

# UC Agriculture & Natural Resources

## Proceedings of the Vertebrate Pest Conference

### Title

A novel technology for the control of rodents

### Permalink

<https://escholarship.org/uc/item/1v11b7b4>

### Journal

Proceedings of the Vertebrate Pest Conference, 21(21)

### ISSN

0507-6773

### Authors

Grech, Nigel M.  
Dawson, William  
Putman, Rory  
[et al.](#)

### Publication Date

2004

# A Novel Technology for the Control of Rodents

Nigel M. Grech

Rodetrol Technologies, Visalia, California

William Dawson

School of Mathematics and Science, Sheffield Hallam University, U.K.

Rory Putman

Dept. of Environmental Biology, Manchester Metropolitan University, U.K.

Steven Havers

Pest Management Consultants, Basingstoke, U.K.

**ABSTRACT:** An alternative rodent control technology is presented. The patented discovery that specific plant-derived structural carbohydrate polymers are inhibitory to the water retentive mechanisms of rodents is discussed. Specifically, it has been discovered that when natural complex structural carbohydrates are formulated into a palatable pellet, target species of rodents (rats, mice, and ground squirrels), after ingesting the polymers, become less active and eventually die after 3 - 10 days. Captivity and *in situ* tests on the Norway rat have indicated the lethal dose for rats to be approximately 35 - 50 g consumed over a period of 72 - 96 hours, whereas for house mice it is 7 - 10 g over the same period. Captive trials on California ground squirrels have indicated a similar lethal dosage to that of rats, specifically 35 - 50 g consumed over 72 - 96 hours. The commercial product is exempt from registration in many countries including the U.S. This paper discusses laboratory and field test results on rodents to date and field use experiences.

**KEY WORDS:** California ground squirrel, EPA-exempt, house mouse, *Mus musculus*, Norway rat, plant structural carbohydrates, *Rattus norvegicus*, rodent control, Rodetrol, *Spermophilus beecheyi*

Proc. 21<sup>st</sup> Vertebr. Pest Conf. (R. M. Timm and W. P. Gorenzel, Eds.)  
Published at Univ. of Calif., Davis. 2004. Pp. 258-262.

## INTRODUCTION

Conventional control of rodents generally relies heavily on anticoagulant rodenticides (Corrigan 2001). Anticoagulants rodenticides are classified as extremely hazardous or highly hazardous to humans and other non-target species (WHO 2003), and the newer second-generation anticoagulants have led to a dramatic increase in the incidence of accidental ingestion as reported to poison control centers in the United States. Sadly, 87% of those accidental exposures in the United States are to children under 6 years of age (Eisemann and Petersen 2002). Further, the widespread use of anticoagulants has led to the development of anticoagulant resistance in many parts of the world (Corrigan 2001, MacNicoll et al. 1996, Quy et al. 1998). The environmental impact of anticoagulants including effects on non-target species and secondary poisoning of predators and scavengers (Hegdal and Blaskiewicz 1984) recently led to the EPA promulgating the "Reregistration Eligibility Requirements" for selected rodenticides (US EPA. 2003). These requirements were designed to reduce the risks associated with the use of anticoagulant rodenticides and embody certain proposals such as a reduction in the amounts of active ingredients in products and/or a reduction in their application rates. If ratified, broadcast baiting applications of certain anticoagulant rodenticides (such as diphacinone) would be restricted to an active ingredient content of 0.001%.

In California, the use of Proposition 65 pesticides (reproductive toxins) has declined by approximately 40% over the last 10 years, as has the use of cholinesterase-inhibiting pesticides. Concomitantly, there has been an increase in the use of "biopesticides" in California by over 70% in the last 10 years (Anon. 2003). The inherent toxicity of rodenticides requires that they are used in strict accordance

with state and federal pesticide use guidelines, and as such, their application is highly regulated. Globally, the pesticide industry is increasingly seeking safer and less toxic alternatives as a result of an increasing consumer reluctance to accept high toxicity pesticides (Anon. 2002).

Of all the common rodents, the commensal rodents are by far the most damaging to mankind. Environmentally, rodent species are classified as highly invasive with a high potential to eliminate or displace indigenous species (Anon. 2004). Commensal rodents have a great ability to adapt to rapidly changing environments, which, in part, has led to their global success. Rodents have some unique physiological features such as the inability to vomit, the capacity to consume large quantities of food during a single feeding (measured as a percentage of body mass), and a greatly enlarged cecum (which allows for partial microbially-mediated cellulose digestion). Rodents are less efficient than ruminants at digesting plant-derived structural carbohydrates. Our research has determined that certain structural carbohydrates, when formulated correctly, will have a negative impact on commensal rodents, as well as on other rodents such as the California ground squirrel (*Spermophilus beecheyi*). In this paper, we report on studies of a novel, patented, non-poisonous, biodegradable rodent control technology called Rodetrol (Eradirat and Eradimouse in Europe). It is derived from specific plant structural carbohydrates and is administered in a chemically unaltered form. Rodetrol is a minimum-risk pesticide and qualifies for exemption from registration in accordance with the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) regulations. EPA's Pesticide Registration Notice (PR Notice 2000-6) identifies exempted active and inert ingredients (U.S. EPA 2000).

## METHODS

### Laboratory Studies

Good Laboratory Practice (GLP) efficacy tests were conducted at several laboratories around the world according to generally accepted rodenticide testing protocols during the period 1996 - 2003 (Quy 1996; Sayre 2001; Hoyer 2002; Morgan 2002a,b,c; Morgan and Eason 2003). In the tests described herein, we used similar methodologies for rats, mice and Ground squirrels involving the use of wild-caught Norway rats (*Rattus norvegicus*), feral house mice (*Mus musculus*), and California ground squirrels (*Spermophilus beecheyi*) following generally accepted guidelines for rodenticide testing (Anon 1990). Feeding studies were conducted with a minimum of 15 animals per treatment group, in individual cages. All animals were maintained on 12-hour diurnal cycles at temperature range between 21 - 25°C. Animals had free access to water for the entire duration of the tests. All animals were allowed several days (usually 7) to acclimatize to the cages, during which time they were fed either rodent pellets (Harlan Teklad Protein Rodent Maintenance Diet 2014, Avon, IN). Animals were divided into treatment groups and controls, with a similar proportion of males and females in each group. All animals used were within a 10% range of the average body mass per gender group. One treatment group was fed Rodentol pellets (rat pellets for rats and ground squirrels, and mouse pellets for mice) in the absence of any other food. Control groups were either starved or fed the laboratory rat maintenance diet. All animals had free access to water. In the rat and mouse experiments, daily feed and water consumption was recorded, as well as body weight changes over the experimental period. At death, treated rats underwent an autopsal examination. Three animals from each control group were euthanized and also subjected to examination. In the ground squirrels tests, only mortality was recorded.

### Field Studies

#### *Eston, Natal, South Africa*

A tomato packing facility was identified as having a persistent Norway rat infestation. Poisons could not be used at this location, due to the risks associated with contamination of the food chain and non-target toxicity. The facility supported high numbers of rats because of the abundant harborage and food availability. Rat feces were abundant around the facility, which was a serious public health issue in regard to microbial contamination.

Areas of activity and heavy infestation were identified and noted. Night-vision cameras were installed to monitor rat activity at specific locations over a 10-day period. Rodentol pellets (100 g/tray) in bait trays were placed in at least 20 locations around the facility (access points, identified rat trails). Rodentol pellets (100 g) were also wrapped in parafilm (America National Can, Neenah, WI) and placed in the roof (30 bait packages) and in rat nest holes (25). Rat activity was monitored every night between 1900 and 2000 hrs. Rodentol bait was replenished daily in the bait trays.

#### *Cory Waste Processing, Southend, U.K.*

A municipal refuse accumulation site, used as an accumulation point for primarily supermarket and grocery

waste, had a recurrent Norway rat problem. Previous use of 0.005% bromadiolone (Deadline, Liphatech) had not been effective at eradicating the infestation. The site had abundant alternative food sources available. In 1998, pre-baiting was initiated at 19 baiting points (1,500 g oats/ station) to assess the activity of the rats. After a first week of pre-baiting, Rodentol was presented during the second, third, and fourth weeks (1,500 g/station). A final post-baiting period was initiated, again with oats, during the fifth week to assess residual rodent activity.

## RESULTS

### Laboratory Studies

All female rats in the Rodentol treatments died (Table 1), whereas 14 of the 15 males died. Two days after presenting the Rodentol bait, the rats' fecal pellets became larger in size and lighter colored. One or two days prior to death, Rodentol-treated animals developed body tremors and became lethargic, followed by comatosis. At death, the Rodentol-treated rats had lost on average 42 g (male) and 34 g (female) of their body mass, whereas the control groups gained an average of 23 g (male) and 17 g (female). The starved controls lost an average of 19 g of body mass. One starved control animal died on Day 3 (Table 1), whereas all the other starved controls remained alive until the end of the experiment. In the Rodentol treatments, weight loss was mainly as a result of water loss, as body fat deposits were still present in Rodentol-treated animals, although less so than in the fed controls. All treated rats consumed less during the first few days of exposure to the Rodentol, but intake rates recovered to pre-treatment rates 2 - 3 days prior to death. All animals exhibited a significant reduction in water consumption. Further, red blood cell densities were higher in the treated animals, indicative of blood hypovolemia. Autopsy results (Table 2) indicated cecal enlargement and compaction, reduced urine volume, blood hypovolemia, and liver tissue ischemia. In the mouse trials, all treated mice died (Table 3). At death, the Rodentol-treated mice had lost on average 32% (male) and 27% (female) of their body mass, whereas the control groups lost an average of 3% (male) and 4% (female). The starved controls lost an average of 16% of body mass. Three starved control mice died by Day 6 (Table 3), whereas all the other starved controls remained alive until the end of the experiment. All treated ground squirrels died by Day 9 (Table 4), and at death both males and females had exhibited considerable mean weight loss (58 g and 46 g, respectively).

### Field Trial

#### *Eston, Natal, South Africa*

The Rodentol pellets were eaten at all locations in and around the tomato packing facility, although the applications in the nests appeared to be the most consistently consumed. After Rodentol was applied, there was an initial increase in rat activity 3 to 5 days after application, followed by a reduction in rat activity after approximately 6 to 7 days post-application (Figure 1). By Day 10, there was no further activity observed, and a concomitant reduction in the damage to packed tomatoes in the facility was recorded (personal observation by the packhouse manager). Several burrows



**Table 1. Mean daily feed intake, rat mortality, mean body mass change and mean daily intake in laboratory rats.**

Rat Treatment Group	Total Mortality at Day 8	Mean Body Mass Loss or Gain at Day 8 or Death (- or + In. g.)	Mean Daily Water Consumption (ml)	Mean Daily Rodetrol or Feed Consumption (g)
Female Treatment n = 15	15 a	-34	8 a	7 a
Female Control n = 15	0 c	+17	17 b	19 b
Male Treatment n = 15	14 a	-42	12 b	9 a
Male Control n = 15	0 c	+23	37 c	27 b
Male Starved Control n = 15	1 b	-19	43 c	Not Applicable

Values followed by the same letter in the same column are not significantly different from one another at P = 0.05 according to the standard Student's t-test

**Table 2. Summary of the major autopsal observations on rats from laboratory tests.**

Treatment Group	Body fat	Red blood cell density	Urine Production	Large Intestines	Cecum
Rodetrol treated male	Little	9.5 x 10 <sup>12</sup> cells/L	Little	Full, blood present in some specimens	Distended
Rodetrol treated female	Little	8.8 x 10 <sup>12</sup> cells/L	Little	Full	Distended
Control male	Abundant	5.8 x 10 <sup>12</sup> cells/L	Normal	Full	Normal
Control female	Abundant	5.5 x 10 <sup>12</sup> cells/L	Normal	Full	Normal
Control starved	Little	4.7 x 10 <sup>12</sup> cells/L	Normal	Empty	Normal

**Table 3. Mouse mortality, mean body mass change and mean daily feed intake in laboratory mice.**

Mouse Treatment Group	Total Mortality at Day 6	Mean % Body Mass Loss or Gain at Day 6 or Death	Mean Days to Death	Mean Daily Rodetrol or Feed Consumption (g)
Female Treatment n = 15	15 a	-27	4.3 a	1.4 a
Female Control n = 15	0 c	-4	NA	3.4 b
Male Treatment n = 15	15 a	-32	4.8 b	1.45 a
Male Control n = 15	0 c	-3	NA	4.1 b
Male Starved Control n = 15	3 b	-16	5.5 c	Not applicable

Values followed by the same letter in the same column are not significantly different from one another at P = 0.05 according to the standard Student's t-test

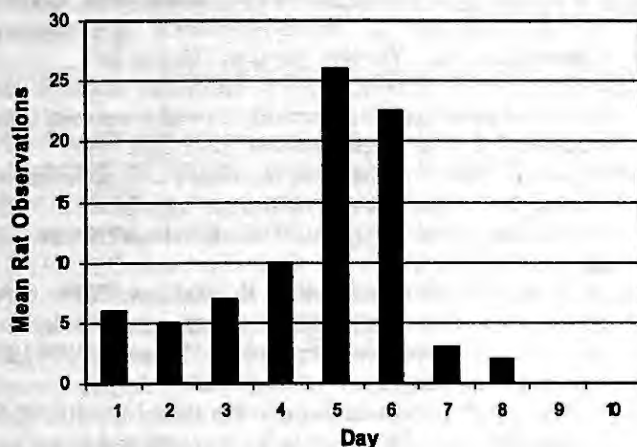
**Table 4. Ground squirrel mortality and mean body mass change.**

Ground Squirrel Treatment Group	Total Mortality at Day 9	Mean body mass loss or gain at Day 9 or death (- or +)
Female Treatment n = 15	15 a	-46
Female Control n = 15	0 c	-17
Male Treatment n = 15	15 a	-58
Male Control n = 15	0 c	-23
Male Starved Control n = 15	1 b	-34

**Table 5. Field rat control trial. Field pre-baiting take, Rodentol take, and post-baiting take recorded at a waste processing facility in the U.K.**

Baiting Week	Mean amount of oats consumed weekly (g) per bait station (n = 19)	Mean amount of Rodentol consumed weekly (g) per bait station (n = 19)
1 (prebait)	271 a	
2		291 a
3		246 a
4		28 b
5 (postbait)	0 c	

Values followed by the same letter in the same column are not significantly different from one another at P = 0.01 according to the standard Students t-test.



**Figure 1. Nocturnal activity of rats in a tomato packaging facility following Rodentol applications; South Africa. Rat observations are the mean for each of 5 locations, recorded between 1900 and 2000 hrs. Rodentol treatment was presented on Day 2.**

were excavated and observed for the presence of dead rats; 4 were found and examined. All had Rodentol pellets in their nests, and when dissected, exhibited evidence of ingested Rodentol in their hindgut.

#### **Cory Waste Processing, Southend, U.K.**

Initial prebaiting at the refuse accumulation site resulted in a large bait take (>200 g/bait station). Subsequent Rodentol bait take was equally large during the second week and third weeks (>200g/bait station). By the fourth week, the Rodentol weekly take had been reduced to <30 g/bait station. The operator of the site commented on the reduction in visual rat activity. Post-baiting with oats during the fifth week resulted in zero bait take (Table 5).

#### **DISCUSSION**

Laboratory trials showed that Rodentol, when presented in no-choice tests, required approximately 5 - 7 days to kill Norway rats, 4 - 9 days to kill ground squirrels and 2 - 5 days to kill mice. Field studies indicated that rat control is satisfactory even where abundant alternative food sources are available.

These studies have indicated that Rodentol bait pellets can kill rodents and are consistent with previous studies on field control of rats and mice with Rodentol (Havers 2000a,b; Havers 2001; Spurr et al. 2004), where satisfactory field

rodent control was achieved. Laboratory studies indicated that 20 - 30 g of the material is required over the course of 2 - 4 days to kill mature Norway rats. In these experiments, the lethal dose range required to kill a rat is 0.05 - 0.3 g/g body mass (mean LD<sub>50</sub> for male rats = 0.1 g/g). Whisson et al. (2000) reported that when California ground squirrels, similarly sized to the rats used in this test, were fed a 0.01% diphacinone bait, animals required up to 36 g/animal over 3 feedings before death ensued, 9 days after the first exposure. Interestingly, the overall mean mortality rates in these studies were <65%. Similarly for house mice (*Mus musculus*), previous tests measured mortality when fed chlorophacinone and diphacinone oat baits in no-choice tests (Rowe and Redfern 1968). In these tests, mouse mortality was less than 50% when fed for 3 days at 0.025% chlorophacinone or diphacinone. Lund (1971) fed groups of 20 mice 0.025% chlorophacinone oat bait. Mortality was <5% for feeding periods of 1 - 5 days, 90% for 10 days, and 95% after 21 days. One mouse consumed 906 mg/kg of bait and survived.

These studies indicated an acceptable level of field rat and mouse control when the Rodentol pellets were applied. These studies concur with previous studies (Sayre 2001; Morgan 2002a,b; Hoyer 2002) in which comprehensive testing indicated acceptable levels of control of commensal rodents. Recently, preliminary field tests performed on California ground squirrels have indicated that the Rodentol product is also effective at killing these important pests (Grech 2003, unpubl. data).

Unlike conventional rodenticides, which are based on compounds with high mammalian toxicity (Corrigan, 2001, World Health Organization 2003), Rodentol thus far is appears specific to rodents and is not toxic to non-target species (Morgan 2003c). The preliminary evidence indicates a mode of action based primarily on a disruption to the water retentive mechanisms of the cecum and hindgut (Morgan and Eason 2003). As such, acquired tolerance to this material is unlikely. Rodentol offers an alternative non-toxic approach to rodent control.

#### **LITERATURE CITED**

- ANON. 1990. Guidelines on Efficacy Testing of Rodenticides. Part three/A3/Appendix 3, formerly working document 10/2. PSD/HSE Registration Handbook for Pesticides, Biocides and Plant Protection Products. Health & Safety Executive, U.K.
- ANON. 2002. Pesticides and You. 22(4). The Pesticide Coalition, Washington D.C.
- ANON. 2003. Annual registrations of cholinesterase inhibiting pesticides, proposition 65 pesticides and biopesticides in California 1994-2003. California Environmental Protection

- Agency, Sacramento, CA. 72 pp. ([www.cdpr.ca.gov/](http://www.cdpr.ca.gov/))
- ANON. 2004. The Invasive Species Specialist Group (ISSG). <http://issg.org/database>.
- CORRIGAN, R. M. 2001. Rodent control: a practical guide for pest management professionals. GIE Media, Cleveland, OH. 355 pp.
- EISEMANN, J. D., AND B. E. PETERSEN. 2002. Human poisonings and rodenticides: evaluation of incidents reported to the American Association of Poison Control Centers. Proc. Vertebr. Pest Conf. 20:290-294.
- HAYERS, S. J. 2000a. Brief notes on the control of an infestation of house mice (*Mus domesticus*) in an inner city school. Field Report July 2000, Pest Management Consultants, Basingstoke, U.K.
- HAYERS, S. J. 2000b. Brief notes on the control of anti-coagulant resistant rats (*Rattus norvegicus*). Field Report April 2000, Pest Management Consultants, Basingstoke, U.K.
- HAYERS, S. J. 2001. The use of Eradirat in the control of the brown rat (*Rattus rattus*) in an urban environment. Field Report April 2001, Pest Management Consultants, Basingstoke, U.K.
- HEGDAL, P. L., AND R. W. BLASKIEWICZ. 1984. Evaluation of the potential hazard to barn owls of Talon (brodifacoum bait) used to control rats and house mice. Environ. Toxic. Chem. 3(2):167-179.
- HOYER, K. 2002. Effects of Eradirat on rats when fed ad libitum. 40 CFR 160-FIFRA. GLP Study GLP31755, Celsis Laboratory Group, St. Louis, MO.
- LUND, M. 1971. The toxicity of chlorophacinone and wafarin to house mice (*Mus musculus*). J. Hyg. (Cambr.) 69(10):69-72.
- MACNICOLL, A. D., G. M. KERINS, N. J. DENNIS, AND J. E. GILL. 1996. The distribution and significance of anticoagulant-resistant Norway rats (*Rattus norvegicus*) in England and Wales, 1988-95. Proc. Vertebr. Pest Conf. 17:179-185.
- MORGAN, D. R. 2002a. Efficacy of Eradimouse against Norway rats. Landcare Research Contract Report LC0203/054. Landcare Research, Lincoln, New Zealand. 32 pp.
- MORGAN, D. R. 2002b. Efficacy of Eradimouse and Eradirat against feral house mice and Norway rat. Landcare Research Contract Report LC0102/109. Landcare Research, Lincoln, New Zealand. 26 pp.
- MORGAN, D. R. 2003c. Susceptibility of non-target species to Eradirat and Eradimouse— a summary report. Landcare Research Contract Report LC0203/124. Landcare Research, Lincoln, New Zealand. 9 pp.
- MORGAN, D. R., AND C. T. EASON. 2003. Efficacy of Eradirat against Sprague-Dawley laboratory rats and assessment of clinical effects and mode of action. Landcare Research Contract Report LC0203/061. Landcare Research, Lincoln, New Zealand. 34 pp.
- QUY, R. J. 1996. Evaluation of the palatability of Eradirat rodenticide products. Evaluation report, Central Science Laboratory, Slough, London, U.K.
- QUY, R. J., A. D. MACNICOLL, AND D. P. COWAN. 1998. Control of rats resistant to second-generation anticoagulant rodenticides. Proc. Vertebr. Pest Conf. 18:262-267.
- ROWE, F. P., AND R. REDFERN. 1968. Laboratory studies on the toxicity of anticoagulant rodenticides to wild house mice (*Mus musculus* L.). Ann. Appl. Biol. 61(2):322-326.
- SAYRE, R. W. 2001. Evaluation of the efficacy and mechanism of Eradimouse on the Norway rat (*Rattus norvegicus*) in a 15-day, no-choice test. Study No. N01024, Genesis Laboratories, Inc., Wellington, CO.
- SPURR, E. B., G. A. MORRIS, AND D. R. MORGAN. 2004. Field efficacy of Eradimouse for rodent control at a New Zealand pig farm. Landcare Research Contract Report LC0304/147. Landcare Research, Lincoln, New Zealand. 24 pp.
- U.S. EPA. 2000. Pesticide Registration (PR) Notice 2000-6. Office of Pesticide Programs, U.S. Environmental Protection Agency, Washington D.C. 10 pp.
- U.S. EPA. 2003. Reregistration Eligibility Decision (RED), Office of Prevention, Pesticides and Toxic Substances, U.S. Environmental Protection Agency, Washington D.C. EPA Publication No. 738-R-2003. 225 pp.
- WHISSON, D. A., T. P. SALMON, AND W. P. GORENZEL. 2000. Reduced risk anticoagulant baiting strategies for California ground squirrels. Proc. Vertebr. Pest Conf. 19:362-364.
- WORLD HEALTH ORGANIZATION. 2003. The WHO classification of pesticides by hazard and guidelines to classification 2000-2002. International Programs on Chemical Safety, World Health Organization Publications, Geneva, Switzerland. 62 pp.