without blood**. As a consequence, species that cannot vomit (eg, horses and rabbits) are likely to be at increased risk for severe clinical signs and potentially death.

Once liberated, the phosphine gas, which carries a **very distinct decaying-fish or garlic odor**, is quickly absorbed from both the GI tract and respiratory mucosa. The gas poses substantial risk to humans, and owners should be instructed to transport pets to the clinic with open windows for ventilation, in case spontaneous vomiting occurs.

Staff exposure should be limited as much as is practically possible, and adequate ventilation should be established and maintained at all times. Any symptomatic human should be evaluated by a medical professional and human poison control contacted out of an abundance of caution.

Once systemically absorbed, phosphine gas causes extensive oxidative damage to multiple organ systems. It may also disrupt aerobic respiration and cellular energy production at the mitochondrial level, and it may inactivate acetylcholinesterase.

Clinical signs of phosphide poisoning can include **lethargy, severe GI distress that may be hemorrhagic** in nature, **ataxia, tremors, seizures, hyperesthesia, pulmonary edema (tachypnea or dyspnea), pallor, shock, tachycardia or bradycardia, renal and hepatic damage, disseminated intravascular coagulopathy, and methemoglobinemia.** Clinical signs can occur within minutes after exposure or, rarely, take up to 24 hours to appear. In severe cases, cardiovascular collapse and death may occur within 5 hours after exposure.

Heavy metal poisoning from zinc, magnesium, or aluminum is unlikely due to the concentration of the metals in these products. A few agricultural grain fumigant products that contain 50–88% aluminum, magnesium, or zinc phosphide have potential for heavy metal toxicosis if a large enough ingestion occurs and the patient survives the initial phosphine gas exposure.

**Pre-decontamination care and decontamination:**

- Raising of gastric lumen pH
    - Feeding should be avoided because food stimulates gastric acid secretion, lowering pH.
    - Magnesium hydroxide (5–15 mL/animal, PO) is favored for rapid effect, transitioning to aluminum hydroxide for more sustained effect.
    - Animal owners may give calcium carbonate antacids orally before transport.
- Induction of emesis in a controlled, well-ventilated environment, within 1–2 hours after ingestion, in clinically normal patients only
- Gastric lavage, with caution, may be considered in clinically affected patients.
- A single dose of activated charcoal (1–2 g/kg, PO as aqueous slurry) with a cathartic, in patients that are clinically normal and have a low risk of aspiration

**Diagnostic tests:**

- Baseline CBC, serum biochemical analysis, urinalysis, venous blood gas analysis
- Coagulation profile in severely affected patients
- ECG, if cardiovascular signs are noted
- Thoracic radiographs, if respiratory changes are noted
- Close monitoring of vitals and blood pressure
- Monitoring of serum biochemical analysis and venous blood gas analysis in clinically affected patients
- Liver profile 3–5 days after ingestion

**Treatment:**

- Antacids (aluminum hydroxide, magnesium hydroxide, or calcium carbonate) for 3–5 days
- Gastroprotectants (proton pump inhibitors, $H_2$ blockers, sucralfate, or combination thereof) for 5–7 days in clinically normal patients, or for 14–21 days in clinically affected patients
- IV fluid therapy for 24 hours or until clinical signs resolve
- N-acetylcysteine in clinically affected patients or in cases of large exposure, to act as a free radical scavenger, diluted to 5% or less and given IV through a 0.2-mcm filter (140–280 mg/kg loading dose, followed by 70 mg/kg, IV, every 6 hours for 7–17 doses, depending on clinical progression)
- Methocarbamol (55–220 mg/kg, IV, to effect, or as a CRI at 10 mg/kg/h) as needed for tremors
- Anticonvulsants as needed for seizures
- Hepatoprotectants as needed
- Supportive care as determined by clinical course

The prognosis is favorable in dogs that remain clinically normal 12 hours after ingestion of the phosphide and in dogs that are clinically affected without progression of clinical signs over 24 hours. The prognosis is guarded in cases of multiorgan involvement. In a single retrospective study across 5 years and 362 patients, the survival rate was found to be 98.3% (1).

## Key Points

- **Phosphides convert into phosphine gas in the stomach, resulting in corrosive damage to the stomach and oxidative damage to multiple organ systems.**
- **Fumes from vomitus are toxic to humans. Before induction of emesis, do not give food, but instead give magnesium hydroxide or aluminum hydroxide.**
- **Induction of emesis should take place outside or in a well-ventilated area with antiemetics to follow.**
- **Heavy metal toxicosis is unlikely from phosphides.**

## For More Information


- Knight MW. Zinc phosphide. In: Peterson ME, Talcott PA, eds. *Small Animal Toxicology*. 3rd ed. Elsevier; 2013:853-863.
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- Gupta RC, Chae H, Ezell KB. Non-anticoagulant rodenticides. In: Gupta RC, ed. *Veterinary Toxicology: Basic and Clinical Principles*. 4th ed. Elsevier; 2025:611-627.

## References

1. Gray SL, Lee JA, Hovda LR, Brutlag AG. Potential zinc phosphide rodenticide toxicosis in dogs: 362 cases (2004-2009). *J Am Vet Med Assoc*. 2011;239(5):646-651. doi:10.2460/javma.239.5.646