195

ZINC PHOSPHIDE—ITS DEVELOPMENT AS A CONTROL AGENT FOR BLACK-TAILED PRAIRIE DOGS

UNITED STATES DEPARTMENT OF INTERIOR
FISH AND WILDLIFE SERVICE
Special Scientific Report—Wildlife No. 195

UNITED STATES DEPARTMENT OF THE INTERIOR
Fish and Wildlife Service

ZINC PHOSPHIDE—ITS DEVELOPMENT AS A CONTROL AGENT FOR BLACK-TAILED PRAIRIE DOGS

By

Howard P. Tietjen

Denver Wildlife Research Center
Denver, Colorado 80225

Special Scientific Report—Wildlife No. 195
Washington, D.C. • 1976

CONTENTS

	Page
ABSTRACT	iv
THE PROBLEM	1
DEVELOPMENT OF ZINC PHOSPHIDE BAIT	2
Laboratory Bioassays	2
Grain Acceptance Test	2
LD_{50} and Initial Bait Acceptance Bioassay	2
Bait Development Bioassays	3
Secondary Hazard Bioassays	4
Treatment of prairie dogs	4
Treatment of minks	4
Field Evaluations—Efficacy and Hazards	5
General Methods	5
Design and Results of Individual Trials	6
Trial I	6
Trial II	7
Trial III	7
Trial IV	7
Hazards to Nontarget Species During Trials	7
Birds	7
Carnivores	8
Discussion and Conclusions	8
Field Evaluations—Zinc Phosphide Residues in Vegetation	9
Experimental Design, Treatment, and Sampling	10
Residue Analyses	10
Results of Residue Experiments	11
Application of the Method	12
ACKNOWLEDGMENTS	13
LITERATURE CITED	14

ABSTRACT

A program was undertaken to develop zinc phosphide as a replacement for the more hazardous toxicants currently available to control black-tailed prairie dogs (*Cynomys ludovicianus*). Laboratory grain acceptance tests, LD_{50} determinations, and bait development bioassays led to selection of 2% zinc phosphide-treated oats. In 30-day secondary hazard bioassays, minks (*Mustela vison*) showed no effects from eating entire carcasses of prairie dogs killed with this bait. Four field trials at 15 black-tailed prairie dog colonies in Montana, Colorado, and Nebraska resulted in consistently high reductions in prairie dog activity (76–96%) when colonies were prebaited and the bait was applied in surface bait spots at the low rate of 4 g per burrow. Extensive surveys during these trials revealed no primary or secondary hazards to any nontarget species. Experiments to measure zinc phosphide residues in range vegetation demonstrated that baiting, even at a much higher rate than 4 g per burrow, caused virtually no environmental contamination. This series of studies produced a recommended method for control of black-tailed prairie dogs: prebaiting followed by one surface application, at 4 g per burrow, of a bait formulated from steam-rolled oats, 2% zinc phosphide, and 1% corn oil. All evidence from the laboratory and field tests indicates that this treatment is effective and safe, and that the risk of its resulting in either primary or secondary intoxication of nontarget vertebrates, including black-footed ferrets (*Mustela nigripes*), is remote.

THE PROBLEM

In general, grazing of rangeland vegetation by black-tailed prairie dogs (*Cynomys ludovicianus*) is no longer considered a serious economic problem in the western United States. Nevertheless, prairie dog colonies still exist on many National Grasslands and Forests, on other lands administered by Federal agencies such as the Bureau of Land Management, National Park Service, and Bureau of Indian Affairs, and on many State lands and private holdings. Large areas of rangelands could be overgrazed if all these colonies were allowed to expand uncontrolled. Therefore, the need still exists for some efficient method of reducing local prairie dog populations.

Most of the available information on control methods for black-tailed prairie dogs was published before the mid-1950's and is now rather outdated (Merriam 1901, 1908; Lantz 1905, 1909, 1916, 1918; Spaulding 1912; Swenk 1915; Bell and Piper 1915; Bell 1918, 1921; Melton 1922; Johnson 1925; Stephl 1927; Cates 1937; Ward and Spencer 1947; and USDI 1956). In summary, black-tailed prairie dogs could be easily controlled by grain baits treated with thallium, strychnine, or 1080 (sodium monofluoroacetate); the latter was recommended in the late 1940's as the principal rodenticide for this species (refer to Atzert [1971] for a contemporary review of 1080). For all practical purposes, research to develop other control methods for prairie dogs ceased at this time.

Although 1080 is probably the most efficient acute toxicant ever developed for controlling commensal and field rodents, its potential for causing primary and secondary hazards to nontarget wildlife and domestic stock severely restricts or precludes its use in many damage situations. Thallium and strychnine have never been restricted as closely as 1080, but grain baits containing these toxicants are in certain situations also considered extremely hazardous to many nontarget species. In 1964, the Leopold Committee strongly recommended that 1080 be banned as a rodenticide and be replaced with "other chemicals which are not readily transmitted to scavenging animals" (Leopold 1964:39). This Committee's recommendations were carried one step further when, on 8 February 1972, Presidental Executive Order 11643 (later amended by Executive Order 11870, 18 July 1975) defined specific limitations under which chemical agents may be used on Federal lands or in other Federal programs authorized by law. The Order specifically restricts the use of toxicants that cause any secondary poisoning effects and further states that all damage control programs "shall be conducted in a manner which contributes to the conservation and protection, to the greatest degree possible, of the Nation's wildlife resources." These restrictions are important, but they are subject to interpretation and they apply only to Federal jurisdiction. Although thallium may no longer be used as a rodenticide in the United States, many States still permit the use of both stychnine and 1080 for prairie dog control, and strychnine-treated grain is still Federally registered for this purpose.

The problem of safe and efficient control of black-tailed prairie dogs is compounded by the presence of black-footed ferrets (*Mustela nigripes*), an endangered species which is closely associated with black-tailed prairie dogs and is at least partially dependent on them as a food source (Cahalane 1954; Hillman 1968; Henderson et al. 1969; Sheets and Linder 1969; Fortenbery 1972; Linder et al. 1972; Sheets et al. 1972; and Snow 1972). Programs to control black-tailed prairie dogs with 1080 run a clear risk of secondary exposure and death of ferrets. Therefore, safer control methods are needed.

The development, evaluation, documentation, and registration of a control method takes considerable time and resources, especially if the proposed technique or material is new and supporting data on its efficacy and hazards are not already available. Faced with the problem of quickly developing a safe method for controlling black-tailed prairie dogs, my research group turned to an older rodenticide that has proved fairly effective and largely free of secondary hazards: zinc phosphide. We felt that the problem could be solved if we found a formulation and application technique for zinc phosphide-treated grain that was: (1) well accepted by prairie dogs, (2) consistently lethal to a large percentage of the baited population, (3) safe to predators or scavengers that might eat animals killed by the bait, (4) unlikely to kill other grain-eating species near baited prairie dog colonies, (5) noncontaminating to soil and vegetation, and (6) economical to formulate and apply.

This report details the development and testing of a formulation and application technique that meets these criteria. I have reported the development process step by step because I believe it illustrates the kind of concern, not only for efficacy, but for consistency and safety to nontarget species and the environment that is essential in today's control methods research.

DEVELOPMENT OF ZINC PHOSPHIDE BAIT

The development and evaluation of a zinc phosphide-treated grain bait for use against black-tailed prairie dogs followed a set of standardized procedures developed or adapted by the Fish and Wildlife Service's Wildlife Research Center at Denver, Colorado. The test regime consists of a series of experiments designed to evaluate effectiveness and hazards and to determine the degree to which candidate rodenticides meet criteria for registration by the U.S. Environmental Protection Agency. The series is largely sequential, with the results of one step governing the factors chosen for testing in the next step.

Laboratory Bioassays

Grain Acceptance Test

In this preliminary test, the objective was to establish the kind of grain carrier that black-tailed prairie dogs preferred. Five untreated grains—white wheat, milo maize, rolled barley, steam-rolled oats, and oat groats—were each presented to five individually caged test animals for 3 consecutive days. Each test animal received 50 g of grain per day, plus laboratory chow pellets and water *ad libitum*; grain consumption was recorded daily. The experimental design was a 5 × 5 Latin square (Snedecor and Cochran 1971).

Analysis of variance and Scheffé's test (Scheffé 1959), based on mean grain intake over 3 days, indicated only one statistically significant relationship: the animals ate less wheat than they did the other four grains ($P = 0.10$). However, the data showed that they clearly preferred rolled barley, steam-rolled oats, and oat groats (Table 1). Because of more consistent daily consumption, oat groats was selected as the standard bait carrier for all laboratory bioassays and initial field trials.

LD_{50} and Initial Bait Acceptance Bioassay

The LD_{50} (the dose calculated as lethal to 50% of a test population) of zinc phosphide to black-tailed prairie dogs was determined by the

Table 1. *Grain acceptance test: consumption of untreated grain bait carriers by black-tailed prairie dogs (five animals per grain).*

Grain tested	Mean consumption per animal (g)			
	Day 1	Day 2	Day 3	3-day mean
White wheat	5.14	11.89	5.34	7.45
Milo maize	11.55	13.11	6.47	10.37
Rolled barley	12.87	12.73	16.52	14.04
Steam-rolled oats	10.11	18.86	14.20	14.39
Oat groats	11.19	17.24	19.24	15.89

Table 2. *Initial bait acceptance and bait development bioassays: Acceptance and mortality when various concentrations of zinc phosphide-treated oat groats were offered to black-tailed prairie dogs (10 animals per concentration).*

Bait concentration % Zinc phosphide	Amount of bait (g) containing one LD_{50} dose[a]	Mean no. of LD_{50} doses[a] consumed on Day 1	Total mortality (%)
	Initial bait acceptance bioassay		
0.91	1.59	2.34	70
	Bait development bioassays (Phase I)		
0.75	1.87	1.28	70
1.0	1.48	2.02	90
1.5	0.98	3.60	80
2.0	0.71	4.18	90
3.0	0.61	4.00	100
	Bait development bioassays (Phase II)		
2.0	0.67	5.05	100
2.0	0.70	4.31	90
2.0	0.62	5.80	100
2.0	0.51	6.18	80

[a] Based on an LD_{50} of 18.0 mg/kg and the mean body weight for each group of 10 prairie dogs.

method of Thompson (1947) and Weil (1952). The zinc phosphide[1] was administered in a corn oil suspension by oral gavage. All test animals were made to fast overnight, treated, and offered laboratory chow pellets and water *ad libitum* during a 14-day posttreatment observation period. The LD_{50} value for a mixed sample of males and females was 18.0 mg/kg of body weight (95% confidence limits, 12.00–27.01 mg/kg).

Following the LD_{50} determination, zinc phosphide was evaluated on the carrier in an initial acceptance bioassay to develop baseline data on acceptance and mortality. The concentration of the zinc phosphide on the bait, 0.91%, was equivalent to one LD_{50} (18.0 mg/kg) on one-tenth the average daily consumption of the untreated oat groat carrier (1.589 g). The bait was formulated by applying a 1.0% zinc phosphide-corn oil suspension to the oat groats and mixing until the coating was equally distributed and relatively dry. Each of 10 individually caged prairie dogs was offered 40 g of fresh bait per day for 3 days or until death; survivors were observed 4 days longer. Consumption of bait and mortality were recorded daily. Laboratory chow pellets and water were freely available in each cage before, during, and after the 3-day test.

The 10 animals in this test consumed a mean of 2.3 LD_{50} doses the first day they were offered bait (range, 0.4–7.4 LD_{50} doses), and 70% of them had died by the 4th day (Table 2). These results indicated that zinc phosphide was sufficiently active against black-tailed prairie dogs to warrant further testing.

Bait Development Bioassays

This second evaluation of zinc phosphide bait was designed to identify the most efficient concentration for field testing. Again, the criteria were acceptance and mortality. The concentration of zinc phosphide was the only variable tested; the bait carrier (oat groats), formulation (corn oil-zinc phosphide suspension overcoated on the carrier), and test protocol were the same as those used in the initial bait acceptance bioassay. The development bioassays were conducted in two phases: Phase I tested several incremental concentrations to compare acceptance and mortality; Phase II focused on the most efficient concentration isolated during Phase I to determine if the results were consistently repeatable.

Five concentrations of zinc phosphide were evaluated during Phase I (Table 2), but only those in the mid-high range of 1.5–3% produced good acceptance and relatively short times

[1]Throughout all tests, the zinc phosphide used was technical grade material containing 94% active ingredient. All concentrations reported are based on weight of active zinc phosphide present.

until death. Because the 2% concentration was accepted well, it was selected for further study in Phase II (Table 2). These tests provided additional evidence that the 2% bait was well accepted; the four groups of test animals consumed from 4.3 to 6.2 LD_{50}'s the first day, and 80–100% died. At the end of this test sequence, the 2% bait was specified as the standard for all further laboratory studies and initial field trials.

Secondary Hazard Bioassays

This experiment was conducted to determine if killing prairie dogs with the 2% bait would result in secondary poisoning of carnivores, specifically, the endangered black-footed ferret. To represent ferrets, we used the closely related domestic mink (*Mustela vison*).

Treatment of prairie dogs.—To supply the prairie dogs needed to sustain the long-term secondary hazard study, and to further test the consistency of results with the 2% bait, seven additional bioassays were conducted. The carrier, formulation, and test protocol were the same as those used in preceding bioassays, except that test groups contained 5 animals rather than 10. All prairie dogs that died from zinc phosphide poisoning during the 3-day test or the 4-day posttest observation period were tagged for identification and immediately frozen until fed to minks; survivors were disposed of.

The test animals showed marked variations in bait consumption, but except for test 3 (in which disturbance caused the animals to eat little food of any kind), acceptance and mortality were similar to those in previous tests with the 2% bait. Excluding test 3, the six groups consumed a mean of 3.6–7.9 LD_{50}'s the first day, and 28 (93%) of the 30 animals died (Table 3). These results provided additional evidence of this bait's consistent effectiveness against black-tailed prairie dogs.

Treatment of minks.—We tested 10 individually caged adult minks, 3 males and 2 females each for the treated and the control diet. To prepare the treated diet, all dead prairie dogs were thawed and the skin and feet were removed. The remaining carcass, including the complete digestive tract, was ground into a hamburger-like consistency. This material was thoroughly homogenized with standard commercial mink ration, at a ratio of 2.5:1, and frozen until fed. Each test mink received 200 g per day of the treated diet, which contained 71.5% (143 g) ground prairie dog, for 30 consecutive days. After treatment the mink were given 200 g per day of untreated ration during a 15-day posttest observation period. Each control mink received 200 g per day of untreated mink ration throughout the 45 days. Water was freely available to all animals but no other food was provided.

During the test, the theoretical maximum daily dose of zinc phosphide for each test mink (assuming no detoxification or elimination by the prairie dogs) was 16.8 mg, or a mean of 13.6 mg/kg based on the minks' pretest weight of 1.24 kg. Over 30 days, this amounted to 506 mg, or a mean of 407 mg/kg, per mink.

Table 3. *Preliminary bioassays for secondary hazard study: Acceptance, mortality, and intake of toxicant when 2% zinc phosphide-treated oat groats were offered to black-tailed prairie dogs (five animals per test).*

Test no.	Amount of bait (g) containing one LD_{50} dose[a]	Mean no. of LD_{50} doses[a] consumed on Day 1	Total mortality (%)	Total zinc phosphide consumed by animals that died (mg/kg)	
				Mean	Range
1	0.73	4.36	100	85.5	8.7–225.1
2	0.61	5.90	100	105.5	3.5–156.9
3	0.66	1.84[b]	40[b]	36.2	8.8–110.7
4	0.74	6.92	80	140.0	45.0–341.9
5	0.57	3.64	100	117.2	59.6–198.3
6	0.66	7.93	100	141.9	81.4–209.7
7	0.51	6.18	80	126.5	60.7–279.7

[a] Based on an LD_{50} of 18.0 mg/kg and the mean body weight for each group of five prairie dogs.

[b] Poor results attributed to repeated disturbance of test animals; data not used, but the two animals killed were fed to minks.

There was no observable reaction, acute or subacute, in the test minks during the 30-day test or the 15-day posttest observation period. Throughout the test period, all five mink continued to eat a full 200-g daily ration consisting mainly of zinc phosphide-treated prairie dog. The control group also maintained a consistent intake of 200 g per day of untreated diet. We concluded that long-term feeding on entire carcasses of black-tailed prairie dogs killed with 2% zinc phosphide bait caused no gross secondary effects.

Field Evaluations—Efficacy and Hazards

Field tests were divided into four distinct yet interrelated trials to evaluate as many variables as possible. The primary objectives of the total program were to assess the efficacy of the 2% zinc phosphide grain bait as influenced by: (1) application rate, (2) prebaiting, (3) type of carrier, (4) baiting site (on the surface or in burrows), and (5) test locality (geographic variation).

General Methods

The four trials were conducted in summer and fall at prairie dog colonies in Montana, Colorado, and Nebraska. In each trial, three to five well-separated colonies were used, one for each baiting treatment being evaluated, plus one reference (untreated) colony to define baseline activity levels during the pre- and post-treatment periods. Before baiting, each colony was marked off into one to eight test cells, roughly similar areas of 0.4-1.2 ha containing different numbers of burrows (single tunnel systems). To eliminate bias, all burrows in each colony, including those apparently inactive, were treated as active. For comparison, the reference colony was treated exactly like the others, except that the "bait" used was untreated oats.

Before baiting, all reference colonies and most treatment colonies were prebaited with untreated grain. Prebaiting is a standard method of increasing bait consumption by conditioning the target animals to the new food source. The prebait is untreated grain of the same type as the bait carrier and is applied in the same way and at the same rate. Baiting is delayed until the test population is eating all or most of the prebait; in these trials this process required only 24 h.

For all but one treatment (in trial IV), the prebait and bait were applied on the surface in small bait spots. This is a standard hand-baiting method in which a measured spoonful of bait is dropped from waist height to cover about 0.1 m^2 of ground surface. For prebaiting and baiting, one bait spot was applied at each burrow (active or inactive), either on the edge of the mound surrounding the burrow entrance or on a nearby feeding area.

During the pre- and post-treatment periods, prairie dog activity was measured in each cell by plugging all burrows (active and inactive) with dry cow chips and recording the number that had been reopened after 48 h. The effectiveness of the various treatments was evaluated by comparing the pre- and post-treatment counts in each colony and calculating the percentage by which activity had increased or decreased.

The following treatment schedule was developed during trial I and followed during trials II, III, and IV:

Pretreatment period

- Day 1. Survey the area in and around each colony to be used (both reference and treated). Verify that there are no signs of black-footed ferrets. Count and remove any dead animals found.
- Day 1-2. Divide all colonies into test cells and mark off the cells with stakes.
- Day 2. Close and tally all burrows in each cell.
- Day 4. Survey each cell and record the number of burrows reopened. Apply prebait in colonies where the experimental design calls for it.

Treatment period

- Day 5. In each treated colony, apply zinc phosphide bait as specified by the experimental design; apply additional prebait in the reference colony.

Posttreatment period

Day 8. At 72 h after baiting, close and tally all burrows in each cell.

Day 10. Survey each cell and record the number of burrows reopened. Survey the area in and around each colony; record the number and species of all dead animals found.

Design and Results of Individual Trials

The variables compared in the four trials and the results of each are summarized in Table 4. No geographic variation was shown. Preliminary mathematical evaluations of the data indicated that the method used to measure efficacy (counts of burrows reopened before and after baiting) provided a reliable estimate of treatment-related reductions in prairie dog activity. Statistical analysis (Chi^2 contingency tests) comparing actual and expected values revealed a consistent pattern that defined significant differences ($P<0.05$) between pre- and post-treatment activity in the treated colonies and none in the reference colonies. As in the laboratory tests, analysis of the results of each trial defined the variables to be tested in the next trial.

Trial I.—This first trial was conducted in July and August 1972 at three prairie dog colonies on the Charles M. Russell National Wildlife Range, Lewistown, Montana. The colonies were prebaited, and the bait formulation used was the one that proved successful in the laboratory (2% zinc phosphide in corn oil on oat groats). The trial was designed to compare a high and a low rate of bait application. The high rate, about 14 g or 1 heaping tablespoon per burrow, was the rate used successfully in prairie dog baiting programs with strychnine or 1080. The low rate, about 4 g or 1 heaping teaspoon per burrow, was an amount calculated from laboratory data as ample for maximum control. In bioassays (Tables 2 and 3), prairie dogs normally ate 3–5 g of 2% bait during the first 24 h it was exposed. Because 4 g is equivalent to about 6 LD_{50}'s for an average-size adult, we reasoned that this lesser amount should be more than enough to kill any prairie dog that ate even half of it, but should reduce the amount of surplus bait and thus the hazards to nontarget species.

Table 4. *Field efficacy trials: Change in activity at black-tailed prairie dog colonies after single baitings with 2% zinc phosphide-treated oats, various formulations and application methods.*

Trial	Colony	Type of oats: prebait & bait	Adhesive for bait	Applition rate and site[a]		Pre-baited?	Total no. in colony		Total no. of burrows active in 48-hr count		% change in activity between pre- and post-treatment counts	
							Cells	Burrows	Pre-treat.	Post-treat.	Range among cells	Mean for colony
I	1 Ref	Groats	—	High	S	Yes	6	377	300	268	+ 4.9 to -22.7	-10.7
	2 Tr'd	Groats	Corn oil	High	S	Yes	8	420	316	46	-77.2 to -97.9	-85.4
	3 Tr'd	Groats	Corn oil	Low	S	Yes	7	617	474	57	-85.2 to -95.5	-88.0
II	4 Ref	Groats	—	Low	S	Yes	4	267	110	126	+24.0 to + 6.6	+12.7
	5 Tr'd	Groats	Corn oil	Low	S	Yes	4	389	221	38	-71.8 to -91.8	-82.8
	6 Tr'd	Groats	Corn oil	Low	S	No	3	305	226	63	-58.5 to -81.3	-72.1
III	7 Ref	Rolled	—	Low	S	Yes	4	465	157	147	+12.1 to - 9.1	- 6.1
	8 Tr'd	Rolled	Corn oil	Low	S	Yes	3	211	132	5	-91.6 to -100	-96.2
	9 Tr'd	Rolled	Corn oil	Low	S	No	3	190	106	47	-38.9 to -76.0	-55.7
	10 Tr'd	Groats	Corn oil	Low	S	Yes	3	236	127	29	-69.6 to -84.4	-77.2
	11 Tr'd	Groats	Corn oil	Low	S	No	3	259	154	87	-31.1 to -59.6	-43.5
IV	12 Ref	Rolled	—	Low	S	Yes	1	219	148	146	- 1.4	- 1.4
	13 Tr'd	Rolled	Alcolec S	Low	S	Yes	1	131	75	18	-76.0	-76.0
	14 Tr'd	Rolled	Corn oil	Low	S	Yes	1	129	82	13	-84.0	-84.0
	15 Tr'd	Rolled	Corn oil	High	B	Yes	1	376	194	116	-40.2	-40.2

[a] Application of prebait and bait: High rate = 14 g/burrow; low rate = 4 g/burrow; S = placed in spot on surface; B = placed in burrow.

In both treated colonies, prairie dog activity was significantly reduced after baiting, but there was no significant difference in the amount of reduction between the high and low application rates (Table 4). At the high rate, uneaten bait, often 5-10 g per burrow, was visible at bait spots for up to a week. At the low rate, the only remains of bait visible at 24 h were small amounts of chaff. The similarity in effectiveness of the two rates and the obvious reduction in hazards to nontarget species with the low rate led to the first tentative bait-treatment standard: 2% zinc phosphide on oat groats, low application rate.

Trial II.—This trial was conducted in September 1972 at three other prairie dog colonies on the Charles M. Russell National Wildlife Range. The oat groat bait was applied at the low rate, and prebaiting was compared with no prebaiting to determine if this extra step was necessary.

Prairie dog activity was reduced significantly more in the treated colony that was prebaited than in the one that was not (Table 4). This led to a second tentative bait-treatment standard: 2% zinc phosphide on oat groats, low application rate, prebaited.

Trial III.—This trial was conducted in October 1972 at five colonies at Fort Carson (Colorado Springs), Colorado. Oat groats were compared with the less expensive steam-rolled oats as a carrier for the 2% bait at the low application rate, and prebaiting was again compared with no prebaiting.

In this four-way comparison, activity was reduced significantly more in the two treated colonies that were prebaited than in the two that were not, but there were no significant differences between the oat groats and the steam-rolled oats as a bait carrier (Table 4). These results further supported the theory that prebaiting increases efficacy and showed that the cheaper rolled oats could be used. This led to a third tentative bait-treatment standard: 2% zinc phosphide on steam-rolled oats, low application rate, prebaited.

Trial IV.—This trial was conducted in August and September 1973 at four colonies on private rangelands near Sydney, Nebraska. This final series was designed to evaluate two possible improvements in the formulation of the rolled oat bait and the method of applying it: (1) The corn oil adhesive was compared with another commercial adhesive, Alcolec S[2], and (2) the surface bait-spot method at the low rate, used for the corn oil and Alcolec S baits, was compared with application of the bait inside the burrow entrance, a possible way of further reducing surplus bait on the surface. The high application rate was used for in-burrow baiting because the bait scattered widely as it was tossed down the burrow entrance.

There was no significant difference in post-treatment activity at the two colonies where corn oil was compared with Alcolec S and bait was applied on the surface, but activity was reduced significantly more at these two colonies than at the one where the bait was applied inside burrows (Table 4). These results led to the final recommended bait-treatment standard: 2% zinc phosphide on steam-rolled oats, formulated with 1% corn oil, applied on the surface in bait spots, low application rate, prebaited.

Hazards to Nontarget Species During Trials

A variety of nontarget wildlife species were active in the vicinity of test colonies during the four trials. Extensive surveys in and around all colonies before, during, and after zinc phosphide baiting showed no evidence of either primary or secondary hazards to any species.

Birds.—Large numbers of birds, mostly horned larks (*Otocoris alpestris*) and mourning doves (*Zenaidura macroura*), were frequently seen in or near treated colonies. Horned larks were often seen walking or standing on prairie dog mounds and, during baiting, were sometimes seen picking up and then dropping treated oats at bait spots. Examination of these spots showed that the larks had broken up some of the oats but apparently had eaten little or none. Observations at treated colonies, on surrounding rangelands, and at stock-watering tanks in the general vicinity failed to show any sick or recently dead birds of any species.

[2] American Lecithin Co., Inc. Reference to trade name does not imply U.S. Government endorsement of commercial products.

Carnivores.—Most of the zinc phosphide-killed prairie dogs died inside their burrows, but 25 were found on the surface 6–8 h after bait exposure, mostly in trial I in the colony treated at the high application rate. By design, these carcasses were left undisturbed, and all were missing the following morning. Signs in the vicinity indicated that they had been taken by coyotes (*Canis latrans*) or badgers (*Taxidea taxus*). For several days after baiting, badger activity increased in all treated colonies, probably because of the increase in a ready food supply—dead prairie dogs. In spite of clear signs that badgers were digging up and eating large numbers of zinc phosphide-killed prairie dogs, no badgers, and no coyotes or other carnivores, were found sick or dead during extensive ground searches in the treated colonies and surrounding areas.

Discussion and Conclusions

These four interrelated field trials provided clear evidence that 2% zinc phosphide-treated oats is an effective, economical, and safe treatment for control of black-tailed prairie dogs, and thus a logical replacement for strychnine and 1080. However, any toxic bait has some potential for primary and secondary hazards, and one may still ask if additional safeguards are needed to protect nontarget species, either from the bait or from carcasses of animals that have eaten it. A variety of responses are possible with zinc phosphide baiting, depending on several interacting factors. These include (but are not limited to): (1) the comparative toxicity of zinc phosphide to the target and various nontarget species, (2) its rate of detoxification or elimination when ingested by these species, (3) the amount of bait the various species eat, (4) the numbers and distribution of animals killed, (5) the amount of surplus bait that remains on the surface after the control operation, (6) the time this residual bait remains available and toxic, (7) the food habits and preferences of nontarget species, and (8) the numbers and distribution of nontarget species on treated areas. Some of the zinc phosphide studies published over the last 30 yr illustrate this broad spectrum of primary and secondary responses.

Hayne (1951), in a series of laboratory tests with zinc phosphide-treated cracked corn, concluded that this particular bait type was extremely hazardous to pheasants (*Phasianus colchicus*). The U.S. Fish and Wildlife Service (1964) reported the accidental poisoning of several hundred geese at Tule Lake, California, after treated fields were burned, exposing residual zinc phosphide-treated oat groats. Reports from Europe (Gotink and van Hulsen 1952) showed similar primary hazards to geese, ducks, and other wildlife that encountered grain baits. On the other hand, Siegfried (1968), who studied primary hazards to crowned guinea fowl (*Numida melagris*) and laughing doves (*Stigmatopelia senegalensis*) and secondary hazards to spotted eagle owls (*Bubo africanus*), observed adverse effects only once, when a sample of 4 out of 12 doves that had fasted for 30 h died after eating zinc phosphide-treated wheat. Unstressed guinea fowl and doves either totally ignored the bait or left it untouched after a brief initial inspection. Hines and Dimmick (1970) reported that bobwhite quail (*Colinus virginianus*) were repelled by zinc phosphide-treated oats and that all test birds remained healthy and vigorous at the conclusion of their tests.

Rudd and Genelly (1956) reported that it may take several days for ingested zinc phosphide to completely break down in the gut of target animals and that secondary poisoning is a definite hazard during that period. Doty (1945) stated that domestic cats and mongoose (*Herpestes* sp.) were not affected when fed rats killed with zinc phosphide, but Chitty (1954) reported secondary hazards when cats were fed rats killed with 5% zinc phosphide (a high concentration). Chitty remarked that the evidence for cats and other carnivores being killed by secondary poisoning is conflicting but believed that such kills undoubtedly take place under certain conditions. Brock (1965), who fed zinc phosphide-killed rodents to gopher snakes (*Pituophis catenifer*), reported regurgitation but no other response. Evans (1966, unpublished annual report, Denver Wildlife Research Center), who fed zinc phosphide-killed nutria (*Myocaster coypus*) to bald eagles (*Haliaeetus leucocephalus*), black vultures (*Coragyps atratus*), minks, cats, and dogs found that the only animals acutely poisoned were one cat and one dog that ate stomach contents. However, he considered the hazards to free-ranging cats and dogs as negligible. Evans et al. (1970) reported that golden eagles (*Aquila chrysaetos*), great horned owls (*Bubo virginianus*), and coyotes that received multiple feedings of zinc phosphide-killed black-tailed jackrabbits (*Lepus californicus*) showed no visible symptoms of secondary intoxication. Schitoskey (1975), in a series of acute secondary hazard tests, found that kit foxes (*Vulpes macrotis*) survived repeated feedings of Kangaroo rats (*Dipodomys* sp.) each dosed with 480 mg of zinc phosphide (equivalent to 3 LD_{50}'s per fox). Two of the test foxes consumed nine LD_{50} doses in 3 days and survived. The author partially attributed this lack of secondary effects on the strong emetic properties of zinc phosphide. Most of these secondary hazard studies indicate that toxic residues are not widely distributed in the carcasses of animals killed by zinc phosphide baits but are limited to the alimentary tract.

The complete and undiluted stomach contents of a prairie dog could contain enough zinc phosphide to cause secondary reactions in black-footed ferrets. However, hazards are considered minimal because zinc phosphide is subject to *in situ* detoxification in carcasses and the stomach and contents are not a preferred food of the ferret (Henderson et al. 1969). Our 30-day secondary hazard bioassay showed no chronic secondary intoxication in minks, even though they readily ate food prepared from entire carcasses, including the alimentary tract.

It is difficult to be completely objective in reviewing the published data on primary and secondary hazards of zinc phosphide baiting because of the many intrinsic and extrinsic variables that prevent absolute conclusions. For each positive case where hazards were shown, there is often an equally significant negative one where they were not. Under these circumstances, one basic observation may be more valid than the conclusions (pro or con) made for any individual study; if all safety precautions are followed and careful attention is paid to how, when, and where zinc phosphide-treated baits are formulated and applied, operations can be carried out without undue risks to nontarget species.

In our studies, the primary and secondary hazards to the numerous domestic and wild species that coinhabit the rangelands with black-tailed prairie dogs were not investigated beyond the scope I have described. However, if prairie dog colonies are treated according to the final recommended bait-treatment standard for 2% zinc phosphide-treated steam-rolled oats, several factors should contribute to low primary and secondary hazards: (1) the food habits, preferences, and feeding patterns of the domestic and wild nontarget species; (2) the relatively low concentration of zinc phosphide in the bait; (3) the small amount of bait applied per unit area; (4) the widely scattered bait distribution pattern; and (5) the short time most of the bait is exposed. Taking these into consideration, we believe that the baiting treatment we recommend will present no significant hazards to nontarget species, and we make no recommendation that these species be removed or kept out of prairie dog colonies scheduled for treatment. But we do emphasize that the method described should be carefully followed and used with due attention to the welfare of nontarget species and the environment.

Field Evaluations—Zinc Phosphide Residues in Vegetation

The recommendations made at the completion of the field efficacy trials called for 2% zinc phosphide-treated steam-rolled oats to be dropped from a measure onto a surface bait spot near each prairie dog burrow. This method of application should theoretically produce minimum environmental contamination. Zinc phosphide acts as a toxicant by liberating toxic phosphine gas (PH_3) when it is hydrolyzed; phosphine is highly reactive, and the end products are simply zinc complexes and phosphates. Since the vegetation around prairie dog burrows, when it occurs, is low-growing grasses and small forbs, few dropped bait particles should lodge there. Most should rapidly fall through to the ground. The work of

Hilton and Robison (1972) and Hilton et al. (1972) showed that zinc phosphide is rapidly and thoroughly decomposed on contact with soil. However, there was still the possibility that some zinc phosphide could be eroded from the bait during application or weathering and deposited on vegetation, still in its original, potentially toxic form. Experiments were therefore conducted to measure zinc phosphide residues in rangeland vegetation after baiting.

Experimental Design, Treatment, and Sampling

Two experiments were conducted, both in rangeland areas where field efficacy trials were run. In each experiment, treated oat baits (2% zinc phosphide, 1% corn oil) were hand broadcast over the entire surface of three fenced exclosures, and zinc phosphide residues were measured in vegetation from these and from a fourth untreated exclosure at four sampling intervals—1, 15, 30, and 60 days after bait distribution. In experiment I, conducted in August-October 1972 at the Charles M. Russell National Wildlife Range, Montana, oat groat baits were broadcast in separate exclosures at three multiples (1X, 3X, and 10X) of the high bait application rate (14 g, 42 g, and 140 g per 0.1 m^2). In experiment II, conducted in October-November 1973 at Fort Carson, Colorado, steam-rolled oat baits were broadcast at the same three multiples of the low application rate (4 g, 12 g, and 40 g per 0.1 m^2). At the Russell Wildlife Range, the habitat type was a park-like short- and mid-grass prairie association dominated by perennial grasses, annual and perennial forbs, and sagebrush (*Artemesia* sp.); surrounding areas were dominated by monotypic stands of ponderosa pine (*Pinus ponderosa*). At Fort Carson, the type was transition zone short- and mid-grass rangeland with many complex subcommunities; the area where the residue experiments were run was dominated by needlegrass (*Stipa* spp.), wheatgrass (*Agropyron* spp.), and annual and perennial forbs.

The eight experimental exclosures (four on each study area) were wire-fenced rectangles measuring 3.1 × 3.7 m (10 × 12 ft). Each exclosure was divided into three replicate cells about 3.1 × 1.2 m, and each cell was gridded into 40 vegetation sampling units of 0.1 m^2 each. At each sampling interval, five previously unsampled 0.1-m^2 units in each cell were selected by a predetermined random number sequence. Vegetation in these five units was marked off with a 0.1-m^2 hoop, clipped off at 2.5 cm above ground level, and combined to form one composite sample per cell. The 12 samples taken at each interval (four exclosures, three cells each) were labeled, frozen, and stored until analysis.

Residue Analyses

Analyses were conducted at the Denver Wildlife Research Center by the method developed by Okuno et al. (1975). The composite vegetation samples were thawed and cut into about 1-cm lengths. To minimize losses of zinc phosphide or cross contamination through excessive handling, they were not blended or milled to increase homogeneity but were thoroughly mixed by hand. Each sample was analyzed by sealing a 1-g aliquot in a flask, acidifying to hydrolyze zinc phosphide to phosphine, sampling the gas from the headspace, and measuring it by gas chromatography with a flame photometric detector. This method detects 0.01 ppm or less of zinc phosphide in 1 g.

As Okuno et al. (1975) pointed out, when zinc phosphide residues are measured in vegetation samples, recoveries vary widely with the type and condition of the plant material. Recoveries cannot be reliably calculated by fortifying treated samples ("overspiking") because the residual zinc phosphide in such samples is not evenly distributed. Under these conditions, the best method for determining recoveries appears to be spiking of untreated vegetation; if the untreated samples are taken from the same area as the treated samples and are stored and handled in the same way, the type and condition of the two plant samples should differ only slightly. In these experiments, recoveries were estimated by taking vegetation samples from the untreated exclosures just before bait distribution, spiking them with 0.5 μg of zinc phosphide, and analyzing by comparison with a secondary standard (Okuno et al. 1975). During experiments I and II recoveries averaged 55% (range, 53–59%). The meaning of these figures is uncertain, however. Recoveries from spiked samples may be low because the zinc phosphide generated by hydrolysis is being immediately sorbed on the sample surface. A recovery correction for treated field samples would be appropriate for only that unknown proportion of the loss that results from the sorption of phosphine. Therefore, the residue values reported here are not corrected for recovery.

Results of Residue Experiments

The residue values found are listed in Table 5. Low levels of zinc phosphide were detected in vegetation from the untreated exclosures in both experiments. To determine if this contamination was present before baiting or resulted from the analysis, an additional series of samples was collected just before bait distribution in experiment II. Since these pretreatment samples were negative for zinc phosphide, we assumed that the low levels in samples from the untreated exclosures resulted from mechanical transfer of zinc phosphide, probably from handling or wind drift from treated areas.

In general, zinc phosphide levels in vegetation from the three replicate cells of an exclosure

Table 5. *Field residue experiments: Zinc phosphide residues (measured as phosphine) in samples of rangeland vegetation taken at intervals from exclosures where 2% zinc phosphide-treated oats were broadcast at 1, 3, and 10 times the high- and low-application rates.*

Experiment	Exclosure	Amount of bait applied (g/0.1 m²)	Cell	Zinc phosphide in vegetation (ppm) at intervals before and after baiting				
				Pre-treat.	Day 1	Day 15	Day 30	Day 60
I (High rate)	1 Untreated	0	A		0.03[a]		0.02[a]	
			B				0.03[a]	
			C		0.02[a]		0.02[a]	
	2 Tr'd, 1X	14	A		2.6		2.0	
			B		2.0		1.6	
			C		2.3		1.3	
			Mean		2.3		1.6	
	3 Tr'd, 3X	42	A		8.5		2.4	
			B		4.3		6.6	
			C		1.9		4.2	
			Mean		4.9		4.4	
	4 Tr'd, 10X	140	A		73.4	10.0	22.1	3.4
			B		91.9	7.6	20.7	3.1
			C		81.7	26.4	11.7	5.6
			Mean		82.3	14.7	18.2	4.0
II (Low rate)	5 Untreated	0	A	ND[b]	0.04[a]	Tr[c]	Tr[c]	Tr[c]
			B	ND[b]	Tr[c]	Tr[c]	Tr[c]	Tr[c]
			C	ND[b]	Tr[c]	Tr[c]	Tr[c]	Tr[c]
	6 Tr'd, 1X	4	A		0.8	0.3	0.2	0.03
			B		0.9	0.2	0.1	0.05
			C		0.3	0.1	0.1	0.07
			Mean		0.7	0.2	0.1	0.05
	7 Tr'd, 3X	12	A		0.6	0.7	1.8	0.04
			B		1.4	0.6	1.0	0.2
			C		1.6	0.6	0.7	0.1
			Mean		1.2	0.6	1.2	0.1
	8 Tr'd, 10X	40	A		5.7	2.3	5.6	0.4
			B		4.9	1.8	2.4	0.6
			C		6.0	1.2	0.8	39.0
			Mean		5.5	1.8	2.9	13.3

[a] Contamination from unknown causes. Sample was run at least twice; results of last run are given.
[b] ND = not detected
[c] Tr = trace, detected but <0.01 ppm.

differed little at any given interval and showed a steady decrease from day 1 through day 60 (Table 5). There were exceptions—figures were higher in some cells than in companion cells or than in the same cell at earlier intervals. One 60-day sample (experiment II, exclosure 8, cell C) had a particularly high value, indicating strong contamination. The several unexpectedly high values may have reflected nonhomogeneity or cross contamination of the samples, or "hot spots" resulting from unequal bait distribution or wind drift against the exclosure walls. Although they suggest that more care should have been taken to avoid contamination in spacing and treating the exclosures, they have little practical significance in view of the low residue levels found throughout both experiments.

Because the entire surface of treated exclosures was baited, the residue values in Table 5 represent the level of zinc phosphide contamination in the middle of a treated bait spot, not that in an area where prairie dog colonies have been baited. The density of a typical colony is about 100 burrows per ha. If each burrow in such a colony were treated with one 0.1-m^2 bait spot, only 0.001 of the area would be baited and the mean residue levels in vegetation (Table 5) would be reduced by a like amount. On a colony basis (dividing by 1000), none of the values exceeded our proposed tolerance of 0.1 ppm zinc phosphide in rangeland vegetation even in the exclosure baited at 10 times the high rate. Thus, even within the limits of a treated colony, the probability that vegetation would retain significant zinc phosphide residues after one baiting at the recommended low rate, or even 10 times that rate, is essentially nil.

In addition, our field trials showed that when bait is applied at the low rate, prairie dogs eat virtually all of it within 24 h. Most of the zinc phosphide remaining on the few uneaten bait particles should readily hydrolize with weathering or contact with the soil; the phosphine released would rapidly dissipate into the atmosphere or react with vegetation or soil to form phosphates (Hilton et al. 1972). The pattern of residues in all treated exclosures—highest on day 1, steadily decreasing through day 60—suggests that plant residues result mainly from surface contamination with zinc phosphide, not absorption or translocation. Therefore, residual zinc phosphide on vegetation could likewise be expected to disappear with weathering. The following conclusion is indicated: Baiting black-tailed prairie dog colonies with 2% zinc phosphide-treated oats at the low application rate will result in very little short-term, and no detectable long-term, contamination of the environment.

Application of the Method

Several bait-treatment standards were evaluated during the extensive laboratory and field investigations of zinc phosphide. It is essential during the transition from the research to operational phases that the recommended standards be closely adhered to and adopted as instructions for use. A properly formulated bait, adequate prebaiting, and an application rate keyed to the individual problem situation are all important in achieving good control. The bait must be of high quality—well accepted and uniformly toxic—and just enough must be applied to expose all prairie dogs. Thus, a balance must be struck between underbaiting, which leads to marginal results, and overbaiting, which is uneconomical, potentially hazardous to nontarget species, and may lead to unnecessarily high zinc phosphide residues in range vegetation.

ACKNOWLEDGMENTS

I am deeply indebted to the following individuals for their assistance in the field phases of the zinc phosphide-prairie dog study: Frank R. Martin (retired) and William C. Kranz of the Charles M. Russell National Wildlife Range, Lewistown, Montana; Robert P. Kelly, Ernest J. Giese, Jr., and other staff members of the Fish and Wildlife Service Region 6 ADC office in Lincoln, Nebraska; and Durward Davis, Post Agronomist, Fort Carson (Colorado Springs), Colorado.

Special thanks are directed to my many colleagues at the Wildlife Research Center at Denver for their assistance in the supporting roles that made this a successful study—Glenn A. Hood, G. Keith LaVoie, John K. Crier, Robert E. White, Iwao Okuno, Richard A. Wilson, Sr., William H. Higgins, Charles P. Breidenstein, Margaret B. Musgrove, Ruth E. McNeal, and Ann H. Jones.

LITERATURE CITED

Atzert, S. P. 1971. A review of sodium monofluoroacetate (compound 1080) its properties, toxicology, and use in predator and rodent control. U.S. Fish Wildl. Serv., Spec. Sci. Rep. Wildl. 146. 34 pp.

Bell, W. B. 1918. Cooperative campaigns for the control of ground squirrels, prairie-dogs, and jack rabbits. Yearbook, U.S. Dep. Agr. for 1917:225-233.

Bell, W. B. 1921. Death to the rodents. Yearbook, U.S. Dep. Agr. for 1920:421-438.

Bell, W. B., and S. E. Piper 1915. Extermination of ground squirrels, gophers and prairie dogs in North Dakota. N.D. Agr. Exp. Sta. Circ. 4. 11 pp.

Brock, E. M. 1965. Toxicological feeding trials to evaluate the hazard of secondary poisoning to gopher snakes, *Pituophis catenifer*. Copeia 2:244-245.

Cahalane, V. H. 1954. Status of the black-footed ferret. J. Mammal. 35(3):418-424.

Cates, E. C. 1937. Control of rodent pests in Montana. Mont. State Coll. Ext. Serv. Bull. 151. 32 pp.

Chitty, D. 1954. Control of rats and mice, vol. 1. Oxford Univ. Press, London. 305 pp.

Doty, R. E. 1945. Rat control on Hawaiian sugarcane plantations. Hawaiian Planter's Rec. 49:71-239.

Evans, J., P. L. Hegdal, and R. E. Griffith, Jr. 1970. Methods of controlling jackrabbits. Proc. Vertebr. Pest Conf. 4:109-116.

Fortenbery, D. K. 1972. Characteristics of the black-footed ferret. U.S. Fish Wildl. Serv., Resour. Publ. 109. 8 pp.

Gotink, W. M., and F. W. van Hulsen 1952. Massale Wildsterfte door Vergift met Zinkphosphide (Muizentarwe). Tijdschrift voor Diergeneeskunde 77:214-219.

Hayne, D. W. 1951. Zinc phosphide: Its toxicity to pheasants and effect of weathering upon its toxicity to mice. Mich. Agr. Exp. Sta. Quart. Bull. 33(4):412-425.

Henderson, F. R., P. F. Springer, and R. Adrian 1969. The black-footed ferret in South Dakota. S. D. Dep. Game, Fish, Parks, Pierre. 37 pp.

Hillman, C. N. 1968. Field observations of black-footed ferrets in South Dakota. Trans. N. Am. Wildl. Nat. Resour. Conf. 33:433-443.

Hillman, C. N. 1971. The black-footed ferret. S. D. Conserv. Dig. 38(4):5-7.

Hilton, H. W., and W. H. Robison 1972. Fate of zinc phosphide in the soil-water environment. J. Agr. Food Chem. 20(6):1209-1213.

Hilton, H. W., W. H. Robison, A. H. Teshima, and R. D. Nass 1972. Zinc phosphide as a rodenticide for rats in Hawaiian sugarcane. Proc. Congr. Int. Soc. Sugarcane Technol. 14:561-570.

Hines, T., and R. W. Dimmick 1970. The acceptance by bobwhite quail of rodent baits dyed and treated with zinc phosphide. Proc. Annu. Conf. Southeast. Assoc. Game Fish Comm. 24:201-205.

Johnson, G. E. 1925. Control of mammals injurious to agriculture in Kansas. Kans. State Agr. Coll. Exp. Sta. Circ. 120. 10 pp.

Lantz, D. E. 1905. Kansas mammals in their relations to agriculture. Kans. State Agr. Coll. Exp. Sta. Bull. 129:331-404.

Lantz, D. E. 1909. Use of poisons for destroying noxious mammals. U.S. Dep. Agr. Yearbook for 1908:421-432.

Lantz, D. E. 1917. Destroying rodent pests on the farm. Yearbook U.S. Dep. Agr. for 1916:381-398.

Lantz, D. E. 1918. Rodent pests of the farm. U.S. Dep. Agr. Farmers' Bull. 932. 23 pp.

Leopold, S. A. 1964. Predator and rodent control in the United States. Trans. N. Am. Wildl. Nat. Resour. Conf. 29:27-49.

Linder, R. L., R. B. Dahlgren, and C. N. Hillman 1972. Black-footed ferret-prairie dog interrelationships. Pages 22-37 *in* Symposium on Rare and Endangered Wildlife in the Southwestern United States. (N.M. Chap. Wildl. Soc. and Am. Fish Soc.)

Melton, B. J. 1922. Killing prairie dogs in Oklahoma. Okla. A & M Coll. Ext. Div. Circ. 162. Gen. Ser. No. 16. 7 pp.

Merriam, C. H. 1901. Directions for the destruction of prairie dogs. U.S. Dep. Agr. Div. Biol. Surv. Circ. 32. 2 pp.

Merriam, C. H. 1908. Directions for the destruction of prairie dogs. U.S. Dep. Agr. Circ. 32, 2nd revised ed. 3 pp.

Okuno, I., R. A. Wilson, and R. E. White 1975. Determination of zinc phosphide in range vegetation by gas chromatography. Bull. Environ. Contam. Toxicol. 13(4):392-396.

Rudd, R. L., and R. E. Genelly 1956. Pesticides: their use and toxicity in relation to wildlife. Calif. Dep. Fish Game Bull. 7. 209 pp.

Scheffé, H. 1959. The analysis of variance. John Wiley & Sons, New York. 477 pp.

Schitoskey, F. Jr. 1975. Primary and secondary hazards of three rodenticides to kit fox. J. Wildl. Manage. 39(2):416-418.

Sheets, R. G., and R. L. Linder 1969. Food habits of the black-footed ferret (*Mustela nigripes*) in South Dakota. Proc. S. D. Acad. Sci. 48:58-61.

Sheets, R. G., R. L. Linder, and R. B. Dahlgren 1972. Food habits of two litters of black-footed ferrets in South Dakota. Am. Midl. Nat. 87(1):249-251.

Siegfried, W. R. 1968. The reactions of certain birds to rodent baits treated with zinc phosphide. Ostrich 39(3):197-198.

Snedecor, G. W., and W. G. Cochran 1971. Statistical methods. Iowa State Univ. Press, Ames. 539 pp.

Snow, C. 1972. Black-footed ferret (*Mustela nigripes*). Bu. Land Manage. Habitat Manage. Ser. for Endangered Wildl., Rep. 2. 23 pp.

Spaulding, M. H. 1912. The control of prairie dogs and ground squirrels. Mont. State Coll. Agr. Exp. Circ. 20:9-26.

Stephl, O. E. 1927. The prairie dog in Montana. Mont. State Coll. Ext. Serv. Bull. 83. 13 pp.

Swenk, M. H. 1915. The prairie dog and its control. Univ. Neb. Agr. Exp. Sta. Bull. 154 Vol. 28, Art. 2. 38 pp.

Thompson, W. R. 1947. Use of moving averages and interpolation to estimate median-effective dose. Bacteriol. Rev. 11:115-145.

U.S. Fish and Wildlife Service 1964. Pages 59-60 *in* Pesticide-wildlife studies; 1963: a review of Fish and Wildlife Service investigations during the calendar year. U.S. Fish Wildl. Serv. Circ. 199.

U.S. Department of the Interior 1956. Prairie dogs and their control. U.S. Fish Wildl. Serv., Wildl. Leafl. 357. 4 pp.

Ward, J. C., and D. A. Spencer 1947. Notes on the pharmacology of sodium fluoroacetate. J. Am. Pharm. Assoc., Sci. Ed. 36:59-62.

Weil, C. S. 1952. Tables for convenient calculation of median-effective dose (LD_{50} or ED_{50}) and instructions in their use. Biometrics 8(3):249-263.

As the Nation's principal conservation agency, the Department of the Interior has responsibility for most of our nationally owned public lands and natural resources. This includes fostering the wisest use of our land and water resources, protecting our fish and wildlife, preserving the environmental and cultural values of our national parks and historical places, and providing for the enjoyment of life through outdoor recreation. The Department assesses our energy and mineral resources and works to assure that their development is in the best interests of all our people. The Department also has a major responsibility for American Indian reservation communities and for people who live in island territories under U.S. administration.

GPO 833-306

UNITED STATES
DEPARTMENT OF THE INTERIOR
FISH AND WILDLIFE SERVICE
EDITORIAL OFFICE
AYLESWORTH HALL, CSU
FORT COLLINS, COLORADO 80523

POSTAGE AND FEES PAID
U.S. DEPARTMENT OF THE INTERIOR
INT 423

U.S.MAIL