The Toxicity of Chlorophacinone and Diphacinone to Deer Mice
Author(s): Rex E. Marsh, Walter E. Howard, Ronald E. Cole
Source: *The Journal of Wildlife Management*, Vol. 41, No. 2 (Apr., 1977), pp. 298-301
Published by: Allen Press
Stable URL: http://www.jstor.org/stable/3800608
Accessed: 26/05/2011 17:39

THE TOXICITY OF CHLOROPHACINONE AND DIPHACINONE TO DEER MICE

REX E. MARSH, Wildlife and Fisheries Biology, University of California, Davis 95616
WALTER E. HOWARD, Wildlife and Fisheries Biology, University of California, Davis 95616
RONALD E. COLE, Wildlife and Fisheries Biology, University of California, Davis 95616

Abstract: Oat groat baits containing 0.00125, 0.0025, 0.005, and 0.01, and 0.02 percent concentrations of the anticoagulant rodenticides chlorophacinone (2-[*p*-chlorophenyl)-1-phenylacetyl]-1,3-indandione), or diphacinone (2-diphenylacetyl-1,3-indandione), were offered to 400 individually caged deer mice (*Peromyscus maniculatus*), with and without the availability of supplemental food, to determine their comparative effectiveness as control agents. No significant difference occurred in mortality with or without supplemental food. In the 2-day tests, only 0.02 percent chlorophacinone gave 100 percent mortality. Diphacinone produced 70 to 90 percent mortality in the 4-day tests with concentrations of 0.0025 percent or higher, while chlorophacinone gave 100 percent mortality at all concentrations of 0.0025 percent or above. The collective results of chlorophacinone ($P < 0.01$) were significantly more effective than the comparable concentrations of diphacinone on both the 2- and 4-day tests. The mortality difference between the 2- and 4-day results was significant ($P < 0.01$) for both compounds.

J. WILDL. MANAGE. 41(2):298–301

The use of anticoagulant rodenticides for the control of field rodents has received increased attention because they are effective, less likely to create problems to nontarget species than some acute toxicants, and the difference in cost is no longer a limiting factor for many situations. The authors have previously reported on the excellent control of deer mice in northern California conifer forests by baiting with the anticoagulant diphacinone (2-diphenylacetyl-1,3-indandione) at a concentration of 0.01 percent on oat groats (Howard et al. 1970:220). The use of 0.01 percent chlorophacinone (2-[(*p*-chlorophenyl)-1-phenylacetyl]-1,3-indandione) as an effective means of controlling deer mice has been more recently discussed (Passof 1974, Marsh et al. 1974).

Before chlorophacinone was marketed in the United States, diphacinone proved effective for forest rodent control because it is more active per unit of weight and requires fewer multiple feedings than other anticoagulants (Bentley and Larthe 1959, Gates 1957) which were available. Chlorophacinone, an anticoagulant chemically quite similar to diphacinone, also produces death with minimal feedings in some species. Rowe and Redfern (1968) indicate that chlorophacinone is approximately 2.5 times more active (per unit weight of chemical) than diphacinone when tested on house mice (*Mus musculus*). Since chlorophacinone was successfully used for field rodents in Europe prior to its introduction into the United States, we decided to compare it with diphacinone as a potential agent for the control of deer mice in forest habitat.

This study has been supported in part by grants from the California State Division of Forestry; Public Health Service Grant CC 00262 from the Center for Disease Control, Atlanta, Georgia; and Vector Biology and Control of the World Health Organization, Geneva, Switzerland. We wish to thank the Nease Chemical Company and the Chempar Chemical Company for providing the chemicals used in this study, which was carried out in the Institute of Ecology, University of California, Davis.

METHODS AND MATERIALS

Four hundred deer mice (*Peromyscus maniculatus*) raised in our laboratory from

parental stock captured in northern California coniferous zone were divided into 40 groups (5 males and 5 females per group) and housed in individual laboratory cages measuring 22.9 × 27.9 × 20.3 cm and fed pelleted Purina laboratory chow. After a 3-day acclimation period, these mice were offered crimped oat groats treated with one of five concentrations (0.00125, 0.0025, 0.005, 0.01 and 0.02 percent) of either diphacinone or chlorophacinone for either 2 or 4 days. The powdered technical material (approximately 95 percent pure) was suspended or partially dissolved in a 1:1 (by weight) of vegetable lecithin and U.S.P. No. 9 mineral oil, and then mixed with the crimped oat groats by a slurry method. Twenty of the 40 test groups were offered both the test bait and laboratory chow. The remaining 20 groups were offered only the bait.

The amount of treated and supplemental food offered to each rodent always exceeded their daily intake requirement. Each day all uneaten treated and supplemental food was weighed and replaced with fresh food in clean test bowls. Water was available ad libitum. Upon completion of the 2- or 4-day feeding schedule, the mice were fed pelleted laboratory chow and maintained for 21 days or until death.

RESULTS

The results are presented in Table 1 and Fig. 1. Chlorophacinone resulted in a greater mortality than diphacinone in every test from the lowest to the highest concentration when the same concentrations are compared. When collectively analyzing the mortality data from the 2- or 4-day tests using Wilcoxin's signed rank test (Mack 1967:130), there was a highly significant difference ($P < 0.01$) between chlorophacinone and diphacinone. There was also a highly significant difference ($P < 0.01$) in mortality with both materials when comparing the 2-day against the 4-day feedings. No significant difference in mortality between the two chemicals was apparent when analyzing each of the 4 highest concentrations separately for the 4-day test periods.

As a point of interest, even when supplemental laboratory chow was present, most animals still ate substantially more of the treated oat groats than chow, except for one group on 0.0025 percent diphacinone-treated bait, where there was an unexplained reduction in the amount of treated bait consumed. The mice that were given a free choice of either the treated bait or laboratory chow selected the treated bait for an average of 65 percent of their diet. The diphacinone-treated baits were chosen for 62 percent of the diet, and 69 percent of the diet consisted of chlorophacinone baits rather than laboratory chow. The differences in acceptance between the two compounds are not significant but do indicate that acceptance of both compounds was good compared to that of laboratory chow and that no obvious rejection of either compound was apparent at even the highest concentration. The mortality percentages were not affected significantly by the presence or absence of supplemental laboratory chow so both feeding situations are grouped together in Table 1 and Fig. 1.

As with all anticoagulants, there is a delay from the time of the first ingestion until mortality occurs. This period varies slightly depending on the number of ingestions and the amount consumed. Table 1 gives the range of number of days until death and the averages for each compound and concentration thereof. The differences in time to death measured in increments of days (24 hours) between the lowest and highest concentrations were not great with either anticoagulant. No significant differ-

Table 1. Percent mortality and range in number of days until death of 40 groups of 10 deer mice each (sexes equal) fed weighed amounts of crimped oat groats treated with 5 concentrations of diphacinone or chlorophacinone for 2 or 4 days with or without free choice of supplemental laboratory ration.

Rodenticide	Number of test days	Supplemental food present	Percent mortality: number of days until death (average days)[a]				
			Percent concentration of rodenticide				
			0.00125	0.0025	0.005	0.01	0.02
Diphac.	2	Yes	0	20:6(6.0)	10:5(5.0)	40:4–5(4.8)	40:4–6(5.2)
Chloro.	2	Yes	30:6–7(6.3)	30:5(5.0)	50:5–7(5.6)	60:4–6(5.0)	100:4–9(5.6)
Diphac.	2	No	0	20:5(5.0)	30:4–5(4.7)	20:6(6.0)	60:4–7(5.8)
Chloro.	2	No	20:2–6(4.0)	60:5–7(5.8)	60:5–6(5.3)	50:5–6(5.2)	100:4–8(6.0)
Total on 2-day tests							
Diphac.			0	20:5–6(5.5)	20:4–5(4.8)	30:4–6(5.2)	50:4–7(5.6)
Chloro.			25:2–7(5.4)	45:5–7(5.6)	55:5–7(5.5)	55:4–6(5.1)	100:4–9(5.8)
Diphac.	4	Yes	60:6–12(7.5)	80:5–8(5.9)	50:3–9(6.2)	90:5–7(5.9)	80:5–7(5.6)
Chloro.	4	Yes	70:5–9(6.1)	90:5–8(6.4)	100:4–8(5.7)	100:5–8(5.9)	100:4–7(5.4)
Diphac.	4	No	50:5–7(6.0)	90:4–7(5.4)	80:4–7(6.0)	80:5–8(7.2)	80:5–7(6.0)
Chloro.	4	No	60:5–7(6.2)	100:5–8(6.1)	90:4–7(5.7)	90:3–11(6.2)	100:4–8(5.4)
Total on 4-day tests							
Diphac.			55:5–12(6.8)	85:4–8(5.6)	65:3–9(6.1)	85:5–8(6.3)	80:5–7(5.8)
Chloro.			65:5–9(6.1)	95:5–8(6.3)	95:4–8(5.7)	95:3–11(6.0)	100:4–8(5.4)

[a] In calculating the number of days to death the sample size is small when only a small percentage had died.

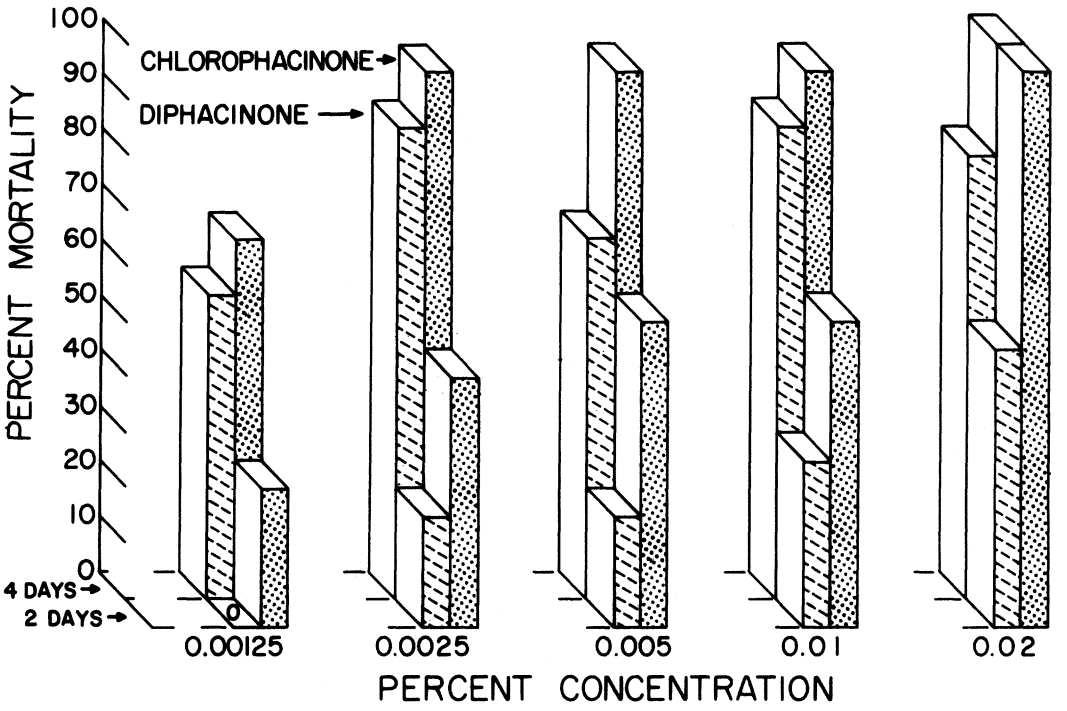

Fig. 1. Percent mortality of 20 groups of 20 deer mice each (sexes equal) offered crimped oat groats treated with 1 of 5 concentrations of chlorophacinone or diphacinone for 2 or 4 days. Half of each group were offered only toxic bait and half were offered a free-choice of treated bait or laboratory chow, but they are grouped together because there was no significant change in percent mortality or days to death.

ences in time to death could be shown between chlorophacinone and diphacinone.

Unlike most acute rodenticides, which are characterized by rather well defined lethal levels based on a single dose to arrive at the mg/kg to prove lethal to 99 percent of the population, the chronic action of anticoagulants depends both on the number of consecutive feedings and the concentration of the active ingredient in the bait. Consequently the number of days of feeding is critical and considerable variation in percent mortality can be expected from only 2- or 4-day exposures, especially at the lower concentrations. This is why 100 percent mortality is not more frequent in Table 1. With longer feeding periods of either diphacinone or chlorophacinone, all of the concentrations would be expected to approach 100 percent mortality.

We did not explore concentrations greater than 0.02 percent because under field conditions we had already determined that under conditions of good weather 0.01 percent diphacinone-treated oat groat bait broadcast at 0.36 kg/ha (2 lb/acre) achieved effective control of forest-dwelling deer mice (Howard et al. 1970).

CONCLUSIONS

These data suggest that the mortality differential between the two rodenticides is based on either a greater degree of susceptibility by deer mice to chlorophacinone, or that chlorophacinone is generally more active per unit weight in a shorter period of time than diphacinone, or possibly both. Such efficacy indicates that chlorophacinone has the potential of surpassing the degree of deer mouse control previously experienced with diphacinone. Under forest conditions it is difficult to be certain that a single broadcast application of bait would be available and acceptable to deer mice for more than a couple of days, particularly at the time of year when dense fog, rain or snow are frequent occurrences. Inclement weather often leaches a toxicant from the bait or renders the bait unacceptable to the mice because of moisture uptake. Snow may cover the bait making it difficult for the mice to find lethal amounts. Even a slight advantage in the reduction of the number of days deer mice must consume the bait to provide a high degree of mortality is most important. Our data favor chlorophacinone over diphacinone, although both can be effective toxicants for deer mice. Chlorophacinone's subsequent field use based on these laboratory studies now substantiates our earlier supposition.

LITERATURE CITED

BENTLEY, E. W., AND Y. LARTHE. 1959. The comparative rodenticidal efficiency of five anticoagulants. J. Hyg. 57(2):135–149.

GATES, R. L. 1957. Diphacin, new anticoagulant rodenticide. Pest Control 25(8):14,16.

HOWARD, W. E., R. E. MARSH, AND R. E. COLE. 1970. A diphacinone bait for deer mouse control. J. Forestry 68(4):220–222.

MACK, C. 1976. Essentials of statistics for scientists and technologists. Plenum Press, New York. 174pp.

MARSH, R. E., P. C. PASSOF, AND W. E. HOWARD. 1974. Anticoagulants and alpha-naphthylthiourea to protect conifer seeds. Pages 75–83 *in* H. C. Black, ed. Wildlife and Forest Management in the Pacific Northwest. Oregon State University, Corvallis.

PASSOF, P. C. 1974. Rodent control activities when direct seeding forest lands in northern California. Pages 127–131 *in* W. V. Johnson, ed. Proceedings Sixth Vertebrate Pest Conference, University of California, Davis.

ROWE, F. P., AND R. REDFERN. 1968. Laboratory studies on the toxicity of anticoagulant rodenticides to wild house mice (*Mus musculus* L.). Ann. Appl. Bio. 61(2):322–326.

Received 18 August 1972.
Accepted 26 January 1977.