TOXICANTS AND FUMIGANTS

ACROLEIN

Use: The trade name for this fumigant is Magnacide "H "®. It is commonly used to control aquatic weeds in canals, ditches, ponds, and water systems. It is now a registered fumigant for controlling burrowing rodents (i.e. California ground squirrels, pocket gophers). Efficacy studies have shown that Acrolein is effective in the control of fleas on rats. This is important for applications conducted in association with plague suppression. The liquid fumigant is injected directly into the burrow under pressure.

History: 1948 the Shell Oil Company formulated the current compound from petroleum; earlier compounds from other sources were formulated as early as 1828. Baker Performance Chemicals received a California Special Local Needs 24c registration for acrolein as a burrow fumigant for control of ground squirrels in 1993.

Characteristics: Acrolein is a general biocide that is a highly volatile, colorless, pungent liquid which is very irritating to the eyes at concentrations of less than 1 part per million (ppm). It is water soluble and should not be contaminated with any foreign material at any time. The boiling point is 52.7^0C, the flash point is -25^0C, and has a specific gravity of 0.847.

Pharmacology: Poisoning may occur from vapor inhalation or swallowing the material in the liquid state. Irritation to the eyes may occur from contact with the vapor.

Toxicity: The active ingredient, acrolein, is a general cell toxicant that kills through its sulfhydryl reactivity, which destroys vital enzyme systems in plant cells. It is a general biocide that exhibits considerable variability in toxicity towards fish. Some fish species are very sensitive to this material.

Exposure to skin can cause severe irritation with chemical burns resulting from prolonged contact with the skin surface. If the material is swallowed, acute gastrointestinal distress will occur in association with difficulty in breathing and edema. If the material is inhaled, irritation of the respiratory system will occur along with headaches and eye irritation. Further symptoms such as (Pulmonary edema) may be slow in developing (up to 24 hours following exposure).

Statement of Practical Treatment

Call a physician immediately in all cases of suspected poisoning; California Poison Control Center can be contacted at 1-800-222-1222.

Internal: If the material has been swallowed, induce vomiting immediately. This may be done by introducing a finger into the throat or by giving warm salt water (1 tablespoon of salt to a glass of water). Repeat until vomit fluid is clear.

Never give anything by mouth to an unconscious person. Keep patient prone and quiet. If inhaled, get victim into fresh air immediately and give artificial respiration if breathing has stopped.

External: If spilled on skin, remove all contaminated clothing and wash skin with soap and running water. If material gets into the eyes, wash immediately with running water for at least 15 minutes. For eyes, get medical attention immediately.

Statement of Practical Use

Acrolein is a Restricted Use Category-1 economic poison that can only be sold from and applied by a certified applicator or person under their direct supervision. Acrolein is a material requiring special transportation needs so the Highway Patrol and the Dept. of Motor Vehicles should be contacted for transportation requirements and proper documentation. The local county agricultural commissioner should be contacted to obtain a Restricted Material permit and certification prior to the purchase, storage, or use of the material. A label condition requires that California Fish and Game shall be notified 48 hours prior to the application of this product to determine if any known threatened or endangered species are listed as being in the application area.

ALUMINUM PHOSPHIDE

Use: Hydrogen phosphide (PH_3) is a highly poisonous and flammable gas that is released from certain phosphorous compounds when they react with moisture. When generated from aluminum phosphide tablets, pellets or bags, hydrogen phosphide becomes a general biocide that is used to control insects in stored grains, nut meats, processed foods, and for rodent burrow fumigation.

History: Aluminum phosphide was formulated in 1930 by DEGESCH, a German company. It is formulated under many trade names including Phostoxin®, Detia®, Rotox®, Fumitoxin®, Gastoxin®, and Phostek®.

Characteristics: Aluminum phosphide has an odor similar to garlic, which is characteristic of an impurity of carbide. It is virtually insoluble in water and fats, but may spontaneously ignite when it contacts liquid water. The boiling point of hydrogen phosphide is 87.4^0C. It is slightly heavier than air, with a vapor density of 1.184. Aluminum phosphide should be stored in its original metal container until used.

Pharmacology: Poisoning may occur from inhalation of gas, but also by swallowing the tablet or pellets.

Toxicity: Hydrogen phosphide barely penetrates the skin; it is a poison that blocks important physiological systems in the body cells.

Symptoms may occur immediately or within hours depending on the amount of gas inhaled. Typical symptoms of a light exposure would be fatigue, buzzing in the ears, nausea, pressure in the chest, and uneasiness. When these symptoms occur, the victim should be placed in fresh air. Greater volumes of gas inhaled will result in a quicker onset of symptoms that include general fatigue, nausea, stomach-intestine discomfort, with vomiting and diarrhea. Further exposure will result to equilibrium loss, strong chest pains, dispend, and death.

Hydrogen phosphide does not penetrate the skin very well, but is absorbed through the mouth, nose and mucus membranes. Once in the blood stream hydrogen phosphorous acid and phosphate block important ferment systems in the body's cells. In high concentrations, it alters the hemoglobin by forming methemoglobin which blocks hemolysis. Aside from acute poisoning (single influence of high exposures) there is the possibility of sub acute poisoning by consecutive influence of smaller dosages. Chronic poisoning has not been observed in humans or animals.

The human nose can detect quantities of hydrogen phosphide as small as 1.4 ppm. Tests have shown that, hazardous industrial exposure levels have not been observed in the field when the material is applied as a rodent burrow fumigant according to the label and proper safety clothes are worn.

Statement of Practical Treatment

Call a physician immediately in all cases of suspected poisoning, (CPCS http://www.calpoison.org 1-800-222-1222). If swallowed, drink one or two glasses of water and induce vomiting by touching the back of the throat with a finger, or if available, by administering syrup of ipecac. Do not induce vomiting or give anything to an unconscious person. If inhaled, remove the person to fresh air, immobilize and keep warm. Sustain breathing artificially if necessary.

Statement of Practical Use

Aluminum phosphide is a Restricted Use Category-1 economic poison that can only applied by a "Certified" applicator. The local county agricultural commissioner should be contacted to obtain a Restricted Material permit and certification prior to the purchase, storage, or use of the material. Since aluminum phosphide is not selective, any animals in the fumigated burrow are likely to be affected. Some endangered species are found in rodent burrows. Check for label restrictions and with the California Department of Pesticide Regulation to see if aluminum phosphide use restrictions are in effect in the treatment area. Prior to transporting Aluminum phosphide on public roads, the California Highway Patrol CHP and the Department of Motor Vehicles DMV should be contacted. They will provide information concerning the required documentation, and current restrictions. The restrictions may vary depending upon the trade name and quantity transported. Public agencies including county agricultural commissioners are not exempt from these requirements administered by the CHP and DMV.

ANTICOAGULANTS

Use: The anticoagulants represent a group of rodenticides which will have the same mode of action, although they may differ in other characteristics. Anticoagulants are widely used for commensal rodent control, with an estimated 95% of all commensal rodent baiting currently conducted with anticoagulants. Anticoagulants have also gained a prominent place in the control of field rodents. Their importance has grown with the loss of many of the acute rodenticides formerly available.

Terminology can be somewhat confusing. Anticoagulants can be separated into two distinct groups: first generation anticoagulants (warfarin, chlorophacinone, and diphacinone), and second generation anticoagulants (brodifacoum, bromadiolone, and difethialone).

All six rodenticides are used in bait products to control rats and mice in and around buildings. Only chlorophacinone and diphacinone are registered for agricultural uses. Brodifacoum and diphacinone have island conservation uses that are managed by the Fish and Wildlife Service (FWS). First generation anticoagulants are used for the control of certain field rodents; ground squirrels, pocket gophers, voles. Second generation anticoagulants have the ability to control warfarin resistant rats and house mice, and are also considered single feeding anticoagulants.

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 researching the cause of "Sweet Clover Disease" in cattle. Moldy sweet clover, *Mililotus alba*, hay was found to contain a powerful anticoagulant. Dicumarol was identified and later developed for human use to prevent the formation of blood clots. In April 1948, J.A. O'Connor described the first successful use of the anticoagulant, dicoumarin, for the control of rats under field conditions. Dr. Link and staff continued their line of research and synthesized warfarin (Compound 42) which is a significantly stronger anticoagulant to be marketed as a rodenticide, and this resulted in a whole new approach to rodent control and a dramatic change in our ability to control commensal rats and mice.

Since the development of warfarin, a number of other anticoagulants have been developed and marketed. Most had characteristics which made them equal or superior to their predecessors. Today we have a number of excellent anticoagulants which are available in an array of bait formulations for commensal rats and mice and, to a lesser extent, for native field rodents.

Characteristics: Anticoagulant rodenticides are divided into chemical groups based on their chemical structure: the hydroxycoumarins (e.g. warfarin, brodifacoum, and bromadiolone) and the indandiones, such as pival, diphacinone, and chlorophacinone. Several additional anticoagulants have been developed in other countries and were never marketed in the United States.

Warafin resistance in rats and mice seemed to be a significant and growing problem, having been identified in the United States in the early 1970's, first in Norway rats and later in house mice and roof rats. This genetically linked warfarin resistance meant cross-resistance in varying degrees to all of the early anticoagulants. This concern over resistance stimulated developmental research in Europe on new anticoagulant rodenticides. This new research led to the development and marketing of brodifacoum, bromadiolone, and difethialone which are more potent materials and are referred to as second generation anticoagulants. This means that now we have two functional groups of anticoagulants; the first generation, which represented all of the early anticoagulants, and the second generation anticoagulants, which are effective on warfarin resistant rats and mice.

The first generation anticoagulants are more chronic in their action, requiring multiple feedings over several days to a week or more to produce death. The second generation, brodifacoum, bromadiolone, and difethialone are much more toxic to rodents and, unlike the first generation materials, their acute toxic values differ relatively little from the sum of their chronic toxic values. This means that a single feeding can produce death with the second generation materials if a sufficient amount of bait is consumed. Death, however, is still delayed by 5-6 days, as with all anticoagulants. In commensal rodent control, the second generation materials are much more apt to control rodents which are marginal or reluctant feeders or one time feeders. Many pest control operators have found that they have fewer call-backs with these so called single-feeding anticoagulants.

In all anticoagulants death is delayed for several days following the ingestion of a lethal dose. This delayed action provides a safety advantage as it provides time to administer an antidote if identified and save pets, livestock, and people who may have ingested the bait. Vitamin K_1 is the antidote for anticoagulants, and if administered soon enough after ingestion can reverse the action of the anticoagulant.

The slow action of anticoagulants has another 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 anticoagulant baits used today are commercial ready to use baits; or are available mixed from some County Agricultural Commissioner offices. Also available are block type paraffin baits where the anticoagulant has been mixed with melted paraffin and molded into blocks. Block type baits confine multiple feedings into a self contained unit; they are more readily placed in strategic locations where legally permissible; bait deterioration from insects and molds is retarded.

FIRST GENERATION ANTICOAGULANTS

Warfarin, 3-(alpha-acetonylbenzyl)-4-hydroxycoumarin, the first of the marketed rodenticides, has relatively limited sales today. The more potent anticoagulants have replaced warfarin. Warfarin, however, remains one of the safest of all the anticoagulants. Where potential hazards to certain nontarget species are great, for example, the presence of dogs, warfarin baits would be the least apt to create problems. Birds generally are quite resistant to warfarin compared to some of the other nontarget species. Since warfarin has

the shortest half-life of any anticoagulant, the potential for secondary hazards to predators and scavengers is greatly reduced. While warfarin was once used for several of our native field rodents, none are registered today. All current warfarin baits are registered for commensal rodents. A soluble sodium salt of warfarin is also available for water baits.

Pival® (pindone), 2-pivalyl-1, 3-indandione, is also one of the early anticoagulants. Like warfarin, its use presents fewer potential hazards to nontarget species. The sodium salt, pivalyn, is one of the most popular for preparing water baits. Pival had some insect and mold inhibiting characteristics which give it some advantages over warfarin. The registrant failed to respond to EPA's re-registration data call-in, therefore the registration of this material was suspended in 1994.

Fumarin (coumafuryl), 3-(alpha-acetonylfurfury)-4-hydroxycoumarin, was another early anticoagulant. The registration of this material was suspended in 1988. In EPA's re-registration process, so many new data were required that Union Carbide apparently decided such expenditures could not be justified on the basis of their market. The loss of fumarin did not have a significant impact on rodent control, but it represents the loss of the first of the safer anticoagulants, which is disturbing. Since then, we have also lost PMP (valone®), 2 isovaleryl-1,3-indandione. PMP, for the most part, was used in tracking powders for commensal rodents. Although fumarin was used in field rodent control early on, its loss and the loss of PMP are of no consequence to current field rodent control as presently practiced.

Diphacinone, 2-diphenylacetyl-1,3-indandione, was first of the first generation anticoagulants which was substantially more toxic to rodents. Because of this increased toxicity, commensal rodent baits could be prepared at the concentration of 0.005% as opposed to the 0.025% commonly used for warfarin, fumarin and pival. This increased toxicity and longer half-life made diphacinone more effective than the earlier materials for the control of field rodents, e.g. meadow voles and group squirrels. Concentrates were readily available for bait formulation and remain so today. Diphacinone continues to be used for commensal rodents and is important for ground squirrel control in the west and for meadow vole control in the apple industry. Depending on the use and application method, diphacinone is sometimes prepared at double the normal strength, i.e. 0.01%. Its increased toxicity and prolonged half-life, as would be expected, brings with it potential increased risk in both primary and secondary poisoning to nontarget species. Dogs are especially sensitive to diphacinone. Other than for dogs, potential hazards are considered minimal and most can be substantially mitigated through use practices.

Chlorophacinone, 2-[(p-chlorophenyl) phenylacetyl]-1,3-indandione, came onto the market in the 1960's and is similar to diphacinone in its toxicity. Some rodent species may be slightly more sensitive to chlorophacinone than diphacinone (e.g. deer mice). Chlorophacinone was initially marketed for commensal rodents, like all of its predecessors. Later it was registered for several field rodents and continues to be used in substantial amounts, especially for ground squirrel and meadow vole control. Chlorophacinone is the first anticoagulant to be marketed as an oil concentrate, which greatly facilitated the preparation of commensal rodent baits. Dry concentrates of 0.1% and 2.0% are also available; the latter is most often used in preparing field rodent baits in California. The potential primary and secondary hazards associated with chlorophacinone are considered

relatively minimal, but do exist. Such hazards are considered comparable to those of diphacinone.

SECOND GENERATION ANTICOAGULANTS

Brodifacoum is the most potent rodenticide currently available for commensal rodents. It is available in 0.005% pellet formulations and in wax blocks. It will control warfarin resistant rats and mice. Because of its acute toxicity, a lethal dose can be obtained in a single feeding, although death is delayed for 4 to 6 days. Research has demonstrated that brodifacoum is very effective for a number of field rodents, however, in California, it is only registered for commensal rodents in and around buildings. It is marketed under trade names such as Talon®, Havoc® and Weather Block®.

Bromadiolone, is commercially available in a variety of 0.005% bait formulations, including paraffinized pellets and wax blocks. It is not quite as toxic to rodents as brodifacoum; however, these differences are of a more academic than practical nature. Bromadiolone will kill warfarin resistant rats and mice; however, there are some isolated cases which suggest that house mice may be developing a resistance to bromadiolone. Experimentally, bromadiolone has been evaluated for several field rodent species with promising results. Bromadiolone, however, is not registered for any field rodent. It is marketed under a number of trade names (e.g. Maki® and Contrac®) for commensal rodent control in and around buildings.

Difethialone, is a new second generation anticoagulant belonging to the chemical family called hydroxy-4 benzothiopyranones developed by Lipha Tech. Technically, it is the thio derivative of brodifacoum. From information available, it appears to be more toxic than bromadiolone and more closely comparable to brodifacoum. Its toxicity to both rats and mice has led the manufacturers to pursue registration for baits containing only 0.0025% active ingredient. Difethialone is currently registered for commensal rodent control.

Pharmacology: In very basic terms, anticoagulants have two actions; they reduce the clotting ability of the blood and cause damage to the capillaries (tiny blood vessels). Death results from consuming a sufficient quantity of bait which may be over a span of several days, or as short a period as one day for the newer second generation anticoagulants. With few exceptions, death is apparently painless.

The rate of blood clotting is gradually reduced and blood is lost until death occurs. Animals killed by anticoagulants show an extreme lack of color of the skin, muscles, liver and heart. In addition, evidence of internal hemorrhage may be found in any part of the body, but it is generally in the abdominal cavity and, to a lesser extent, in the thoracic cavity. The blood that remains in the heart and vessels is grossly thin and forms a poor clot or no clot. The animal exhibits increased weakness due to blood loss; appetite and body weight is not specifically affected. Hematomas are common.

First generation anticoagulants, as formulated in baits, must be fed upon several times over a period of days. With field rodents, diphacinone and chlorophacinone feeding does not have to be on consecutive days, but several feedings should take place within a 10 day

period with no more than about 72 hours between feedings. For this reason it is important that adequate bait is available at all times to satisfy the repeated feedings and to achieve a high degree of control. With warfarin, which has a shorter half-life, the feedings must generally be at least every other day, but it is preferable that they be daily. The blood of animals that feed infrequently or run out of bait to feed upon will revert back to normalcy.

As previously indicated, most anticoagulants are more toxic to rodents if consumed in small amounts over several days, and thus the sum of the chronic doses is much lower than the single acute lethal dose, especially for the first generation anticoagulants. This generalization is less applicable for the newer, second generation anticoagulants where there may be little difference between the acute and chronic lethal dose.

Brodifacoum and bromadiolone can produce a high percent of mortality in some rodent species with a single overnight feeding, although multiple feeding of lesser amounts is equally effective. This allows their use to be similar to that used for acute rodenticides, that is, the application of relatively small amounts of bait, offered one time only.

Toxicity: For reasons yet unexplained, anticoagulants vary in their toxicity to different species. With rare exceptions in other parts of the world, rodents, in general, are very susceptible to all anticoagulants, although house mice are usually less susceptible than Norway rats to most anticoagulants. Toxicity information for specific nontarget animals is wanting; data are currently available for only a few species. Toxicity data is made more complex by the fact that the sum of daily chronic and acute LD_{50} values may be dramatically different and the sum of 5-day chronic values different from 10-day chronic data.

Anticoagulant generalizations must be made with care, because many exceptions occur. For example, birds generally are very resistant to high doses of warfarin, but this does not hold true for other common anticoagulants, especially for the second generation group. Swine, as another example, are more susceptible to warfarin than they are to diphacinone, while dogs are quite the reverse.

Accidental deaths to humans from anticoagulants are extremely rare; however, it is possible for death to occur in those with suicidal intent. The fact that vitamin K_1 is an effective antidote for all anticoagulants, makes accidental ingestion cases treatable in humans and domestic animals. In the more serious cases, K_1 treatment may be augmented by blood transfusion.

CHLOROPHACINONE

Species	LD50 Acute Oral mg/kg
Deer mouse	0.49
Norway rat	5.0
Roof rat	15.0
Vampire bat	7.5
Mallard duck	> 100.0

Ring-necked pheasant	> 100.0
Red-winged blackbird	430.0

DIPHACINONE

Species	LD50 Acute Oral mg/kg
Rat	~3
Dog	0.8-7.5
Coyote	0.6
Cat	14.7
Pig	150.0
Rabbit	35.0

Anticoagulants are generally well accepted in baits since they are used in very low concentrations. They produce no bait and/or toxic shyness. Because of the slow action of anticoagulants, rodents are incapable of associating their illness with the bait. The lack of bait shyness means that the timing of ground squirrel control to correlate with the earliest date when they are feeding exclusively on seed is less critical than with acute toxicants which produce bait shyness. Baiting with anticoagulants in bait stations can often be started before ground squirrels have completely switched from eating green forage to eating seed. Pre-baiting provides little or no control advantage with anticoagulant baits, but pre-bait placed in bait stations until the squirrels become accustomed to feeding in the stations may be economically sound for it will generally reduce the amount of bait needed later to achieve control.

Primary Hazard to Nontarget Species: Potential hazards to nontarget species may result when some nontarget animal gains access to and consumes a substantial amount of bait. Prior to the registration of any rodenticide, such possibilities are extensively reviewed. Only those rodenticides and application methods which do not create significant hazards are permitted. Potential primary hazards are mitigated through the concentration of rodenticide in the bait, the kind and type of bait used, extra precautions such as dying the bait and, probably the most important, the method and rate of application. The use of bait stations, which exclude animals larger than the target species, is one recommended procedure for offering anticoagulant bait to ground squirrels and has even been used for meadow voles. Thinly broadcasting the bait is another acceptable method for those rodent species that have the ability to forage thoroughly or widely in the vicinity of their burrow systems. In order to mitigate some anticipated problems, anticoagulant rabbit baits, if need be, can be exposed only from dusk to dawn. When anticoagulant baits are applied according to directions, only rarely will a nontarget animal be affected. The applicator must, however, assess every control situation to ascertain potential problems and, if an atypical situation presents itself, bait application may have to be abandoned in favor of some alternative control approach such as burrow fumigation, trapping or cultural control.

Secondary Toxicity: Potential secondary toxicity cannot be ruled out because in some of the laboratory or caged studies certain predators and scavengers have been affected by feeding on poisoned rodents since the rodent carcasses contain some anticoagulant in its tissues. Since the amount is small, multiple feedings are usually required. The sensitivity of the predator or scavenger species will also influence the potential secondary poisoning risk. The interpretation of laboratory studies must be done with great care since under field conditions many factors relative to feeding behavior come into play for both the target and nontarget species which often effectively negate much of such hazard. With some 40 years of anticoagulant use of field rodents, the preponderance of scientific evidence suggests that secondary toxicity hazards of chlorophacinone and diphacinone under field conditions are minimal when baits are applied according to label directions. The larger body size (i.e. weight) of the predator or scavenger compared to the rodent prey, coupled with the dilution factor associated with the predator's large feeding range, and the fact that its diet would normally represent a composite of contaminated and non-contaminated prey items, greatly reduces the odds of a lethal threshold ever being achieved. Practitioners should not become complacent because of past experiences, and should be on a constant alert for suspected secondary poisoning. Burying or disposing of poisoned carcasses can greatly reduce secondary poisoning risks. Suspected incidents should be reported immediately to the proper authorities.

Species at Risk: In most situations, relatively few species are at risk. Those at the greatest potential risk are anticoagulant susceptible seed eating rodents smaller in size than the target species and living or feeding within the same area. The dominance of some of our pest rodents over others precludes extensive overlaps of home ranges of certain other species, but some overlap does occur. For example, deer mice or harvest mice may occur in the same vicinity as ground squirrels and are, therefore, at risk. While the ground squirrel may be dominant, confrontation is avoided by the fact that deer mice and harvest mice are mostly nocturnal and ground squirrels are entirely diurnal.

Anticoagulant Resistance: Resistance to first generation anticoagulants first appeared in Scotland in 1958, then in other parts of the United Kingdom and elsewhere in Europe and in the United States. Resistance has been identified in Norway and roof rats, and in house mice. The resistance problem in rats has received more study in the U.S. than has resistance in house mice. Most authorities agree that house mouse resistance is far more prevalent than is rat resistance. Animals resistant to warfarin show resistance, at least to some degree, to all other first generation anticoagulants.
Since brodifacoum and bromadiolone are available to use for rat and mouse control in and around buildings in place of the early anticoagulants, resistance is not as serious as it was when first discovered. However, some resistance to the second generation anticoagulants has been identified. Because of this, good resistance management practices must be adopted. Fortunately, for commensal rodent control, we also have cholecalciferol and bromethalin as useful alternatives to the anticoagulants.

Anticoagulant resistance has recently shown up in meadow voles populations in commercial artichoke fields. In this case, annual treatment with one anticoagulant was practiced for over 20 years. Resistance management needs to be part of any anticoagulant, or other rodenticide, treatment program. Careful use of all products is essential. Prophylactic use of anticoagulants is discouraged. If necessary, use non-toxic bait to

monitor for rodent present. Switch to toxic baits when evidence of feeding occurs.

Statement of Practical Treatment

If swallowed: Call a physician or Poison Control Center (CPCS) immediately, 1-800-222-1222. Drink one or two glasses of water and induce vomiting by touching back of throat. Do not induce vomiting or give anything by mouth to an unconscious person.

Note to physician: Intramuscular and oral administration of Vitamin K_1, combined with blood transfusions, is indicated as in the case of hemorrhage caused by overdoses of biohydroxycoumarin (dicumarol).

AVITROL

Use: Avitrol® is a bird management chemical registered for blackbirds, cowbirds, starlings, grackles, house sparrows, and feral pigeons as a flock-frightening repellent. Treated bait is usually 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 perform aerial distress displays and often frighten the other birds in the flock and cause them to leave.

Avitrol® products are for use by or under the supervision of government agencies or pest control operators. They are not for sale to the public. A Restricted Materials Permit and necessary certification shall be obtained from the county agricultural commissioner prior to the purchase, storage or use of this material.

History: Avitrol® is the registered trademark of the Avitrol Corporation for the chemical 4-aminopyridine. The synthesis of this chemical was first reported by Koenigs and Gredner in 1931, but its unique action on birds was not reported until 1964 by Goodhue. Its utility for controlling damage by birds in some situations was demonstrated within the next year by Goodhue and Aumgartner.

Characteristics: The chemical name for Avitrol® is 4-aminopyridine. It is a white crystalline, odorless, water soluble material; is stable in light, and melts at 158 C.

Pharmacology: Avitrol® is an acutely toxic substituted pyridine. 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.

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

In mammals, the following symptoms are produced: hyper excitability, salivation, tremors, lack of muscular coordination, convulsions, cardiac or respiratory arrest, and death. Initial effects are usually noted in 10-15 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.

In red-wing blackbirds, a lethal dose of Avitrol® is necessary to produce distress behavior.

In one test, secondary poisoning did not occur when a cat was fed 51 sparrows in 4 days that were killed with 19 times the lethal dose of Avitrol®.

TOXICITY TABLES

MAMMALS

Species	Route*	Carrier	LD_{50}(mg/kg)
White Rat, *Rattus Norvegicus*	po	Water	32-32.5 (HC1)**
	po	Water	28 (CH1)
	po	Water	20
	ip	Water	6.50
White Mouse, *Mus musculus*	ip	Water	14.7
	ip	Water	10
	ip	Water	9
	sc	Water	5
	iv	Water	7
Hog, *Sus scrofa*	po	Grain	17.8 (HC1)
Dog, *Canis familiaris*	po	Grain	11.9
	po	Capsule	4.0
	po	Water	3.7
	im	Water	3.5
Rabbit, *Oryctolagus, cuniculus*	dm	Water	327

BIRDS

Species	Route*	Carrier	LD_{50}(mg/kg)
Mallard, *Anas platyrhynchos*	po	Propylene glycol	4.2
Sparrow Halk, *Falco sparverious*	po	Propylene glycol	5.6
Domestic Chicken, *Gallus gallus* (2-3 wk)	po	Water	15(HC1)
Ring-necked Pheasant, *Phaisianus colchicus* 4 wk old	po	Propylene glycol	7.5
	po	Propylene glycol	5.6
Ring-billed Gull, *Larus delawarensis*	po	Water	8(HC1)
Common Pigeon, *Columba livia*	po	Water	20(HC1)
	po	Propylene glycol	7.5
Mourning Dove, *Zenaiduva macroura*	po	Propylene glycol	8.1
Robin, *Turdus Migratorius*	po	Propylene glycol	4.2
Starling, *Sturnus vulgaris*	po	Water	14(HC1)
	po	Pellet	< 6
	po	Propylene glycol	4.9
Black-billed Magpie, *Pica pica*	po	Propylene glycol	2.4
Common Crow, *Corvus brachyrhynchos*	po	Propylene glycol	2.4

Yellow-billed Magpie, *Pica nuttali*	po	Propylene glycol	2.4
Boat-tailed Grackle, *Cassidix mexicanus*	po	Propylene glycol	3.2
Brown-headed Cowbird, *Molothrus ater*	po	Propylene glycol	4.2
Common Grackle, *Quiscalus quiscula*	po	Propylene glycol	2.4
Red-winged Blackbird, *Agelaius phoeniceus*	po	Water	8.5
	po	Water	3.2(HC1)
	po	Propylene glycol	2.4
	im	Propylene glycol	2.4
Shiny Cowbird, *Molothrus bonariensis*	po	Propylene glycol	< 1.0
Tricolored Blackbird, *Agelaius tricolor*	po	Propylene glycol	4.2
House Finch, *Carpodacus mexicanus*	po	Propylene glycol	5.6
Golden-crowned Sparrow, *Zonotrichia atricapilla*	po	Propylene glycol	5.6
House Sparrow, *Passer domesticus*	po	Propylene glycol	7.5
	po	Water	4.0
	po	Water	3.8
	po	Water	3.6
	dm	Acetone	> 100
White-crowned sparrow, *Zonotrichia leucophrys*	po	Propylene glycol	5.6

Table Key
po = per os (by mouth),
im = intramuscular;
ip = intraperitioneal
iv = intravenous;
sc = subcutaneous;
dm = dermal
**HC1 = Hydrochloride salt

Statement of Practical Treatment

If swallowed: If the patient is unconscious, maintain breathing and heart beat (CPR: cardiopulmonary resuscitation). Contact your local California Poison Control Center, hospital or physician immediately, 1-800-222-1222.

If patient is conscious: Induce vomiting by stimulating the back of throat with finger. Never give anything by mouth to an unconscious person. Contact your Poison Control Center, hospital or physician immediately.

If in eyes: Flush with plenty of water. Get medical attention if irritation persists.

GAS CARTRIDGE

Use: Gas cartridges are devices designed to give off carbon monoxide and other poison gases and smoke when ignited. They are used as a rodent fumigant in burrows.

History: Gas cartridges were developed by the former Bureau of Biological Survey more than 50 years ago. They are currently manufactured and supplied by the Pocatello Supply Depot of the USDA-APHIS-ADC Idaho State Office. Other types have been developed, manufactured and sold by private firms.

Characteristics: Gas cartridges are made of cardboard cylinders filled with sodium nitrate, charcoal, and inert ingredients. The contents are ignited with a fuse provided. Care should be taken to avoid fire hazards at locations used. For example, dry grasses on rangeland, and methane or natural gas which may be present in or around structures.

Pharmacology: When ignited, smoke and toxic gases are emitted. Carbon monoxide gas, similar to that from an automobile exhaust, is a major product. In humans, 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 about the same on animals.

Toxicity: Two hundred parts per million of carbon monoxide in inhaled air may produce symptoms of poisoning in a few hours, and 100ppm can cause unconsciousness and death in four hours.

Statement of Practical Treatment

Remove victim to fresh air and keep him lying down. If breathing has stopped, apply artificial respiration. Use oxygen inhalator only at the direction of a physician. Call a physician immediately.

Gas Cartridges Directions: With ¼ inch screwdriver blade or large nail punch a hole in the end cap and shove deeply into the cartridge to loosen material. Insert ¾ of the fuse into hole with ¼ projecting from cartridge for lighting. This procedure assures proper lighting of the cartridge, as they are designed to burn slowly, in order to create carbon monoxide gas and minor amounts of other gases for exterminating purposes. The cartridge is therefore difficult to ignite and it should have as much fuse as possible inside for ignition.

Preparing the cartridge as described is safe and no gas is created until the cartridge is burning steadily. Place the prepared cartridge in the burrow hole, light the fuses, and when cartridge starts to burn, shove deeper into the hole with a stick or rod. Then quickly cover hole with a shovel full of soil or a clump of sod. Nearby burrows might begin to smoke. If so, cover them with soil or treat them if the burrow system is large.

STRYCHNINE

Use: Strychnine is currently registered for underground use only for controlling pocket gophers. Any agricultural use requires a Restricted Materials Permit that can be obtained through the agricultural commissioner. . Historically, strychnine was used for controlling many birds and mammals, including skunks and coyotes. Since 1988 these uses have been banned. Only underground uses are allowed.

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 2.0-2.7% total alkaloid found in the seeds. The seeds were used for killing dogs, cats and birds in Europe at least as early as 1640.

Characteristics: Strychnine, a white crystalline powder, is available in an alkaloid form. It has a characteristic bitter taste. Strychnine alkaloid 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.

A number of commercial baits are available for pocket gopher control. Pure strychnine is only available to commercial bait formulators.

Pharmacology: Strychnine reacts the quickest (excluding fumigants) of the commonly used rodenticides. It is not cumulative, not absorbed through normal intact skin, has a very slight odor, has very high toxicity, and is somewhat variable in action against target animals. Strychnine enters the blood very rapidly and acts on the central nervous system. The time of action depends upon the condition of the stomach, that is, whether empty or full and the nature of the food present. Animals with little in their stomach react more quickly to strychnine than those that have fed recently. Symptoms may appear from five to thirty 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. The required below ground applications limit the potential for secondary poisoning.

Toxicity

Primary - In general, strychnine is somewhat less toxic to gallinaceous birds than other life forms. LD_{50} values range from a low of 0.70 and 0.75 mg/kg for coyotes and desert kit fox; from 1.5 mg/kg for black tailed prairie dogs, to 27.0 mg/kg for nutria; and from 16.0 mg/kg for chuckar partridge to 24.7 mg/kg for ring-necked pheasants. LD_{50} for mallard ducks, 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 are rats.

Secondary - Strychnine is not assimilated into tissues or bone as in the case with Compound 1080; however, residues in the gastrointestinal tract of animals poisoned with lethal doses are known to be potentially hazardous if the gastrointestinal tract is consumed. Secondary poisoning of raptors and mammalian predators is relatively rare.

Horses and hogs show no hesitation in eating strychnine baits. Cattle and sheep are more reluctant to accept baits. Geese and ducks show no reluctance in eating strychnine baits. However, gallinaceous game birds and domestic poultry are less susceptible to strychnine than most rodents.

Species	Strychnine Alkaloid (Approximate Oral LD50 in mg/kg)*
Desert kit fox	0.75
Coyote-dog	0.70
California ground squirrel	19.90
Black-tailed prairie dog	1.50
Northern pocket gopher	8.30
Banner-tailed kangaroo rat	3.70
Meadow vole	6.80
California meadow mouse	22.10
White rat (male)	14.00
White rat (female)	5.80
Norway rat (wild)	12.00
Black rat	10.10
Polynesian rat	6.80
Nutria	27.00
Black-tailed jackrabbit	4.40
Mule deer	17.0-24.00
Mallard duck	2.90
Canada goose	4.00
Golden eagle	-5.00
Coturnix quail	22.60
Chuckar	16.00
Pheasant	24.70
Mourning dove	over 5.12
Pigeon	21.30
English sparrow	4.18
Robin	over 10.00
House finch	5.60
Bullfrog	2.21

*The values given under this heading are calculated projected theoretical values based upon known toxicity data.

Statement of Practical Treatment

If swallowed: Call a physician or Poison Control Center (CPCS) immediately, 1-800-222-1222. If less than ten (10) minutes have passed since the poison was taken, give one or two glasses of water and induce vomiting by touching back of throat with finger. Repeat until vomit fluid is clear. Have patient lie down in quiet, darkened room and keep warm and quiet.

Do not induce vomiting or give anything by mouth to an unconscious person. If inhaled: remove victim to fresh air. Apply artificial respiration if indicated. If on skin: remove contaminated clothing and wash affected areas with soap and water. If in eyes: flush eyes with plenty of water. Get medical attention if irritation persists.

ZINC PHOSPHIDE

Use: Zinc phosphide has been used on grain baits and bait blocks to successfully control meadow voles, pocket gophers, ground squirrels, Norway rats, Polynesian rats, cotton rats and nutria. In some areas of California, zinc phosphide baits have been partially or completely rejected by ground squirrels and meadow voles and, at times, control has been erratic. Commercial baits are available for a variety of rodent pests.

A restricted Use Permit and necessary certification shall be obtained from the county agricultural commissioner prior to the purchase, storage or use of the material.

History: Zinc phosphide appears to have been first synthesized by Margral in 1740 and was first used as a rodenticide by the Italians in 1911-12. Extensive use of zinc phosphide in the United States did not occur until 1942-43 when the availability of strychnine became uncertain due to World War II.

Characteristics: 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. 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 (garlic-like), this characteristic seems to attract rodents, particularly rats, and apparently makes the bait unattractive to some other animals. Bait/toxic shyness commonly occurs in rodents receiving a sublethal dose on first exposure.

In general, zinc phosphide is less toxic than "1080" or strychnine. It is the slowest acting of the commonly used acute rodenticides.

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 some protection against moisture, and, therefore, may increase its field stability. Under field conditions, zinc phosphide baits may remain toxic for several months until eroded by weathering or decomposition of the carrier or the grain is removed by insects. In one instance, zinc phosphide treated bait, exposed in the field for two to three months and ten to twelve inches of rain, continued to maintain some toxicity.

When zinc phosphide is dusted onto wet baits sometimes used in rat and vole control, such as cubed fresh fruits and vegetables, it probably breaks down within a few days and the bait themselves soon lose their attractiveness.

On moist soil, especially acid soils, zinc phosphide breaks down 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 indicating that hazards exist via this route when grain baits are applied for rodent control in crop situations. Zinc phosphide is registered for certain crop uses.

Pharmacology: When zinc phosphide comes into contact with dilute acids in the stomach, phosphine (PH_3) is released and this substance 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 exposure is prolonged for several days, intoxication occurs similar to that observed with yellow phosphorous in which the liver is heavily damaged. The surface of the liver is spotted and discolored. Prolonged exposure to phosphine can produce chronic phosphorous poisoning. To this extent, zinc phosphide may possess some characteristics of cumulative toxic materials. However, bait/toxic shyness prevents this from occurring when used in rodent control.

Early symptoms of zinc phosphide poisoning are: nausea, vomiting (yielding black stomach contents and 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 three 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, and most other rodents, show no sign of distress until a short terminal death agony occurs. A large percent of rodents such as ground squirrels will die in their burrows.

Toxicity: Zinc phosphide is poisonous to some degree to all animals. The odor and dark color of zinc phosphide (supposedly safety factors) may be of little importance 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 contacting it.

Since zinc phosphide apparently is not stored in the muscle or other tissues of poisoned animals, there is no true secondary poisoning with this rodenticide. Other animals can be affected if they eat enough of the gut content of rodents recently killed with zinc phosphide. Such secondary toxicity is very rare and, in fact, zinc phosphide is considered one of the safest rodenticides from a secondary point of view.

Use care in handling zinc phosphide concentrate and treated bait. If zinc phosphide baits are prepared in the open air, phosphine generated from grain bait offers little hazard. When quantities of bait are prepared within a bait mixing plant, safeguards against continued exposure to low concentrations of phosphine 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 semi-solid, are used in some preparations and since phosphorous is soluble in certain fatty oils, the poison may be absorbed in small amounts through the skin. Continued exposure

to phosphorous absorption may result in toxic manifestations at some later time.

Toxicity Tables

The following toxicity tests in Tables 1, 2, 3 and 4 were conducted at the Wildlife Investigations Laboratory, California Department of Fish and Game.

Acute toxicity data was obtained from tests utilizing both the technical poison and "grain bait" formulations. Chronic toxicity information was derived from tests of grain bait formulations only. These tests involved a single feeding daily for five consecutive days. The dosage level in the grain bait experiments was determined by the number of kernels administered. Each bird in each series received the same number of kernels.

TABLE 1

Zinc Phosphide Acute Toxicity mg/kg			
Species	MLD	LD_{50}	LD_{100}
Snow goose	5-10	8.75	5-10
White-fronted goose	0-5	7.5	10-15
Mallard	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

TABLE 2

Zinc Phosphide Treated Grain Bait Acute Toxicity			
1%			
Species	MLD	LD_{50}	LD_{100}
Snow goose	200-300K	260K	300-400K
White-fronted goose	200-300K	310K	300-400K
Mallard	> 100K	< 100K	< 100K
Pheasant	> 75K	--	--
Quail	0-15K	17.5K	15-25K
Dove	--	--	--

Zinc Phosphide Treated Grain Bait Acute Toxicity			
2%			
Species	MLD	LD_{50}	LD_{100}
Snow goose	--	--	--

White-fronted goose	--	--	--
Mallard	0-50K	--	0-50K
Pheasant	> 50K	--	--
Quail	0-5K	7.5K	5-15K
Dove	10-20K	--	30K

TABLE 3

Zinc Phosphide Treated Grain Bait Chronic Toxicity			
1%			
Species	MLD	LD_{50}	LD_{100}
Snow goose	50-100K	115K	100-200K
White-fronted goose	50-100K	--	< 100K
Mallard	25-50K	57.5K	50-100K
Pheasant	25-50K	42.5K	50-75K
Quail	> 10K	--	10K
Dove	--	--	--

Zinc Phosphide Treated Grain Bait Chronic Toxicity			
2%			
Species	MLD	LD_{50}	LD_{100}
Snow goose	--	--	--
White-fronted goose	--	--	--
Mallard	0-25K	--	< 25K
Pheasant	--	--	--
Quail	--	--	--
Dove	--	--	--

TABLE 4

Species	Approximate LD_{50} in mg/kg
Desert kit fox	93.00
Dog and cat	40.00
California ground squirrel	33.10
Black-tailed prairie dog	18.00
Northern pocket gopher	6.80
Banner-tailed kangaroo rat	8.00

Deer mouse	40.50
Meadow vole	18.00
California meadow vole	15.70
Muskrat	29.90
Wood rat (LD_{100})	25.00
White rat	55.50
Black rat	21.00
Norway rat (wild)	27.00
Roof rat	2.90 + 40.50
Polynesian rat	23.00
Nutria	5.55
Black-tailed jackrabbit	8.25
Cow	50.00
Mallard	13.00
Snow goose	8.80
White-fronted goose	7.50
California quail	13.50
Pheasant	8.80
Mourning dove	34.20
Partridge	26.70
Pheasant	8.80-26.70
Red-winged blackbird	23.70
Chicken	20.00-30.00
Man (estimated MLD)	40.00

Table Key
MLD= Level at which first losses occurred
LD_{50} = Level of toxicant fatal to half the animals
LD_{100} = Level 100% mortality resulted
K= Kernels of toxic grain
The symbol < means the LD is less than the quoted figure
The symbol > means the LD is greater than the quoted figure

Statement of Practical Treatment

Any person applying zinc phosphide products and experiencing signs and symptoms such as nausea, abdominal pain, tightness in the chest, or weakness, should be seen by a physician immediately.

If Swallowed: Immediately call Poison Control Center (CPCS) or physician, 1-800-222-1222 or transport the patient to the nearest hospital. Do not drink water. Do not administer anything by mouth or make the patient vomit unless advised to do so by a

physician.

If In Eyes: Flush with plenty of water. Get medical attention if irritation persists.

If On Skin: Remove contaminated clothing and wash affected areas with soap and water.

REFERENCES AND ADDITIONAL READING

Baker, Rex O. 1992. Exposure of Persons to Phosphine Gas from Aluminum Phosphide Application to Rodent Burrows. Proc. Vertebrate Pest Conference 15:312-321.

Bennett, Gary W., et al. 1988. Truman's Scientific Guide to Pest Control Operations. 4th Edition. Purdue University/Edgell Communications Project. 495 pp.

Buckle, A.P. and R.H. smith (Eds.) 1994. Rodent Pests and Their Control. CAB International, Wallingford, UK. 405 pp.
Hayes, Wayland J., Jr. 1982. Pesticides Studied in Man. Williams & Wilkins, Baltimore. 672 pp.

Hone, J. and H. Mulligan. 1982. Vertebrate Pesticides. Department of Agriculture, New South Wales, Science Bulletin 89. 130 pp.

Hudson, Rick H., Richard K. Tucker, and M.A. Haegele. 1984. Handbook of Toxicity of Pesticides to Wildlife. USDI, Fish and Wildlife Service, Resources Publication 153. Washington, D.C. 90 pp.

Jacobs, William W. 1990. Required Use of Protective Bait Stations in the U.S Proc. Vertebrate Pest Conference 14:36-42.

Jacobs, Willlian W. 1992. Vertebrate Pesticides No Longer Registered and Factors Contributing to Loss of Registration. Proc. Vertebrate Pest Conference 15:142-148.

Kaukeinen, Dale. 1982. A Review of the Secondary Poisoning Hazards Potential to Wildlife from the Use of Anticoagulant Rodenticides. Proc. Vertebrate Pest conference 10:151-158.

Marsh, R.E. 1988. Relevant Characteristics of Zinc Phosphide as a Rodenticide. Pp. 70-74. In: Proceedings, Eighth Great Plains Wildlife Damage Control Workshop, Rapid City, South Dakota, April 28-30, 1987. USDA Forest Service, Rocky Mountain Forest and Range Experiment Station, Fort Collins, Colorado. General Technical Report RM-154. 231 pp.

Marsh, R.E. 1988. Current (1987) and Future Rodenticides for Commensal Rodent Control. Bulletin of the Society of Vector Ecologists, 13(1):102-107.

Marsh, Rex E. 1992. Reflections on Current (1992) Pocket Gopher Control in California. Proc. Vertebrate Pest Conference 15:189-195.

O'Connell, Ross A. and Jerry P. Clark. 1992. A Study of Acrolein as an Experimental Ground Squirrel Burrow Fumigant. Proc. Vertebrate Pest Conference 15:326-329.

Record, C. Raymond and Rex E. Marsh. 1988. Rodenticide Residues in Animal Carcasses and Their Relevance to Secondary Hazards. Proc. Vertebrate Pest Conference 13:163-168.

Salmon, Terrell P., W. Paul Gorenzel and Walter J. Bently. 1982. Aluminum Phosphide (Phostoxin) as a Burrow Fumigant for Ground Squirrel Control. Proc. Vertebrate Pest Conference 10:143-146.

Timm, Robert M. (Ed.). 1983. Prevention and Control of Wildlife Damage. Nebraska Cooperative Extension Service, Lincoln, NE. 640 pp.