

What are rodenticides?

Rodenticides are pesticides that kill [rodents](#). Rodents include not only rats and mice, but also squirrels, woodchucks, chipmunks, and other animals. Although rodents play important roles in nature, they may sometimes require control. They can damage crops, violate housing codes, transmit disease, and in some cases cause ecological damage.¹

Rodents, humans, dogs and cats are all mammals, so our bodies work in very similar ways. Rodenticides may have the same type of effect when eaten by any mammal. They can also affect birds. Rodenticides are usually formulated as baits, which are designed to attract animals. Flavorings may include fish oil, molasses or peanut butter. Baits used in agriculture and natural areas may contain ground meat, vegetables, grains, or fruits.² These may be attractive to [children](#) and [pets](#), so they should never be used or stored within their reach. Tamper-resistant bait stations make it even more difficult for accidents to happen. For ways to prevent exposures to children, pets, and wildlife, see the information below about what you can do to reduce risks.



How many kinds of rodenticides are there?

There are many different [active ingredients](#) registered as rodenticides in the United States. They can be grouped together according to how they work. Many rodenticides stop normal blood clotting; these are called anticoagulants. Bromadiolone, chlorophacinone, difethialone, diphacinone, brodifacoum, and warfarin are all anticoagulants. There are a number of rodenticides that are not anticoagulants, and these work in different ways. This fact sheet will discuss [zinc phosphide](#), bromethalin, cholecalciferol, and strychnine.

How toxic are rodenticides?

All rodenticides can be toxic when eaten.^{3,4,5,6} See Table 1 on page 2. Most rodenticides are also toxic when inhaled and when they come into contact with skin. The exceptions include warfarin, which is low in toxicity when inhaled or if skin contact occurs.⁶ Strychnine, cholecalciferol, and zinc phosphide are relatively low in toxicity upon skin contact.^{3,5,7} Bromethalin is moderately toxic for dermal exposure.⁴

How do anticoagulants work?

Our livers make a special enzyme that allows our bodies to recycle Vitamin K. Our bodies need Vitamin K to make the blood clotting agents that protect us from bleeding too much. Anticoagulants stop this enzyme from doing its job. Our bodies store an extra supply, but if we are exposed to enough anticoagulant, the supply will run out and internal bleeding may begin.⁸

NPIC fact sheets are designed to answer questions that are commonly asked by the general public about pesticides that are regulated by the U.S. Environmental Protection Agency (US EPA). This document is intended to be educational in nature and helpful to consumers for making decisions about pesticide use.

Table 1. ACUTE TOXICITY CLASSIFICATION - RODENTICIDES

	Oral	Inhalation	Dermal	Primary Eye Irritation	Primary Skin Irritation
Warfarin ^{3,6}	Moderate - High toxicity	Not significant	Not significant	No data	No data
Chlorphacinone ⁴	High toxicity	High toxicity	High toxicity	Non-irritating	Non-irritating
Diphacinone ⁴	High toxicity	High toxicity	High toxicity	Moderate irritation	Slight irritation
Bromadiolone ⁴	High toxicity	High toxicity	High toxicity	Low irritation	Minimally irritating
Difethialone ³	High toxicity	High toxicity	High toxicity	Mild irritant	Non-irritating
Brodifacoum ⁴	High toxicity	High toxicity	High toxicity	Minor irritation	Mild irritant
Bromethalin ⁴	High toxicity	High toxicity	Moderate toxicity	Slight irritation	Non-irritating
Cholecalciferol ³	High toxicity	Very low toxicity	Low toxicity	No data	No data
Zinc phosphide ⁵	High toxicity	High toxicity	Low toxicity	Slight irritation	Non-irritating
Strychnine ⁷	High toxicity	High toxicity	Low toxicity	Highly irritating	Non-irritating

Classification categories were modeled after the U.S. Environmental Protection Agency, Office of Pesticide Programs, Label Review Manual, Chapter 7: Precautionary Labeling. <http://www.epa.gov/oppead1/labeling/lrm/chap-07.pdf>

Warfarin was the first anticoagulant rodenticide.¹ It was registered for use in 1950.⁴ Warfarin was discovered in moldy sweet clover that had made a herd of cattle sick. Researchers found that a fungus had converted a chemical that occurs naturally in the clover to a more toxic chemical.⁹ Warfarin was the most widely used rodenticide until many rodents began to become resistant to it. This led to the development of new rodenticides.⁹

Which anticoagulants require more feedings to work?

Warfarin, chlorphacinone, and diphacinone generally require that an animal eat multiple doses of the bait over several days. These are known as multiple-dose anticoagulants. Single-dose anticoagulants, such as brodifacoum, bromadiolone, and difethialone are more toxic. One day's feeding can deliver a toxic dose.⁴

Table 2. Summary of common rodenticides

Rodenticide	Type	Chemical class	Days of feeding needed
Warfarin	Anticoagulant	Hydroxycoumarin	multiple
Chlorphacinone	Anticoagulant	Indandione	multiple
Diphacinone	Anticoagulant	Indandione	multiple
Bromadiolone	Anticoagulant	Hydroxycoumarin	single
Difethialone	Anticoagulant	Hydroxycoumarin	single
Brodifacoum	Anticoagulant	Hydroxycoumarin	single
Bromethalin	Non-anticoagulant	other	single
Cholecalciferol	Non-anticoagulant	Vitamin D3	multiple or single
Zinc phosphide	Non-anticoagulant	other	single
Strychnine	Non-anticoagulant	other	single

Single-dose anticoagulants are more toxic because they bind more tightly to the enzyme that makes blood-clotting agents. They can also interfere with other steps in Vitamin K recycling. Second-generation, or single-dose anticoagulants, are not easily excreted from the body, and they can be stored in the liver.¹⁰ Most of these rodenticides are not allowed to be marketed to non-licensed applicators for residential use.¹¹ Instead of classifying anticoagulants into “first generation” or “second generation”, many sources refer to them as multiple-dose or single-dose rodenticides because it is less confusing.

What are some of the other rodenticides?

There are a number of rodenticides that work differently than anticoagulants. These are currently used within the United States: bromethalin, cholecalciferol, zinc phosphide, and strychnine. Each of these pesticides works in a different way.

Bromethalin was first registered by the U.S. Environmental Protection Agency (EPA) in 1984.⁴ It stops the cells in the central nervous system from producing energy. The nerve cells swell, this puts pressure on the brain, and paralysis and death soon follow.¹² The major breakdown product of bromethalin is more toxic than bromethalin itself. The varying ability of different species to break down bromethalin may explain why it is more toxic to some animals than others.¹² Bromethalin is considered a single-dose rodenticide.⁴



Cholecalciferol was first registered as a rodenticide in the United States in 1984.⁴ Cholecalciferol is vitamin D₃.¹³ Vitamin D helps the body maintain calcium balance by enhancing absorption of calcium from the gut and kidneys.¹³ Toxic doses of cholecalciferol lead to too much calcium in the blood, which can affect the central nervous system, muscles, the gastrointestinal tract, cardiovascular system, and the kidneys.¹³ The body's ability to maintain proper calcium levels must be overwhelmed before cholecalciferol becomes toxic. Rodents must eat several doses of this rodenticide.⁴ This causes a time lag between exposure and signs of toxicity.¹³ Although pets have gotten sick from eating cholecalciferol, poisonings of people are very rare.¹⁴

Zinc phosphide was first registered in 1947.¹ It changes into phosphine gas in the presence of water and acid. The phosphine gas is very toxic; it blocks the body's cells from making energy, and the cells die.¹⁵ Phosphine exposure is particularly damaging to the heart, brain, kidney, and liver.¹⁵

Strychnine was first registered in 1947, but it was used for many years before then.¹⁶ It can only be used below ground. Products with more than 0.5% strychnine are restricted; they are only sold to certified applicators.¹⁶ Strychnine comes from the seeds of certain plants, *Strychnos nux-vomica* and *Strychnos ignatii*.¹⁷ It affects the cells in the spinal cord by causing nerve cells to fire more readily, which leads to muscle spasms. Given a sufficient dose, the spasms cause breathing paralysis and death.¹⁷

What are signs of rodenticide poisoning?

Always follow label instructions and take steps to minimize exposure. If any exposure occurs, be sure to follow the First Aid instructions on the product label carefully. Some products contain blue or green dye that helps determine whether a child or pet has handled or eaten the product.^{4,18} For additional treatment advice, contact the Poison Control Center at 1-800-222-1222. If you wish to report an incident to the [National Pesticide Information Center](https://www.npic.gov), please call 1-800-858-7378.

Anticoagulant rodenticide exposure can lead to uncontrolled bleeding in any part of the body, but this is not always obvious. Difficulty breathing, weakness, and lethargy have been seen in animals poisoned with anticoagulant rodenticides. Less common signs include coughing, vomiting, stools marked with blackened, tarry blood, paleness, bleeding from the gums, seizures, bruising, shaking, abdominal distention and pain.⁹ Because the stored clotting agents have to run out, signs may be delayed for up to five days following exposure.⁸ Children usually eat small amounts and may never show signs of poisoning. Signs in people include sudden bleeding from the nose, gums, or skin. Internal bleeding can also occur.¹⁰

Bromethalin ingestion causes muscle tremors, seizures, heightened sensitivity to light or noise, and hyperexcitability if the animal eats more than a lethal dose. The onset of signs depends on the dose. If a lethal dose is eaten, signs may develop 8 to 12 hours or several days after ingestion and progress over a period of a week or longer. In this case, animals lose their ability to control their hind legs or sense where their hind legs are. Animals may also vomit, lose interest in food, or adopt strange postures. They may fall into a coma.^{12,19} People may also have altered mental status.²⁰

Cholecalciferol can be toxic from routine or one-time exposure.¹³ Signs in animals include weakness, depression, and loss of appetite. Signs progress to include vomiting, increased thirst, more frequent urination, dehydration, and constipation.¹³ Vomiting, diarrhea, loss of appetite, and depression may develop within 12 to 36 hours after exposure and the kidneys may fail within one or two days. Survivors may have permanent damage to kidneys and muscles. Signs of poisoning may last for weeks because cholecalciferol can be stored in the body and its breakdown products are removed slowly.²¹ Exposed people experience unusual thirst and increased urination. They may suffer heart and kidney damage if the increase in calcium levels lasts long enough.¹⁴

Zinc phosphide may cause vomiting within an hour of ingestion. However, signs of toxicity may be delayed for 4 hours and in some cases longer than 18 hours. The vomit may smell like garlic and may contain blood. Other signs of toxicity include anxiety, discomfort leading to frantic pacing, staggering and weakness, difficulty breathing, and convulsions.^{15,22} Humans also experience vomiting, excitement, chills, shortness of breath and coughing, delirium, convulsions, and coma. Breathing in zinc phosphide dust or phosphine gas given off by zinc phosphide may cause anxiousness and extreme difficulty breathing.¹⁴

Strychnine poisoning causes involuntary muscle spasms in both people and animals. These spasms can be severe, and include extreme extension of the limbs. Signs can begin within 15 minutes in people and within two hours in animals after eating strychnine. Death is caused by impaired breathing.^{14,17}

What if pets and wildlife eat rodents that have been poisoned?

Rodenticide baits are made to attract animals. Pets and wildlife may take the bait if they find it. When an animal eats the bait directly, it is called primary poisoning. Secondary poisoning is caused by eating poisoned prey. It may also be called relay toxicosis. See the fact sheet on [Ecotoxicology](#). For ways to prevent exposures, see the information about what you can do to reduce risks.

The rodenticides with high secondary poisoning risks to birds such as hawks and owls include difethialone and brodifacoum (see Table 2).²³ The rodenticides that pose the greatest secondary poisoning risks for wild mammals, dogs and cats include chlorophacinone, diphacinone, and all of the single dose rodenticides. Bromethalin and cholecalciferol may pose secondary risks but these risks have not been studied as extensively.²

Table 3. Secondary poisoning risks to birds and mammals²

Rodenticide	Secondary risk to birds	Secondary risk to mammals
Warfarin	slight risk	low risk
Chlorophacinone	slight risk	high risk
Diphacinone	moderate risk	high risk
Bromadiolone	moderate risk	high risk
Difethialone	high risk	high risk
Brodifacoum	high risk	high risk
Bromethalin	possible (insufficient data)	low risk
Cholecalciferol	low risk	low risk
Zinc phosphide	low risk	slight risk
Strychnine ^{17,24}	possible (insufficient data)	possible (insufficient data)

Single-dose anticoagulants pose a greater risk to animals that eat poisoned rodents.²⁵ If the rodent continues to feed on the single-dose anticoagulant after it eats a toxic dose during the first day, it may build up more than a lethal dose in its body before the clotting factors run out and the animal dies. Residues of single-dose anticoagulants may remain in liver tissue for many weeks, so a predator that eats many poisoned rodents may build up a toxic dose over time.²⁶ However, even the multiple-dose anticoagulants may be poisonous to animals who eat poisoned rodents.²

Strychnine has caused secondary poisoning in pets that ate poisoned rodents.¹⁷ Zinc phosphide may cause secondary poisoning in pets, but only when the stomach of the rodent still contains intact pellets of the rodenticide. Zinc phosphide breaks down quickly so the rodent must be very recently dead or just dying in order for the zinc phosphide to pose a secondary poisoning risk.¹⁵

What can I do to reduce the risks?

Always follow label instructions and take steps to avoid exposure. Keep all rodenticides out of the reach of children and pets, whether they are in use or in storage. Because of the flavorings and attractive odors in these products, dogs may ***dig them up***, working hard to get to them. ***Choose the right bait station*** for your needs around the home. Some of them are resistant only to children. Some are resistant to children and pets; others are resistant to children, pets and the weather. The EPA has been taking action to reduce risk by requiring bait stations in sensitive areas and by limiting the most toxic active ingredients available on the homeowner market.¹¹

Many rodenticide baits can be toxic to wildlife if they are eaten, or if an animal eats a rodent that was recently poisoned. If you choose to use a rodenticide outdoors, always follow label instructions. To reduce risks of secondary poisoning for pets and wildlife, search for, collect, and dispose of poisoned rodents. Use gloves when disposing of dead rodents to avoid contact and secure trashcan lids to minimize pet or wildlife access to poisoned rodents. If you suspect an ***animal may have been poisoned***, please contact NPIC at 1-800-858-7378 to talk with a Pesticide Specialist.



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You may find that there are other things you can do to control rodents, in addition to using rodenticides. Find out what kind of rodent you have and [learn](#) about its habits, abilities, likes and dislikes. Consider trapping, try to block entry points, and remove any food and water sources. This is called [Integrated Pest Management \(IPM\)](#).

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References

1. *Analysis of rodenticide bait use*; U.S Environmental Protection Agency, Office of Prevention, Pesticides, and Toxic Substances, U.S. Government Printing Office: Washington, DC, 2004.
2. Erickson, W.; Urban, D. *Potential Risks of Nine Rodenticides to Birds and Nontarget Mammals: a Comparative Approach*; U.S Environmental Protection Agency, Office of Prevention, Pesticides, and Toxic Substances, Office of Pesticide Programs, U.S. Government Printing Office: Washington, DC, 2004.
3. Tomlin, C. D. S. *The Pesticide Manual: A World Compendium*, 13th ed.; British Crop Protection Council: Alton, UK, 2003; pp 307-308, 1028-130.
4. *Reregistration Eligibility Decision (RED) Rodenticide cluster*; EPA 738-R-98-007; U.S. Environmental Protection Agency, Office of Prevention, Pesticides, and Toxic Substances, Office of Pesticide Programs, U.S. Government Printing Office: Washington, DC, 1998.
5. *Reregistration Eligibility Decision (RED) Zinc Phosphide*; EPA 738-R-98-006; U.S. Environmental Protection Agency, Office of Prevention, Pesticides, and Toxic Substances, Office of Pesticide Programs, U.S. Government Printing Office: Washington, DC, 1998.
6. *R.E.D. Facts Warafin*; EPA 738-F-91-111; U. S. Environmental Protection Agency, Office of Prevention, Pesticides, and Toxic Substances, Office of Pesticide Programs, U.S. Government Printing Office: Washington, DC, 1991.
7. *Reregistration Eligibility Decision (RED) Strychnine*; EPA 738-R-96-033; U.S. Environmental Protection Agency, Office of Prevention, Pesticides, and Toxic Substances, Office of Pesticide Programs, U.S. Government Printing Office: Washington, DC, 1996.
8. Campbell, A. ; Chapman, M. Anticoagulant rodenticides. *Handbook of poisoning in dogs and cats*; Blackwell Science: Oxford, England, 2000; pp 64-73.
9. Murphy, M. J.; Talcott, P. A. Anticoagulant rodenticides. *Small Animal Toxicology*, 2nd ed.; Elsevier Saunders: St. Louis, MO, 2006; pp 565, 570-571.
10. Watt, B. E.; Proudfoot, A. T.; Bradberry, S. M.; Vale, J. A. Anticoagulant rodenticides. *Toxicol. Rev.* 2005, 24 (4), 259-269.
11. *Rodenticide Products for Consumers*; U.S. Environmental Protection Agency, Office of Prevention, Pesticides, and Toxic Substances, Office of Pesticide Programs. <http://www.epa.gov/pesticides/reregistration/rodenticides/consumer-prod.html> (accessed June 2011), updated June 2011.

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12. Roder, J. D. *Veterinary Toxicology*; Butterworth Heinemann: Boston, 2001; pp 84, 106-108, 123.
13. Rumbelha, W. K. Cholecalciferol. *Small Animal Toxicology*, 2nd ed.; Peterson, M. E.; Talcott, P. A., Eds.; Elsevier Saunders: St. Louis, MO, 2006; pp 629-642.
14. Reigart, J. R.; Roberts, J. R. Miscellaneous rodenticides: Red squill and cholecalciferol. *Recognition and Management of Pesticide Poisonings*, 5th ed.; EPA 735-R-98-003; U.S. Environmental Protection Agency, Office of Prevention, Pesticides, and Toxic Substances, Office of Pesticide Programs, U.S. Government Printing Office: Washington, DC, 1999; pp 179-180.
15. Albretsen, J. C. Zinc phosphide. *Clinical Veterinary Toxicology*; Plumlee, K. H., Ed.; Mosby, Inc.: St. Louis, MO, 2004; pp 456-459.
16. *R.E.D Facts Strychnine*; EPA 738-F-96-033; U.S. Environmental Protection Agency, Office of Prevention, Pesticides, and Toxic Substances, Office of Pesticide Programs, U.S. Government Printing Office: Washington, DC, 1996.
17. Talcott, P. A. Strychnine. *Small Animal Toxicology*, 2nd ed.; Peterson, M. E.; Talcott, P. A., Eds.; Elsevier Saunders: St. Louis, MO, 2006; pp 1076-1082.
18. Means, C. Anticoagulant rodenticides. *Clinical Veterinary Toxicology*; Plumlee, K. H., Ed.; Mosby, Inc.: St. Louis, MO, 2004; pp 444-446.
19. Dorman, D. C. Bromethalin. *Small Animal Toxicology*; Peterson, M. E.; Talcott, P. A., Eds.; Elsevier Saunders: St. Louis, MO, 2006; pp 609-618.
20. Pasquale-Styles, M. A.; Sochaski, M. A.; Dorman, D. C.; Krell, W. S.; Shah, A. K.; Schmidt, C. J. Fatal bromethalin poisoning. *J. Forensic Sci.* 2006, 51 (5), 1154-1157.
21. Morrow, C. K.; Volmer, P. A. Cholecalciferol. *Clinical Veterinary Toxicology*; Plumlee, K. H., Ed.; Mosby, Inc.: St. Louis, MO, 2004; pp 448-451.
22. Knight, M. W. Zinc phosphide. *Small Animal Toxicology*; Peterson, M. E.; Talcott, P. A., Eds.; Elsevier Saunders: St. Louis, MO, 2006; pp 1101-1118.
23. Saravanan, K.; Kanakasabai, R. Evaluation of secondary poisoning of difethialone, a new second-generation anticoagulant rodenticide to barn owl, *Tyto alba* Hartert under captivity. *Indian J. Exp. Biol.* 2004, 42, 1013-1016.
24. Schmutz, J. K.; Rose, K. A.; Johnson, R. G. Hazards to raptors from strychnine poisoned ground squirrels. *J. Raptor Res.* 1989, 23 (4), 147-151.
25. *Risk Mitigation Decision for Ten Rodenticides*; U.S. Environmental Protection Agency, Office of Prevention, Pesticides, and Toxic Substances, Office of Pesticide Programs, U.S. Government Printing Office: Washington, DC, 2008.
26. Eason, C. T.; Murphy, E. C.; Wright, G. R. G.; Spurr, E. B. Assessment of risks of brodifacoum to non-target birds and mammals in New Zealand. *Ecotoxicology* 2002, 11, 35-48.