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EFFECTS OF ORGANOPHOSPHORUS INSECTICIDES ON SAGE GROUSE IN SOUTHEASTERN IDAHO

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Abstract: Die-offs of sage grouse (*Centrocercus urophasianus*) were verified in southeastern Idaho in 1981. We captured 82 apparently healthy grouse to quantify the effects of organophosphorus insecticides (OP's) and other pesticides on sage grouse in sagebrush (*Artemisia* spp.) bordering agricultural lands in July 1985 and 1986. Grouse were fitted with radio collars and tracked through part of each summer. At least 18% of 82 radio-tagged grouse in 1985–86 subsequently occupied fields at the time they were sprayed with OP insecticides dimethoate or methamidophos. Cholinesterase (ChE) assays of brains and residue analysis of crop contents indicated that 5 and 16% of the marked sample died from OP's in 1985 and 1986, respectively. Approximately 200 sage grouse were present in a block of alfalfa sprayed with dimethoate; 63 of these were later found dead and ChE activity in 43 brains suitable for assay were depressed >50%. Maximum residues in crop contents of dead grouse were 18 μg/g methamidophos and 30 μg/g dimethoate. Intoxicated or dead grouse were observed in or near 6 fields sprayed with dimethoate or methamidophos in 1985–86. Twenty of 31 intoxicated grouse radiotagged after being found in dimethoate-sprayed (1986) alfalfa died. Our study indicates that certain pesticides have the potential for adversely affecting grouse populations.

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Replacement of organochlorine insecticides (OC's) with shorter-lived chemicals such as organophosphorus (OP), carbamate, and other compounds alleviated many problems with persistence and bioaccumulation of lipid-soluble OC's (Blus 1982, Wiemeyer et al. 1984). Additional research revealed that serious effects, resulting from different modes of action, are also associated with use of the newer compounds, particularly from a short-term perspective where acute or subacute toxicity (Hill and Fleming 1982, Grue et al. 1983, Henny et al. 1985) and reduction in the food base are major concerns (Rands 1985, Potts 1986).

Initial evidence that OP's caused mortality of sage grouse was noted in 1981 when a die-off occurred near a potato field sprayed with methamidophos. Brain ChE activity of 5 sage grouse collected when intoxicated (sick, immobile, and showing signs of OP poisoning) and later sacrificed ranged from normal to 61% inhibition (E. F. Hill, Fish and Wildl. Serv., pers. commun.). Data collected in 1983 indicated depression of 40 to 65% in brain ChE activity of grouse collected in a potato field shortly after spraying with methamidophos. These preliminary findings and previous unverified reports of die-offs suggested a potentially serious situation and led to radio-telemetry studies in southeastern Idaho in 1985–86. The purpose of our study was to determine and quantify effects of OP's on a population of sage grouse.

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STUDY AREA AND METHODS

The study area was located in southeastern Idaho near Mud Lake, Monteview, Hamer, and Camas in Jefferson County and Arco in Butte County (Fig. 1). This area provided summer range for sage grouse (Gates 1983, Connelly et al. 1988). Major agricultural crops included small

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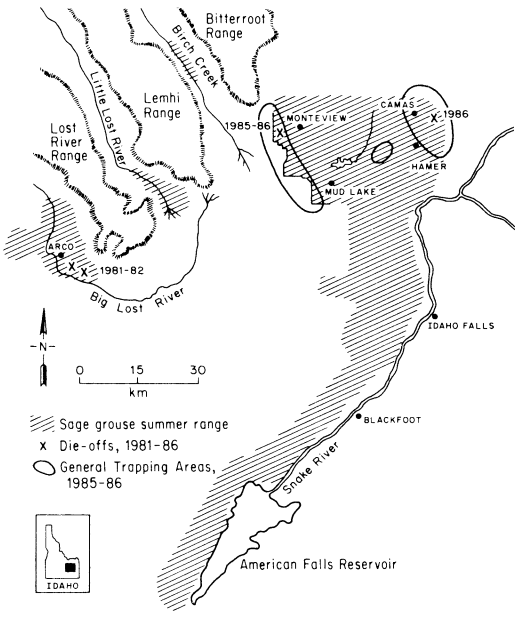

Fig. 1. Area of sage grouse study where die-offs occurred.

grains, potatoes, and alfalfa; many fields were bordered by sagebrush. Field work was conducted from April to August 1985 and from May to early September 1986. Spraying regimes employed by farmers in the study area were those normally used there.

From 9 to 26 July 1985, 39 apparently healthy sage grouse (30 juv and 9 ad F) were captured by night-lighting (Giesen et al. 1982) in sagebrush near alfalfa fields, and fitted with radio collars (Amstrup 1980, Dunn and Braun 1985). An intoxicated grouse captured in a sprayed alfalfa field was also radiotagged.

We captured apparently healthy sage grouse from 7 to 29 July 1986 by night-lighting. Radio collars were attached to 31 sage grouse taken near alfalfa fields and 12 sage grouse taken near potato fields; 31 grouse found intoxicated in sprayed fields were also radiotagged. In 1985 and 1986, individual grouse were located 2–14 times/week until the signal was permanently lost, the bird died or was collected, or the study was terminated (23 Aug 1985 and 3 Sep 1986 with a subsequent collection of 3 birds on 17 Sep 1986). Survival of radio-tagged grouse was recorded in grouse-days (i.e., 1 grouse surviving 1 day). Habitat was recorded each grouse-day that we located a bird; radio locations were verified by triangulation of several readings taken within 0.5 km of each grouse. Most radio tracking was done from trucks equipped with a null-peak system; some tracking was done on foot and from fixed-wing aircraft. Searches for radio-tagged grouse were not random; rather, we concentrated our work in areas where the grouse were last seen and expanded our search area to look for missing grouse.

Grouse found dead and those shot were placed on ice and frozen within 4 hours of collection. For analysis, grouse were thawed at room temperature and their brains were removed after medial bisection. We performed ChE assays on each half of the brain and values were averaged.

Although 2 different spectrophotometers were used, standardized methods for ChE assays (Ellman et al. 1961) with subsequent modifications (Hill and Fleming 1982) were used throughout the study. All assays were performed at 25 C and brains of apparently healthy (control) sage grouse were assayed concurrently with those of grouse exposed to OP's. Controls consisted of hunter-killed grouse and road-killed individuals picked up in non-agricultural areas. Precilip standard (Boehringer Mannheim, Indianapolis, Ind.) with an acceptable range of ChE values for freeze-dried human serum was used for quality assurance. The standard was tested ≥ 1 time/day that grouse brains were assayed to ensure that the spectrophotometer was properly calibrated and that our procedures resulted in accurate readings. In both years, ChE assays of the standard indicated our results were in the acceptable range listed by the manufacturer.

Cholinesterase activity is expressed as micromoles of substrate (acetylthiocholine iodide) hydrolyzed/minute/g of brain tissue. Control ChE activity is expressed as the mean $\pm$ 2 standard deviations (SD). Exposure of individual grouse to an anti-ChE compound is indicated when the ChE level is $\leq$ the control $\bar{x} - 2$ SD, and anti-ChE exposure is postulated as the cause of death with inhibition $\geq 50\%$ (Ludke et al. 1975, Hill and Fleming 1982).

Crop or gizzard contents of sage grouse collected in 1985 and 1986 were homogenized, extracted, and analyzed for OP's at the Patuxent Wildlife Research Center or the Environmental Protection Agency, Corvallis Environmental Research Laboratory, with a gas chromatograph equipped with an electron capture detector (White et al. 1982; E. J. Kolbe, Fish and Wildl. Serv., pers. commun.; R. S. Bennett, Environ. Prot. Agency, pers. commun.). Approximately

Table 1. Proportion of time radio-tagged sage grouse were located in various habitats in summer range, southeastern Idaho, 1985–86.

Yr Statistics	Grouse-days and individuals located in fields				
	Alfalfa	Potatoes	Other crops	Non-cropland	Totals
1985	119		31	145	775
n	31		11	35	39
$\bar{x}$	3.9		2.6	4.1	19.9
SD	3.9		1.9	2.7	12.4
Range	1–17		1–6	1–11	1–45
1986	342	96	44	390	1,476
n	27	9	7	41	43
$\bar{x}$	13.3	10.7	3.7	9.5	34.3
SD	9.8	8.1	3.3	10.1	17.6
Range	1–34	1–21	1–10	1–43	1–57

10% of the residue analyses was confirmed with a mass spectrometer. Recovery of dimethoate or methamidophos from spiked samples ranged from 70 to 90%; residues were not corrected for recovery values. The lower limit of quantification ranged from 0.1 to 0.5 μg/g; residues were expressed on a wet weight basis.

Survival functions of radio-collared sage grouse were estimated with the Kaplan-Meier (product limit) non-parametric estimator (Lee 1980). Using this method we estimated the probability of grouse surviving beyond a specified time, to a specific date or number of days since marking. A staggered entry scheme was used in estimating the survival function (Pollock et al. 1989) to preserve the relationship between the survival function and the calendar date. A Chi-square test was used to compare survival of adult and juvenile grouse that were radiotagged when apparently healthy or intoxicated.

RESULTS

General Movements

Each of 39 healthy sage grouse radiocollared near alfalfa fields in 1985 were tracked from 1 to 45 days for 775 grouse-days (Table 1). Thirty-one grouse were observed in cropland; 19 and 15% of the grouse-days were recorded in cropland and alfalfa, respectively.

The 43 healthy sage grouse radiocollared in 1986 were divided into 2 groups: grouse captured near alfalfa ($n = 31$) or potatoes ($n = 12$). All grouse were trapped in sagebrush within 0.5 km of cropland. Each grouse trapped near alfalfa or potato fields was tracked from 1 to 57 days and 12 to 41 days, respectively (Table 1). Grouse captured near alfalfa spent 33% of the total grouse-days in cropland (31% in alfalfa), while those captured near potato fields spent 32% of the grouse-days in cropland (25% in potato fields).

During 1985 and 1986 85% of the 82 radio-tagged grouse were located ≥1 time in cropland, and the other 15% remained near cropland through much of the tracking period. Maximum distances sage grouse moved from sagebrush into cropland were 2.3 and 3.9 km in 1985 and 1986, respectively; these grouse remained in cropland for several weeks. By late August 1986, a few grouse moved back to sagebrush; some were 4 km from the nearest cropland. The daily activity pattern of about 90% of the radio-tagged sage grouse suggested feeding in cropland and roosting and loafing in nearby sagebrush. Because individuals were not located on 62 and 41% of the grouse-days in 1985 and 1986, respectively, their use of cropland and other habitats was much higher than recorded.

Intoxication and Mortality

Six of 39 (15%) grouse radiocollared when apparently healthy in 1985 later occupied a 240-ha alfalfa field (AB alfalfa) sprayed with dimethoate on 5 August; all 6 became intoxicated and 2 birds died with 62 and 73% inhibition of brain ChE activity (Table 2). The 4 intoxicated birds could not walk or fly; they were emaciated, had diarrhea, frequently salivated, and sometimes uttered faint vocalizations. These signs are characteristic of anticholinesterase compounds such as OP's and carbamates. The biochemical lesion is phosphorylation or carbamylation of acetylcholinesterase and resultant accumulation of acetylcholine that induces problems with the nervous system (O'Brien 1960). Four intoxicat-

Table 2. Brain cholinesterase (ChE) activity of sage grouse, controls compared to birds collected or found dead in summer in or near southeastern Idaho cropland, 1985 and 1986.

				Brain ChE			
				% change from control[a]		% of grouse	
Y	*n*	OP[b]	Condition	$\bar{x}$	Range	Exposed[c]	With ≥50% inhibition
1985	2	DI	Dead	−67.3	−72.5–−62.1	100	100
1985	3	—	Dead[d]	+0.6	−9.8–13.7	0	0
1985	5	DI	Shot	−34.2	−36.8–−31.0	100	0
1985	11	—	Shot	+7.1	−61.1–37.9	9	9
1985	2	DI	Sick	−66.5	−66.8–−66.3	100	100
1986	43	DI	Dead	−73.6	−90.3–−50.6	100	100
1986	2	ME	Dead	−40.8	−42.8–−38.7	100	0
1986	8	DI	Shot	−13.9	−30.2–6.2	25	0
1986	1	—	Dead[e]		−7.8	0	0

[a] Results of control ChE assays ($\bar{x}$ micromoles of substrate [acetylthiocholine iodide] hydrolyzed/min/g of brain tissue ± 2 SD) were 12.54 ± 2.18 for 11 birds in 1985 and 15.30 ± 3.34 for 7 birds in 1986.
[b] Known exposure to methamidophos (ME) or dimethoate (DI) listed when known; — = no known exposure to organophosphorus insecticides (OP's).
[c] Less than control $\bar{x}$ − 2 SD.
[d] Includes roadkill, predator kill, and undetermined cause of death.
[e] Roadkill.

ed grouse recovered after approximately 1 week and left the alfalfa field; these birds appeared normal but had 31–35% inhibition of brain ChE activity when shot on 14 or 23 August (Table 2).

Three intoxicated grouse without radio collars were located in 1985 during a field search in AB alfalfa for radio-tagged grouse. Two grouse were captured and sacrificed (8–9 Aug); brain ChE activity was inhibited 66 and 67%. The third intoxicated grouse found on 9 August was fitted with a radio collar. It recovered and seemed healthy when shot on 23 August; however, its brain ChE was still inhibited 37% (Table 2). On 6 August, 2 grouse without radio collars were shot on the ground in sagebrush near AB alfalfa. The brain ChE activity of 1 grouse was normal but the other showed 61% inhibition (Table 2).

In 1985, residue analysis of the gizzard contents of 3 grouse adversely affected by dimethoate sprayed on AB alfalfa (2 found dead and 1 sacrificed 3–4 days post-spray) revealed that only 1 grouse had residues of dimethoate (0.2 μg/g); crops of all 3 were empty. Seven grouse that were shot, including 5 that had recovered from OP intoxication in AB alfalfa and were collected 9 or 18 days post-spray, contained no residues of dimethoate in crop or gizzard contents.

Nine of 43 (21%) grouse radiocollared when healthy in 1986 later occupied fields sprayed with OP's. Eight of the 9 became intoxicated and 7 died from OP's. Five juveniles died after being sprayed with dimethoate in AB alfalfa at 0600 on 1 August; an adult female in the same field left shortly after spraying and showed no signs of intoxication. On 5 August, 2 partially eaten juvenile grouse were found buried in or near a potato field that was sprayed with methamidophos the previous day; these birds were probably eaten by a coyote (*Canis latrans*). A radio-tagged adult male that occupied a small alfalfa field sprayed with dimethoate on 6 August was intoxicated for several days; this was the only sick grouse found in the field adjacent to AB alfalfa.

We observed 100 sick or dead grouse around 3 alfalfa and 2 potato fields that were sprayed with OP's in 1986; the major die-off occurred in the AB alfalfa fields where we noted dead grouse in 1985. A flock of about 200 sage grouse occupied the AB alfalfa sprayed on 1 August; about 30 intoxicated and dead grouse were observed on 2 August with the last verified OP mortality occurring there on 12 August. We found 63 dead sage grouse in the AB alfalfa; these included 5 grouse radiotagged when healthy, 20 radiocollared when intoxicated, and 38 birds without radios (Table 3). In the large block of AB alfalfa sprayed with dimethoate on 1 August, we radiotagged 29 sage grouse found intoxicated; 20 of these apparently died from dimethoate and 10 deaths were verified by brain ChE assays.

Intoxicated sage grouse in the AB fields exhibited the same signs noted in 1985. Most of the sick grouse attempted to move into sagebrush. At least 2 grouse fell to the ground from

Table 3. Incidence of organophosphorus-related mortality of sage grouse by age and sex, southeastern Idaho, 1986.

Marking Physical condition	No. grouse					
	F		M		Unknown sex	Unknown sex and age
	Ad	Juv	Ad	Juv	Juv	
Radiotagged						
Healthy	11 (0)[a]	9 (3)	4 (0)	9 (4)	10 (0)	
Intoxicated	4 (4)	11 (5)	1 (1)	14 (9)		1 (1)
Unmarked	(1)	(7)	(1)	(13)	(3)	(13)

[a] Grouse radiotagged with organophosphorus insecticide-induced mortalities in parentheses.

flight. Most grouse died in or at the edge of the AB alfalfa, but 2 grouse radiotagged when intoxicated died in sagebrush 0.8 and 1 km from the field border. Avian and mammalian predators were attracted to the dead and dying grouse. We found 17 depredated carcasses in or near the AB fields ≤2 weeks after spray.

Assays of brains of 43 sage grouse found dead in AB alfalfa in 1986 revealed 51–90% inhibition of ChE activity (Table 2). Brains of 9 depredated grouse were suitable for ChE assay; activity was depressed from 51 to 86%. Of the 9 grouse that were radiocollared in AB alfalfa when intoxicated and subsequently recovered, 5 shot on 3 September had brain ChE activity inhibited from 9 to 30%; 3 other grouse shot on 17 September had brain ChE activity that ranged from −13 to 6% of control values. Unlike the 2 grouse that died from OP's in 1985, some of the birds in 1986 died soon after spraying; crops of 16 of 18 grouse found on 2 August contained alfalfa. Dimethoate residues in crop contents of 12 grouse found dead the day after spray ranged from 3 to 30 μg/g.

Two depredated radio-collared grouse that were found buried in or near a potato field the day after it was sprayed with methamidophos had brain ChE activity depressed 39 and 43% and crop contents of 1 grouse contained 18 μg/g methamidophos; these were the only 2 suspected OP mortalities during this study that had <50% inhibition of ChE activity.

Survival Analysis

Survival analysis of the 39 sage grouse radiotagged when apparently healthy in 1985 indicated that the probability of these grouse dying during the 45-day tracking period was 0.25 (mortality = 1 − survival); however, only 2 (juv) of 9 documented deaths (1 ad F and 8 juv) were related to OP intoxication (probability of dying from OP's = 0.10). Four radio-tagged grouse were killed by predators, 2 by farm machinery, and 1 died from an unknown cause; ChE activity in brains of 2 of these grouse was similar to control values. Two young killed by farm machinery died the day after they were trapped and were not included in the mortality estimates. Of the 7 deaths unrelated to OP poisoning, 5 occurred from 10 to 27 July and 2 occurred in early August. As a result of the short range of the transmitters (<1.3 km) and related problems, signals from 17 grouse were lost before the study ended; thus, the mortality values are minimal estimates with low precision.

Of the 43 sage grouse radiocollared when healthy in 1986, 10 died (7 from OP's) before the end of the study with an overall mortality rate of 0.32. The probability of a grouse dying during the 72-day study from OP poisoning was 0.25. Aside from the 7 juvenile grouse that died from OP's, 3 additional radio-tagged grouse (2 juv and 1 ad F) were depredated on 15 and 20 August and 17 September. Although these 3 grouse were located in cropland from 3 to 20 days, there is no evidence of their exposure to OP sprays and their brains were not available for ChE assays. Radio collars were removed from apparently healthy grouse on 1 August (1 grouse) and 2 August (4 grouse); these units were then placed on intoxicated grouse.

The probability of mortality for 31 grouse, radiotagged when intoxicated in alfalfa from 25 July to 7 August 1986, was 0.76 to 12 August when the die-off from dimethoate in AB alfalfa apparently ended and 0.78 to 3 September when several of these grouse were collected. Dimethoate apparently accounted for deaths of 20 of these grouse; ChE activity was inhibited >50% in brains of 10 birds. Mortality of grouse instrumented when intoxicated was highest in 8 marked in AB alfalfa on 2 August (1 day post-spray) and all died by 5 August; 12 of 21 grouse radiocollared when intoxicated on 3–7 August

died from 4 to 12 August and 1 was depredated on 1 September. The grouse that died on 12 August was depredated; however, its brain ChE activity was inhibited 55.3%. The longer range (2.0–2.5 km) of the transmitters used in 1986 resulted in more efficient tracking compared to 1985; nevertheless, signals of 5 grouse were lost before the end of the study.

Age Effects.—Concerning sage grouse radiotagged when healthy in 1986, juveniles were more likely to die from OP poisoning than adults (Table 3); 7 of 28 juveniles died compared with zero of 15 adults ($P < 0.05$). There was no significant difference ($P > 0.05$) in survival of adults and juveniles radiocollared when intoxicated; however, all 5 adults died compared with 14 of 25 juveniles. Two adults were among 38 nonradioed birds that probably died from dimethoate in the AB alfalfa fields; however, sex and age of 13 birds were unknown. Considering the 6 grouse radiotagged when healthy and subsequently sprayed in AB alfalfa, an adult female showed no ill effects but all 5 juveniles died. The first 2 grouse radiocollared when sick were found in several cm of water in an alfalfa field on 25 July; the field was sprayed with dimethoate 2 days previously and was subsequently flood irrigated. One grouse flew from the field the same day it was radiotagged and the other left the field the next day.

DISCUSSION

Generally, sage grouse in southeastern Idaho are migratory (Dalke et al. 1963, Connelly and Ball 1983); movements to summer range, including cropland, begin in June. Maximum movement of adult sage grouse from winter range to summer range was 82 km (Connelly and Markham 1983, Connelly et al. 1988). Distances moved from nests to summer range by 6 females with broods ranged from 3 to 21 km (Gates 1983). Gates (1983) also noted that 82% of 22 sage grouse trapped and marked on leks subsequently moved to irrigated cropland. Based on this study and previous work by Gates (1983), most of the Idaho population uses cropland for summer range; such use increases sharply during extended periods of extremely hot and dry weather (J. W. Connelly, Id. Dep. Fish and Game, pers. commun.). In our study area, spraying crops with pesticides is initiated in late spring, but most applications occur in July and August at the height of cropland use by sage grouse.

The die-offs during our study were apparently the first verified records for wildlife losses that resulted from dimethoate application. There are no toxicity data relating to sage grouse tolerance to OP insecticides. Factors that increased risk of OP's to sage grouse were their use of alfalfa fields for feeding, roosting, and loafing, and their extensive feeding on alfalfa foliage after spraying.

The conditions associated with methamidophos application to potatoes that result in risk to sage grouse were similar to those associated with dimethoate applications to alfalfa. Some sage grouse used potato fields extensively during this study. The crops of grouse shot or found dead in potato fields contained foliage of weeds and small amounts of insect material; sage grouse may occasionally eat potato leaves (J. W. Connelly, Id. Dep. Fish and Game, pers. commun.). We are uncertain whether repellency of dietary methamidophos to experimental birds (Stromborg 1986) is an important factor mediating toxicity to wild sage grouse, especially in view of the 18 μg/g methamidophos detected in crop contents of a sage grouse. Although the acute toxicity of methamidophos is higher than for dimethoate (Hudson et al. 1984), we located only 1 record of a die-off of wild birds (house sparrow [*Passer domesticus*] and killdeer [*Charadrius vociferus*]) from this compound (Smith 1987). On the basis of survival of about 35% of the sage grouse found intoxicated, some of the sick birds may have survived effects of OP's had they not been depredated. In any case, OP exposure was considered the primary cause of death when ChE assay results and residues were available for verification. Although the 2 depredated sage grouse found in or near the potato field sprayed with methamidophos had brain ChE activity depressed <50%, recent experimental evidence supports the probability that their deaths resulted from the spraying. Japanese quail (*Coturnix japonica*) were critically intoxicated when euthanized 1 hour after receiving an oral dose of the OP dicrotophos; however, brain ChE activity was inhibited about 40% (Hill 1989).

In other studies, half-time of dimethoate and methamidophos on plants was <4 days; however, low residues of these systemic insecticides may persist for several weeks (Szeto et al. 1984, Westcott et al. 1987). Thus, intoxicated sage grouse in cropland may be exposed to additional residues of OP's when ChE reversal is initiated and the grouse resume feeding on contaminated foliage. Sublethal depression of ChE activity in

the brain did not have lasting physiological effects in experimental birds in earlier studies (Metz 1958, Glow and Rose 1966, Banks and Russell 1967), but more recent studies present evidence that OP's similar to dimethoate and methamidophos are capable of inducing long-term effects (Farage-Elawar and Francis 1987, 1988). There are few data for free-ranging birds (Hill and Fleming 1982). European starling (*Sturnus vulgaris*) nestlings exhibited 19% mortality within 48 hours of receiving a dose of the OP dicrotophos compared with no mortality among controls; thereafter, survival of dosed and control young was similar for 1 month (Stromborg et al. 1988). We found no short-term effects after recovery of locomotive abilities by grouse, but the sample size was small and mortality was the only factor considered. The approximate time for renewal of ChE activity in intoxicated sage grouse in this study was similar to the 26-day recovery period (from 55 to 64% inhibition to within 2 SD of the control $\bar{x}$) measured for 5 avian species given diets containing dicrotophos (Fleming and Grue 1981).

Our findings suggest that OP's may adversely affect sage grouse populations, but this study only involved that segment of the population whose summer range included cropland. The mortality rate and sublethal intoxication of our marked population, induced by OP's and possibly other pesticides used in the area, was probably underestimated because sage grouse were radiotracked only during part of the season when OP's and other pesticides were applied, signals were lost from a number of grouse before the study terminated each year, radio collars were removed from 5 healthy birds in 1986 for use on intoxicated grouse, and some unrecorded exposure of marked grouse may have occurred between radio locations because the birds were not tracked continuously.

MANAGEMENT IMPLICATIONS

In all fields where grouse were affected, the spray pilot reported the maximum allowable rates of dimethoate (0.56 kg active ingredient [ai]/ha) and methamidophos (1.13 kg ai/ha) were applied. Use of the minimal recommended application rates of 0.37 kg ai/ha for dimethoate and 0.85 kg ai/ha for methamidophos may reduce the hazard to grouse. General wildlife repellents are being tested by ≥1 chemical company for use with pesticides; successful short-lived repellents may deter sage grouse from ingesting contaminated foliage and may force them to leave sprayed fields. Die-offs of sage grouse and other species of birds including ring-necked pheasants (*Phasianus colchicus*) and gray partridge (*Perdix perdix*) are possible throughout much of their range in Idaho and in other states where cropland is available. The situation may worsen if intensive spraying of OP's on small grains is expanded in efforts to control the newly invading Russian wheat aphid (*Diuraphis noxia*). Our study provides evidence for claims that pesticides are at least partially responsible for declining populations of upland game birds in the United States and Europe; however, additional data are needed for verification.

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