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Effects of Chlorophacinone on Captive Kestrels

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This study was designed to assess the potential secondary poisoning hazard of chlorophacinone to American kestrels (Falco sparverius L.). Secondary poisoning caused by consumption of anticoagulant-contaminated food has been reported in golden eagles (Aquila chrysaetos) (Savarie et al. 1979), captive barn owls (Tyto alba) (Mendenhall and Pank 1980, and screech owls (Otus asio) (Merson et al. 1984).

Chlorophacinone (2 (P-chlorophenyl)-1-phenylacetyl) 1-3 indandione) is a slow-acting anticoagulant rodenticide currently registered in Canada for ground squirrel control and for control of small rodents in grain storage facilities and orchards. Permanent chlorophacinone baiting stations have been used to control rodents in reclamation and afforestation programs and in tree nurseries in seven Canadian provinces (Radvanyi 1980). Since American kestrels are seasonally dependent on mice, voles and other small mammals (Craighead and Craighead 1956, Heintzelman 1964, Hart 1972, Callopy 1973), we sought to evaluate their susceptibilty to secondary poisoning resulting from the ingestion of chlorophacinone-contaminated prey.

MATERIALS AND METHODS

To determine baseline lethal dosage levels for the kestrel experiments, preliminary multiple-choice feeding trials were conducted on 83 meadow voles (Microtus pennsylvanicus). The voles were permitted to feed ad libitum on whole oat groats treated with 2.0% chlorophacinone concentrate (0.01% by weight), laboratory chow and water until they died. The test mice consumed an average accumulative total of 11.4 grams of the treated grain before dying in 6 days (5.7 + 0.3 days). This quantity of consumed grain would carry on it 1.14 mg of active chlorophacinone or about 53 mg of 2.0% chlorophacinone concentrate. The 53.0 mg of rodenticide was considered the average quantity lethal to individual Microtus and the basis for dose levels later administered to the kestrels.

Experiments to assess potential poisoning hazards of chlorophacinone to American kestrels were conducted in two phases. In Phase 1, encapsulated rodenticide was administered by oral intubation to kestrels to assess direct effects. In Phase 2 experiments, kestrels were fed rodenticide-poisoned mice to assess secondary effects. All of the experiments were conducted at the Macdonald Raptor Research Centre of McGill University, Montreal, Quebec, which maintained a captive colony of American kestrels for research purposes. The test birds were individually housed in outdoor pens (1.2 x 2.4 x 2.4 m) each equipped with an upper and lower rope perch, a water pan, partial roof and a one-way mirror window to permit behavioural observations.

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In Phase 1, two dose-response studies were conducted, each of three weeks duration. We hypothesized that if captive kestrels were to survive three weeks of direct rodenticide exposure, then death from secondary poisoning in wild kestrels would not likely occur.

In both Phase 1 experiments, No. 5 Libby gelatin capsules (1/4" x 3/8") were used to administer the rodenticide and placebo to kestrels. Chlorophacinone was added to the gelatin capsules using a vibrating spatula and then weighed on an analytical balance. Treatment capsules contained either 53.0 mg or 18.0 mg of 2.0% chlorophacinone. The placebo capsules contained freeeze-dried hamburger. Placebo capsules were given to both control and treatment groups to stimulate digestive processes. The capsules were administered to the kestrels daily at 0900 hrs by oral intubation. Test birds were observed in random sequence for five minutes daily immediately after dosing and all behavioural traits recorded. The birds were then each fed their daily diet of two day-old chicks. Water was available ad libitum. Kestrels which died during the experiments were frozen immediately. With one exception (Kestrel #654), all live birds were sacrificed upon termination of the study and frozen for subsequent gross pathological examination.

The first experiment was conducted from 04-31 July, 1980, using twelve adult kestrels. Four birds (three males and one female) were randomly assigned to each of three treatment groups (low dose, high dose and control). The low dose group received 53.0 mg/day of 2.0% chlorophacinone (three placebo capsules and one rodenticide capsule. The high dose group received 159.0 mg/day of 2.0% chlorophacinone (one placebo capsule and three rodenticide capsules). The control group received four placebo capsules.

Twelve juvenile kestrels were used in the second experiment conducted from 15 August - 04 September 1980. These birds were approximately nine months old but were of greater mean weight than the adult kestrels used in the first experiment. Four birds (three male and one female) were randomly assigned to each of three treatment groups. Here the low dose group received 18.0 mg/day of 2.0% chlorophacinone (one placebo capsule and one rodenticide capsule). The high dose group received 53.0 mg/day of 2.0% chlorophacinone (one placebo capsule and one rodenticide capsule). Control birds received two placebo capsules daily.

In preparation for Phase 2, 1520 wild mice (1020 Microtus, 410 Peromyscus and 90 Clethrionomys) were live-trapped near Edmonton, Alberta, and Montreal, Quebec. Of the Alberta sample, 420 were fed 2.0% chlorophacinone-treated oat groats (0.01% by weight) until they died, after which they were frozen at -20°C. The remaining nonpoisoned mice were killed with CO^2 gas and frozen.

The Phase 2 feeding trials were conducted between 10 August and 09 October 1981. Ten adult American kestrels (five males and five females) were randomly assigned to each of three treatment groups.

Kestrels in the high dose group each received a poisoned mouse daily for 21 consecutive days. The low dose group birds received a poisoned mouse every third day and a nonpoisoned mouse on each intervening day for 61 days. Each kestrel of the control group received a nonpoisoned mouse daily for 61 days. All birds were fed at 0900. At 1800 hrs, unconsumed portions of the mice (usually the hind legs and tail) were removed. The prey species were randomized daily and thawed prior to feeding to the kestrels. After feeding, five-minute behavioural observations were made in random sequence. Behavioural traits recorded included location and movements of the kestrels and the location and condition of the food item. Following the experiment, all test birds were sacrificed with CO_2 gas and frozen for subsequent gross pathological examination.

RESULTS AND DISCUSSION

All kestrels treated with chlorophacinone showed physical and behavioural changes regardless of the dosage administered; the control birds did not. Drooping of one or both wings was the first sympton observed in the rodenticide-poisoned kestrels in Phase 1 experiments. Subsequent symptoms in these same tests included: loss of voice, course breathing, frequent closing of the eyes, loss of balance, decreased flying ability, diarrhea and loss of appetite. In the final stages, the poisoned birds bled from the trachea, beak and nares and spent increasing amounts of time on the ground unable to fly. With the exception of kestrel #654 which survived the tests, once wing drooping occurred in these birds, the symptom persisted throughout the experiment until the birds died. Wing drooping did not occur in Phase 1 control birds.

Direct toxicity effects of the three treatment levels used in Phase 1 are summarized in Table 1. In tests administering 159.0 mg/day, dosages were higher than that which a kestrel might encounter when feeding on individual poisoned mice in the field, but our aim was to show a chlorophacinone dose-response and a "worst-case" scenario. The response of adult kestrels to the rodenticide treatment was variable. One of four adult birds treated at 159.0 mg/day level died after 14 days. After 21 days treatment, the three surviving birds in the high-dose group were in such poor health that they were sacrificed. All adult and juvenile kestrels subjected to 53.0 mg/day died after a mean of 16.5 and 10.3 days respectively. No significant differences (one-way ANOVA, $p = 0.05$) were found between time of death for juvenile and adult kestrels treated at this dosage level. In the 18.0 mg/day juvenile group, three of the four birds died after a mean of 11 days of treatment. The surviving bird in this low dose group, while having shown all the symptoms of poisoning intoxication, recovered completely after ten days once removed from the treatment procedure of the three week experiment. During the recovery period, normal wing posture, vocaliation, appetite and ability to fly were restored.

Adult kestrels treated at 159.0 mg/day began wing drooping after 14 days (mean). Adult and juvenile birds treated at 53.0 mg/day

Table 1. Phase 1 toxicity effects of 2.0% chlorophacine concentrate administered to adult and juvenile American kestrels over a 21 consecutive day period.

Dosage group	Kestrel #	Sex	Initial weight (g)	Final weight (g)	Weight change (g)	Fate	Days to death
Adults							
Control	550	M	105.8	90.6	-15.3	S	
	578	M	105.5	90.2	-15.3	S	
	583	F	126.1	109.2	-16.9	S	
	593	F	112.0	110.6	-1.4	S	
Mean			112.3	100.1	-12.2		
±S.E.			±4.6	±5.6	±3.6		
159 mg/	492	M	89.2	90.2	+1.0	S	
day	582	M	105.7	93.6	-12.1	S	
	595	M	111.0	81.8	-29.2	D	
	538	F	102.6	92.2	-10.4	S	
Mean			102.1	89.2	-12.7		
±S.E.			±4.6	±2.6	±6.2		
3 mg/	453	M	101.9	93.7	-8.2	D	14
ay	473	M	100.9	98.6	-2.3	D	16
	580	M	107.4	89.8	-17.5	D	21
	594	F	125.0	102.7	-22.3	D	15
5 d Mean			108.8	96.2	-12.6		16.5
±S.E.			±5.6	±2.8	±4.5		±1.6
Juveniles							
Control	653	M	143.0	128.2	-14.8	S	
	691	M	112.0	111.4	-0.6	S	
	694	F	105.0	110.0	+5.0	S	
	695	F	113.0	110.5	-2.2	S	
Mean			118.0	115.0	-3.2		
±S.E.			±8.4	±4.4	±4.2		
53 mg/	651	M	115.0	108.3	-6.7	D	9.0
day	683	M	130.0	104.3	-25.7	D	12.0
	686	M	120.0	91.4	-28.6	D	9.0
	671	F	150.0	116.7	-33.3	D	11.0
Mean			128.8	105.2	-23.8		10.3
±S.E.			±7.7	±5.3	±5.8		±0.8
18 mg/	654	M	130.0	129.9	-0.1	R	
day	666	M	123.0	95.3	-27.7	D	16.0
	698	M	110.0	100.8	-9.2	D	11.0
	743	F	112.0	95.7	-16.3	D	6.0
Mean			118.8	105.4	-13.3		11.0
±S.E.			±4.7	±8.3	±5.8		±2.9

R - Recovered; S - Sacrificed; D - Died

exhibited drooping wings after 7.5 and 5 days (mean) respectively. These values are not significantly different ($t = 2.18$, d.f. = 7, $p = 0.05$). Wing drooping in juvenile kestrels treated with 18.0 mg/day began after 6.5 days. An inverse relationship between dosage and days to wing drooping is suggested in juvenile birds but not in adults. While wing drooping is an expected respose in elevated environmental temperatures, as occurred during the Phase 2 experiments, here it can only be considered to signal the birds' response to effects of the intoxicant.

All birds treated directly with chlorophacinone displayed anticoagulant poisoning and massive hemorrhaging in one or several of the nine tissues or organs examined (Table 2). In 73% (11/15) of the birds, six or more of the organs examined were affected. Hemorrhaging occurred in the nares, beak, trachea, abdominal walls, pectoral muscles, thoracic cavity, heart, lungs, liver and kidney. The incidence of hemorrhaging in tissues and organs in adult birds appeared to be similar for both 159.0 mg/day and 53.0 mg/day dosage levels. However, in juvenile kestrels, tissue and organ hematomas appeared to increase with elevated dosages of rodenticide. None of the control birds died during this experiment. One bird appeared to have a slight congestion of blood in the lungs, but all other organs appeared normal.

Table 2. Phase 1 incidence of hemorraging in tissues and organs of kestrels administered chlorophacinone directly.

	Adult kestrels								Juvenile kestrels						
	on 159 mg/day				on 53 mg/day				on 53 mg/day				on 18 mg/day		
No.	492	582	595	538	453	473	580	594	651	683	686	671	666	698	743
Fate	S	S	D	S	D	D	D	D	D	D	D	D	D	D	D
Sex	M	M	M	F	M	M	M	F	M	M	M	F	M	M	F
Tissues/organs															
Nares/beak	X	X			X		X	X	X	X		X	X	X	X
Trachea		X	X		X	X	X	X	X	X	X	X		X	X
Abdominal walls	X	X		X	X	X	X	X	X	X	X	X			X
Pectoral muscles	X	X					X	X				X	X	X	
Thoracic cavity					X		X	X			X				X
Heart	X	X		X			X			X	X		X		
Lungs	X				X			X	X	X	X	X		X	X
Liver	X	X				X	X	X	X	X	X	X	X	X	
Kidney		X			X		X	X	X	X	X	X	X	X	X

The data illustrate a variable response to direct chlorophacinone treatment. The majority of adult kestrels treated at the higher dose of 159.0 mg/day did not die earlier than the adult birds at 53.0 mg/day. In fact, three of the high dose adults survived the experiment but were sacrificed on the assumption that they would not recover. Juvenile kestrels subjected in 53.0 mg/day of 2.0% chlorophacinone did show symptoms of rodenticide intoxication earlier and died earlier than did adult birds treated with the same daily dosage. In addition, hemorrhaging of heart, liver and kidney appeared to occur more frequently in juvenile kestrels than in adults. Perhaps rodenticide poisoning in birds is age-related as in mammals (Rowe and Redfern 1967).

In the Phase 2 study, evidence of rodenticide intoxication was considerably less pronouned than in Phase 1. Feeding trials showed that the kestrels did not discriminate between mice of different species or between poisoned and nonpoisoned mice. No birds fed poisoned mice died during these experiments. While wing drooping was observed in all three groups, this behaviour may have been a stress response brought on by unusually warm temperatures during the second week of the experiment. Wing drooping in Phase 2 birds was periodic and did not persist to the end of the experiment. It occurred in 7 of 10 birds on the high dose group, in 9 of 10 birds in the low dose group and in 6 of 10 control birds.

No American kestrels fed poisoned mice died in this experiment but hematomas on the pectoral muscles, lungs, liver and heart were observed. For both poisoned mice feeding regimes, heart lesions were the most frequent, occurring in 6 of 10 birds in Group 1 and in 8 of 10 birds in Group 2 (Table 3). Only 2 of 10 control birds displayed small heart lesions caused possibly in the sacrificing procedure. Other types of hematomas occurred in more than 50% of the treated birds.

All Phase 2 captive kestrels fed either on poisoned mice for 21 days or on a combination of poisoned mice and nonpoisoned mice for 61 days survived and behaved normally but did hemorrhage in one or more organs. Whether these pathological effects alone or in combination with other environmental stresses would cause decreased survival in wild kestrels is unknown. Should kestrels consume enough poisoned food in the wild, starvation brought on by impaired flight (wing drooping), hemorrhaging of various tissues and organs would accelerate onset of death. Mendenhall and Pank (1980) noted hemorrhaging throughout owl carcasses resulting from anticoagulant rodenticide secondary poisoning. Townsend et al. (1981) suggested that tawny owls occupy areas where poisoned bait stations are left in position for up to three months would not likely consume a lethal dose of warfarin-contaminated mice, but that sublethal effects could occur. Conversely, Merson et al. (1984) show that a wild American kestrel in an orchard treated with brodifacoum was not in danger of secondary poisoning. This may be because rodenticides are generally used in orchards for only several weeks

Table 3. Incidence of hemorrhaging in tissues and organs in Phase 2 kestrels.

	Group 1 1 poisoned mouse daily for 21 days	Group 2 1 poisoned mouse every third day for 61 days	Group 3 1 nonpoisoned mouse daily for 61 days
Tissues/organs			
Nares/beak	2	3	-
Trachea	-	-	-
Abdominal walls	-	1	-
Pectoral muscles	-	3	2
Thoracic cavity	1	1	-
Heart	6	8	2
Lungs	4	-	-
Liver	2	3	-
Kidney	-	-	-

and, therefore, contaminated food is not available for a long period of time.

Rudolph (1982) suggests that in order to conserve time required for food searching, kestrels transport prey directly to the nest occupants to be torn apart and consumed, thus freeing to the hunting individual more time for search manouvers. For this same reason, larger vertebrate prey species are more often cached than immediately eaten. In Alberta, invertebrate prey species (grasshoppers) do not peak until several weeks after fledglings hatch. Feeding of small rodents (poisoned mice) directly to young kestrels at this time could occur.

The course, fate, metabolites and persistence of rodenticides under laboratory and field situations are poorly understood. Cahill and Crowder (1979) used 14_C to label diphacinone administerd by gavage to laboratory mice. Females excrete 48.8% and males 69.9% of the administered dosage within 24 hours. Diphacinone was not stored by any organ or tissue, including liver. No comparable study outlining distribution and fate of chlorophacinone in primary or secondary animals appears to have been done. In the current study, if 50% of the chlorophacinone had been eliminated from the poisoned mice via feces and urine, and if a kestrel consumed two or more poisoned mice a day, the predator would likely become subject to poisoning effects similar to our 59.0 mg/day-dosed birds in Phase 1 with concomitant 6-8 internal organs being effected.
This study attempted to determine whether prolonged field use of chlorophacinone to control rodents poses a threat in American kestrels. Behavioural and gross pathological changes and death occurred in kestrels directly subjected to chlorophacinone in dosages as small as one-third that required to kill a meadow vole. The data in Phase 2 of the study suggests that if poisoned mice

were continually available for more than three weeks and if kestrels ate more than one poisoned mouse a day, sublethal secondary poisoning or even death might occur.

Caution is urged if permament chlorophacinone baiting stations are to be used in areas where kestrels depend almost entirely on a vertebrate diet and where high concentrations of nesting and juvenile kestrels occur. The hazards of secondary poisoning to avian predators can be minimized in Canada by limiting the period of rodent control to late fall and winter months when most of tree-girdling damage by harmful rodents occurs and while most migrant raptors are absent.

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