MSD MANUAL
Veterinary Manual

PROFESSIONAL VERSION

Organic Herbicides Toxic to Animals

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Anilide, Acetamides, or Amide Compounds Toxic to Animals

These herbicides (propanil, cypromid, clomiprop, bensulide, dimethenamid) are plant growth regulators, and some members of this group are more toxic than others. Hemolysis, methemoglobinemia, and immunotoxicity have occurred after experimental exposure to propanil.

Aromatic/Benzoic Acid Compounds Toxic to Animals

The herbicides in this group (chloramben, dicamba, and naptalam) have a low order of toxicity to domestic animals, and poisoning after normal use has not been reported. Environmental persistence and toxicity to wildlife is also low. Recently, dicamba has been blamed for polluting ~4% of US soybean fields because this herbicide is volatile, meaning it spreads to nearby areas. It is estimated that ~70% of plants die following dicamba exposure, and honey production is down 40%-–50% in areas where the chemical is used.

The signs and lesions are similar to those described below for the phenoxy acid derivatives. There is no suitable antidote. Supportive and symptomatic treatment is recommended.

Bipyridyl Compounds or Quaternary Ammonium Herbicides Toxic to Animals

The bipyridyl compounds (diquat, paraquat) produce toxic effects in the tissues of exposed animals by development of free radicals. Tissues can be irritated after contact. For example, mouth lesions have been seen after contact with recently sprayed pastures. Skin irritation and corneal opacity occur on external exposure to these chemicals, and inhalation is dangerous. Animals, including people, have died as a result of drinking from contaminated containers.

Paraquat and diquat have somewhat different mechanisms of action. Diquat exerts most of its harmful effects in the GI tract. Animals drinking from an old diquat container showed anorexia, gastritis, GI distention, and severe loss of water into the lumen of the GI tract. Signs of renal impairment, CNS excitement, and convulsions occur in severely affected individuals. Lung lesions are uncommon.

Paraquat has a biphasic toxic action after ingestion. Immediate effects include excitement, convulsions or depression and incoordination, gastroenteritis with anorexia, and possibly renal involvement and respiratory difficulty. Eye, nasal, and skin irritation can be caused by direct contact, followed within days to 2 weeks by pulmonary lesions as a result of lipid-membrane peroxidation and thus destruction of the type I alveolar pneumocytes. This is reflected in progressive respiratory distress and is evident on necropsy as pulmonary edema, hyaline membrane deposition, and alveolar fibrosis.

There is no specific treatment. Because these chemicals are absorbed slowly, intensive oral administration of adsorbents in large quantities and cathartics is advised. Bentonite or Fuller's earth is preferred, but activated charcoal will suffice. Toxicity of paraquat is enhanced by deficiency of vitamin E or selenium, oxygen, and low tissue activity of glutathione peroxidase. Therefore, vitamin E and selenium with supportive therapy may be useful in early stages of intoxication. Excretion may be accelerated by forced diuresis induced by mannitol and furosemide. Oxygen therapy and fluid therapy are contraindicated.

Carbamate and Thiocarbamate Compounds Toxic to Animals

These herbicides (terbucarb, asulam, carboxazole, EPTC, pebulate, triallate, vernolate, butylate, thiobencarb) are moderately toxic; however, they are used at low concentrations, and poisoning problems would not be expected from normal use. Massive overdosage, as seen with accidental exposure, produces signs similar to those induced by the insecticide carbamates, with lack of appetite, depression, respiratory difficulty, mouth watering, diarrhea, weakness, and seizures. Thiobencarb has induced toxic neuropathies in neonatal and adult laboratory rats. It appears to increase permeability of the blood-brain barrier. There is no suitable antidote. Supportive and symptomatic treatment is recommended.

Dinitrophenolic Compounds Toxic to Animals

Several substituted dinitrophenols alone or as salts such as dinitrophenol, dinitrocresol, dinoseb, and binapacryl are highly toxic to all classes of animals (LD_{50} 20–100 mg/kg body wt). Poisoning can occur if animals are sprayed accidentally or have immediate access to forage that has been sprayed, because these compounds are readily absorbed through skin or lungs. Dinitrophenolic herbicides markedly increase oxygen consumption and deplete glycogen reserves. Clinical signs include fever, dyspnea, acidosis, tachycardia, and convulsions, followed by coma and death with a rapid onset of rigor mortis. Cataracts can occur in animals with chronic dinitrophenol intoxication. In cattle and other ruminants, methemoglobinemia, intravascular hemolysis, and hemoproteinemia have been seen. Exposure to these compounds may cause yellow staining of the skin, conjunctiva, or hair.

An effective antidote for dinitrophenol compounds is not known. Affected animals should be cooled and sedated to help control hyperthermia. Use of physical cooling measures (eg, cool baths or sponging and keeping the animal in a shaded area) are recommended. Atropine sulfate, aspirin, and antipyretics should not be used. Dextrose-saline infusions in combination with diuretics and tranquilizers such as diazepam (not barbiturates) are very useful. Phenothiazine tranquilizers are contraindicated. IV administration of large doses of sodium bicarbonate (in carnivores), parenteral vitamin A, and oxygen therapy may be useful. If the toxin was ingested and the animal is alert, emetics should be administered; if the animal is depressed, gastric lavage and treatment with activated charcoal should be performed.

In ruminants with methemoglobinemia, methylene blue solution (2%–4%, 10 mg/kg, IV, three times a day, during the first 24–48 hours) and ascorbic acid (5–10 mg/kg, IV) are useful.

Imidazolinones Toxic to Animals

Imidazolinone herbicides include imazapyr, imazamethabenz-methyl, imazapic, imazethapyr, imazamox, and imazaquin. These are selective broad-spectrum herbicides. Imidazolinone herbicides caused slight to moderate skeletal myopathy and/or slight anemia in dogs during 1-years dietary toxicity studies with three structurally similar imidazolinones (imazapic, imazaquin, and imazethapyr). There is no evidence of any adverse effect on reproductive performance or of fetal abnormalities in rats or rabbits. There is no suitable antidote. Supportive and symptomatic treatment is recommended.

Organophosphate Compounds Toxic to Animals

Organophosphate compounds such as glyphosate, glufosinate, and bensulide are broad-spectrum, nonselective systemic herbicides. Glyphosate and glufosinate exist as free acids, but because of their slow solubility they are marketed as the isopropyl amine or trimethylsulfonium salts of glyphosate and the ammonium salt of glufosinate. They are toxic to fish. Previously, it was thought sprayed forage was attractive to cattle for 5–7 days after application, but this causes little or no problem because immediate toxicity of glyphosate is likely very low at most environmental concentrations. Recent assessments have been focused on carcinogenicity and genotoxicity of glyphosate. Within the past decade, evidence started to accumulate in the scientific literature that glyphosate might have other toxic effects. Recent epidemiologic studies examined pesticide exposure and the risk of developing neurodegenerative diseases, such as Parkinson and Alzheimer disease in humans. This could result from oxidative stress as well as mitochondrial inhibition in neurons. Whether pesticide exposure increases the risk for developing such diseases is still unknown, because the effects are subtle and cumulative over years of exposure.

Dogs and cats show eye, skin, and upper respiratory tract signs when exposed during or subsequent to an application to weeds or grass. Nausea, vomiting, staggering, and hindleg weakness have been seen in dogs and cats exposed to fresh chemical on treated foliage. The signs usually disappear when exposure ceases, and minimal symptomatic treatment is needed. However, formulations of these compounds may lead to hemolysis and GI, cardiovascular, and CNS effects due to presence of the

surfactant polyoxyethyleneamine. Treatment should include washing the chemical off the skin, evacuating the stomach, and tranquilizing the animal. Massive exposure with acute signs due to accidental poisoning should be handled as an organophosphate poisoning.

Bensulide, listed as a plant growth regulator, has an oral LD_{50} in rats of 271–770 mg/kg; in dogs, the lethal dose is >200 mg/kg. The most prominent clinical sign is anorexia, but other signs are similar to those caused by 2,4-D poisoning.

Phenoxy Acid Derivatives Toxic to Animals

These acids and their salts and esters (2,4-D [2-4-dichlorophenoxyacetic acid], dalapon, dichlorprop [2,4-DP], 2,4,5-T [2,4,5-trichlorophenoxyacetic acid], 2,4-DB, MCPA, MCPB, mecoprop, and silvex) are commonly used to control undesirable plants. As a group, they are essentially nontoxic to animals, except silvex which is unusually very toxic. When large doses are fed experimentally, general depression, anorexia, weight loss, tenseness, and muscular weakness (particularly of the hindquarters) are noted. Large doses in cattle may interfere with rumen function. Dogs may develop myotonia, ataxia, posterior weakness, vomiting, diarrhea, and metabolic acidosis. The oral LD_{50} for 2,4-D and 2,4,5-T in dogs is 100–800 mg/kg. Even large doses, up to 2 g/kg, have not been shown to leave residues in the fat of animals. These compounds are plant growth regulators, and treatment may result in increased palatability of some poisonous plants as well as increased nitrate and cyanide content.

The use of 2,4,5-T was curtailed because extremely toxic contaminants, collectively called dioxins (TCDD and HCDD), were found in technical grade material. TCDD is considered carcinogenic, mutagenic, teratogenic, and fetotoxic, and is able to cause reproductive damage and other toxic effects. Although manufacturing methods have reduced the level of the contaminants, use of this herbicide is very limited worldwide.

Treatment is usually symptomatic and supportive. IV fluids should be given to promote diuresis. Adsorbents and drugs that aid in restoration of liver function are recommended.

Polycyclic Alkanoic Acids or Aryloxyphenoxypropionic Compounds Toxic to Animals

Members of this group (diclofop, fenoxaprop, fenthiaprop, fluazifop, haloxyfop) have moderately low toxicity (acute oral LD_{50} in rats 950 mg/kg to >4,000 mg/kg), except for haloxyfop-methyl (LD_{50} ~400 mg/kg). These compounds are more toxic if exposure is dermal. The dermal LD_{50} of diclofop in rabbits is only 180 mg/kg. There is no suitable antidote. Supportive and symptomatic treatment is recommended.

Protoporphyrinogen Oxidase Inhibitors Toxic to Animals

Protox inhibitors may be diphenyl ether (DPE) or non-diphenyl ether (non-DPE) such as nitrofen and oxadiazon. In the past few years, numerous other non-oxygen-bridged compounds (non-DPE protox inhibitors) with the same site of action (carfentrazone, JV 485, and oxadiargyl) have been marketed. Protox inhibitors have little acute toxicity and are unlikely to pose an acute hazard in normal use. These compounds increase porphyrin levels in animals when administered orally; the porphyrin levels return to normal within a few days. There is no suitable antidote. Supportive and symptomatic treatment is recommended.

Substituted Anilines Toxic to Animals

The most commonly used herbicides of this group are alachlor, acetochlor, butachlor, metolachlor, and propachlor. Low doses in rats and dogs do not produce any adverse effects, but longterm exposure in dogs causes liver toxicity and affects the spleen. Ocular lesions produced by alachlor are considered to be unique to the Long-Evans rat, because the response has not been seen in other strains of rats or in mice or dogs.

Compared with other substituted anilines, propachlor is severely irritating to the eye and slightly irritating to the skin. Propachlor produces skin sensitization in guinea pigs. High doses of propachlor produce erosion, ulceration, and hyperplasia of the mucosa and herniated mucosal glands in the pyloric region of stomach and hypertrophy and necrosis of the liver in rats. In dogs, there is poor diet palatability, which results in poor feed consumption and weight loss. There is no suitable antidote. Supportive and symptomatic treatment is recommended.

Triazines and Triazoles Toxic to Animals

Triazines and triazoles have been used extensively as selective herbicides. These herbicides are inhibitors of photosynthesis and include both the asymmetric and symmetric triazines. Examples of symmetric triazines are chlorostriazines (simazine, atrazine, propazine, and cyanazine), the thiomethyl-s-triazines (ametryn, prometryn, terbutryn), and methoxy-s-triazine (prometon). The commonly used asymmetric triazine is metribuzin.

These herbicides have low oral toxicity and are unlikely to pose acute hazards in normal use, except ametryn and metribuzin, which may be slightly to moderately hazardous. They do not irritate the skin or eyes and are not skin sensitizers. The exceptions are atrazine, which is a skin sensitizer, and cyanazine, which is toxic by the oral route. Sensitivity of sheep and cattle to these herbicides is appreciably high. The main signs are anorexia, hemotoxia, hypothermia, locomotor disturbances, irritability, tachypnea, and hypersensitivity. Simazine is excreted in milk, so it is of public health concern. Atrazine is more toxic to rats but comparatively less toxic to sheep and cattle than simazine. When cultured human cells are exposed to atrazine, splenocytes are damaged; bone marrow cells are not affected. Atrazine induces liver microsomal enzymes and is converted to *N*-dealkylated derivatives. In contrast to simazine, it is not excreted in milk. There is no suitable antidote. Supportive and symptomatic treatment is recommended.

Triazinylsulfonylurea or Sulfonylurea Compounds Toxic to Animals

Toxicity of this group of herbicides (chlorsulfuron, sulfometuron, ethametsulfuron, chloremuron) appears to be quite low. The oral acute LD_{50} in rats is in the range of 4,000–5,000 mg/kg. The dermal acute LD_{50} in rabbits is ~2,000 mg/kg. There is no suitable antidote. Supportive and symptomatic treatment is recommended.

Triazolopyrimidine Compounds Toxic to Animals

Triazolopyrimidine herbicides include cloransulam-methyl, diclosulam, florasulam-methyl, flumetsulam, and metosulam. The acute oral toxicity is very low. There is no suitable antidote. Supportive and symptomatic treatment is recommended.

Ureas and Thiourea Compounds Toxic to Animals

The ureas and thioureas (polyureas) are available under different names such as diuron, fluometuron, isoproturon, linuron, buturon, chlorbromuron, chlortoluron, chloroxuron, difenoxuron, fenuron, methiuron, metobromuron, metoxuron, monuron, neburon, parafluron, siduron, tebuthiuron, tetrafluron, and thidiazuron. Of these, diuron and fluometuron are the most commonly used in the USA, whereas isoproturon is mostly used in other countries. In general, these compounds have low acute toxicity and are unlikely to present any hazard in normal use, except tebuthiuron, which may be slightly hazardous. Cattle are more sensitive to polyurea herbicides than sheep, cats, and dogs.

Signs and lesions are similar to those described for the phenoxyacetic herbicides. The substituted urea herbicides induce hepatic microsomal enzymes and may alter metabolism of other xenobiotic agents. Altered calcium metabolism and bone morphology have been seen in laboratory animals. Recovery from diuron intoxication is quick (within 72 hours), and no signs of skin irritation or dermal sensitization have been reported in guinea pigs. After repeated administration, hemoglobin levels and RBC counts are significantly reduced, while methemoglobin concentration and WBC counts are increased. Increased pigmentation (hemosiderin) in the spleen is seen histopathologically. Linuron in sheep causes erythrocytosis and leukocytosis with hypohemoglobinemia and hypoproteinemia, hematuria, ataxia, enteritis, degeneration of the liver, and muscular dystrophy. In chickens, it leads to weight loss, dyspnea, cyanosis, and diarrhea. It is nontoxic to fish. Fluometuron is less toxic than diuron. In sheep, depression, salivation, grinding of teeth, chewing movements of the jaws, mydriasis, dyspnea, incoordination of movements, and drowsiness are commonly seen. On histopathology, severe congestion of the red pulp with corresponding atrophy of the white pulp of the spleen and depletion of the lymphocyte elements have been reported. The acute LD_{50} of isoproturon in rats is similar to that of diuron.

Polyurea herbicides have been suspected to have some mutagenic effects but do not have carcinogenic potential. In general, these compounds do not cause developmental and reproductive toxicity, except for monolinuron, linuron, and buturon, which are known to cause some teratogenic abnormalities in

experimental animals. There is no suitable antidote. Supportive and symptomatic treatment is recommended.

Other Herbicides Toxic to Animals

Bromacil and terbacil are commonly used methyluracil compounds. Toxic doses of bromacil can be hazardous, especially for sheep, but no field case of toxicity has been reported. The nitrile herbicides, ioxynil and bromoxynil, may uncouple and/or inhibit oxidative phosphorylation. Ioxynil, presumably because of its iodine content, causes enlargement of the thyroid gland in rats.

A number of substances are used as defoliants in agriculture. For example, sulfuric acid is used to destroy potato haulms and two closely related trialkylphosphorothioates (DEF and merphos) to defoliate cotton. A notable feature of the latter is that it produces organophosphate-induced delayed neuropathy in hens. Chlomequat is used as a growth regulator on fruit trees. The signs of toxicity in experimental animals indicate that it is a partial cholinergic agonist.

For More Information

- Also see pet health content regarding poisoning.

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