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Toxicology of Rodenticides And Their Relation to Human Health

Frank S. Lisella*, Ph.D., Keith R. Long†, Ph.D. and Harold G. Scott‡, Ph.D.

Many sanitarians and other environmental health workers are directly involved with programs related to rodent control; therefore, they should have a relatively thorough knowledge of the chemicals used against rodents. The purpose of this paper is to present the *salient toxicological characteristics* of some of the rodenticides of public health importance. Because of the length of this subject, the information will be presented in two separate articles.

Rodents, Rodenticides and History

The history of our civilization is replete with references to man's continual battle to eliminate or reduce populations of commensal rodents. Although precise evidence is lacking, it has been postulated that problems with rodents occurred when cave-dwelling Paleolithic man stored the products of the hunt. With the advent of Mesolithic seed storing and Neolithic agriculture, primitive man faced increased pilfering by rodents. Excessive mouse populations were reported in the literature of the Mediterranean and the Near East. So feared were these animals whose numbers overwhelmed the land that the mouse became part of religious ceremony and effigy.[1] Chinese literature is reported to contain similar references to excessive mouse populations. The early records of European rodent problems are a voluminous mass of separate and incomplete documentations. It is known, however, that the Black Death (bubonic plague) wiped out a large population in Europe in the 14th Century. This disease gradually retreated to a few endemic centers, one of which was in inner China. From here in the 1880's, the plague began to creep down to the seaboard and was spread by rats and their fleas to other parts of the world.

Infestations with rodents were documented in 14 major areas in France between 1792 and 1899. Later rodent populations of massive proportions on a country-wide basis were noted between 1900 and 1935. An infestation in 1801 was noteworthy in that it almost wiped out the crops in many parts of Western France. The disturbance created by this was sufficient to bring about a site visit by a commission of educated men appointed by the French Academy under the Revolutionary Government. In 1903, 4 million acres in France were ravaged and the crops thereon were injured or totally destroyed. In 1909, there were several large rodent infestations, but the largest began in 1912 and reached its peak the following year. In 1913, over 1,200,000 acres of land were attacked by rodents. Finally, after a loss of approximately 80 million francs, the French Government appropriated 750,000 francs for rodent control. Thus, chemical rodenticides, such as strychnine, zinc phosphide, barium carbonate, and arsenic oxide slowly came into use.[2] These chemicals are highly toxic to man, domestic animals and natural predators, thus investigators were continually on the lookout for safer materials. The high cost of these chemicals coupled with

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the fact that their use was not accompanied by environmental improvements led to only token control of rodent infestations. Research investigators, such as Pasteur, Perrien and others theorized that the oscillation in rodent populations was associated with the presence of some microbial agent in the animals. Pasteur isolated an organism which he labeled "Pasteurella" (probably tularensis) and inoculated wild rabbits in one region of France. The organism apparently multiplied and subsequently reduced the rabbit population. Later attempts to use the same procedure were blocked by authorities in that country. Despite this setback, however, Pasteur became the first known individual to employ biological agents as "pesticides."

In 1892 in Germany, Loeffler isolated an organism from an epidemic among white mice in his laboratory. He subsequently called this microbe *Bacillus typhimurium*. A field trial in Thessaly, Greece as a means of demonstrating the effectiveness of his technique in an area heavily populated with rodents met with varying degrees of success.

In 1893, Danysz, while working at the Pasteur Institute in France examined an epidemic among voles and wood mice. He was able to isolate an organism later called "Danysz Virus" from which propagations were made and used for rodent control in France and elsewhere. The years 1892-1914 were characterized by a great wave of experiments and the use of rat and mouse "viruses" as a means of controlling rodents biologically. New impetus was given to the need for the development of economical, efficient rodenticides during this period by another important discovery — that the disease known as Black Death or plague was carried by rats.

Rodents played an important role in the history of the United States. In the early history of the colony of Virginia, Captain John Smith recorded that in 1609:[1]

> In searching our cashed corn, wee found it halfe rotten and the rest so consumed with the many thousand rats increased from the ships, that we knewe not how to keepe that little wee had. This did drive us all to our wits ende; for there was nothing in the country but what nature afforded.

Later, in 1617, he wrote of rats again:[1]

> . . . they spared not the fruits of the plants, or trees, nor the very plants themselves, but ate them up. . . . At last it pleased God, but by what means it is not knowne, to take them away.

In 1866, the U. S. Government through its Bureau of Biological Survey became concerned with crop damage by rodents. A rodent infestation of massive proportions took place in the Humbolt Valley of Nevada in 1907, where alfalfa, hay, root crops, and potatoes were all devoured. The direct loss was thought to have reached $240,000. Shade trees and even Lombardy poplars were completely girdled. Sometime prior to 1914 sylvatic plague became known in California. This imposed new responsibilities on those individuals concerned with rodent control. Prior to this point, rodent poisoning campaigns attained local and temporary success by concentrating efforts on valuable agricultural lands. But, the spread of plague from the sylvatic environment to the urban environment became a matter of concern. One of the ways this disease could be checked effectively was through the use of chemical rodenticides (ectoparasite measures were not known at this time). Thus, the use of chemical rodenticides became widespread. Those compounds that met with limited success in Europe, such as arsenic, strychnine, barium carbonate, and zinc phosphide were used on a wide scale basis in this country. In 1935-1936, the U. S. Biological Survey distributed 900 tons of poison bait to 45 different states in the Union. In 1937-1938, the Survey supervised the use of rodenticides over 29 million acres of land.

Gradual improvements in our technology, living standards, disease prevention measures, and the continual "invasion" of human populations into rural areas placed high demands on the development of effective rodenticides. The

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role of the Public Health Service in rodent control programs, and the entry of the United States into World War II accelerated the need for effective chemical rodenticides. The compounds that were subsequently developed, and a few of the earlier rodenticides will be discussed in this paper, and the relationship of these chemicals to human health will be pointed out.

Categories of Toxicity of Rodenticides

For the purpose of this report, the rodenticides described herein will be categorized in accordance with the criteria established in the Federal Insecticide, Fungicide and Rodenticide Act. Thus, those compounds with a single dose LD_{50} of 50 mg/kg of body weight or less when administered orally to both male and female rats that have been fasted for 24 hours will be listed as "highly toxic." Compounds with an LD_{50} of between 50 and 500 mg/kg of body weight will be noted as "moderately toxic." Those with an LD_{50} of 500 to 5000 mg/kg of body weight will be noted as having a "low order" of toxicity. In the case of the "moderately toxic" and "low order" compounds, the LD_{50} values were arrived at by the same method as was established for the "highly toxic" materials.[3] Actually under the provisions of the act, a fourth category of toxicity is established (greater than 5000 mg/kg of body weight) but this category has little application to rodenticides and will not be discussed further.

Highly Toxic Rodenticides

1. *Thallium or Thallium Sulphate*

Thallium was first discovered by Crookes in 1861. It was later introduced as a therapeutic measure for the prevention of "night sweats" associated with tuberculosis. It was in this connection that it was observed that ingestion of this chemical caused a rapid and almost complete loss of hair.[4] Thallium acetate has been used at an oral dosage of 8 mg/kg to cause depilation in children. This practice led to serious poisoning in about 1 case in 20, and to death in some instances.[5] The chemical is readily absorbed through both the skin and gastrointestinal tract. It is distributed rapidly and nearly uniformly to all parts of the body. Elimination of the material occurs very slowly through the gastrointestinal tract and the kidneys. In renal clearance studies in the rabbit, it has been found that approximately 60 percent of the thallium filtered by the glomeruli is reabsorbed in the tubules.[6] There is no known antidote for thallium poisoning, but dithizon, a chelating agent has been reported successful in treating 5 out of 6 severely ill children.[7] It is generally felt that EDTA or BAL (Dimercaprol) are not useful in the treatment of thallium intoxication.

As early as 1920, 2 percent thallium sulphate was used in Germany as a rodenticide. "Zelio paste" and granules were quite popular at that time. In 1931, "Thalgrain" was distributed over 10 California counties to control ground squirrel infestations. This preparation contained about 1 percent thallium sulphate and caused an outbreak of thallotoxicosis in humans.[6] In the period between 1935 and 1955, 778 persons were reported to have been poisoned by this material, of which 46 (6%) died of thallotoxicosis. Of this number (778), 21 persons were poisoned by thallium-based rodenticides, and five of these individuals died. The vast majority of poisoning cases were due to the ingestions of thallium-based insecticides or to the ingestion of the material for therapeutic purposes.[4]

Thallium sulphate is a white crystalline substance which is stable in air and partly soluble in water. The chemically pure substance is apparently tasteless and odorless. The chief advantage of thallium sulphate as a rodenticide is its ready acceptance in water solutions of food baits by Norway rats, roof rats, and house mice. It is used at a 0.5 percent level in solid baits and a 2 percent concentration in liquid baits.

The material has been marketed under the brand names "Gizmo Mouse Killer," "GTA Rat Bait," "Martins Rat Stop," etc. The ingestion of these specific products by humans has been reported in the literature.[4] In recent years, efforts have been directed toward the addition of tar-

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tar emetic to thallium-based rodenticides as a safety factor. The effectiveness of this procedure is subject to question. The fact that the material is highly toxic, odorless, that it can penetrate the unbroken skin, and that it may cause "secondary poisoning" (i.e., animals eating poisoned rats or mice might in turn be poisoned) has led to the decline of this material as a rodenticide. In addition, many states have imposed restrictions to the effect that thallium-based rodenticides cannot contain more than 1 percent of the active ingredient. Further, in most instances, the material must be applied by a licensed pest control operator. The Federal Government imposed new restrictions on the use of thallium sulphate in 1965, when interpretation number 22 of the Federal Insecticide, Fungicide and Rodenticide Act was issued. This restricted the use of thallium to qualified Federal, State or local governmental personnel. This action virtually eliminated the use of this chemical as a rodenticide.

2. *Sodium Monofluoroacetate (Sodium Fluoroacetate, 1080)*

This is a white, oderless, and tasteless compound that is soluble in water. The chemical was originally synthesized in 1944 by chemists at the Department of the Interior's Fish and Wildlife Research Center in Denver, Colorado.[8] The material is fast acting, usually producing symptoms in rats in 20 minutes or less and killing in 1 to 8 hours. The poison, which has an LD_{50} of 3-7 mg/kg, is apparently not cumulative, and rats that survive sub-lethal doses display neither aversion to, nor serious tolerance of the material. Because of the possibility of "secondary poisoning," the use of 1080 in solid bait materials is not recommended. It should be used only in liquid concentrations as the dosage can be more accurately controlled and the containers more easily recovered. The use of this material is restricted to licensed pest control operators, and it is currently labeled for use by these individuals only. The recommended concentration of 1080 in water is about 0.3 percent or 12 grams per gallon of water. Nigrosine, a black water-soluble dye is usually incorporated in the powder by the manufacturer as a safety measure. If the solution evaporates, however, the remaining residue is stable and highly poisonous.

Human poisoning usually begins with nausea and apprehension about two hours after the ingestion of the material. Although the chemical is not absorbed through the unbroken skin, it may be absorbed through cuts or abrasions. It is rapidly absorbed from the gastrointestinal tract and appears to interfere with acetate metabolism by an ill-defined mechanism. The chemical acts on either the cardiovascular or nervous systems or both in all species and on the skeletal muscles in some species. In man, the action on the central nervous system produces epilepti-form convulsive seizures followed by severe depression.[7] Twenty-two cases of poisoning by this material with 12 deaths have been reported. In another instance, four men died from the consumption of 1080 which had been stored in soft drink or whiskey bottles. Also, four suicides have occurred as a result of drinking 1080. Five confirmed deaths and three possible deaths of small children have been traced to the consumption of 1080 solutions from souffle cups or from chewing on empty cups containing dried residues of the poison.[9]

In recent years much research time and effort has been spent in developing an effective method for treating 1080 poisoning. The treatment regimen usually involves cautious control of the convulsions, and gastric lavage. An antidote (glycerol monoacetate) has been successful in treating poisoned monkeys, but it has not been used in man.[5] At the present time, however, there does not seem to be a successful antidote for this poison.

Currently, 1080 can be purchased and applied only by licensed pest control operators or other qualified persons. But, the fact that there is not a successful antidote, plus the advent of safer rodenticides has led to a decline in the use of this chemical within the past 10 years.

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3. *Fluoroacetamide (1081)*

Fluoroacetamide is another one of the fluoracetic derivatives that produces toxic symptoms in rats. It was first suggested as a potential rodenticide in 1955 by English researchers, when it was pointed out that it is safer to manufacture and handle than 1080.[10] The material is reported to have an oral LD_{50} of 13-14 mg/kg. Thus, it is extremely toxic to humans. Research in England has indicated that the onset of warning symptoms is much slower than 1080 poisoning, an important factor as far as human health is concerned.

The chemical was first registered by the U. S. Department of Agriculture in 1963. It is restricted to use by licensed pest control operators, and has had poor acceptance among the pest control industry. At this time, there are no known cases of poisoning associated with the ingestion of fluoroacetamide.

4. *Gophacide (Bay 33819)*

This is a recently developed organophosphate rodenticide. It has an estimated oral LD_{50} of 4-8 mg/kg for man, thus its use must be carefully supervised. At the present time Gophacide is labeled for use by pest control operators against pocket gophers only. It is not permitted to be used for domestic rodent control purposes. The effect of this chemical on mammals and birds varies with the species concerned. In field trials in Georgia, Gophacide at a level of 0.25 percent in baits of yellow corn meal, hamburger, or dog food gave good control of Norway rats.[11] Thus, it may eventually be used for this purpose if the current restrictions are waived. Specific toxicological information about this product is not yet available, although it is reported to act like organophosphorus compounds in general; i.e., it inhibits cholinesterase. If this is the case, suitable antidotes are available.

5. *Strychnine*

Strychnine is the principal alkaloid present in nux vomica, the seeds of a tree native to India. The term "nux vomica" has been improperly translated as "emetic nut." Actually strychnine is not an emetic, and the word "vomica" means depression or cavity, a feature of the strychnos seed attributed by legend to the digital imprint of the Creator.[12]

Nux vomica was introduced into Germany in the sixteenth century as a rodenticide. Its use as a rodenticide in the form of "rat biscuits" exists on a small scale today. The chemical has in the past been a source of accidental poisoning in children.

In 1966 a total of 181,626 oz. of strychnine were imported into the United States, in comparison to 190,310 oz. in 1965 and 182,600 oz. in 1964.[13] With regard to nux vomica, none was imported in 1964, 9,005 lb. in 1965, and 269,540 lb. in 1966. The use of the majority of these products has been attributed to the drug industry.

Strychnine produces exitation of all portions of the central nervous system. When the concentration of the chemical in the CNS reaches a critical level, impulses travel without restriction throughout the nervous system. The material is a powerful convulsant, and the convulsion has a characteristic motor pattern. Death results from medullary paralysis, which is due primarily to the hypoxia resulting from the periods of impaired respiration. Treatment involves the gastric lavage into which potassium permanganate has been incorporated. CNS depressants, such as short-acting barbiturates are also employed.[12]

Strychnine is reported to have an LD_{50} of 8 mg/kg. Its use as a rodenticide is minimal, however, because rats develop "bait shyness" rapidly due to the bitter taste. Mice will frequently ingest baits such as 1% strychnine-coated wheat. As reported earlier, the use of this chemical for rodent control has declined in the United States. Its use is prohibited by law in Great Britain.

6. *Zinc Phosphide*

Zinc phosphide is a heavy, dark grey powder that is chemically stable and insoluble in water. The chemical has the faint odor of phosphorus due to the slow release of phosphine through hydrolysis. This material has been used for

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many years as a rodenticide and is effective against all species of rats and mice. It is reported to kill rodents in less than 24 hours, with a mean lethal time of 6-12 hours. Dogs and cats do not appear to be susceptible to poisoning by this chemical, and this has been attributed to the emetic qualities of the zinc.[5] The LD_{50} value of zinc phosphide is 40 mg/kg, and two fatal human cases have been attributed to the ingestion of this material. In another case, the affected individual inhaled zinc phosphide dust, became ill, but eventually made a successful recovery. Treatment in the case of poisoning is symptomatic.

The qualities that make zinc phosphide unattractive to other species of mammals (odor, taste and color) appear to make it attractive to rodents. It is normally applied at a concentration of 1 percent by weight in blended and mixed baits. As a safety factor, tartar emetic (antimony potassium tartrate) should be added to the poison prior to mixing the bait. Oils and fats used as binders increase absorption of the material by rodents; likewise, the health hazards to human beings are increased.[14] Despite the availability of a wide variety of less toxic materials, this chemical is still the rodenticide of choice by many pest control operators in the southeastern United States.

7. *Elemental Phosphorus*

Elemental phosphorus exists in two forms, one of which is granular, non-absorbable, and non-toxic. The other is a white or yellow waxy form that is highly poisonous and will burn on contact with water or even moist air.[12] The material is widely sold in a paste form for both insecticidal and rodenticidal purposes. Phosphorus paste is often used as a poison bait for cockroach control. In this application, it is common practice to spread the material on bread which has been placed in strategic locations. This chemical cannot be mixed with baits because of the fire hazard. It must be mixed with materials containing liquids such as molasses, water, oil, fat or various solvents. Poisonings have resulted because of the practice of spreading this material along with molasses, etc. on bread. For the most part, these poisonings have been associated with children in low socioeconomic groups.

The toxic effects of phosphorus result from the ingestion of the material. Contact of the skin with yellow phosphorus causes burns, but dermal absorption is not known to lead to systemic poisoning. A dose of 15 mg may be severely toxic to a human and 50 mg may be fatal.[7] After ingestion, the phosphorus exerts a toxic action within the gastrointestinal tract, followed later by injury to the liver, muscles, myocardium, kidneys, and central nervous system. Vomiting may be fairly prompt after ingestion, and the vomitus which is luminescent, has a characteristic garlic odor. In general, these symptoms are followed by a series of more profound illness. Cardiovascular collapse results from both the metabolic difficulties and a direct influence by phosphorus on the myocardium and blood vessels. Death usually follows a period of delirium and coma.[15] Continued contact with minute amounts of phosphorus can result in "phossy jaw," destroyed jawbone, once a serious industrial hazard to personnel employed in the match industry. Treatment usually involves the use of copper salts to form a semi-metallic coating over the phosphorus particles, thus preventing further absorption.

At this time, this material is rarely used as a rodenticide.

8. *Arsenic (Arsenic Trioxide)*

This chemical which is also known as white arsenic or arsenious oxide is a white crystalline powder. In its technical form, it has been used in solid baits for Norway rats and roof rats in a 3 percent concentration by weight. The degree of toxicity of the material varies directly with the particle size of the powder, and the toxicant in micronized form has been effective at a strength of 1.5 percent by weight in solid baits. In general, the arsenical rodenticides are effective against Norway and roof rats, but not against house mice. This factor, coupled with its inherent toxicity to man has led to a decline in the use of this material as a rodenticide.

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Arsenic is absorbed chiefly by the respiratory and gastrointestinal tracts. A dose of 5-50 mg is toxic to humans, and a dosage of 128 mg has proven fatal.[7] Gastrointestinal discomfort is usually experienced within an hour after the poison is taken although the discomfort may be delayed if food is in the stomach. Constriction of the throat and difficulty in swallowing may be the first symptoms to appear. Skeletal muscle cramps, diarrhea, and convulsions may occur. In severe poisoning, death can occur within an hour, but the usual time interval is 24 hours.[12] Treatment usually involves gastric lavage and the administration of BAL (Dimercaprol).[7]

Arsenic compounds are the most important single cause of accidental deaths associated with pesticides. In 1956, this chemical was the cause of 35 percent of the deaths caused by pesticides. Seventy-four percent of the cases in 1956 involved children five years of age or younger.[7] Pest control operators in general have avoided the use of this material as a rodenticide.

9. *Barium Carbonate*

This chemical was used as a rodenticide, but because of the availability of more suitable compounds, its use as a rodenticide today is practically nonexistent.

The ingestion of barium carbonate causes severe colic, diarrhea and hemorrhage. Death may result from cardiac arrest.[12]

Part II of this article will be concerned with those rodenticides which have a moderate degree of toxicity and with those compounds which have a low order of toxicity.

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Successful use of helicopters in Vietnam rescue missions has led to consideration of using helicopters to rescue traffic accident victims here at home. In a recent issue of *Traffic Safety*, the National Safety Council says that experimental programs in Texas, Pennsylvania and Chicago indicate that while helicopters will be useful, they probably will not become the mainstay in emergency services. Although increased speed is an important advantage over ground ambulances, cost of helicopters and problems of landing in crowded city streets are drawbacks. In addition, further accidents may be caused by drivers looking at the helicopter operations, instead of paying attention to their driving.

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