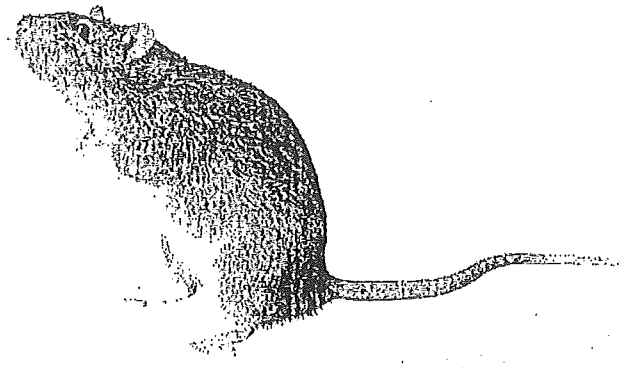
GENERAL VERTEBRATE STUDY PACKET



Supplementary Study Information Packet for the General Vertebrate Pesticide Examination and the Rodent Control Pesticide Examination

Idaho State Department of Agriculture

DESCRIPTION OF ACTIVE INGREDIENTS

Compiled by **Robert M. Timm**

The following pages contain information concerning the active ingredients commonly found in pesticides registered for control of wildlife damage. In general, each description follows a standard format that includes information about the compound's use, history, physical and chemical properties, pharmacology, and toxicity.

Toxicity is defined using the following abbreviations:

For toxicity by oral exposure,
LDL_0 = lowest dose reported to be lethal to an animal

LD_{50} = dose lethal to 50% of the animals tested

LD_{100} = dose lethal to 100% of the animals tested

MLD = minimum lethal dose, or LDL_0.

Doses are typically reported as mass of compound per mass of animal (mg/kg).

For toxicity by inhalation,
LCL_0 = lowest concentration reported to be lethal to an animal

LC_{50} = concentration lethal to 50% of the animals tested

TCL_0 = lowest concentration, for any given period of time, reported to produce any toxic effect.

A toxicity expressed as LC_{50} = 300mg/m^3/30M signifies that 50% of the population can be expected to die when exposed to a concentration of 300 mg per cubic meter of air for 30 minutes.

For certain compounds, much of the descriptive information has been taken from the *Vertebrate Pest Control Handbook* (1986) as revised by Jerry P. Clark, and from the 1975 edition of the same publication compiled by Dell O. Clark. Both volumes were published by the California Department of Food and Agriculture, Sacramento. I thank Jerry Clark and Dell Clark for permission to use this material. Other references used are listed under "For Additional Information" following the description of many compounds, and under "References" at the end of this section. The publications by Spencer (1981), Sweet (1993), and Worthing (1991) were particularly helpful. Rex E. Marsh (University of California-Davis) and Kathleen Fagerstone (USDA-APHIS-Denver Wildlife Research Center) reviewed a draft of this section and provided many helpful comments, additions, and corrections. Additional review was provided by Bill Erickson (US-EPA); Ed Schafer, Jr., Peter Savarie, and Paul Woronecki (USDA-APHIS-Denver Wildlife Research Center); Russ Mason (Monell Chemical Senses Center, University of Pennsylvania); Scott Hygnstrom (University of Nebraska-Lincoln), and Gary Larson (USDA-APHIS-Animal Damage Control). I greatly appreciate their help in improving the accuracy and usefulness of this section.

ACROLEIN

Chemical Name

Acrolein, 2-propenal, acrylaldehyde

Trade Name

Magnacide®

Use

Developed originally as an aquatic herbicide, it is useful in the control of submerged and floating weeds in freshwater ponds and other impoundments.

History

The original research leading to registration of acrolein as an aquatic herbicide was conducted by Shell Chemical Co., which patented the material in 1959 in the United States. Baker Performance Chemicals received a California Special Local Needs 24(c) registration for acrolein as a burrow fumigant for control of ground squirrels in 1993.

Properties

Acrolein is a colorless, highly volatile liquid with a pungent odor. It is moderately soluble in water at 20°C. It is miscible in the lower alcohols, ethers, hydrocarbons, acetone, and benzene. At room temperature, it is noncorrosive to iron or steel. At low doses, it irritates the throat and eyes, thus serving as its own warning agent.

Toxicity

Skin contact causes chemical burns in humans. A concentration of 1 ppm in air produces detectable eye and nose irritation in 2 to 3 minutes and is intolerable after 5 minutes.

Oral LD_{50} values for acrolein have been reported as follows:

Species	Acute Oral LD_{50} (mg/kg)[a]
Mouse	40 mg/kg
Rat	46 mg/kg
Rabbit	7 mg/kg

Toxicity by inhalation has been reported as follows:[a]

Species	TCL_0	LCL_0	LC_{50}
Mouse			66 ppm/6H
Rat			8 ppm/4H[b]
			300 mg/m^3/30M
Human	1 ppm	153 ppm/10M	

[a]Sweet (1993), unless otherwise noted
[b]Lewis and Sweet (1985)

For Additional Information

Lewis, R. L., and D. V. Sweet. 1985. Registry of Toxic Effects of Chemical Substances. 1983-84 Supplement. US Dep. Health and Human Serv., Public Health Serv., Centers for Disease Control, Nat. Inst. Occupational Safety and Health, Cincinnati, Ohio.

O'Connell, R. A., and J. P. Clark. 1992. A study of acrolein as an experimental ground squirrel burrow fumigant. Proc. Vertebr. Pest Conf. 15:326-329.

ALUMINUM PHOSPHIDE

Chemical Name

Aluminum phosphide

Trade Names

Phostoxin®, Detia® Rotox®, Fumitoxin®, Gastoxin®, PhosTek®

Use

A fumigant for certain burrowing rodents and moles, it is also used to control insects in stored products.

History

Aluminum phosphide was introduced as a fumigant for stored products in the early 1930s by Dr. Werner Freyberg, Chemische Fabrik. Its formulation into molded tablets or pellets is a rather recent development. This material was registered for mammal control in 1981, although the compound has been used for this purpose in some other countries for a much longer time.

Properties

Aluminum phosphide forms dark gray or yellowish crystals. For mammal control, it is formulated into 3-g tablets or 600-mg pellets. A typical formulation contains 56% to 57% active ingredient plus 26% ammonium carbamate, 3% paraffin, and 14% to 15% aluminum oxide. Aluminum phosphide reacts with atmospheric moisture to release phosphine (PH_3) gas, the active ingredient. Phosphine is colorless and has a slight carbide-like odor. At some concentrations it is flammable or explosive. In formulations which contain ammonium carbamate, this compound hydrolyzes to release CO_2 and ammonia. Aluminum phosphide should be stored in its original metal container until used.

Toxicity

Phosphine gas is a potent mammalian toxicant. At a concentration of 1,000 ppm, it is lethal to humans after a few breaths. At 400 ppm, it is lethal in 30 minutes.[a] It is immediately dangerous to life or health at 200 ppm.[b] At a concentration of 1 ppm, it can be lethal to some rats within 24 hours.[c]

The following toxicity values are given for phosphine gas:

Species	Inhalation LCL_0[d]
Mouse	380 mg/m^3/2H
Cat	70 mg/m^3/2H
Human	1000 ppm/5H

For Additional Information

Baker, R. O. 1992. Exposure of persons to phosphine gas from aluminum phosphide application to rodent burrows. Proc. Vertebr. Pest Conf. 15:312-321.

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[a]Spencer (1981)
[b]Berg (1983)
[c]Lewis (1979)
[d]Sweet (1993)
For complete citations, see References at the end of this section.

ANTICOAGULANTS

Chemical Names

See below

Trade Names

See below

Use

Anticoagulants are a group of widely used rodenticides; an estimated 95% of all commensal rodent control is conducted with anticoagulants. They are separated into two functional groups, first-generation and second-generation anticoagulants. Those of the second generation have the ability to control warfarin-resistant rats and house mice, and they are also considered single-feeding anticoagulants.

First generation anticoagulants are also used for the control of certain field rodents, including ground squirrels, pocket gophers, and voles. Some field rodent and rabbit registrations are specific to local needs of various states, and they are extensively used to protect agricultural crops and forest trees. At present, none of the second generation anticoagulants are registered for control of field rodents or rabbits.

History

Warfarin, the first anticoagulant rodenticide, had its beginning in 1943 when Dr. Karl Paul Link and his co-workers of the Biochemistry Department, University of Wisconsin, were attempting to determine the cause of "Sweet Clover Disease" in cattle. Moldy sweet clover hay was found to contain a powerful anticoagulant. The first result of the research was the development of dicumarol, which is used to prevent the formation of blood clots in humans. Dr. Link's staff continued the line of research and synthesized warfarin (Compound 42) which is a much more potent anticoagulant than dicumarol. In April 1948, J. A. O'Connor described the first successful use of an anticoagulant compound, dicoumarin, for controlling rats under field conditions.

Pindone, coumafuryl, and valone soon followed warfarin on the market, with diphacinone and chlorophacinone marketed somewhat later. The last two compounds were, by far, more toxic than the earlier materials; hence, the concentration in baits was reduced by some fivefold. Of the earlier anticoagulants, coumafuryl (Fumarin®) and valone (PMP®) are no longer marketed.

The second generation anticoagulants, bromadiolone and brodifacoum, were developed some years later specifically to combat warfarin resistance. The newest of the second-generation anticoagulants, difethialone, has been in development for a number of years and is nearing registration in the United States.

Characteristics

Anticoagulants used as rodenticides are chemically separated into two general groups: the hydroxycoumarins (such as warfarin) and the indandiones (pindone, valone, diphacinone, and chlorophacinone). The second generation materials (bromadiolone, brodifacoum, and difethialone) are closely akin to the hydroxycoumarin group. Table 1 lists the anticoagulants in current use or about to be registered in the United States.

All first-generation anticoagulants, also known as multiple-dose rodenticides, relied on their cumulative toxic effect. They were substantially more toxic if consumed in small doses over a period of several days than if consumed in one large amount (for instance, the 5-day cumulative LD_{50} is substantially lower than the acute LD_{50}). The baits are formulated so that rodents have to feed a minimum of 3 to 5 days before a lethal dose is achieved; death follows after several additional days.

In order to achieve this multiple feeding, the bait must be made available on a continuous basis until the desired control is reached. Prior to the development of anticoagulants, all rodenticides were acute (single dose) materials; hence, the introduction of warfarin required a whole new concept of bait application. Bait trays or bait boxes had to be designed to hold substantial amounts of bait and strategically located so that all rodents in an area had access to ample bait for repeated feedings until death.

Bromadiolone, brodifacoum, and difethialone, all second generation materials, are much more potent, with relatively low acute LD_{50}s for rodents, making them effective for the control of warfarin-resistant rats and mice. When formulated at their current concentrations, they have the ability to kill a high percentage of the rodent population in a single feeding, hence their designation as single-feeding anticoagulants. The effects of these compounds are also cumulative and will result in death after several feedings of even small amounts.

As in the case of all anticoagulants, death is delayed for several days following the ingestion of a lethal dose. This delayed action has a decided safety advantage because it provides time to administer the antidote and save pets, livestock, and of course, people who may have accidentally ingested the bait. Vitamin K_1 is the antidote for anticoagulants and, if administered soon enough after intake, can reverse the action of the anticoagulant. Diphacinone, chlorophacinone, and all of the second-generation materials

Table 1. Anticoagulants used in the United States.

Common Name and Typical Trade Names	Chemical Name	Usual Types of Formulations			Percent Active Ingredient Used in Food Bait
		Food Bait	Liquid	Tracking Powder	
Hydroxycoumarins					
Warfarin (d-Con and others)	3-(α-acetonylbenzyl)-4-hydroxycoumarin	X	X		0.025
Brodifacoum (Talon®, Havoc®)*	3-[3(4'-bromo[1,1'biphenyl]-4-yl)-1,2,3,4-tetrahydro-1-naphthalenyl]-4-hydroxy-2H-1-benzopyran-2-one	X		0.005	
Bromadiolone (Maki® Contrac®)*	3-[3-(4'-bromo[1,1'biphenyl]-4-yl)-3-hydroxy-1-phenylpropyl]-4-hydroxy-2H-1-benzopyran-2-one	X			0.005
Difethialone*	[(bromo-4'-[biphenyl-1-1']-yl-4)3-tetrahydro-1,2,3,4-napthyl-1]3-hydroxy-4,2H-1 benzo-thiopyran-2-one	X			0.0025
Indandiones					
Chlorophacinone (RoZol®)	2-[(p-chlorophenyl)phenylacetyl]-1,3-indandione	X		X	0.005
Diphacinone (Ramik®, Contrax-D®)	2-diphenylacetyl-1,3-indandione	X		X	0.005
Pindone (Pival®, Pivalyn®, Contrax-P®)	2-pivalyl-1,3-indandione	X	X		0.025

*Second-generation anticoagulants especially useful for the control of warfarin-resistant rats and mice.

persist in animals and will often require prolonged veterinary or medical treatment.

The slow action of anticoagulant baits has another great advantage in that the target animal is unable to associate its illness with the bait eaten. Therefore, bait shyness or toxicant shyness does not occur.

Most of the anticoagulant baits used today are commercial ready-to-use baits; very few individuals prepare their own baits from concentrates as they commonly did 20 years ago. Ready-to-use bait increases the cost of rodent control but avoids past problems of incorrect bait concentrations and poor bait formulation, which often led to poor control.

Some anticoagulants are available as tracking powders and others as sodium salts that are water-soluble, allowing their use as water baits.

In the early 1960s the practice began of mixing anticoagulant grain baits with melted paraffin and molding it into cans or cartons to form block-type paraffin baits. These became commercially available a few years later and were promoted for sewer rat control or for other rodent-infested areas with moisture and high humidity. Now there are molded or extruded paraffin-type baits made from most of the current anticoagulants. Block-type baits have several advantages: they confine multiple feedings of bait into one unit; if permitted by the label, they can be placed in strategic locations where bait boxes with loose grain or pelleted bait would be difficult to place; and bait deterioration from insects and molds is retarded.

Anticoagulant Resistance

The resistance of rats to warfarin was first noted in Scotland in 1958, some years following its repeated use. Shortly thereafter, anticoagulant resistance was identified in both rats and house mice in other European countries. It was identified somewhat later in the United States, where it has since been demonstrated in many regions and major cities. All three species of commensal rodents are implicated. Resistance has not been found in field rodents.

Resistance arises from genetic mutation or recombination, sometimes of a single gene, and levels of resistance vary among individual animals. A high degree of resistance will render control with warfarin virtually impossible. Rats and mice that are resistant to warfarin also show some resistance to all first generation anticoagulants. Where resistance is apparent, switch to a second generation anticoagulant or to another rodenticide with a different mode of action.

Whether resistance will eventually extend to all second-generation anticoagulants remains to be seen; some isolated instances of resistance to bromadiolone have been reported.

Pharmacology

All anticoagulants have two actions; they reduce the clotting ability of the blood and cause damage to the capillaries (tiny blood vessels). The rate of blood clotting gradually decreases and blood loss leads to an apparently painless death.

Animals killed by anticoagulants often have no color in the skin, muscles, or viscera. Evidence of hemorrhage may be found in any part of the body, but usually only in one location. The blood that remains in the heart and vessels is very thin and forms a poor clot or no clot. The animal exhibits increasing weakness though appetite and body weight are not specifically affected. Hematoma (a local swelling or tumor filled with blood) formation beneath the skin is often more common than free hemorrhage.

Repeated daily doses of the anticoagulants greatly increases their effective toxicity. Feeding does not have to be on consecutive days, but several feedings should occur within a 10-day interval with no longer than 48 hours between feedings. Plenty of bait must be made available at all times to achieve adequate control.

Toxicity

The susceptibility to anticoagulants varies considerably among species and among anticoagulants. For this reason, generalizing often leads to erroneous conclusions. Since all anticoagulants are cumulative in toxicity, they have the ability to kill any warm-blooded animal if consumed in sufficient amounts for a long enough period. Materials with the highest toxicity and the longest half-lives present the greatest lethal potential with fewer feedings. Compounds with the longest half-lives need not be consumed daily; a lapse of several days between feedings will not alter the outcome.

Many drugs increase the effects of anticoagulants; among these are the broad-spectrum antibiotics, the barbiturates, and the salicylates. Observations of rats treated with chlordane and DDT show the opposite effect; they stimulate the metabolism of warfarin, thus decreasing its toxicity. Susceptibility to anticoagulants seems to increase with age.

Anticoagulants tend to accumulate in the liver and gradually dissipate over a period of time, depending on the initial accumulations and successive does. Where large doses of anticoagulants are ingested, substantial amounts may pass through the animal unassimilated.

Precautions should be taken to prevent children, pets, and livestock from gaining access directly to anticoagulant bait. Baits should be placed in areas inaccessible to nontarget animals or in tamper-resistant bait stations. A single substantial ingestion of diphacinone, chlorophacinone, or any of the second-generation anticoagulant baits may, for example, place a dog in jeopardy, requiring veterinary attention. When used according to label instructions, there is little potential hazard to nontarget species.

Secondary hazard associated with predator or scavenger animals consuming rodent carcasses is minimal in commensal rodent control. It can be of somewhat greater concern when anticoagulants are used for field rodent control. Occasionally a farm dog is known to consume fresh vole or ground squirrel carcasses over several days and begin to show signs of anticoagulant intoxication. With quick and proper veterinary attention, the dog can usually be saved. Although secondary poisoning has been demonstrated in the laboratory for various species, its occurrence in the wild appears very low, with few documented cases where use recommendations were followed.

Toxicity information for many animals is not readily available. A man with suicidal intent induced serious illness by ingesting 1.7 mg warfarin per kg per day for 6 consecutive days. This corresponds to eating almost 1 pound of bait (0.025% warfarin) per day. All signs and symptoms were caused by hemorrhage, and following multiple small transfusions and massive doses of vitamin K, recovery was complete.[a]

The following toxicity data have been reported:

Brodifacoum	Species	Acute Oral LD_{50} (mg/kg)[b]
	House mouse	0.4 - 0.86
	Norway rat	0.27
	Roof rat	0.65 - 0.73[c]
	Meadow vole	0.72[d]
	Pine vole	0.36[d]
	Rabbit	0.29
	Pig	0.5 - 2.0
	Dog	0.25 - 1.0
	Cat	~25
	Chicken	10 - 100

Bromadiolone	Species	Acute Oral LD_{50} (mg/kg)[d,e]
	House mouse, albino	1.75
	Norway rat, albino	1.125
	Rabbit	1.0

Chlorophacinone	Species	LD_{50} (mg/kg) (presumably from single oral dose)[a]
	Vampire bat	7.5
	House mouse	1.06[d]
	White (lab) rat, on meat	2.1
	Norway rat, albino	2.1 - 20.5[d]
	Roof rat	15.0
	Deer mouse	0.49
	Rabbit	50[d]
	Mallard	>100
	Ring-necked pheasant	>100
	Red-winged blackbird	430

Difethialone	Species	Acute Oral LD_{50} (mg/kg)[f]
	House mouse (wild)	0.47
	Norway rat (wild)	0.51
	Roof rat (wild)	0.38
	Pig	2 - 3
	Bobwhite quail	0.264

Diphacinone	Species	Acute Oral LD_{50} (mg/kg)	Chronic Oral LD_{50} (mg/kg/day)
	House mouse	141 - 340[d]	
	Mouse	340[g]	
	Rat	1.9[d] - ~3[h]	(lab rat) 0.1[h]
	Rat	3 - 17[g]	
	Rabbit	35[k]	0.25 for 14 days[d]
	Dog	0.8 - 15[d,h,i]	
	Coyote	0.6[j]	
	Cat	5 - 15[d]	
	Pig	150[h]	

Pindone	Species	Acute Oral LD_{50} (mg/kg)	Chronic Oral LD_{50} (mg/kg/day)
	Rat	280[d]	
	Rat, albino	50[d]	
	Rabbit		0.52 for 7 days[d]
	Dog		2.5[h]

Warfarin	Species	Acute Oral LD_{50} (mg/kg)	Chronic Oral LD_{50} (mg/kg/day)
	Mouse	374[d]	0.6 for 3 - 9 days[a]
	Rat	3.0[d]	0.4 for 4 - 15 days[a]
	Rat	50-100[l]	1 for 5 days[l]
	Rabbit	800[d]	30.0 for 6 - 15 days[a]
	Swine	3[l]	0.05 for 7 days[l]
	Dog	20[d] - 50[l]	5 for 5 - 15 days[l]
	Cat	6 - 40[d]	3.0 - 5.0 for 10 days[a]
	Cat	5 - 50[l]	1 for 5 days[l]
	Ruminants	-	200 for 12 days[l]
	Chicken	1,000[d]	

[a]Clark (1986)
[b]Talon Technical Bulletin, ICI Americas, Inc., (1987)
[c]Dubock and Kaukeinen (1978)
[d]Hone and Mulligan (1982)
[e]Maki Technical Bulletin, Chempar Chemical Co. Inc. (no date)
[f]Lechevin and Poché (1988)
[g]Humphreys (1988)
[h]Spencer (1981)
[i]Technical Bulletin, Velsicol Chemical Co. (1977)
[j]Savarie et al. (1979)
[k]Humphreys (1988)
[l]Osweiler et al. (1985)

For complete citations, see References at the end of this section.

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AVITROL®

Chemical Name

4-aminopyridine

Trade Name

Avitrol®

Use

Avitrol® is a bird management chemical registered for use as a flock-frightening repellent. It is usually formulated as a grain bait. Treated bait is diluted with untreated bait so that only a few birds in a flock ingest a treated particle of bait. Affected birds emit distress cries and/or perform visual displays that often frighten the other birds in the flock, causing them to leave.

Avitrol® has been used for feral pigeons, house sparrows, and for certain blackbirds and cowbirds in and around structures. In agricultural situations, crows, starlings, grackles, cowbirds, and blackbirds are most frequently the targeted species.

Avitrol® products are for use by or under the supervision of government agencies or certified control operators. Avitrol® is not for sale to the public.

History

Avitrol® is the registered trademark of the Avitrol Corporation for the chemical 4-aminopyridine. The synthesis of this chemical was first reported in 1931, and its unique action on birds was reported in 1964 by Goodhue. Its utility for controlling damage by birds in some situations was demonstrated in 1965 by Goodhue and Baumgartner.

Characteristics

4-aminopyridine is a white crystalline, odorless, water-soluble material. It is stable in light and melts at 159°C.

Pharmacology

Avitrol® is an acutely toxic substituted pyridine that affects the nervous system in a manner similar to that of organophosphates and carbamates; however, Avitrol® is not a cholinesterase inhibitor. In most bird species, a lethal dose of Avitrol® is necessary to produce distress behavior.

Toxicity

Birds and mammals appear equally sensitive to Avitrol® intoxication. LD_{50} values are generally less than 10 mg/kg.

Birds ingesting the material become disoriented, emit distress calls, and exhibit erratic flight, tremors, and convulsions before death. Distress usually begins in about 15 minutes and lasts 20 to 30 minutes in most species. Some species, such as pigeons, do not emit distress calls.

In mammals, the following symptoms are produced: hyperexcitability, salivation, tremors, muscular incoordination, convulsions, cardiac or respiratory arrest, and death. Initial effects are usually noted in 10 to 15 minutes and death often occurs 15 minutes to 4 hours later. Occasionally the tremor and/or convulsive stages are accompanied by audible vocalizations produced by strong, involuntary contractions of the diaphragm.

Documented reports of secondary poisoning following Avitrol® use have been very limited. When birds are offered undiluted Avitrol® baits, there may be potential hazards to dogs, cats, and raptors that consume unassimilated Avitrol® in gut contents. In field use, only individual scavengers such as magpies and crows appear to be have been impacted.

Toxicity Table for Avitrol®[a]

Species	Acute Oral LD_{50} (mg/kg)[a,b]	Acute Oral LD_{50} (mg/kg) for HCl*[a,b]
MAMMALS		
White rat	20	28 - 32.5
Hog		17.8
Dog	3.7 - 4.0	11.9
BIRDS		
Mallard	4.22	
American kestrel	5.62	
Domestic chicken (2-3 wk. old)		15
Coturnix quail	7.65 - 8.05	
Ring-necked pheasant (4 wk. old)	5.62 - 7.50	
Ring-billed gull		8
Common pigeon		20
White-winged dove	7.5	
Mourning dove	8.10 - 8.50	
American robin	4.22	
Starling	4.90 - 6.0	14
Black-billed magpie	2.37	
Common crow	2.37	
Yellow-billed magpie	2.37	
Boat-tailed grackle	1.7 - 7.1	
Brown-headed cowbird	4.22	
Common grackle	2.37	
Red-winged blackbird	1.78 - 8.50	3.2
Shiny cowbird	< 1.00	
Tricolored blackbird	4.22	
House finch	5.62	
Golden-crowned sparrow	5.62	
House sparrow	3.00 - 7.50	
White-crowned sparrow	5.62	

* HCl = Hydrochloride salt of 4-aminopyridine.

[a]Schafer et al. (1973)
[b]Schafer et al. (1983)

For complete citations, see References at the end of this section.

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BONE TAR OIL

Trade Name

Magic Circle Deer Repellent®

Use

As an area (odor) repellent to protect trees, shrubs, and other plant materials from deer.

History

Developed as a deer repellent at State College Laboratories, Pennsylvania, during the 1950s. Magic Circle Deer Repellent® was registered and marketed by State College Laboratories, a division of J. C. Ehrlich Chemical Company until 1993.

Properties

The active ingredient, bone tar oil (also called bone oil), is a dark, viscous liquid with a distinct odor. It is produced by the destructive distillation of animal bones. The commercial product contains 93.75% active ingredient.

Toxicity

The human oral LDL_O is 500 mg/kg. The oral LDL_0 for rats is 800 mg/kg.[a]

[a]Sweet (1993)

For complete citations, see References at the end of this section.

BROMETHALIN

Chemical Name

N-methyl-2,4-dinitro-*N*-(2,4,6-tribromophenyl)-6-(trifluoromethyl) benzenamine

Other Name

Bromethalin

Trade Names

Assault®, Vengeance®, Trounce®

Use

A single-dose rodenticide developed for the control of Norway rats, roof rats, and house mice.

History

The toxic nature of certain classes of diphenylamines was researched by Eli Lilly Research Laboratories in the 1970s. Laboratory and field research on bromethalin progressed into the early 1980s, at which time it was registered by EPA.

Properties

Bromethalin is a pale yellow, odorless crystalline solid. Its melting point is 151°C. Bromethalin is insoluble in water but is soluble in many organic solvents.

Pharmacology

Both acute and chronic effects can occur following ingestion. Acute effects include tremors, one or two episodes of clonic convulsions, prostration, and death usually within 18 hours. These effects occur when technical bromethalin is administered in a soluble form and given at a dose two-fold or greater than the LD_{50}, or by generous bait consumption.

Chronic effects, which include lethargy, hind-leg weakness, loss of muscle tone and paralysis, occur with a single dose equal to the LD_{50}, with multiple smaller doses, or with sublethal dietary administration. Several acute and dietary studies have shown that, at sublethal doses, these effects are reversible if administration of the toxicant is discontinued.

Experiments in the physiological and biochemical mechanisms of action suggest that bromethalin uncouples oxidative phosphorylation in central nervous system mitochondria. This could lead to a decreased production of ATP, a diminished activity of Na + /K + ATP-ase, and a subsequent fluid buildup manifested by fluid-filled vacuoles between the myelin sheaths. This vacuole formation in turn leads to an increased cerebrospinal fluid pressure and increased pressure on nerve axons, yielding a decrease in nerve impulse conduction, paralysis, and death.

No secondary hazard is known. Rodents consuming a sublethal dose are not reported to become bait shy.

Toxicity

The toxicity of bromethalin is as follows:

Species	Acute Oral LD_{50} (mg/kg)[a]
Laboratory mouse	5.25 - 8.13
Laboratory rat (Norway)	2.01 - 2.46
Roof rat	6.6
Rabbit	13.0
Dog	4.7
Cat	1.8
Monkey	5.0
Adult quail	4.6
	Chronic Oral LD_{50}[a]
Quail	210 ppm
Mallard	620 ppm

[a]Jackson et al. (1982)

Bait placement and timing are crucial in achieving effective rodent control with bromethalin bait. Bait should be renewed at intervals of several days. Continuous bait availability (as with anticoagulants) is not required, but bait needs to be exposed long enough to allow all animals in the area to feed. Bait quantity will be about one-third that used with anticoagulants, since an animal ingesting a lethal dose does not feed again.

For Additional Information

Dorman, D. C. 1992. Bromethalin rodenticide toxicosis. Pages 175-178 *in* R. W. Kirk and J. D. Bonagura, eds. Current veterinary therapy XI: small animal practice. W. B. Saunders Co., Philadelphia.

Jackson, W. B., S. R., Spaulding, R. B. L. Van Lier, and B. A. Dreikorn. 1982. Bromethalin—a promising new rodenticide. Proc. Vertebr. Pest Conf. 10:10-16.

Spaulding, S. R., and H. Spanring. 1988. Status of bromethalin outside the United States. Proc. Vertebr. Pest Conf. 13:64-69.

Van Lier, R. B. L., and L. D. Ottosen. 1981. Studies on the mechanism of toxicity of bromethalin, a new rodenticide. Toxicol. 1(1):114.

CAPSAICIN

Chemical Name

8-methyl-n-vanillyl-6-nonenamide

Trade Name

Hot Sauce Animal Repellent®

Use

Capsaicin is the material that makes peppers (red, jalapeño, and others) hot. It is also the active ingredient in a repellent used to protect ornamental trees and shrubs, dormant fruit and nut trees, and nursery stock. It is registered for controlling damage by deer, rabbits, and meadow and pine mice. It is also registered as an active ingredient in several dog repellents (Halt®, Dog Shield®) and is frequently used by mail carriers and meter readers to ward off aggressive dogs. It has recently been proposed for federal registration to repel attacking bears and moose.

History

Developed as a repellent by Miller Chemical and Fertilizer Corporation, capsaicin was federally registered by the EPA in 1980.

Properties

Capsaicin (from oleoresin of capsicum) is a liquid with a boiling point of 220°C. It is soluble in vegetable oil and in various solvents. The repellent formulation is a brown to reddish homogeneous liquid at room temperature, containing natural fats and waxes.

Pharmacology

Capsaicin is an extreme irritant to skin, eyes, and other tissues. Exposure to skin causes dermal irritation. Exposure to eyes can cause superficial keratitis and conjunctivitis. Ingestion can cause severe irritation to the stomach or lungs. When handling the concentrate, use rubber or plastic gloves and a face shield.

Toxicity

The acute oral LD_{50} for the mouse is reported to be 47.2 mg/kg.[a]

[a]Sweet (1993)

For Additional Information

Mason, J. R., N. J. Bean, P. S. Shah, and L. Clark. 1991. Taxon-specific differences in responsiveness to capsaicin and several analogues: correlates between chemical structure and behavioral aversiveness. J. Chem. Ecol. 17(12):2539-2551.

Rogers, L. L. 1984. Reactions of free-ranging black bears to capsaicin spray repellents. Wildl. Soc. Bull. 12:59-61.

ALPHA-CHLORALOSE

Chemical Name

1,2-0-(2,2,2-trichloroethylidene)-α-D-glucofuranose

Other Names

chloralose, glucochloralose, A-C

Trade Names

Alphakil®, Murex, Somio

Use

This compound has recently been approved in the United States for use as an immobilizing agent for waterfowl, coots, and pigeons. It is not classified as a pesticide, but rather as a drug. Federal approval for its use by USDA-APHIS personnel and their trained designees was granted by the US Food and Drug Administration in 1992.

History

Alpha-chloralose has been used in baits for stupefying pigeons and house sparrows in the United Kingdom since 1959. It has also been employed in colder climates of European countries as a rodenticide. It has been formulated at about 0.5% in grain baits for birds, but up to 4% active ingredient as a rodenticide against house mice.[a] The compound has also been used as a general anesthetic in laboratory animals, and as an activating agent in electroencephalography in humans.

Properties

This compound is a crystalline powder with a melting point of 187°C. It is soluble in ether and in glacial acetic acid. Its solubility in water is 1 g in 225 ml at 15°C.

Pharmacology

Alpha-chloralose is a centrally active drug with both stimulant and depressant properties on the central nervous system. In birds, alpha-chloralose acts as a hypnotic, rendering birds easier to capture. In rodents, it retards metabolism, lowering body temperature to a degree that may be fatal to small mammals such as house mice in cold climates (below 15°C.).

In mice, ataxia occurs in 5 to 10 minutes following first ingestion, and feeding ceases within 20 minutes. Mice may recover at air temperatures above 15°C. Birds will recover if left alone.[d]

In small doses, the compound increases motor activity and causes myoclonic movements, which may progress eventually to deep anesthesia as dosage increases. It selectively depresses the normal arousal response. The electroencephalogram (EEG) of anesthetized animals, however, suggests that cortical neurons are in a "subconvulsive state."[a]

Alpha-chloralose is metabolized to chloral, which is converted to trichlorethanol, a central nervous system depressant. Hepatic formation of a glucuronide then occurs to form pharmacologically inactive urochloralic acid, which is excreted in the urine.[b]

Early clinical effects are mild ataxia followed by hyperexcitability. Some cats may become quite aggressive. In severe poisoning, the early signs rapidly give way to posterior weakness, prostration, increased salivation, shallow respiration, weak pulse, and hypothermia. Affected animals appear to lose sensitivity to pain but have increased reactivity to touch, sound, or electrical stimulus.[c] A sort of "psychic blindness" is described in dogs[b], in which affected animals do not respond to normal stimuli or familiar surroundings. Fatally poisoned animals appear to die of respiratory failure.[c]

Toxicity

Birds are generally less resistant than mammals to alpha-chloralose. Under experimental conditions, cats have refused to voluntarily consume baits containing the compound, while dogs have eaten 4 g/kg without lethal effects.[b] The following toxicity values have been reported:

Species	Acute Oral LD_{50} (mg/kg)
House mouse	190[d] - 300[e]
Norway rat	300[d] - 400[e,f]
Cat	100[e] - 250[f]
Goose, canada	54[g]
Mallard	42[d,f]
Coot	47 - 58[h]
Pigeon	178[d,f] - 215[g]
Crow	42[e]
Starling	75[e]

[a]Spencer (1981)
[b]Lees and Pharm (1972).
[c]Hayes (1982)
[d]Hone and Mulligan (1982)
[e]Osweiler et al. (1985)
[f]Sweet (1993)
[g]Woronecki (1992)
[h]Woronecki, unpubl.

For complete citations, see References at the end of this section.

For Additional Information

Cornwell, P. B. 1969. Alphakil—a new rodenticide for mouse control. Pharmaceut. J. 202:74-75.

Lees, P., and Y. Pharm. 1972. Pharmacology and toxicology of alpha-chloralose: a review. Vet. Rec. 91:330.

Woronecki, P. P., R. A. Dolbeer, and T. W. Seamans. 1990. Use of alpha-chloralose to remove waterfowl from nuisance and damage situations. Proc. Vertebr. Pest Conf. 14:343-349.

Woronecki, P. P., R. A. Dolbeer, and T. W. Seamans. 1992. Alpha-chloralose efficacy in capturing nuisance waterfowl and pigeons and current status of FDA registration. Proc. Vertebr. Pest Conf. 15:72-78.

CHLOROPICRIN

Chemical Names

Chloropicrin, trichloronitromethane, nitrochloroform

Trade Names

Larvacide®, Chlor-o-pic®, and others

Use

Chloropicrin is a fumigant for controlling rats and mice in structures or burrows. It has been found to be an effective area repellent for house mice. It is also used as a fumigant for insect control in soil and stored grain and cereal products.

History

Its first use as an insecticide was suggested in 1908. It was widely used in chemical warfare (tear gas) from 1916 to 1918.

Properties

Chloropicrin is a heavy colorless liquid with a boiling point of 112°C. It is not flammable. It is soluble in water and miscible with various organic solvents. Chloropicrin is easily detected at very low concentrations because of its characteristic odor and tear gas effect. Hence, it is used as a warning agent in other fumigants. Because of this effect, it may drive rodents out of structures or burrows before they receive a lethal amount.

Pharmacology

The compound is very irritating to mucous membranes, causing tear production. Chloropicrin injures the medium and small bronchi. Pulmonary edema is the cause of death.[a] Because of its irritating properties, it is not usually lethal to humans as long as an escape route exists.

Toxicity

Acute oral LD_{50} for Norway rats is 250 mg/kg.[b] The following toxicities for inhalation of the compound have been reported:

Species	LCL_0	LC_{50}
Mouse		66 mg/m^3/4H[b]
Human	2,000 mg/m^3/M[b]	

[a]Osweiler (1985)
[b]Sweet (1993)

For complete citations, see References at the end of this section.

For Additional Information

Tigner, J. R., and W. A. Bowles. 1964. Chloropicrin tested as an area repellent for house mice. J. Wildl. Manage. 28:748-751.

CHOLECALCIFEROL

Chemical Name

9,10-seocholesta-5,7,10(19)-trein-3 betaol

Other Names

Cholecalciferol, Vitamin D_3

Trade Names

Quintox®, Rampage®

Use

Cholecalciferol is a single-dose or multiple-dose rodenticide.

History

Laboratory and field research of this compound as a rodenticide was conducted relatively recently, although rodenticidal properties of calciferol, a related compound, have been known for some time. Registration as a rat and mouse bait was granted to Bell Laboratories in late 1984 and Motomco Ltd. in early 1985.

Properties

In pure form, cholecalciferol is a light brown resin with a melting point of 84° to 85°C. It is not soluble in water but will dissolve in some organic solvents.

Pharmacology

Cholecalciferol is metabolized in the liver to 25-hydroxycholecalciferol and then in the kidney to 1-alpha, 25-dihydroxycholcalciferol. It causes mobilization of calcium from the bone matrix to plasma. Victims die from hypercalcemia. Time to death is 3 to 4 days after receiving a lethal dose. Bait shyness is said not to occur. Once a rodent consumes a lethal dose, all food intake is claimed to cease. This effect is unlike that of anticoagulants, in which rodents continue to consume bait after they have ingested a lethal dose.

Toxicity

Originally, the acute oral LD_{50} for laboratory mice was reported as 42.5 mg/kg; for laboratory rats, 43.6 mg/kg.[a] For dogs, the reported acute oral LD_{50} was 80 mg/kg.[b] More recent information based on use of formulated products suggests the LD_{50} values for rats and for dogs are in the range of 10 to 15 mg/kg[c], or perhaps lower. Dogs and cats are more susceptible to this compound than are some rodents, and precautions must be taken to prevent their direct access to the bait. Secondary toxicity from feeding on poisoned rodents has not been demonstrated.

[a]Technical Release, Bell Laboratories Inc. (1983)
[b]Sweet (1993)
[c]Personal communication, William Erickson

For Additional Information

Beard, M. L., G. O. Maupin, A. M. Barnes, and E. F. Marshall. 1988. Laboratory trials of cholecalciferol against *Spermophilus variegatus* (rock squirrels), a source of human plague (*Yersinia pestis*) in the southwestern United States. J. Environ. Health 50(5):287-289.

Brown, D. L., and E. F. Marshall. 1988. Field evaluation of Quintox® (cholecalciferol) for controlling commensal rodents. Proc. Vertebr. Pest Conf. 13:70-74.

Dorman, D. C., and V. R. Beasley. 1989. Diagnosis of and therapy for cholecalciferol toxicosis. Pages 148-152 *in* R. W. Kirk, ed. Current veterinary therapy X: small animal practice. W. B. Saunders Co., Philadelphia.

Livezey, K. L., D. C. Dorman, S. B. Hooser, and W.B. Buck. 1991. Hypercalcemia induced by vitamin D_3 toxicosis in two dogs. Canine Practice 16(5):26-32.

Twigg, L. E., and B. J. Kay. 1992. Evaluation of Quintox® for control of feral house mice. J. Wildl. Manage. 56:174-185.

DENATONIUM SACCHARIDE

Chemical Name

Benzyldiethyl ([2,6-xylylcarbamoyl]methyl) ammonium saccharide

Trade Name

Ro-pel®

Use

Ro-pel® is a repellent in a liquid formulation, for application to surfaces to prevent animal feeding or gnawing. Ro-pel® is currently registered for use against deer, beaver, rats, mice, tree squirrels, birds, dogs, and cats. The formulated product contains as active ingredients 0.065% denatonium saccharide and 0.035% thymol. Ro-pel® is described to be "extremely bitter and vile tasting."

History

Denatonium saccharide was developed in the 1980s as a chemical agent by Burlington Scientific for the US government, for potential use in making food crops inedible or rendering drug-producing plants useless. It is chemically related to denatonium benzoate (trade name: Bitrex®), another bitter-tasting compound which is currently registered as a dog and cat repellent, and which has recently been incorporated into some commensal rodent baits as a safety precaution to prevent their ingestion by children.

Toxicity

No toxicity data for denatonium saccharide is available, although the toxicity appears to be low to both birds and mammals. For denatonium benzoate (Bitrex®), the acute oral LD_{50} for the rat is 584 mg/kg, and for the rabbit, 508 mg/kg.[a]

[a]Sweet (1993)

For Additional Information

Davis, S. F., C. A. Grover, C. A. Erickson, L. A. Millere, and J. A. Bowman. 1987. Analyzing aversiveness of denatonium saccharide and quinine in rats. Perceptual Motor Skills 64:1215-1222.

Kaukeinen, D. E., and A. P. Buckle. 1992. Evaluations of aversive agents to increase the selectivity of rodenticides, with emphasis on denatonium benzoate (Bitrex®) bittering agent. Proc. Vertebr. Pest Conf. 15:192-198.

Payne, H. A. 1988. Bitrex—a bitter solution to safety. Chem. Ind. Nov. 21 issue. pp. 721-723.

EGG SOLIDS, PUTRESCENT

Trade Names

Deer-Away®

Use

A repellent for protecting conifers, ornamental trees and shrubs, nonbearing fruit trees, and bearing fruit trees before flowering and leafing out and after harvest, from deer and elk.

History

Developed by researchers at the Weyerhaeuser Company during the 1970s, and later manufactured and marketed by McLaughlin Gormley King Company. Deer-Away® is currently marketed by IntAgra, Inc.

Properties

The product is formulated as a concentrate for making a liquid solution, and as a powder. The concentrate consists of two parts. Concentrate 2103 is a brownish paste-like slurry with a slight fermented egg odor. It contains 37% putrescent whole egg solids and 63% inert ingredients. Formula 2104 is a red liquid that contains only inert ingredients. The concentrates have a shelf life of more than 1 year. When combined and diluted, they result in a ready-to-use spray.

Toxicity

A repellent concentrate containing 15% putrescent whole egg solids and 85% inert ingredients was found to have an acute oral LD_{50} for rats of 34,600 g/kg. It had an acute dermal LD_{50} for rabbits of 3,000 mg/kg. No apparent acute or chronic effects were seen in penned deer exposed to the product for several years.[a]

The product may cause browning and drop of old needles on some species of conifers.

[a]Technical Report, Deer-Away (no date)

For Additional Information

Andelt, W. F., K. P. Brunham, and J. A. Manning. 1991. Relative effectiveness of repellents for reducing mule deer damage. J. Wildlife Manage. 55:341-347.

Conover, M. R. 1984. Effectiveness of repellents in reducing deer damage in nurseries. Wildl. Soc. Bull. 12:399-404.

Palmer, W. L., R. G. Wingard, and J. L. George. 1983. Evaluation of white-tailed deer repellents. Wildl. Soc. Bull. 11:164-166.

FATTY ACIDS

(various compounds)

Chemical Names

a) Ammonium soaps of higher fatty acids
b) Sodium salts of mixed fatty acids

Trade Names

a) Hinder®, Repel
b) Bye Deer®

Use

Repellents for protection of various plants from animal feeding. Hinder® is registered as a deer and rabbit repellents for use on fruit trees and vines, vegetables and field crops, ornamentals, nursery stock, forage crops, grain crops, and noncrop areas. This product contains 15% active ingredient. Label directions instruct dilution with water prior to application.

Bye Deer® contains 85% active ingredient, and is labeled for use to "protect established and new plantings of shrubs, small trees, and flowers from damage caused by white-tailed deer."

History

The product now sold as Hinder® was originally developed in the 1950s by Leffingwell Chemical Co. as a spreader-sticker for use on citrus. Its value as a deer repellent was noted by citrus growers. Field tests to evaluate "Spreader Sticker 268" were conducted by Leffingwell in the late 1960s and early 1970s. It was first registered in California as a repellent in 1969 under the trade name "Repel." It received Special Local Needs registration in many states under the trade name "Hinder®" and full federal registration in 1981.

Bye Deer® received federal registration as a deer repellent in April 1993.

Properties

Ammonium soaps of higher fatty acids, as a concentrate, is a brown liquid with an odor of ammonia. It is soluble in water.

Toxicity

For ammonium soaps of higher fatty acids, the acute oral LD_{50} for rats is > 5 g/kg. For rabbits, the acute dermal LD_{50} is > 2 g/kg.[a]

[a]Hinder® Technical Data Sheet, Agricultural Chemicals, UNIROYAL Inc.

For Additional Information

Fargione, M. J., and M. E. Richmond. 1992. The effectiveness of soap in preventing deer browsing. Proc. Eastern Wildl. Damage Control Conf. 5:68-74.

FENTHION

Chemical Name

0,0-dimethyl 0-[3-methyl-4-(methylthio)phenyl] phosphorothioate

Trade Names

Rid-A-Bird® 1100, Baytex, Queletox, Tiguvon, and others

Use

Fenthion is used in Rid-A-Bird® perches as a contact toxicant for birds (starlings, house sparrows, and pigeons). It has also been used as an insecticide.

History

The compound was developed by G. Schrader and E. Schegk and introduced in 1957 as an experimental insecticide by Farbenfabriken Bayer. It has been used to control *Quelea quelea*, a weaver bird, in Africa by spray applications to roosts and nesting colonies.

Properties

In pure form, it is a colorless liquid. The technical material is a yellow to brown oily liquid with a weak garlic odor. It is soluble in water and in most organic solvents.

Pharmacology

Fenthion is an organophosphate compound; the toxic action of these compounds is caused by their inhibition of acetylcholinesterase. Fenthion is readily absorbed through the skin. Death can occur within minutes but normally takes from 2 to 12 hours, depending on the level and mode of exposure.

Toxicity

Fenthion is moderately toxic to mammals but highly toxic to birds. The symptoms of intoxication include lacrimation, salivation, congestion, ataxia, immobility, toxic or chronic convulsions, and death. The toxicity is as follows:

Species	Acute Oral LD_{50} (mg/kg)
MAMMALS	
Mouse	88[a]
Rat	180[a] - 615[b]
Rabbit	150[a]
BIRDS	
Canada goose	12.0[c]
Duck	5.9[a]
American kestrel	1.0 - 1.33[d]
Bobwhite	≤ 4.0[c]
Quail, Coturnux	17.8[b,d]
Quail, California	15.0[c]
Pheasant	17.8[c]
Chicken	15 - 28[b]
Pigeon	1.78[d] - 4.63[a,c]
Mourning dove	2.37[d] - 2.5[c]
Black-billed magpie	4.22 - 5.62[d]
American robin	5.62[d]
Starling	5.30[d] - 17.8[b]
House sparrow	5.62[b] - 22.7[c]
Red-winged blackbird	1.78[b] - 3.50[d]
Common grackle	4.21[b] - 7.50[d]
House finch	~10[c] - 13.3[d]

Acute dermal LD_{50} values are reported as follows: mouse, 500 mg/kg; rat, 330 mg/kg; duck, 44 mg/kg.[a]

The potential for secondary hazards associated with the use of fenthion-treated perches was documented for avian predators and scavengers in 1969. Because of the low mammalian toxicity, the potential hazard for mammalian scavengers or predators is much less. Secondary poisoning of raptors and owls following exterior use of perches in the winter has been reported.

[a]Sweet (1993)
[b]Hone and Mulligan (1982)
[c]Hudson et al. (1984)
[d]Schafer et al. (1983)

For complete citations, see References at the end of this section.

For Additional Information

Jackson, W. B. 1978. Rid-A-Bird® perches to control bird damage. Proc. Vertebr. Pest Conf. 8:47-50.

Schafer, E. W., Jr., W. A. Bowles, Jr., and J. Hurlbut. 1983. The acute oral toxicity, repellency and hazard potential of 998 chemicals to one or more species of wild and domestic birds. Arch. Environm. Contam. Toxicol. 12:355-382.

GAS CARTRIDGES

Chemical Components

Variable, depending on type of gas cartridge.

Trade Names

US Department of Agriculture Gas Cartridge, Giant Destroyer®, Smoke'Em®, Gopher Gasser®, Dexol Gasser®, and others.

Use

Gas cartridges are incendiary devices designed to give off carbon monoxide and other poisonous gases and smoke when ignited. They are used to fumigate burrows of certain rodents and other mammals.

History

Gas cartridges were developed by the former Bureau of Biological Survey more than 30 years ago. One type is manufactured and supplied by the Pocatello Supply Depot, USDA-APHIS-Animal Damage Control, Pocatello, Idaho. Other types were developed and are manufactured and sold by private commercial establishments.

Properties

The current USDA gas cartridge was developed for control of woodchucks, ground squirrels, prairie dogs, and pocket gophers. It contains sodium nitrate, charcoal, and inert ingredients. A similar cartridge was developed and registered by USDA for fumigating coyote dens.

Most gas cartridges are made of cardboard or paper and are ignited with a fuse. Care should be taken to avoid fire hazards at locations of use. Dry grasses, and methane or natural gas, which may be present in or around structures, can make the use of gas cartridges a potential fire hazard.

Pharmacology

Gas cartridges give off smoke and toxic gases when ignited. Carbon monoxide gas is a major product. In humans, the first stage of carbon monoxide poisoning produces a feeling of tightness across the forehead, headache, throbbing at the temples, dizziness, weariness, nausea, vomiting, collapse, and unconsciousness. In the second stage, the blood pressure falls, muscular control is lost, intermittent convulsions may occur, and the victim's breathing becomes shallower, slower, and finally stops. Presumably, carbon monoxide acts similarly on other animals.

Toxicity

Two hundred parts per million of carbon monoxide in inhaled air may produce symptoms of poisoning in a few hours, and 1,000 ppm can cause unconsciousness in 1 hour and death in 4 hours.[a]

[a]Clark (1986)

For complete citations, see References at the end of this section.

For Additional Information

Dolbeer, R. A., G. E. Bernhardt, T. W. Seamans, and P. P. Woronecki. 1991. Efficacy of two gas cartridge formulations in killing woodchucks in burrows. Wildl. Soc. Bull. 19:200-204.

Matschke, G. H., and K. A. Fagerstone. 1984. Efficacy of a two-ingredient fumigant on Richardson's ground squirrels. Proc. Vertebr. Pest Conf. 11:17-19.

Savarie, P. J., J. R. Tigner, D. J. Elias, and D. J. Hayes. 1980. Development of a simple two-ingredient pyrotechnic fumigant. Proc. Vertebr. Pest Conf. 9:215-221.

METHYL ANTHRANILATE

Chemical Names

Methyl anthranilate; 0-aminobenzoic acid methyl ester; 0-carbomethoxyaniline

Trade Name

ReJeX-iT®

Use

Because methyl anthranilate is broadly (if not universally) repellent to birds, it has many potential applications. The development of several of these applications has begun, and the formal registration of a few is imminent. The manufacturer (PMC Specialties Group) anticipates the registration of methyl anthranilate as a bird repellent additive to standing water at airports. The company also anticipates registration of methyl anthranilate as a bird repellent additive to Concover® (Newastecon, Inc.), a product designed as a thin cover for landfill operations. Gulls and crows refuse to forage in areas sprayed with Concover®/methyl anthranilate. The next anticipated use for the compound is application to turf and cover crops as a goose repellent; this registration is expected in 1994.

History

Methyl anthranilate is a GRAS (generally recognized as safe) food flavoring that is approved by the Food and Drug Administration as an additive to both human and livestock feeds. This chemical occurs naturally and is the characteristic odor of Concord grapes. The major US producer is PMC Specialties Group. The company synthesizes the chemical as a precursor ingredient for the manufacture of calcium and sodium saccharin.

The first publication on the bird repellency of methyl anthranilate appeared in *Poultry Science* (Kare and Pick 1960). The following year, methyl anthranilate was patented as a bird repellent. For reasons still not completely understood, methyl anthranilate is a chemical irritant to birds, much as ammonia, formaldehyde, and black pepper are irritants to mammals. Every avian species tested to date, including laughing gulls, ring-billed gulls, starlings, sparrows, waxwings, red-winged blackbirds, grackles, cowbirds, mallards, Canada geese, snow geese, crows, chickens, guinea fowl, pheasants, bobwhite quail, and turkeys will avoid normally preferred foods when these foods are adulterated with methyl anthranilate at concentrations ranging from 0.5% to 1.0% by weight.

Properties

Methyl anthranilate at room temperature is an oily yellowish liquid. It has a fruity or grapelike odor and occurs in neoli, ylang-ylang, bergamont, jasmine, other essential oils, and in grape juice. It can be obtained synthetically by esterifying anthranilic acid with CH_3OH in the presence of HCl. Methyl anthranilate is only slightly soluble in water but is freely soluble in alcohol or ether. It has a boiling point of 256°C, a melting point of 24°C, and a specific gravity of 1.168. It has a vapor pressure of 1 mm at 20°C.

Pharmacology

According to the Materials Data Safety Sheet, the pure substance may be harmful if inhaled, ingested, or absorbed through the skin. The vapor or mist from the concentrated compound can be irritating to the eyes, mucous membranes, and upper respiratory tract. It can cause skin irritation.

Toxicity

Methyl anthranilate is not fundamentally toxic to mammals or birds. It may, however, be moderately toxic to fish. The acute oral LD_{50} for this compound is reported as follows: mouse, 3,900 mg/kg; rat, 2,910 mg/kg; guinea pig, 2,780 mg/kg. For dimethyl anthranilate, the reported LDL_0 for the rat is 3,380 mg/kg.[a]

[a]Sweet (1993)

For Additional Information

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METHYL BROMIDE

Chemical Name

Methyl bromide

Trade Name

Brom-o-gas®

Use

Methyl bromide is used as a fumigant for rats and mice, insects, certain soil pests, and for treating agricultural commodities. It is sometimes formulated to include a small amount of chloropicrin as a warning agent.

History

Methyl bromide was first described by Perkin in 1884. Its early use was medicinal. It was later used in the preparation of methyl compounds employed in the manufacture of aniline dyes and still later as a fire extinguishing agent.

Methyl bromide was first used in California as an insecticide in 1935 by D. B. Mackie, then chief of the Bureau of Entomology and Plant Quarantine, California State Department of Agriculture.

In 1938, C. E. Berry reported success in killing ground squirrels in California by injecting methyl bromide into their burrow systems at the rate of 10 cm^3 per burrow opening. In the same year, it was reported that all life stages of fleas in the rodent burrows could be killed at this same dosage. This was important from the standpoint of plague control.

Properties

Methyl bromide is a colorless liquid which boils at 4.5°C. At ordinary temperatures it is a nonflammable gas, 1 pound of which occupies 3.98 ft^3 (0.25 m^3/kg). Methyl bromide is 3.5 times heavier than air. It has a burning taste and slight chloroform-like smell. Some formulations contain a small amount of chloropicrin as a warning agent.

Pharmacology

Methyl bromide is harmful by inhalation of the vapors; injury may occur by eye or skin contact with the liquid.

In acute exposures by inhalation, the effects are on both the respiratory and central nervous systems. These effects may be somewhat delayed, but rarely for longer than 24 hours in the case of respiratory symptoms, and seldom over 48 hours for symptoms in the central nervous system. The delay in onset of symptoms of intoxication makes toxic exposure to methyl bromide possible before the hazard is appreciated. Repeated exposures to low concentrations will seldom have pulmonary effects, but central nervous system manifestations may occur days or weeks after the initial exposure.

Methyl bromide will act as a lung irritant in the respiratory system. Effects may vary from mild bronchitis to pulmonary edema and respiratory failure. Signs and symptoms may include coughing, chest pain, difficult or painful breathing, and eventually "wet" breathing often complicated by bronchopneumonia.

Central nervous system symptoms usually accompany or follow the respiratory effects by several hours. Signs and symptoms include intense nausea and vomiting, dizziness, blurred vision, staggering gait, and slurred speech. Convulsions are an ominous sign. Following excitation, central nervous system depression may occur. Muscle weakness and respiratory paralysis may also occur.

Exposure to methyl bromide may produce permanent neurological disburbances such as muscular pain, diplodia, dizziness, or even mental deterioration. Persons surviving for more than 48 hours may experience nausea and vomiting for as long as a week and permanent neurological damage.

Since methyl bromide has a very low boiling point, evaporation takes place very rapidly and within seconds it can entirely disappear from the surface of the skin. However, if methyl bromide is spilled on clothing, gloves, or shoes, such coverings may become saturated, and contact with the skin is prolonged. No particular sensation is produced by such skin contact and the individual may be unaware that exposure has occurred. As a result, burns similar to thermal burns may occur. Leather goods such as shoes that become contaminated with methyl bromide should be destroyed.

No person should be permitted to handle methyl bromide while wearing gloves or close fitting clothes. Methyl bromide will penetrate ordinary rubber gloves, so these should not be worn. Where methyl bromide has been spilled on clothing, such clothing should be removed and carefully aerated before being reworn.

Severe corneal burns may result from splashes of the liquid material in the eye. Vapor exposures to the eye will cause irritation, but burns are unlikely.

Toxicity

For humans, the reported inhalation LCL_0 is 60,000 ppm for 2 hours, and the reported TCL_0 is 35 ppm.[a] Single exposures of 1,000 ppm for 30 to 60 minutes are dangerous to life.

Repeated exposures of 100 ppm for 7 hours per day can produce serious poisoning.[b] Humans should wear appropriate respirators whenever the concentration in the air exceeds 17 ppm.[c]

For rats, the reported LC_{50} is 302 ppm for 8 hours[a], and the reported LCL_0 is 3,120 ppm for 15 minutes[d].

Methyl bromide is phytotoxic. Plants with roots in the vicinity of fumigated rodent burrows may be injured or killed.

[a]Sweet (1993)
[b]Clark (1986)
[c]Spencer (1981)
[d]Lewis and Sweet (1985)

For complete citations, see References at the end of this section.

For Additional Information

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NAPHTHALENE

Chemical Name

Naphthalene

Use

An insecticidal fumigant of limited usefulness and somewhat low potency. Because of its pungent odor, it is sometimes used as a mammal repellent, in paticular for repelling unwanted animals from beneath houses or from attics. It is currently registered for rabbits, squirrels, bats, dogs, and birds and is one of two active ingredients in a registered snake repellent.

History

Naphthalene has long been used as a fumigant and repellent for the clothes moth.

Properties

Colorless, flaky crystals with a melting point of 80°C. Insoluble in water; slightly soluble in alcohol; soluble in ether, chloroform, and other organic solvents.

Pharmacology

Prolonged inhalation may cause headache, nausea, vomiting, and sweating, followed by anemia, haematuria, and optic neuritis. Skin contact may cause local irritation and occasionally dermatitis. In accidental ingestions, naphthalene is rapidly absorbed from the gastrointestinal tract of children. Ingestion causes abdominal pain, diarrhea, rapid pulse and respiration, fever and rigors, and excitement leading to unconsciousness, coma, and convulsions.

Some individuals, particularly males with a Mediterranean or African ancestry, may have a severe hemolytic crisis which may be delayed for several days after exposure. Dermal absorption of naphthalene from blankets has been reported to have caused deaths in extremely young Greek infants.

Toxicity

The acute oral LD_{50} of naphthalene for mice is reported to be 533 mg/kg; for rats, 490 mg/kg. The acute oral LDL_0 for cats is reported as 100 g/kg; for dogs, 400 mg/kg; and for a human child, 100 mg/kg.[a]

[a]Sweet (1993)

For complete citations, see References at the end of this section.

RED SQUILL

Other Names

Squill, scilliroside glycosides (active ingredients)

Use

Red squill powders and extracts have been used primarily for the control of Norway rats. Red squill can be used successfully with all food baits that can be finely ground and mixed thoroughly. It is not satisfactory in cubed fruits or vegetables because these materials will not take up enough squill to kill rats.

History

Red squill, often referred to as the sea onion, is a perennial onionlike plant (*Urginea maritima*), belonging to the lily family and native to countries around the Mediterranean Sea. The bulbs, weighing 5 to 6 pounds each, are sliced, dried, and ground to a fine reddish powder. Red squill has been used since ancient times in the Mediterranean area to combat rats. Until 1945 it was difficult to obtain red squill powders of uniform toxicity. Then a method of fortification was developed whereby "weak" squill powders could be made more toxic and still retain the emetic qualities possessed by the original powder.

Properties

Red squill has been available in powdered form, as a liquid extract, and in a paste. The powder is hygroscopic—it absorbs water from the atmosphere and cakes and hardens if exposed to the air. The powder is extremely irritating to the skin, only slightly soluble, has low toxicity, is not cumulative, is not absorbed by the skin, and is relatively safe to humans and nontarget animals that can vomit such as dogs, pigs, and sometimes cats. Its most desirable quality is this natural emetic aciton which causes vomiting in most animals. Since rats are unable to vomit, they are unable to get rid of the bait through emesis.

Red squill has a sharp, unpleasant, bitter taste but is fairly well accepted by Norway rats initially, if not used in proportions greater than 10% of the finished bait material. Most species of domestic animals will reject baits containing red squill. Its bitter taste can be a major disadvantage, however, because it may cause bait shyness in rats consuming a sublethal dose in the initial feeding. Also, red squill bulbs, powders, or extracts deteriorate in rat-killing ability when subjected to temperatures over 80°C. The powder slowly loses toxicity when it remains in contact with the air and retains only one-half of its original potency when stored for 4 years under ordinary warehouse conditions. Toxicity is preserved when the material is kept in hermetically sealed containers.

Pharmacology

The toxic action of red squill depends on the presence of a rodent-toxic glycoside, scilliroside, in the plant. It kills by a digitalis-like action that causes heart paralysis. Most rats die within 12 hours of eating a lethal dose.

Toxicity

Male rats are considerably more resistant to squill than are females. The toxicity of red squill has been reported as follows:

Species	Acute Oral LD_{50} (mg/kg)
Mouse	50[a]
Norway rat (female)	200 - 250[b]
Norway rat (male)	490[c]
Cat	100[c]
Dog	145[c]
Pig	200[c]
Goat	500[c]
Cattle (juvenile)	100[c]
Cattle (adult)	250[c]

Red squill is considered essentially nontoxic to poultry.

[a]Sweet (1993)
[b]Bartik and Piskac (1981)
[c]Clarke et al. (1981)

For Additional Information

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Verbiscar, A. J., T. F. Banigan, and H. S. Gentry. 1986. Recent research on red squill as a rodenticide. Proc. Vertebr. Pest Conf. 12:51-56.

SODIUM CYANIDE

Chemical Name

Sodium cyanide

Use

Sodium cyanide has been used as a fumigant to kill insects or mammals in burrows. Currently it is used in the M-44® device for control of coyotes, foxes, and feral dogs that depredate livestock and poultry or that depredate federally designated threatened or endangered species. One product is also registered for use to control vectors of communicable diseases (such as canids that may be carrying rabies).

History

Sodium cyanide has been used in predator control in the United States since the invention of the Coyote Getter in the 1930s. This device, a precursor of the current M-44® device, utilized a dose of sodium cyanide propelled into the mouth of a coyote by a .38-caliber shell containing a primer. All predacidal uses of sodium cyanide were cancelled by the EPA in March 1972. Sodium cyanide was re-registered for use in the M-44® device in 1975.

Properties

Sodium cyanide is a colorless solid with a melting point of 564°C. It readily absorbs moisture from the air and is very water soluble in water. When in contact with carbon dioxide or acids, it forms hydrogen cyanide (HCN) gas, which is extremely toxic.

Pharmacology

Hydrocyanic acid (HCN) is highly and quickly toxic by contact, ingestion, or inhalation of vapors. It poisons the cytochrome-oxidase system of all cells. Unconsciousness occurs rapidly, followed by convulsions and death within 5 minutes. Extreme caution must be used.

Toxicity

The acute oral LD_{50} of sodium cyanide for rats is 6.44 mg/kg. The reported human acute oral LDL_0 is variously reported as 2.86 mg/kg or 6.56 mg/kg.[a] The acute oral human TDL_0 is reported to be 0.7 mg/kg.[a] For HCN, the threshold limit value/time weighted average is 10 ppm for humans.[b] It is immediately dangerous to life or health at 50 ppm.[c] A concentration of 200 ppm will quickly kill a human.[b] An antidote kit containing amyl nitrate is available and effective if used quickly following exposure.

[a]Sweet (1993)
[b]Spencer (1981)
[c]Berg (1983)

For complete citations, see References at the end of this section.

For Additional Information

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Shult, M. J., C. W. Ramsey, and W. G. Klussmann. 1976. Using the M-44 in coyote control. Texas Agric. Ext. Serv. Pub. MP-1181, College Station, Texas.

SODIUM FLUOROACETATE

Chemical Name

Sodium monofluoroacetate

Other Names

Compound 1080®, 1080, Fluoroacetate

Use

Compound 1080 is a single-dose toxicant currently registered for use in the Livestock Protection Collar for control of coyotes that are depredating sheep or goats in fenced pastures only. It was formerly registered for use against commensal rodents and certain field rodents in some states. It was also formerly used in large meat draw stations for the control of coyotes and other canids.

History

Polish scientists discovered that an organic fluorine compound from "ratsbane" (*Dichapetalum toxicarium*), a West African poisonous plant, had possibilities as an economic poison. The information was disclosed in 1942 to British scientists who sent samples of the compound to the United States. It received the designation "1080" from the invoice number given to it at the Patuxent Wildlife Research Center, where it was first tested as a rodenticide in June 1944. When the compound appeared promising, the remainder was forwarded to the (then) US Fish and Wildlife Service Denver Wildlife Research Center for further testing and development. More recently, the compound was researched for use in the Livestock Protection Collar, a 1080-filled rubber bladder placed around the neck of a goat or sheep, designed to kill an attacking coyote.

Properties

Sodium monofluoroacetate is a white, stable, water soluble, practically tasteless, crystalline compound. It has a faint acetate odor and a mild acid-salty taste. When the powdered material is exposed to air, it takes up water and becomes gummy. It decomposes at approximately 200°C and is unstable above 110°C. The material is extremely soluble in water and relatively insoluble in many organic solvents and in vegetable fats and oils. A black dye has often been added to the compound during manufacturing to distinguish it from substances like sugar and flour.

Degradation. Salts of monofluoroacetic acid are readily absorbed by root tissues and other cellulosic materials, and are decomposed adaptively by soil bacteria, apparently of the genus *Pseudomonas*. Therefore, any sodium monofluoroacetate leached into the soil will likely be held in the upper layers rich in microorganisms, and decomposed by bacteria. In tests conducted in England the compound either exhibited no measurable toxicity from the start or exhibited no measurable toxicity within two weeks, depending upon the soil type, when applied to soils at 10 ppm; it exhibited no measurable toxicity within 11 weeks when applied to soils at 50 ppm.

Translocation. Sodium monofluoroacetate leached into the soil may be taken up by plants. However, only a small amount is translocated upward to the leaves; the rest remains adsorbed in the roots. Plants can decompose the compound. In one experiment plants had decomposed 29% of the absorbed sodium monofluoroacetate after 48 hours of incubation.

Pharmacology

Sodium monofluoroacetate is very rapidly absorbed in the gastrointestinal tract. Symptoms of poisoning may appear in 15 to 45 minutes and death may occur some later time but usually within 24 hours. The compound is not accumulative to any practical degree. Demonstration of symptoms varies widely among species.

In the body, sodium monofluoroacetate, like other monofluoroacetates, is metabolized to highly toxic fluorocitrate. Fluorocitrate blocks the Krebs cycle, the major mechanism for releasing energy from food. The resulting buildup of citrate blocks glucose metabolism, another mechanism for releasing energy from food. The blockage of these processes causes the energy supply to be reduced to a point where cellular permeability barriers are destroyed, resulting in a loss of function and finally cellular death. Eventually, gross organ or organ system disorders are manifested. Death results from cardiac and/or central nervous system failure.

The primary pharmacological action of Compound 1080 may be either in the central nervous system or the heart — or a combination of the two — depending on the subject involved. Central nervous system symptoms are characterized by initial intermittent convulsions followed by depression. The heart symptoms appear later, and in primates, heart action terminates in ventricular fibrillation. There is no practical antidote for 1080 poisoning; treatment is symptomatic. Once fibrillation begins, death is assured.

Compound 1080 is slower in its action than strychnine. A dog poisoned with 1080 becomes extremely excited, frequently howls, and exhibits running fits, nonrecognition of human presence, and action suggestive of fearful hallucinations or hysteria. The terminal phase of 1080 poisoning in a dog

involves a tonic spasm (continuous muscular contractions) followed by running movements executed in a prone position. The course of tonic spasms may subside at times and the dog may appear normal, but ultimately, repeated convulsive anoxic assaults on the respiratory center lead to death through respiratory paralysis. Death is seldom attributable primarily to action on the heart.

Secondary poisoning can be demonstrated under some conditions with sodium monofluoroacetate. However, simple dilution and the fact that animals can metabolize 1080 to nontoxic metabolites and/or excrete a large quantity of a dose prior to death considerably reduces the hazard of acute poisoning via secondary sources.

Toxicity

An outstanding characteristic of the toxicity of Compound 1080 is the extremely wide variation in susceptibility between different species of animals. Dogs have been killed with a dosage of 1080 as small as 0.06 mg/kg, but a Norway rat may require 8.0 mg/kg. Species vary considerably in their response, with primates and birds the least sensitive, and carnivores and rodents generally the most susceptible. The amount necessary to produce lethal effects often varies in different strains of the same species. The LD_{50} is 2.5 to 8 mg/kg for *Rattus norvegicus* and 1 to 4 mg/kg for *Rattus rattus*.[a] In general, birds are more tolerant of 1080 than are most mammals, although there are exceptions.

Dogs and coyotes are particularly sensitive to Compound 1080. Dogs, with an LD_{50} of 0.06 mg/kg, and coyotes at 0.12 mg/kg, can be poisoned secondarily by eating 1080-killed rodents. Chickens and birds in general are not very susceptible to poisoning from Compound 1080, having an LD_{50} in the range of 5 to 10 mg/kg. Compound 1080 is not hazardous to fish; tests have shown fingerling bream and bass to live indefinitely without discomfort in a concentration of 370 ppm. Judging from fatal and near-fatal cases, the dangerous dose to a human is 0.7 to 2.1 mg/kg.[a]

Toxicity Table

Species	Acute Oral LD_{50} (mg/kg)[b]
MAMMALS	
Carnivores	
Bear, *Ursus* sp.	0.5 - 1.0
Domestic cat	0.4 - 0.5[d]
Coyote, *Canis latrans*	0.12[e]
Dingo, *Canis familiaris dingo*	0.11[d]
Dog, *Canis familiaris*	0.05 - 1.0[d]
Desert kit fox, *Vulpes macrotis*	0.22[d]
Domestic ferret, *Mustela putorious*	1.41
Rodents	
Ground squirrels	
Fisher's, *Spermophilus beecheyi fisheri*	0.3
Pocket gophers: Northern, *Thomomys talpoides*	0.33[c]
Rats	
Norway (lab), *Rattus norvegicus* (male)	2.1*
Rattus norvegicus (female)	2.2*
Norway (wild), *Rattus norvegicus*	3.0
Alexandrine, *Rattus rattus alexandricus*	0.5
Black, *Rattus rattus* sp.	0.1
Cotton, *Sigmodon hispidus litteralis*	0.1
Woodrat, *Neotoma intermedia*	1.5
Mice	
Deer mouse, *Peromyscus* sp.	4.0
House mouse, *Mus musculus*	4.0 - 17.0[d]
Miscellaneous spp.	
Meadow vole, *Microtus pennsylvanicus*	0.92
Prairie dog, *Cynomys ludovicianus*	0.3
Lagomorphs	
Black-tailed jackrabbit, *Lepus californicus*	5.55
European rabbit, *Oryctolagus cuniculus*	< 0.8[d]

*Research has shown much variation between strains of laboratory rodents.

[a]Clark (1986)
[b]Values as reported by Atzert (1971) except as otherwise noted
[c]Denver Wildlife Research Center, USDA, files
[d]Hone and Mulligan (1982)
[e]Connolly (1980)

For complete citations, see References at the end of this section.

Precautions

Sodium monofluoroacetate should be used by trained personnel only, and then under conditions that afford maximum security against accidental poisoning. Users of the Livestock Protection Collar are required to complete thorough training prior to certification. The 1080 solution may escape from collars that are punctured or otherwise damaged. Protective safety measures should always be taken when 1080 solutions are handled.

First Aid Treatment for Dogs

Lloyd (1983) notes that in practically all cases, treatment of poisoned dogs is ineffective, and the prognosis of poisoned animals is grave. If a practitioner has the opportunity to treat an affected dog, the best that can be hoped for is palliation or symptomatic relief. Intravenous injections of barbiturates q.s. to control convulsions have been used. Injections of calcium gluconate solutions have also been employed to control tetany, which is thought to be caused by a

lactic acidemia. Attempts to decrease the concentration of citrate have involved the administration of acetate. Monoacetin (glyceryl monoacetate) has been given intramuscularly in doses of 0.55 gm/kg. Additionally, solutions containing 50% alcohol and 5% acetic acid have been given orally in doses of 8.8 ml/kg (Lloyd 1983).

For Additional Information

Atzert, S. P. 1971. A review of sodium monofluoroacetate (Compound 1080) — its properties, toxicology, and use in predator and rodent control. Bur. Sport Fish. Wildl., Div. Wildl. Serv., Spec. Sci. Rep.-Wildl. No. 146, US Dep. Inter., Washington, DC. 34 pp.

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STARLICIDE®

Chemical Name

3-chloro-*p*-toluidine hydrochloride

Other Names

3-chloro-4-methyl benzylnamine hydrochloride, CPTH, DRC-1339

Use

Starlicide® is a slow-acting avicide registered for the control of starlings, blackbirds, pigeons, gulls, ravens, crows, and magpies.

History

This chemical, originally coded DRC-1339 and evaluated by the Denver Wildlife Research Center, was found to be an excellent toxicant for starlings and blackbirds when formulated as a Starlicide® pellet. It received federal registration in 1967 for feedlot uses. Starlicide® is manufactured and distributed by the Purina Mills Company. Registration of a DRC-1339 concentrate has been maintained by USDA-APHIS for use against starlings, blackbirds, and gulls, with additional approvals granted for use against pigeons in 1992 and against ravens, crows, and magpies in 1993. Use of the DRC-1339 concentrate is restricted to USDA-APHIS personnel.

Properties

The technical compound is a pale yellow, crystalline solid material that is very soluble in water and other highly polar solvents; it sublimes at 220°C. If formulated with many grains, potency of the compound may decline significantly when stored. Commercial Starlicide® pellets retain their potency for 6 to 12 months.

Pharmacology

Starlicide® is a slow-acting and apparently painless toxicant in birds and mammals. In sensitive bird and mammal species, death results primarily from uremia (a buildup of uric acid in the blood). Death occurs without convulsions or spasms, and is the result of generalized circulatory impairment in the liver and kidney, and congestion of the major organs. At death, victims' feathers are usually fluffed and their feet tucked inside the feathers of the lower breast.

In most mammals and nonsensitive birds, death results from methemoglobinenemia (a buildup of methemoglobin in the blood). Mammals become listless and comatose before death.

Birds and mammals appear to metabolize or excrete Starlicide® completely within a matter of hours, and the metabolites are also excreted. Known metabolites are nontoxic to birds and mammals. Because the Starlicide® and its metabolites are excreted while birds are still alive, there is no secondary toxicity to any scavengers eating dead birds.

Toxicity

In birds, the average time between ingestion and death is 36 to 60 hours, depending on the amount ingested. Even when the lethal dose level is exceeded many times, death still takes many hours. In most mammals, death occurs in 3 to 12 hours.

The toxicity of Starlicide® varies considerably between bird species. Starlings, blackbirds, and crows are among the most sensitive birds; house sparrows and hawks are nonsensitive. Mammals are generally not sensitive to the toxic effects of Starlicide®.

Toxicity Tables

Species	Acute oral LD_{50} (mg/kg)[a,d]
BIRDS	
Mallard	17.8
Blue-winged teal	31.6
Pintail	> 32
Cooper's hawk	562
Golden eagle	> 100
Marsh hawk	100
Kestrel	> 320
Ring-necked pheasant	10
Coturnix quail	2.24 - 10
Domestic turkey	5.62
Domestic chicken	4[b]
White-winged dove	4.22
Mourning dove	3.16 - 7.50
Pigeon (rock dove)	17.8
Barn owl	4.22
Blue jay	10
Scrub jay	1.78
Black-billed magpie	5.6 - 17.7
Common raven	5.62
Common crow	1.33 - 1.78
American robin	3.16
Starling	3.16 - 4.11
House sparrow	320 - 448
Red-winged blackbird	2.41
Tricolored blackbird	2.74
Boat-tailed grackle	1.00
Common grackle	1.00
Cassin's finch	> 100
House finch	> 225
White-crowned sparrow	> 320

Species	Acute oral LD_{50} (mg/kg)[c]
MAMMALS	
Mouse	2,000
White mouse	960
White rat	1,170 - 1,770
Rat	655[b]
Dog	> 100
Sheep	ca 400
Cow	> 10

[a]DeCino et al. (1966)
[b]Sweet (1993)
[c]Clark (1986)
[d]Schafer et al. (1983)

For complete citations, see References at the end of this section.

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Sterner, R. T., D. J. Elias, and D. R. Cerven. 1992. The pesticide reregistration process: collections of human health hazards data for 3-chloro-*p*-toluidine hydrochloride (DRC-1339). Proc. Vertebr. Pest Conf. 15:62-66.

STRYCHNINE

Chemical Name

2,4a,5,5a,7,8,15,15a,15b,15c-dechydro-4,6-methano-6H,14H-indolo (3,2,1-ij)oxepino(2,3,4-de)pyrrolo(2-3-h)quinolin-14-one

Use

Strychnine is a widely used toxicant registered for use in controlling certain rodent and depredating bird species. In the past, strychnine was commonly used for controlling rodents, depredating birds, and mammals such as skunks and coyotes. Aboveground uses were halted by court action in 1988, but it remains registered and used belowground for control of pocket gophers and, in some states, other species.

History

Strychnine is one of the alkaloids processed from raw dried ripe seed of *Strychnos nux vomica*, a small tree native to India, North Australia, Vietnam, and Ceylon. This alkaloid was discovered by Pelletier and Caventon in 1817. There is 2.0% to 2.7% total alkaloid found in the seeds, which were used to kill dogs, cats, and birds in Europe at least as early as 1640.

Properties

Strychnine, a white crystalline powder, is currently available in an alkaloid form; the sulfate form previously used is no longer registered. Strychnine has a bitter taste. It is almost entirely insoluble in water and very stable; however, it is subject to acid-salt formation, which renders it water soluble and subject to leaching in acid soils.

Pharmacology

Strychnine acts the quickest of the commonly used rodenticides. It is not stored in body tissues nor absorbed through normal intact skin. It has a very slight odor, very high toxicity, and acts somewhat variably on target animals. Strychnine enters the blood very rapidly and acts on the central nervous system. The time of action depends on whether the stomach is empty or full and the nature of the food present. Animals with little in their stomachs react more quickly to strychnine than those that have fed recently. Symptoms may appear from 5 to 30 minutes after ingestion.

Intoxicated animals have frequent tetanic convulsions interspersed with quiescent periods. Ultimately these convulsions lead to death through respiratory failure.

Strychnine is not assimilated into tissues or bone; however, residues in the gastrointestinal tract of animals poisoned with lethal doses are known to be potentially hazardous if the gastrointestinal tract is consumed. With its current belowground application pattern, secondary poisoning is unlikely.

Toxicity

LD_{50} values range between a low of 0.70, 0.75 and 1.5 mg/kg for coyotes, desert kit foxes, and black-tailed prairie dogs, respectively, to a high of 27.0 mg/kg for nutria; and between 16.0 mg/kg for chukar partridge and 24.7 mg/kg for ring-necked pheasants. LD_{50}s for mallards, Canada geese, golden eagles, and house sparrows fall within an approximate range of 3.0 to 5.0 mg/kg.

Livestock are about as sensitive to strychnine as rats.[a] Horses, hogs, geese, and ducks show no hesitation in eating strychnine baits. Cattle and sheep are more reluctant to accept baits. Gallinaceous game birds and most domestic poultry, however, are less susceptible to strychnine than most rodents.

Antidote

The use of general antidotes is feasible and often successful if treatment is initiated soon after exposure. Sodium pentobarbital and sodium amytal both act to reduce the severity of convulsions in humans (see J. Am. Med. Assoc. 100:548-551). Emetics such as 1% to 2% tannic acid are useful but should only be used after the convulsive stage is past. Prompt administration of methocarbamol is useful in treating poisoned dogs. Prognosis: if the patient lives for 24 hours, he or she will probably recover.

Strychnine Alkaloid	Acute Oral LD_{50} (mg/kg)
MAMMALS	
Carnivores	
Cat	0.5[f] - 2.0[c]
Coyote	0.7[b]
Dog	0.5[f] - 1.2[b]
Desert kit fox	0.7[b]
Rodents	
Squirrels	
California ground squirrel	19.9 - 28.0[b]
Black-tailed prairie dog	1.5[b]
Pocket gophers	
Northern pocket gopher	8.3[b]
Rats & Mice	
Rat	3 - 6[e]
Norway rat (wild)	12.0[b]
White mouse (lab)	9.3[b]
White rat (male)	14.0[b]
White rat (female)	5.8[b]
Black rat	10.1[b]
Polynesian rat	6.8[b]
Miscellaneous Rodents	
Banner-tailed kangaroo rat	3.7[b]
California meadow mouse	22.2[b]
Meadow vole	6.8[b]
Nutria	27.0 - 42.0[b]
Lagomorphs	
Black-tailed jackrabbit	4.4[a]
Ungulates	
Cow	15.0[b]
Mule deer	17.0 - 24.0[d]
Horse	2.0[b]
Sheep	7.5[b]
Swine	0.5 - 1.0[c]
Swine	10.0[b]
Primates	
Human	1[c]
Human	15 - 30[c]
Human	30 - 60[e]
AMPHIBIANS	
Bullfrog	2.2[d]
BIRDS	
Ducks and Geese	
Mallard	2.3[d] - 2.9[c]
Canada goose	4.0[a]
Raptors	
Golden eagle	4.8 - 8.1[d]
Galliformes	
Chicken	5.0[c]
Chukar	16.0[d]
Coturnix (Japanese quail)	22.6[d]
Pheasant	24.7[d]
California quail	112[d]
Pigeons and Doves	
Pigeon (rock dove)	21.3[d]
Passerines	
House finch	5.6[b]
Robin	> 10.0[a]
English sparrow	4.2[d]

[a]Clark (1986)
[b]Denver Wildlife Research Center, USDA, files
[c]Hone and Mulligan (1982)
[d]Hudson et al. (1984)
[e]Pinto and Spear (1980)
[f]Sweet (1993)
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THIRAM

Chemical Name

Tetramethylthiuram disulfide

Other Names

TMTD, Arasan

Use

A fungicide which also has been used as a repellent to protect plants from deer, rabbits, rodents, and moles.

History

Developed for fungicidal use in 1931 by E. I. duPont de Nemours and Company. The DuPont trade name, Arasan, is no longer used since other companies are now involved in manufacture and formulation of the fungicide.

Properties

A colorless crystalline material with a melting point of 155° to 156°C. Its solubility in water is 17.4 mg/l. It is slightly soluble in alcohol and in ether; it is soluble in chloroform and in acetone.

Pharmacology

Thiram is of relatively low mammalian toxicity but is reported to cause skin irritation. As a repellent, it is presumed to work by taste, not odor. In some animals, a learned aversion may occur. For humans, ingestion may cause nausea, vomiting, diarrhea, and anorexia, followed by ataxia, hyperexcitability, and hypothermia. Hypotonia may progress to flaccid paralysis and respiratory failure. Skin contact or inhalation may cause irritation of the nose, throat, or skin and may induce an allergic dermatitis.

A related compound, tetraethylthiuram disulfide (Antabuse) blocks the in vitro oxidation of ethanol at the acetaldehyde stage and is used for the treatment of chronic alcoholism.

Persons handling or using thiram should avoid ingestion of alcohol before or after use.

Toxicity

The toxicity of thiram is as follows:

Species	Acute Oral LD_{50} (mg/kg)
Mouse	1,350[a]
Rat	560 - 780[a,b]
Rabbit	210[a]
Sheep	225[c]
Pheasant	673[d]
Poultry	~1,000[c]
Mallard	>2,800[d]

[a]Sweet (1993)
[b]Berg (1983)
[c]Humphreys (1988)
[d]Hudson et al. (1984)

For complete citations, see References at the end of this section.

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ZINC PHOSPHIDE

Chemical Name

Zinc phosphide

Use

Zinc phosphide, at concentrations of 0.75% to 2.0% on grain, fruit, or vegetable baits, has been used successfully against such species as meadow mice, ground squirrels, prairie dogs, Norway rats, Polynesian rats, cotton rats, and nutria. In some areas, zinc phosphide baits have been partially or completely rejected by ground squirrels and meadow mice and at times control has been erratic.

History

Zinc phosphide appears to have been first synthesized by Marggral in 1740 and was first used as a rodenticide by the Italians in 1911. Extensive use of zinc phosphide in the United States did not occur until 1942, when the availability of strychnine became uncertain due to the war.

Properties

Zinc phosphide is a heavy, finely ground gray-black powder that is practically insoluble in water and alcohol. When exposed to moisture, it decomposes slowly and releases phosphine gas (PH_3). Phosphine, which is highly flammable, may be generated rapidly if the material comes in contact with dilute acids. Zinc phosphide concentrate is a stable material when kept dry and hermetically sealed.

Although zinc phosphide baits have a strong, pungent, phosphorous-like odor (garliclike), this characteristic seems to attract rodents, particularly rats, and apparently makes the bait unattractive to some other animals. For many uses of zinc phosphide formulated on grain or grain-based baits, prebaiting is recommended or necessary for achieving good bait acceptance.

In general, zinc phosphide is less toxic than Compound 1080 or strychnine and is slower-acting than either of these compounds.

There is only a small amount of deterioration of zinc phosphide on baits due to the evolution of phosphine gas; therefore, dry baits must be considered to be toxic indefinitely and must be used accordingly. Lecithin-mineral oil, added to zinc phosphide to adhere it to grain bait, offers protection against moisture, and therefore may increase its stability. Under field conditions, zinc phosphide baits may remain toxic for several months until baits are eroded by weathering, the carrier decomposes, or the grain is removed by insects. Physical erosion does not seem to occur rapidly. In one instance, zinc phosphide-treated bait exposed in the field for 2 to 3 months and subjected to 10 to 12 inches (25 to 30 cm) of rain continued to maintain some toxicity.

When zinc phosphide is dusted onto wet baits, such as meats or cubed fresh fruits and vegetables, it breaks down within a few days and the baits soon lose their attractiveness.

In soil, zinc phosphide breaks down rapidly to phosphine, which is either released into the atmosphere or converted to phosphates and zinc complexes.

Translocation of phosphine gas has been demonstrated, but it is rapidly converted to harmless phosphates. There is no evidence that hazards exist via this route when grain baits are used in growing vegetables.

Pharmacology

When zinc phosphide comes into contact with dilute acids in the stomach, phosphine (PH_3) is released. It is this substance that probably causes death. Animals that ingest lethal amounts of bait usually succumb overnight with terminal symptoms of convulsions, paralysis, coma, and death from asphyxia. If death is prolonged for several days, intoxication occurs that is similar to intoxication with yellow phosphorous, in which the liver is heavily damaged. The surface of the liver will be spotted and discolored. Prolonged exposure to phosphine can produce chronic phosphorous poisoning.

Early symptoms of zinc phosphide poisoning are nausea, vomiting (yielding black stomach contents and the smell of phosphine), abdominal pain, chest tightness, excitement, and a feeling of coldness. In fatal cases, there is liver, kidney, and heart damage. The time between ingestion and death is frequently about 30 hours. Victims who are alive after 3 days are said to recover completely. Mild poisoning from breathing minute amounts of phosphine gas can be mistaken for food poisoning because of the diarrhea and stomach pains produced.

Zinc phosphide-poisoned rats show no signs of distress until a short terminal death agony occurs. They typically die in a prone position with their legs and tails outstretched.

Because zinc phosphide is not stored in muscle or other tissues of poisoned animals, there is no secondary poisoning with this rodenticide. The bait, however, remains toxic up to several days in the gut of a dead rodent. Other animals can be poisoned if they eat enough of the gut content of rodents recently killed with zinc phosphide.

Toxicity

Zinc phosphide is poisonous to some degree to all animals. Supposed safety factors such as the odor and dark color may be of little deterrence in some situations. As little as a teaspoonful of bait containing zinc phosphide could cause toxic symptoms in a child to whom the color and odor may not be disagreeable. Therefore, around dwellings, bait should be exposed only in situations that will prevent pets and children from coming into contact with it.

Use extreme care in handling zinc phosphide concentrate and treated bait. If zinc phosphide baits are prepared in the open air, phosphide generated from the moist bait offers little hazard. When quantities of bait are prepared within a bait mixing plant, safeguards against continued exposure to low concentrations of phosphide must be taken. Zinc phosphide dust created by the preparation or handling of baits is also hazardous. Personnel working indoors should wear appropriate respirators and work under exhaust fans. Zinc phosphide baits should not be mixed or distributed with the bare hands. Oils, liquid or semisolid, are used in some preparations. Because phosphorous is soluble in certain fatty oils, it may be absorbed in small amounts through the skin. Continued exposure to phosphorous absorption may result in toxic manifestations at some later time. Rubber or synthetic gloves are preferrable when handling dry zinc phosphide bait formulations but cotton or leather gloves are acceptable.

Zinc phosphide can be used for rat control on almost any food product; however, it (or any other acute toxicant) should not be used on bait materials recognizable as food in the home environment. Do not use on such foods as tomatoes, apples, oranges, or bread, unless they are made unrecognizable by rolling or cubing them.

Toxicity Table 1.[a,b]

Species	Acute Oral LD_{50} (mg/kg)
MAMMALS	
Carnivores	
Cat and dog	20 - 40[c]
Desert kit fox	93.0
Rodents	
Squirrels	
California ground squirrel	33.1
Black-tailed prairie dog	18.0
Pocket gophers	
Northern pocket gopher	6.8
Rats	
Norway rat (wild)	27 - 40[c]
White rat	55.5
Black rat	21.0
Roof rat	2.9 - 40.5
Polynesian rat	23.0
Mice	
Mouse	40[d]
Deer mouse	40.5
Meadow vole	18.0
California meadow mouse	15.7
Other Rodents	
Banner-tailed kangaroo rat	8.0
Muskrat	29.9
Nutria	5.55
Woodrat (LD_{100})	25.0
Other Mammals	
Black-tailed jackrabbit	8.25
Cow	50.0
Human (estimated MLD*)	40.0
Human, female (LDL_0)	80[d]
Pig	20 - 40[c]
BIRDS	
Ducks & Geese	
Mallard	13.0 - 35.7[c]
Snow goose	8.8
White-fronted goose	7.5
Galliformes	
Chicken	20 - 40[c]
California quail	13.5
Partridge	26.7
Pheasant	8.8 - 26.7
Other Birds	
Mourning dove	34.2
Red-winged blackbird	23.7

*MLD is minimum lethal dose or LDL_0

Toxicity Table 2.[a,b]

Species	Zinc Phosphide Acute Oral Toxicity (mg/kg)		
	LDL_0	LD_{50}	LD_{100}
Snow geese	5-10	8.75	5-10
White-fronted geese	0-5	7.5	10-15
Mallards	5-10	13.0	10-20
Pheasant	0-10	8.8	10-15
Quail	5-10	13.5	10-20
Dove	10-20	34.25	10-50

[a]Clark (1986) unless otherwise noted
[b]Hood (1972) unless otherwise noted
[c]Hone and Mulligan (1982)
[d]Sweet (1993)

For complete citations, see References at the end of this section.

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ZIRAM

Chemical Name

Zinc dimethyldithiocarbamate

Other Name

Zerlate

Use

Currently this compound is used as the active ingredient in one rabbit repellent.

History

Ziram was originally developed as an accelerator in rubber vulcanization. It has been used as a fungicide since the early 1930s and later received use as a rodent, rabbit, and bird repellent.

Properties

Ziram is a white, odorless powder with a melting point of 246°C. It is not soluble in water slightly soluble in alcohol and in ether; moderately soluble in acetone; and soluble in dilute alkali, chloroform, and in carbon disulfide. It is stable under ordinary conditions but is decomposed by acids.

Pharmacology

Ziram's repellency is presumably due to taste or physiological discomfort following ingestion. Ziram may irritate the skin and mucous membranes. Persons handling or using ziram should avoid ingestion of alcohol before or after use.

Toxicity

The acute oral LD_{50} for rats is reported to be as low as 267 mg/kg.[a] The acute oral LD_{50} to mice is 480 mg/kg, and to rabbits 400 mg/kg.[a] Dogs reportedly tolerated 25 mg/kg per day for one month but not for one year; they ingested 5 mg/kg per day for one year without ill effects.[b]

[a]Sweet (1993)
[b]Spencer (1981)

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