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Declining ring-necked pheasants in the Klamath Basin, California: I. Insecticide exposure

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A study of organophosphorus (OP) insecticide exposure was conducted on a declining population of ring-necked pheasants (*Phasianus colchicus*) associated with agricultural lands at Tule Lake National Wildlife Refuge (TLNWR) during the summers of 1990–92. Findings at TLNWR were compared with a nearby pheasant population at Lower Klamath National Wildlife Refuge (LKNWR) not subjected to intensive farming or OP insecticide applications. Direct toxicity of anticholinesterase (antiChE) compounds (in this case methamidophos) killed 2 young pheasants (91 and 92% brain acetylcholinesterase [AChE] inhibition), but no deaths of adult radio-equipped hens were ascribed to direct insecticide intoxication. However, within 20 days postspray of OP insecticides, 68% (28 of 41) of the adult pheasants collected at TLNWR were exposed to antiChE insecticides, and exhibited brain AChE inhibition of 19–62%, with 15% (6 of 41) showing ≥55% brain AChE inhibition. The lack of radio-equipped hens dying was unexpected because >50% brain AChE inhibition has been frequently used as a 'diagnostic tool' for evaluating cause of death from antiChE insecticides. No young were radio-equipped, so the extent of the effects of insecticide exposure on the survivorship of young was unknown. It is concluded that insecticide exposure was not the major factor impacting the pheasant population (see Grove *et al.*, in press), although some young were acutely intoxicated. However, the loss of insects killed by insecticide use may have contributed to food shortages of young pheasants, indirectly influencing survival.

Keywords: methamidophos; organophosphorus insecticides; OPs, ring-necked pheasant; radio telemetry.

Introduction

The exotic ring-necked pheasant, first successfully introduced into the United States in 1881 (Bent, 1932; Gabrielson and Jewett, 1940; Lauckhart and McKean, 1956; Laycock, 1966; Weigand and Janson, 1976), thrived for many years in Oregon and other parts of the country. However, in recent decades, most pheasant populations declined markedly (Farris, 1977; Warner, 1981; Warner *et al.*, 1984), with populations in the Klamath Basin of Oregon and California being no exception (Zezulak, 1990). Gradual changes in agricultural land use practices have been generally recognized as one of the primary reasons for these declines. Small diversified farms have been incorporated into larger farms that utilize larger tracts of land for monocultural crops, thus eliminating fence and/or hedgerow habitat between smaller fields. Herbicide use to control economically important weeds in crops removes much of the weedy cover associated with field edges, ditch banks, and disturbed areas. Moreover, cleaner farming removes needed pheasant habitat, forcing the birds to associate with crops that are sprayed annually with organophosphorus insecticides. Exposure to these insecticides is inevitable.

Two study areas were selected in the Klamath Basin, Tule Lake National Wildlife Refuge (TLNWR) and Lower Klamath National Wildlife Refuge (LKNWR), to evaluate the effects of current agricultural practices, including the use of insecticides, on ring-necked pheasant populations. TLNWR is intensively farmed, with major crops being alfalfa, cereal grains, onions, potatoes, and sugar beets. LKNWR was chosen as a reference site for comparison because of its proximate location, reduced agricultural activity, and lack of insecticide use. LKNWR consists of ponds and old fields interspersed with some cereal grains. The primary objective of this study was to determine why the pheasant population at TLNWR was declining. The proximate reason for population decline could be poor adult survivorship, poor recruitment, or a combination of

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both, but ultimately, habitat quality (cover and food) and insecticide exposure could potentially affect recruitment and survivorship. Thus, a study was designed that could identify the patterns of recruitment and adult and juvenile survivorship in the context of habitat quality and insecticide use. To determine the aforementioned patterns, we: (1) estimated the size of the nesting population, (2) evaluated body condition of hen pheasants in the spring, (3) evaluated cover availability, types of cover, and cover growth rates, (4) evaluated nest site placement and success, and (5) evaluated adult hen and brood survival which will be reported in Part II of this series (Grove *et al.*, in press). In addition, the persistence of organophosphorus insecticides was evaluated on soil and crop vegetation which would impact insect populations (potential pheasant food) and pheasants exposed to these insecticides. Insecticide exposure and associated pheasant response is the subject of this report.

Methods

Study area

This study was conducted at TLNWR in northern California, 10 km south of the Oregon border, and 42 km southeast of Klamath Falls, Oregon. Much of the 15 830 ha refuge was once lake bed which was drained at the turn of the century. Now 6910 ha (44%) of the refuge is intensively farmed. Lower Klamath National Wildlife Refuge (LKNWR), located west of TLNWR, was used as the reference area. The 21 691 ha refuge is separated from TLNWR by Sheepy Ridge which rises 244 m above the valley floor, preventing significant pheasant movement between the two study areas. Between 1200 and 2000 ha (5% to 9% of land area) at LKNWR are planted with barley and/or wheat each year.

Capture, radio attachment, and monitoring

Ring-necked pheasants were live-trapped from 1 April to 15 May of 1991 and 1992 using walk-in funnel traps (Bub, 1991) at each refuge. Several areas with visible pheasant activity were pre-baited with cracked corn for one week prior to trap placement. Five sites with the greatest amount of pheasant activity at each refuge were selected for trap placement. Traps were checked and re-baited two or three times a day. Telemetric radios were placed on hens to follow them through the insecticide spray season to evaluate insecticide exposure and possible mortality. Radios were not placed on roosters because pheasants are polygynous and, therefore, hen survival is more important to the population. Captured hens were weighed and checked for general body condition prior to radio attachment. Necklace-designed telemetric radios (Advanced Telemetry Systems, Isanti, MN) weighing 16 g ($\simeq$1.6% of the body weight) were used because of their greater suitablility than backpack radios for studies of pheasant biology (Marcström *et al.*, 1989). The transmitters had mortality switches that doubled the normal pulse rate of 60–70 beats/min if the birds remained motionless for more than 5 hours. Hens were checked daily to identify deaths, but exact locations were not determined until after 15 June. In 1991, hens were located daily from 15 June and 30 August; while in 1992 (because of the increased number of hens monitored), they were located at the two refuges only on alternating days. Hens were located using a vehicle mounted 4-element null-peak antenna system or a 3-element hand-held Yagi antenna. Bearings were taken on each bird, using the null-peak system, from at least two receiver sites, with locations determined using the program XYLOG4 (Dodge and Steiner, 1986). Directional bearing accuracy was determined using techniques described in White and Garrott (1990). Hen survival was closely monitored in both years from April to September, with irregular checks throughout the winter. In 1992, radios of overwintering hens not retrapped and equipped with new radios were monitored until they stopped transmitting, became detached, the hens died, or the hens were collected.

Evaluation of exposure to OP insecticides

Methamidophos, a systemic OP insecticide, was used at TLNWR to control two economically important insect disease vectors, the green peach aphid (*Myzus persicae*) and the potato aphid (*Macrosiphum euphorbiae*) (Radcliffe, 1982). The persistence of methamidophos was examined under field conditions at TLNWR using four potato fields in 1992. Samples of potato leaves and soil were collected from five different sites in potato fields 8212, 8351, 8327, and 8306 prior to methamidophos application and at 2-, 5-, 10-, and 20-day post-spray intervals. The liquid methamidophos concentrate was applied with water on the potato fields at a rate of 1.12 kg active ingredient (a.i.)/28.5 to 95 L/ha (1 lb/3 to 10 gal/acre) as an aerial spray. Field entry for sample collection was 48 hours after insecticide application. The first sample of potato leaf and soil was collected 20 rows into the field, half way down its length, and we moved across the field to collect the remaining four samples at 20-row intervals. About 150 g of potato leaves was collected at each location from the top of potato plants. Approximately 500 g of soil was also collected at each location from areas unshaded by potato plants, but exposed to runoff from the plants during irrigation. Topsoil was collected to a depth of 3 cm. New collection sites were selected at each interval to minimize crop damage. Random sample collections were not made as we were interested in field degradation of the insecticide and not its coverage.

Moribund insects were collected in 1991 from potato field 1818-20 at approximate 2 day intervals until day 34 (insects not collected on 4, 6, 30, and 32 days postspray), beginning 2 days post-spray of methamidophos application. Dead insects were not collected as time of death could not be determined.

Plant, soil, and insect samples were placed on ice and frozen at −20 °C within 2 hours of collection. Plant and soil samples were sent to Patuxent Wildlife Research Center in Laurel, MD for chemical analyses. Percent moisture was determined for plant and soil samples by drying pre-weighed aliquots at 100 °C for 24 hours. The dried samples were cooled in a desiccator. Samples were re-weighed to determine moisture (percent moisture = 1 − (dry weight/pre-weight) × 100). Aliquots of plant and soil samples were homogenized and extracted with 1:1 acetone and methylene chloride as described in Belisle and Swineford (1988) and modified by Patuxent Wildlife Research Center, Analytical Chemistry Group Standard Operating Procedures (1989). The gas chromatograph was equipped with a flame photometric detector, with the lower limit of detection-quantification at 0.5 ppm. Moisture content in potato leaves and soil samples ranged from 85.4% to 87.0% and 37.3% to 42.7%, respectively. Residues were expressed on a wet weight basis as no patterns in moisture content of potato leaves or soil were noted. Recovery of surrogate spike samples (famphur, fenthion, monocrotophos, parathion, and phorate) ranged between 80 and 139%; residues were not corrected for recovery. Pesticides from 6 percent of the plant samples were confirmed using a gas chromatograph/mass spectrometer.

OP insecticide application dates and application rates were recorded for fields sprayed at TLNWR. Adult and juvenile pheasants were collected by shotgun at TLNWR in 1990, 1991, and 1992 at 2-, 5-, 10-, 15-, and 20-day post-spray intervals. Pheasants were collected in and adjacent to potato fields sprayed with methamidophos. However, pheasants could have been exposed to other OP insecticides used in adjacent fields (i.e, disyston and parathion). Adult and juvenile pheasants (reference birds) were collected prior to the start of insecticide applications at TLNWR and LKNWR, and later shot at LKNWR where no insecticide applications occurred. Collected pheasants were tagged and placed on ice for no more than 4 hours prior to freezing at −20 °C. Brain AChE activity was determined using the Ellman method (Ellman *et al.*, 1961) as modified by Hill and Fleming (1982). Each brain was bisected medially and removed, with AChE activity determined for each half and values averaged. AChE activity values were reported in μmoles of substrate (acetylthiocholine iodide) hydrolyzed/minute/gram of brain tissue (wet weight) at 25 °C. Precinorm® E normal enzyme control serum (Boehringer Mannheim Corp., Indianapolis, IN) was used to ensure that the spectrophotometer was responding within an acceptable range for AChE values. Mean brain AChE activity for adult and juvenile control pheasants were calculated, with normal activity expressed as the mean ± 2 SD (Ludke *et al.*, 1975; Zinkl *et al.*, 1979). Brain AChE activity of exposed birds below this threshold (usually ≤20 percent below the mean) was considered inhibited, with inhibition expressed as a percentage of the control mean value.

Gross necropsies were performed on pheasants found dead and collected by shotgun. Food items removed from the upper gastrointestinal (GI) tracts of pheasants were placed in chemically cleaned jars and frozen at −20 °C for later chemical analyses at the Department of Agricultural Chemistry, Oregon State University. Aliquots of GI tract samples were homogenized and extracted with 1:1 acetone and methylene chloride as described in Belisle and Swineford (1988) and modified by Patuxent Wildlife Research Center, Analytical Chemistry Group Standard Operating Procedures (1989). GI tract samples were analyzed for methamidophos, parathion, and disulfoton, using a flame photometric detector equipped gas chromatograph (GC). Confirmation of GC results was conducted on a GC-mass spectrometer. Spiked control samples were used to determine extraction recoveries of the insecticides analyzed. Recoveries of methamidophos spiked samples ranged from 70 to 96%. Residues were reported on a wet weight basis, with the lower limit of quantification at 0.1 ppm. Those samples below the lower limit of quantification, but with insecticides present, were treated as positive detections in the data analysis. Residues were not corrected for recovery rates. The actual insecticide identified in the crop (unabsorbed compound) cannot be responsible for cholinesterase inhibition determined in the brain, but rather it identifies the type and possible levels of insecticide exposure.

Radio telemetry was used to evaluate habitat-specific pheasant exposure to OP insecticides at TLNWR. All fields at TLNWR were not planted with the same crop and, therefore, not all fields were sprayed with the same OP and some were not sprayed at all. Furthermore, all spraying did not occur on the same date. Thus, some pheasants were directly exposed to OPs in fields where they lived while others were not exposed, except for spray drift from adjacent fields. Daily locations of individual radio-equipped hens were mapped to determine habitat used during the spray season and the potential extent of insecticide exposure. Edge habitat was defined as the interface between two or more habitat types, extending 20 m into each habitat. Days exposed were monitored in 10 day intervals. OP insecticide exposure was determined by dividing total days exposed by total days available during the spray season.

Statistical methods

Data were summarized and analyzed using the Statistical Analysis System (SAS, 1985), and were examined for normality using box plots, stem and leaf displays, the Shapiro-Wilk test (Shapiro and Wilk, 1965), and normal probability plots. The data were also tested for equal variances using the F-test. Appropriate transformations or analyses were performed based upon test results.

Results

Persistence of methamidophos on potato leaves and soil

Mean methamidophos residue concentrations of potato leaf samples at 2-, 5-, 10-, and 20-day postspray intervals were 12.7, 9.1, 4.2, and 2.7 ppm, respectively (Fig. 1). The mean half-life of methamidophos applied to potato fields at TLNWR in 1992 was 8.1 days, with about 80% of the insecticide degraded after 20 days. Methamidophos was not detected in any of the soil samples collected at the time potato leaves were sampled.

Methamidophos was detected in 3 of 4 pools of insects sampled from 2 to 8 days postspray (0.83, 0.75, and 0.69 ppm). All 15 insect pools collected between 10 and 34 days postspray had no methamidophos detected (i.e., <0.50 ppm). An incidental collection of insects taken from a field at 1 day postspray ethyl parathion contained 4.98 ppm of the insecticide.

Brain cholinesterase activity of pheasants as measure of OP exposure

Normal brain AChE data from control adult pheasants were combined because no refuge (ANOVA, $P = 0.3225$), season (ANOVA, $P = 0.9476$), sex (Kruskal-Wallis, $P = 0.9590$), or year (Kruskal-Wallis, $P = 0.1302$) differences were detected (Table 1). Brain AChE data for control juveniles were also combined because no differences were detected between age in days (Kruskal-Wallis, $P = 0.2716$), sex (ANOVA, $P = 0.7437$), or years (Kruskal-Wallis, $P = 0.3314$). Adult and juvenile control means were not combined because juvenile pheasants had higher cholinesterase activity (ANOVA, $P = 0.0001$). Following spray applications, 28 of 41 adult pheasants, collected in and around potato fields at TLNWR during 1990–92 field seasons, showed brain AChE inhibition ranging from 19 to 62% (Table 2). Six of these pheasants had brain AChE inhibition $\geqslant$55%, but no adult pheasants (including radio- and non-radio-equipped) were found that died of OP poisoning during the 1990–92 field seasons. Twenty-five of 41 adult pheasants collected within 20 days of spray application had detectable residues of methamidophos or parathion in food items taken from their upper GI tracts (Table 3). Parathion was detected in food from 1 hen collected in a potato field sprayed 10 days earlier with methamidophos. Food from 7 adults contained quantifiable residues of methamidophos, which ranged from 0.18 to 2.10 ppm (wet weight). Parathion concentrations were too low to quantify. An adult hen hand-captured (not usually possible) in 1992 near a potato field sprayed with

Table 1. Brain cholinesterase activity of 'control' adult and juvenile ring-necked pheasants at TLNWR and LKNWR, 1990–92. Activity expressed in μmoles/min/g of brain tissue

Age	N	Mean ± SD (Range)
Adult	40	16.95 ± 1.31 (14.63 – 20.22)
Juvenile	43	20.32 ± 2.51 (13.52 – 25.16)

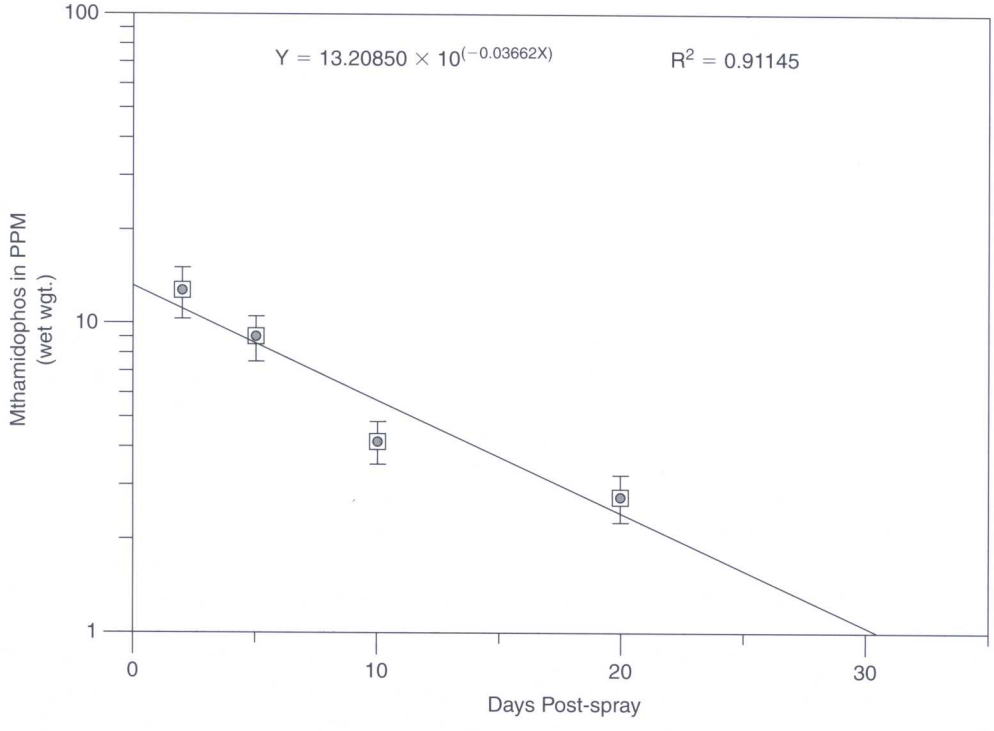

Fig. 1. Degradation of methamidophos in leaf samples collected from potato fields at TLNWR, 1992.

Table 2. Brain cholinesterase inhibition in adult and juvenile ring-necked pheasants from fields sprayed with methamidophos at TLNWR, 1990–92

	Days Post Spray					
Age	0	2	5	10	15	20
Adult	1/1[a] 31[b]	8/9 62 57 56 42 42 41 35 22	6/9 59 35 34 34 30 27	6/9 40 38 37 29 25 22	2/7 60 22	5/6 55 38 24 23 19
Juvenile	–	9/13 92[c] 91[c] 66 41 32 30 29 22 21	11/15 47 46 44 41 41 38 37 27 26 25 24	3/10 40 37 25	2/5 37 30	8/10 39 37 35 32 31 30 23 21

[a]Numerator = number of birds with inhibited brain cholinesterase activity; denominator = number of birds collected.
[b]Percent inhibition of cholinesterase activity for individual birds below 2 SDs of control mean.
[c]All pheasants were collected by shotgun except these 2 young which were found dead in 1990.

Table 3. Residue concentrations in food items from GI tracts of adult and juvenile ring-necked pheasants collected from fields sprayed with methamidophos at TLNWR, 1990-92. Quantifiable residues expressed in ppm (wet weight), with the lowest level of quantitation at 0.1 ppm. Residues below 0.1 ppm defined as detections, but not quantifiable

	Days Post Spray					
Age	0	2	5	10	15	20
Adult						
Detection	1/1M[a]	9/9M	5/9M	7/8M 1/8P	3/7M	0/6
Quantified	–	0.21[b] 0.31 1.34 2.10	0.18	0.36M 0.53M	–	–
Juvenile						
Detection	–	5/7M 2/7D	4/9M	6/8M	1/2M	3/8M
Quantified	–	16.00M[c] 3.90M[c] 0.43M	–	–	–	0.23 0.46

[a]Numerator = number of birds with detected residues; denominator = number of birds analyzed; D = disyston; M = methamidophos; P = parathion.
[b]Numbers denote residue concentrations of individual birds.
[c]All pheasants were collected by shotgun except these 2 young which were found dead in 1990.

methamidophos 2 days earlier, appeared weak, uncoordinated, and was salivating. Food items from the upper GI tract contained 2.10 ppm methamidophos, and brain AChE activity of this hen was inhibited by 41%. Necropsy of this hen and resulting pathology at Oregon State University, Veterinary Diagnostics Laboratory revealed the presence of avian tuberculosis.

Thirty-one of 51 juvenile pheasants, collected in and around potato fields sprayed with methamidophos, had brain AChE inhibited from 21 to 66% (Table 2). Of 32 juvenile pheasants with food items from upper GI tracts analyzed, 17 contained detectable levels of methamidophos and 2 of disyston. Five of the methamidophos detections were quantifiable, ranging from 0.23 to 0.46 ppm (Table 3). The disyston concentrations were not quantifiable. Two juvenile pheasants were found dead from OP poisoning at TLNWR. They were both about 45 days old and died together near a potato field sprayed two days earlier with methamidophos. They showed 91 and 92% brain AChE inhibition (Table 2) with 3.90 and 16.0 ppm methamidophos (Table 3), respectively, in food items taken from their upper GI tracts.

Radio telemetry as a measure of pheasant OP exposure

Roughly half of the telemetric locations of hen pheasants were from grain fields for 1991 and 1992 (Table 4). Edge habitat (the interface between two or more habitat types)

Table 4. Radio-equipped adult hen ring-necked pheasant habitat use at TLNWR, 1991–92 using telemetry locations. Data represents the number of locations in a particular habitat type. Edge habitat is defined as the interface between two habitat types, i.e., fields, marshes. Numbers in parentheses are in percent of observations

Habitat Type	1991	1992
Grain	490 (52.1)	443 (44.1)
Edge	322 (34.2)	436 (43.3)
Potato	70 (7.4)	93 (9.3)
Sugar Beet	22 (2.3)	1 (0.1)
Onion	22 (2.3)	–
Hay	12 (1.3)	–
Marsh	3 (0.3)	32 (3.2)
Total	941 (99.9)	1005 (100.0)

ranked second and potato fields third. Pheasant exposure to OP insecticides was assured because they spent much of their time in croplands. However, not all of the fields within the study area were treated with OP insecticides. Nine of 19 (47%) radio-equipped hens were exposed (present in a field within 30 days after sprayed) to OP insecticides at TLNWR in 1991, and 27 of 32 (84%) hens were exposed in 1992. Spray drift from adjacent sprayed fields was not included in these minimal exposure calculations. Radio-equipped hens were exposed in fields of known application dates to all 3 (disyston, methamidophos, and parathion) OP insecticides used in 1991 and 1992. In fact, some pheasants were exposed to more than one OP insecticide during the year because of staggered spray dates. One hen was exposed to all 3 OPs in 1992. Radio-equipped hen pheasants (during the 30-day post-spray exposure period) were exposed to OP insecticides an average of 24% of 540 available exposure days at TLNWR in 1991 (total exposure days based on OP insecticide dissipation of about 30 days, multiplied by the number of hens available [19 for 1991 and 32 for 1992] during the spray season). Average exposure was 57% of 960 exposure days in 1992.

Discussion

Pheasants and other wildlife at TLNWR were exposed to antiChE insecticides used on refuge agricultural croplands, because alternative foraging habitat was extremely limited at the refuge. Ingestion and inhalation are the most common routes of antiChE exposure. Exposure by ingestion may be by food consumption (i.e., insects, water, and foliage) or preening. Insects collected for 34 days in fields sprayed with OP insecticides at TLNWR in 1991, showed detectable concentrations of both methamidophos and parathion (>0.50 ppm) up to day 8 post-spray. However, concentrations of OPs (below the detection limit) were probably present much longer. Dead and dying insects are certainly easy prey for both adult and juvenile pheasants, and can expose them to either acute or chronic concentrations of OPs. Over 95 percent of radio-equipped hen activity at TLNWR in 1991 and 1992 was associated with agricultural lands. Exposure of hens to OP insecticides doubled between 1991 and 1992, yet no radio-equipped adult hen pheasants died as a direct result of OP intoxication during the two-year telemetry study. This finding is of particular interest because 15% of the pheasants collected at TLNWR had $\geqslant$55% brain AChE inhibition when compared to controls, which is sufficient for diagnosing cause of death (Ludke, *et al.*, 1975; Hill and Fleming, 1982; Grue *et al.*, 1983; Henny *et al.*, 1985; Blus *et al.*, 1989; Hill, 1992; and Hart, 1993). Most birds found dead as a result of antiChE poisoning have shown brain AChE inhibition of $>$50% (usually $>$80–90%) when compared to controls of the same species.

Sublethal effects of OP insecticide exposure to wildlife at TLNWR are of concern when considering possible changes in physiology and behavior, and are discussed in detail in Grue *et al.* (1991). Most pheasants collected in this study appeared normal (Grove *et al.*, in press), except one hen with 41% brain AChE inhibition. She was incoordinated and salivating; but this bird also had avian tuberculosis. Pheasants in a compromised physiological state may be more susceptible to antiChE exposure. Anorexia, disruption of thermoregulation, lethargy, muscle incoordination and piloerection often occur with exposure to antiChE insecticides (Grue *et al.*, 1983; Grue *et al.*, 1991). Expression, duration, and intensity are dose dependent, even though there is variability between compounds. Adult pheasants may have exhibited some of these effects, but telemetry observations were unable to confirm them. Two pheasants (approximately 45 days old) were found dead in 1990 as a result of methamidophos intoxication. Young pheasants may be more sensitive to antiChE insecticides than adults (Hudson *et al.*, 1984; Hill, 1992), but no pheasant young were radio-equipped in this study.

Hen pheasants collected at TLNWR were significantly smaller than hens collected at LKNWR (Grove *et al.*, in press). Later nesting observed in this study in combination with OP exposure (possible anorexia and loss of insects as an important protein source) could have impeded these birds' growth. OP exposure is frequently associated with body weight losses of up to 40% (Grue *et al.*, 1983), with body weight loss during critical periods potentially impeding growth of young (Martin *et al.*, 1996). One radio-equipped hen was found molting extremely late (29 November 1991); inadequate body reserves conceivably delayed feather replacement (Grove *et al.*, in press), but anorexia, resulting from OP exposure, could be partially responsible. Exposure to OP insecticides may also make pheasants more susceptible to predation, but we detected no radio-equipped hen mortalities resulting from predation during the insecticide portion of the study (Grove *et al.*, in press).

Conclusions

Direct mortality of young (two pheasant young found dead) and perhaps indirect effects via reduced prey availability for short periods immediately after OP insecticide application possibly impacted the pheasant population. The magnitude of insecticide effects on juvenile survival was uncertain (young were not radio-equipped), but direct toxicity of radio-equipped adults did not occur. In fact, few adults died during the summer spray season, and most nesting failures of radio-equipped hens (58%) occurred prior to insecticide applications. We conclude that insecticides were not the major factor impacting this declining pheasant population (see Grove *et al.*, in press), although insecticide possibly reduced growth and survival of young.

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