EFFECTS OF HEPTACHLOR- AND LINDANE-TREATED SEED ON CANADA GEESE

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Abstract: A study of Canada geese (*Branta canadensis*) was conducted in the Pacific Northwest from 1978 through 1981. Lowered reproductive success, mortality of adults, and a population decline of resident western Canada geese (*B. c. moffitti*) at the Umatilla National Wildlife Refuge (Umatilla NWR) in Oregon and Washington were associated with the use of heptachlor-treated wheat. Analyses of eggs and tissues from dead geese provided strong evidence that heptachlor was responsible for the mortality and decreased production. Residues of heptachlor epoxide (HE) in brains of *moffitti* found dead in 1978 and 1979 equalled or exceeded the lethal hazard zone of 8–9 μg/g in experimental passerine birds; HE residues of >10 μg/g in sample eggs were associated with low nest success. The breeding population of Canada geese at Umatilla decreased from 129 pairs in 1974 to about 100 pairs in 1979. The heptachlor problem was restricted to the Umatilla area; HE residues were low in tissues and eggs collected at other sites in Oregon, Washington, and Idaho. In September 1979, the use of heptachlor-treated seed was banned in a 1,700-km^2 area that encompassed both sides of the Columbia River near Umatilla. Lindane was substituted for heptachlor in the restricted area in 1979, and its use was extended to much of the Columbia Basin in 1981. Concurrently, reproductive success of geese increased, mortality decreased, and the nesting population increased to 170 pairs by 1983. There was no evidence for either biomagnification of lindane residues from treated seed to goose tissues or eggs or for induction of adverse effects by this compound.

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Treatment of seeds with toxic chemicals to deter pests has resulted in many incidents of wildlife intoxication either through ingestion of the treated seeds or secondarily through consumption of contaminated prey (Turtle et al. 1963, Flickinger and King 1972, Fuchs et al. 1972). Extensive use of heptachlor-treated cereal grains in the Columbia Basin results in adverse effects on wildlife that were first noted in the spring of 1976. Die-offs of ring-necked pheasants (*Phasianus colchicus*), California quail (*Callipepla californica*), and several other species of birds occurred in the Butter Creek area of Oregon (Umatilla and Morrow counties). Unusual mortality of resident Canada geese was first recorded on nesting islands at Umatilla in Oregon and Washington in April 1977. We initiated a study of the effects of heptachlor seed treatments on wildlife in the Columbia Basin in 1978.

This paper is a follow-up on Blus et al. (1979), where we reported that wildlife die-offs in the Columbia Basin were caused by heptachlor seed treatments, and we recommended that a less hazardous chemical such as lindane be used for wireworm (*Ctenicera*) control in this region. We here wish to report on the nesting success and insecticide levels of Canada geese in this same area from 1978 through 1981 and to evaluate what wildlife effects, if any, occurred as lindane replaced heptachlor in the control of wireworms. We thank the many individuals who assisted and encouraged us in this study; especially helpful were J. E. Kurtz and G. M. Constantino and their staff at Umatilla NWR; O. H. Vivion and H. W. Hill, Jr., and their

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STUDY AREAS, SEED TREATMENTS, AND METHODS

Study Areas

In March 1978, two study areas were established on the Columbia River (Fig. 1)—one at Umatilla and another about 65 km upstream in Washington at the McNary Recreation Area (McNary). Research continued at McNary through the 1980 nesting season and at Umatilla through the 1981 season. Nesting data for Umatilla were also obtained for the 1982 and 1983 nesting seasons (J. T. Annear, pers. commun.).

Seed Treatments

Starting about 1975, heptachlor seed treatment was routinely used to prevent wireworm damage on about 121,000 ha of cereal grains in north-central Oregon and was also the major seed treatment in Washington and other areas of the Pacific Northwest. The recommended treatment rate was 57 g of heptachlor per 27 kg of grain (2,000 μg/g). Contaminants in heptachlor include HE and related chlordane compounds (Burrage and Saha 1967). The seed was also treated with a fungicide, usually PCNB, at a rate of 14 to 21 g/27 kg of grain (500–800 μg/g). PCNB also contains HCB as a contaminant. HCB and methylmercury were also used as fungicidal seed treatments in certain areas of the Pacific Northwest. In the McNary area, lindane has been used as the principal seed treatment for wireworm control since at least 1978. In localized areas, farmers treated their wheat with only a fungicide before planting. Seeding rates varied from about 70 kg of wheat planted per ha on dryland to about 100 kg/ha on irrigated land.

Effective August 1979, the states of Oregon and Washington prohibited heptachlor seed treatments in an area of about 1,700 km^2 on either side of the Columbia River near Umatilla (Fig. 1). As of September 1982, production of heptachlor for use as a seed treatment in the United States was prohibited; however, there is a provision for using the existing stocks outside the restricted zone near Umatilla.

Methods

The islands at Umatilla and McNary were searched for nests each year beginning in mid-March. The nest counts at Umatilla were essentially complete each year, whereas only partial counts were made at McNary except in 1980. All nests located at Umatilla and most of those at McNary were marked and their fate determined. Laying dates, clutch size, location, and hatching data were recorded for each marked nest. A sample egg was collected from each clutch in one series of marked nests and the clutch was left intact in the other series. A nest was considered successful if one or more young hatched and left the nest. After nest failure, we examined the remaining eggs in most nests to determine embryonic development.

Collected eggs were weighed, measured, and the stage of development was recorded. The contents were weighed in chemically cleaned glass bottles and frozen. Shell thickness (shell plus membranes) was measured at three sites on the

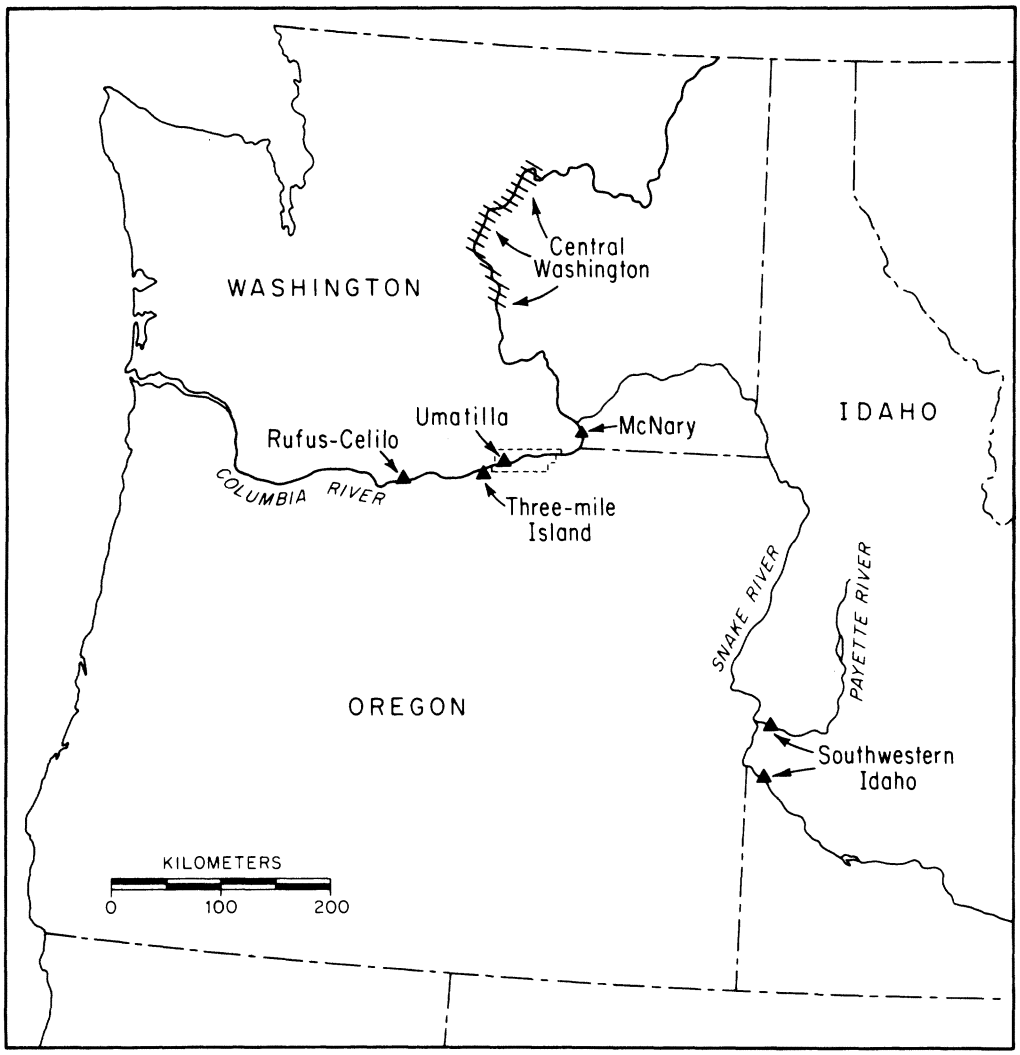

Fig. 1. Study areas and sites where eggs were collected in the Pacific Northwest. Restricted zone (see text) around Umatilla enclosed by dotted lines.

equator of the egg with a micrometer graduated in units of 0.01 mm; an average of the three measurements was taken.

Most geese found dead that were in suitable condition were sent to the U.S. Fish and Wildlife Service National Wildlife Health Laboratory (NWHL) in Madison, Wisconsin, where necropsies were performed. Tissues for histological study were fixed in 10% Formalin®, processed routinely, and stained with hematoxylin and eosin.

Eggs and tissues of geese were analyzed for residues of 18 organochlorines at the Patuxent Wildlife Research Center in Laurel, Maryland. Either homogenized whole samples or subsamples were blended with sodium sulfate to remove moisture, and extracted 7 hours with hexane in a Soxhlet apparatus. Lipids were re-

moved from organochlorine compounds by Florisil column chromatography (Cromartie et al. 1975) for all the samples collected in 1978–80 and for some of the samples collected in 1981. Lipids in most of the 1981–82 samples were removed by a Sep-Pak Florisil cartridge (Waters Assoc., Milford, Mass.).

Organochlorines and polychlorinated biphenyls (PCB's) were separated by silicic acid chromatography (Cromartie et al. 1975) for most of the samples. The organochlorines in the other samples were zoned by silica gel (100–200 mesh, grade 923) column chromatography utilizing the silicic acid solvent systems (Cromartie et al. 1975, Kaiser et al. 1980).

All samples were analyzed for DDE, DDD, DDT, dieldrin, heptachlor epoxide, oxychlordane, *cis*-chlordane, *cis*-nonachlor, *trans*-nonachlor, endrin, toxaphene, HCB (hexachlorobenzene), mirex, and PCB's. Additionally, samples were analyzed for the following pollutants: lindane—1978, 1979, 1980 (some), and 1981–82 (all); PCNB (pentachloronitrobenzene)—1979 (some); α- and β-HCH (hexachlorocyclohexane = benzene hexachloride)—1979 (some) and 1981–82 (all).

Residues were quantified on a gas-liquid chromatograph equipped with a ^{63}Ni detector, automatic sampler, and digital processor. Residues in 7% of the samples were confirmed on a gas chromatograph/mass spectrometer (Kaiser et al. 1980). Recoveries of organochlorine pollutants from fortified tissues of mallards (*Anas platyrhynchos*) ranged from 45 to 65% for *trans*-nonachlor and 77 to 112% for the others; recoveries from spiked chicken eggs were similar to those from mallard tissues. Residue levels were not corrected for recovery. The lower limit of reportable residues was 0.5 μg/g for PCB's and 0.1 μg/g for other pollutants.

Chi-square was used to test the percentage of successful nests; comparisons were made for each study area over several years and between study areas in a given year. One-way analysis of variance (ANOVA) was employed to determine differences in clutch size, eggshell thickness, and number of eggs hatched per successful nest or active nest. The multiple range test with adjustment for unequal sample sizes (Kramer 1956) was used to compare series of means derived from ANOVA. A test of two proportions (Kuebler and Smith 1976) was used in comparing success of 80 nests where a sample egg was collected and where nest fate was established. The two proportions included success of nests where sample eggs contained either ≤ 10.0 or > 10.0 μg/g HE.

RESULTS

Reproductive Characteristics

Canada geese are among the earliest nesters in the study areas; extreme egg-laying dates ranged from mid-February to the third week of April. Temporal aspects of nesting are only approximate because visits to nesting islands ranged from once a week to once every 3 weeks. By mid-March about 25% of the breeding pairs had initiated egg-laying, and nearly all clutches were completed by mid-April. Extreme hatching dates were late March and 24 May. The peak hatching period generally occurred the last half of April; relatively few eggs hatched after 1 May. Temporal aspects of hatching were similar at the two study areas.

Numbers of nests at Umatilla ranged from 126 in 1974 to 129 in 1975 (McCabe 1979); numbers of nests declined to about 114 in 1978 and 102 in 1979 (Table 1). An upturn in the breeding population at Umatilla was noted in 1980 when we counted 118 nests; the number of nests increased each year and reached 170 in

Table 1. Comparison of reproductive success of Canada geese, Umatilla and McNary, 1978–82.

Year	Area	Nests[b] (*N*)	Successful (%)	Clutch size[a]	Mean *N* eggs hatched per nest[a]	
					Successful	Active
1978	Umatilla	104	52	5.47 A	5.15 A	2.65 A
	McNary	52	90	6.13 B	5.67 A	5.05 B
1979	Umatilla	93	53	5.42 A	4.98 A	2.65 A
	McNary	113	88	5.63 A	5.36 A	4.57 B
1980	Umatilla	118	84	5.58 A	5.22 A	4.35 B
	McNary	155	94	5.76 AB	5.03 A	4.68 B
1981	Umatilla	120	77	5.53 A	5.33 A	4.08 B

[a] Means sharing the same letter are not different ($P > 0.05$) from one another.

[b] Total nests located. Nests with unknown fates or clutch sizes excluded when nest success, clutch size, and eggs hatched are calculated. Nest counts at McNary are not representative of total number present except in 1980. The number of nests at Umatilla was 116 in 1982 and 170 in 1983. Counts at Umatilla in 1978, 1979, and 1982 are approximately 10% low because one island was only partially searched in those years.

1983. Nest success at Umatilla also increased ($P < 0.001$) from 52% in 1978 and 53% in 1979 to 84% in 1980 and 77% in 1981. Nest predation was not a factor during the study except on Long Walk Island (Umatilla) in 1981 when several nests were destroyed and a few incubating birds were killed by a mammalian predator. The decrease in nest success from 1980 to 1981 at Umatilla was largely attributable to increased predation of nests on Long Walk Island where 83% of 23 nests were successful in 1980 compared with 45% of 20 nests in 1981. Geese readily desert ground nests when a large mammalian predator is present in the nesting area (Hanson and Eberhardt 1971). In 1974 and 1975, few Canada goose nests were present on Long Walk Island because of a coyote (*Canis latrans*) depredation problem (McCabe 1979). Nest success at McNary was excellent (88–94%) and exhibited no significant changes ($P > 0.10$) from 1978 to 1980. Nest success in 1978 and 1979 was higher ($P < 0.001$) at McNary than at Umatilla. Despite the marked increase in success at Umatilla in 1980, it was still lower ($P < 0.01$) than that recorded at McNary that year.

From 1978 to 1981, mean clutch size of geese at Umatilla ranged from 5.42 to 5.58 eggs, whereas it ranged from 5.63 to 6.13 at McNary (Table 1). The mean clutch of 6.13 eggs measured at McNary in 1978 was higher ($P < 0.05$) than other means except that recorded at McNary in 1980. There were no differences ($P > 0.05$) in the mean number of eggs that hatched per successful nest in the two study areas each year, whereas the mean number of eggs that hatched per active nest at Umatilla in both 1978 and 1979 was lower ($P < 0.05$) than the other means (Table 1). The proportion of eggs that hatched in active Canada goose nests in 1978 and 1979 was lower at Umatilla (50 and 51%) than at McNary (85 and 84%); hatching success improved at Umatilla in 1980 and was similar to that measured at McNary (81 and 82%). The percentage of eggs hatched at Umatilla decreased slightly to 74% in 1981.

At Umatilla, the percentage of embryonated eggs remaining in unsuccessful nests, in relation to the total number of eggs laid, decreased from 21% in 1978, to 11% in 1979, 6% in 1980, and 4% in 1981. Eggs from unsuccessful nests at Umatilla that contained large embryos decreased steadily from 13% of the total egg population in 1978 to 2.5% in 1981. In contrast, unsuccessful nests at McNary contained few embryonated eggs (0.1–2.6% of the total egg population) from 1978 through

Table 2. Residues of insecticides and fungicides on treated wheat seed.

Date	Collection area	Sample location	Residues (μg/g)[a]					
			H	TRCH	γ-HCH	Hg	PCNB	HCB
1978								
13 Sep	Boardman, Oreg. (recent)	Field[b]	105	34		0.16	83	
13 Sep	Boardman, Oreg. (3 weeks)	Field	90	32			26	
13 Oct	Paterson, Wash.	Bag	360	94			298	
5 Oct	Paterson, Wash.	Seeder						81
6 Oct	Paterson, Wash.	Seeder	210	60			178	
5 Oct	Paterson, Wash.	Seeder						245
12 Oct	Irrigon, Oreg.	Pheasant crop[c]	18	6			9	
14 Nov	Paterson, Wash.	Bag			0.31	10.0		
1980								
21 Nov	Rock Island, Wash.	Roadside[d]	40	13		NA		

[a] H = heptachlor, TRCH = *trans*-chlordane, γ-HCH = lindane, Hg = mercury, PCNB = pentachloronitrobenzene, HCB = hexachlorobenzene, NA = not analyzed, blank space = no residue detected (<0.1 μg/g).
[b] Spilled wheat collected from soil surface after recent planting and about 3 weeks after planting.
[c] Crop contents (some treated wheat) of road-killed ring-necked pheasant.
[d] Sample taken from about 400 kg dumped along road; it also contained additional residues (μg/g): heptachlor epoxide—0.47, *cis*-chlordane—2.6, and *cis*-nonachlor—0.17.

1980, and few of these eggs contained large embryos. Two recently hatched goslings were found dead in or around nests at Umatilla in 1978, six in 1979, three in 1980, and five in 1981; no dead goslings were found at McNary. The pattern of gosling mortality at Umatilla did not change appreciably with the improvement in nest success.

Adult Mortality

Spring mortality of adult *moffitti* was rare on the Umatilla nesting islands in 1974 and 1975 when only three fresh carcasses were found (T. R. McCabe, pers. commun.). In contrast, unusual mortality of geese was noted in the nesting areas in April 1977 (E. L. Bowhay, pers. commun.). During our initial visit to the nesting islands at Umatilla in March 1978, we located 30 dead geese. In 1978, a total of 22 *moffitti* and 35 individuals of the smaller overwintering subspecies identified as *B. c. parvipes* and *B. c. taverneri* (R. L. Jarvis, pers. commun.) were found dead at Umatilla. The number of *moffitti* found dead at Umatilla decreased from 22 in 1979, 8 in 1980, 5 in 1981, 1 in 1982 (one visit), to 0 in 1983 (two visits). The mortality figures are minimal because carcass counts were made only during nest searches with the exception of a single search of the nesting islands at Umatilla in February of 1979 and 1980. Of 27 Canada geese necropsied at the NWHL from 1978 through 1981, diagnoses for 14 *moffitti* included predation—3, and undetermined (open)—11. Diagnoses for 13 *taverneri* and *parvipes* included open—5, gunshot—4, lead poisoning—2, peritonitis—1, and aspergillosis—1. Mortality of *moffitti* continued into May in 1978 and 1979. Only five dead *moffitti* were found at McNary from 1978 to 1980.

Eggshell Thickness

Mean eggshell thickness of 183 contemporary eggs of *moffitti* at each site ranged

from 0.630 to 0.663 mm and was slightly less (0.4–5.8%, $P > 0.05$) than the pre-1947 norm of 0.669 mm that was derived from measurement of shells of 12 eggs from the Pacific Northwest that were housed in museum collections.

Organochlorine Residues

Treated Seed.—Generally, analysis of treated seeds revealed a lower residue concentration than the recommended treatment rate (Table 2); this held even for wheat taken from bags or seeders before planting. After planting, the residue profile changes dramatically, particularly after ingestion by birds or other forms of wildlife. The heptachlor is epoxidized, *trans*-chlordane is readily converted to oxychlordane, the PCNB is broken down with no residues detected in tissues, and lindane is also broken down rapidly and is rarely found in wildlife samples. Of the chemicals detected on treated seeds, HCB and mercury (Hg) are the only ones that persist to any extent in an unaltered form after planting.

Eggs.—Of the six areas in the Pacific Northwest where eggs of *moffitti* were collected, HE residues were highest in those from Umatilla (Table 3). However, by 1981, HE residues at Umatilla declined to a point where few eggs contained detectable quantities. Residues were also present in eggs laid in 1977 or before at Umatilla. Geese in the Columbia Basin may incubate old eggs that persist through the winter (Blus and Henny 1980); four of these eggs collected in March 1978 contained from 7.0 to 13.8 μg/g of HE. Except for Three-Mile Island in 1979, where HE was detected in seven of nine eggs, few eggs collected at four other sites in Oregon, Washington, and Idaho (Fig. 1) contained detectable residues. Residues other than HE that were associated with use of heptachlor seed treatments were oxychlordane, *trans*-nonachlor, and *cis*-nonachlor; these were found only in small quantities in eggs collected at Umatilla in 1978 and 1979. By 1980, residues of these three contaminants of heptachlor were not detected in eggs collected at Umatilla nor were they detected at the other five collection sites.

Residues of DDE, a metabolite of DDT, were rarely detected in the samples except at the central Washington collection site where small quantities (≤1.64 μg/g) were detected in 75% of the eggs collected in 1979. Low concentrations of PCB's, HCB, PCNB, DDT, DDD, and dieldrin were detected in a few eggs (Table 3). No residues were detected in the eggs collected in Idaho.

Nest success was highest at Umatilla when sample eggs contained ≤1.0 μg/g of HE, and success decreased only slightly at the 1.1–5.0- and 5.1–10.0-μg/g intervals, whereas success was lower ($P < 0.004$) when sample eggs contained >10.0 μg/g (Table 4). Six of 86 eggs (7%) collected at Umatilla contained >10 μg/g HE, but all of the eggs with high residues were collected in 1978 and 1979 and constituted 15% of the eggs (i.e., nests) sampled those years. Maximum HE residues were 17 μg/g in an egg from a successful nest and 27 μg/g in an egg from an unsuccessful nest. Other organochlorine residues detected in goose eggs at Umatilla were low and were not correlated with nest success. Low residues of several organochlorines were found infrequently at McNary; nest success was not related to residue content of the eggs.

Tissues.—Stickel et al. (1979) fed diets containing heptachlor to four species of passerines, and residues of HE were found in tissues of the treated birds. In experiments where passerines were fed diets containing heptachlor, probable death from HE was indicated when their brains

Table 3. Organochlorine residues in eggs of Canada geese, Pacific Northwest, 1978–81.

Year	Location[b]	N	Residues (μg/g, fresh wet weight)[a] DDE	HE	OXY	HCB
1978	U	21[c]	(1)	2.93 (20)	0.14 (11)	(9)
				1.93–4.47	0.11–0.17	
			ND–0.10	ND–24.29	ND–0.59	ND–0.24
	M	20		(3)		(6)
			ND	ND–0.52	ND	ND–0.57
1979	U	25[d]	(1)	1.49 (22)	(8)	(9)
				1.37–3.05		
			ND–0.11	ND–26.52	ND–0.79	ND–0.34
	M	18[e]		(4)		0.16 (9)
						0.08–0.31
			ND	ND–0.62	ND	ND–1.17
	R-C	17		(5)		
			ND	ND–0.69	ND	ND
	W	20	0.30 (16)[f]			
			ND–1.64			
			0.17–0.54	ND	ND	ND
	TMI	9		0.99 (7)		(1)
				0.23–4.27		
			ND	ND–6.41	ND	ND–0.14
1980	U	22		(10)		(9)
			ND	ND–2.50	ND	ND–2.97
	M	16		(2)		0.10 (8)
						0.05–0.19
			ND	ND–2.33	ND	ND–0.64
	R-C	8		0.14 (4)		(1)
				0.05–0.37		
			ND	ND–0.70	ND	ND–0.71
	TMI	6	(1)	(2)		(2)
			ND–0.19	ND–0.47	ND	ND–0.77
	ID	10	ND	ND	ND	ND
1981	U	24		(5)		(1)
			ND	ND–0.47	ND	ND–0.20

[a] HE = heptachlor epoxide, OXY = oxychlordane, HCB = hexachlorobenzene, ND = no residue detected, geometric mean (number of positive samples), 95% confidence limits, and range are listed for those contaminants detected in ≥50% of the samples. A value halfway between the 0.1 μg/g level of sensitivity and 0 was assigned each ND sample used in statistical calculations.

[b] All locations on the Columbia River except Idaho where collecting was done on the Snake and Payette rivers. U = Umatilla NWR, Oregon and Washington; M = McNary Recreation Area, Washington; R-C = Rufus-Celilo, Oregon; W = central Washington State; TMI = Three-Mile Island, Oregon; and ID = southwestern Idaho.

[c] Following pollutants were also detected (number of positive samples and range [μg/g]): *trans*-nonachlor = TNCH (5, ND–0.24); *cis*-nonachlor = CNCH (1, ND–0.10); polychlorinated biphenyls = PCB's (3, ND–0.74).

[d] TNCH (4, ND–0.29), CNCH (2, ND–0.24).

[e] Dieldrin (1, ND–0.11).

[f] DDT (4, ND–0.22), DDD (1, ND–0.25).

contained residues in the lethal-hazard zone (8–9 μg/g), and death from HE was essentially diagnostic when residues exceeded 9 μg/g. Analysis of brains of 17 *moffitti* found dead at Umatilla in 1978 and 1979 revealed that 1 contained 8 μg/g and 15 contained >9 μg/g of HE (Table 5). In 1980, HE levels in brains of four *moffitti* found dead were below the lethal-hazard zone although one bird contained 5.50 μg/g. In 1981, two of six *moffitti* found dead contained lethal levels of HE, whereas none of the four found dead in 1982 contained HE residues in their brains. Brains of four of seven *moffitti* that were shot in 1979 at Umatilla contained HE; residues in one bird were very high—6.6 μg/g—and were near the lethal-haz-

ard zone. Analysis of two brains of *moffitti* found dead at McNary contained no detectable residues of organochlorines. Of the 30 *parvipes* and *taverneri* that were found dead at Umatilla and analyzed, HE residues were detected in brains of 19 birds. Most of the residues were low and the highest values (3.40 and 4.58 μg/g) were below the lethal-hazard zone. Before initiation of this study, the brain of a Canada goose (*taverneri* or *parvipes*) that was found dead in February 1977 contained a lethal level of HE (G. R. McLeod, pers. commun.).

Residues of other contaminants of heptachlor were also found in tissues of geese. Oxychlordane (OXY) was detected in brains of all geese that died with lethal levels of HE; however, residues were below the lethal level (Stickel et al. 1979). HE/OXY ratios in goose tissues averaged 29:1 and ranged from 15:1 to 50:1. OXY was not detected in samples containing low residues of HE; it initially appeared when HE residues were near 3 μg/g. Residues of *cis*-nonachlor and PCB's were not detected in tissues of geese, although low concentrations were found in a few eggs. Low concentrations of HCB occurred infrequently and generally were present in geese with high levels of HE. Low levels of lindane were found in brains of three *moffitti* that were found dead in 1981, but these residues were not confirmed by mass spectrometry.

DISCUSSION

Heptachlor

The most likely source of HE residues in tissues and eggs of Canada geese was from ingestion of heptachlor-treated wheat seed. The hazards of heptachlor seed treatments to Canada geese and other wildlife are related to concentrated amounts on seed when ingested rather than the small amount of toxicant applied per unit area. Examination of planting practices in the autumn of 1978 revealed that treated grain was exposed and available to geese in dryland and irrigated fields and along roads. Also, excess treated grain may be disposed of in such a manner that it is available to wildlife; about 400 kg of heptachlor-treated wheat was dumped along a rural road near Rock Island, Washington, in October 1980. Some aerial seeding was also noted. The aerially applied seed is exposed on the soil surface until the field is worked, and some seed falls into areas adjacent to the fields. Resident *moffitti* occupy plowed land in irrigated circles and chisel-plowed dryland wheat stubble before and after the planting of wheat (Aug–Oct). Aerial and ground observations near Umatilla in the autumn of 1978 revealed several instances where nearly the entire resident goose flock was feeding in a field that was recently planted with heptachlor-treated wheat. Most overwintering *taverneri* and *parvipes* arrive in the area in mid-November when planting of winter wheat is completed, and they generally migrate north before seeding of the small acreage of spring wheat. Thus, the more numerous overwintering geese are less likely to be exposed to treated wheat than resident geese; this is borne out by their lower residues and lower prevalence of mortality from heptachlor.

Table 4. Relation of heptachlor epoxide residues in sample eggs of Canada geese to nest success, Umatilla, 1978–81.

	Heptachlor epoxide (μg/g, fresh wet weight)			
	≤1.0	1.1–5.0	5.1–10.0	>10.0
Nests (*N*)	48	22	10	6
Successful (%)	83	77	80	17

Umatilla was the area where *moffitti* received greatest exposure to heptachlor; analysis of eggs from southwestern Idaho, McNary, Rufus-Celilo, and central Wash-

Table 5. Residues of organochlorines in brains of Canada geese found dead or shot by hunters, Columbia Basin, 1978–82.

Year	Sex	Age	N[b]	Residues (μg/g, fresh wet weight)[a] HE	OXY	HCB
				B. c. moffitti		
1978	M	Ad	2 (1)	ND–20.37 (1)	ND–1.33 (1)	ND–0.46 (1)
	Unk	Unk	5 (5)[c]	8.00–21.36 (5)	0.46–0.86 (5)	0.19–0.52 (5)
				B. c. taverneri and *parvipes*		
	F	Imm	1 (0)	4.58	0.36	ND
	Unk	Unk	7 (0)	ND–0.25 (4)	ND	ND
				B. c. moffitti		
1979	M	Ad	5 (4)[d]	ND–49.00 (4)	ND–1.30 (4)	ND–0.22 (4)
	F	Ad	1 (1)[e]	15.00	0.48	ND
	F	Imm	2 (2)[f]	16.00–20.00 (2)	0.45–0.78 (2)	0.17–0.20 (2)
	Unk	Ad	1 (1)	10.00	0.32	0.11
	Unk	Unk	2 (2)	11.00–21.11 (2)	0.48–0.99 (2)	0.12–0.86 (2)
	M	Ad	1 (0)[g]	ND	ND	ND
	F	Ad	1 (0)[g]	ND	ND	ND
	M	Imm	2 (0)[g]	1.50–1.80 (2)	ND	ND
	F	Imm	2 (0)[g]	ND–2.60	ND	ND
	Unk	Imm	1 (0)[g]	6.60	0.23	ND
				B. c. taverneri and *parvipes*		
	F	Ad	2 (0)	0.14–0.90 (2)	ND	ND–0.10 (1)
	F	Imm	1 (0)	2.30	ND	ND
	Unk	Unk	10 (0)	ND–0.76 (5)	ND	ND–0.12 (1)
				B. c. moffitti		
1980	F	Imm	1 (0)	5.50	0.26	ND
	Unk	Unk	3 (0)	ND	ND	ND
				B. c. taverneri and *parvipes*		
	M	Ad	2 (0)	ND–3.40 (1)	ND–0.13 (1)	ND
	F	Ad	1 (0)	0.75	ND	ND
	M	Imm	1 (0)	ND	ND	ND
	Unk	Unk	3 (0)	ND–2.70 (2)	ND–0.13 (1)	ND
				B. c. moffitti		
1981	F	Ad	3 (1)[h]	ND–13.00 (1)	ND–0.53 (1)	ND
	M	Imm	1 (1)[i]	22.00	0.52	ND
	Unk	Unk	1 (0)	ND	ND	ND
1982	M	Ad	1 (0)	ND	ND	ND
	Unk	Unk	3 (0)	ND	ND	ND

[a] HE = heptachlor epoxide, OXY = oxychlordane, HCB = hexachlorobenzene, ND = no residue detected.
[b] N = number of brains analyzed; number of geese dying with lethal levels of HE in parentheses.
[c] Contained 0.14 μg/g *trans*-nonachlor (TNCH).
[d] TNCH detected in four samples (0.19–0.68 μg/g) and *cis*-nonachlor (CNCH) in two (0.15–0.16 μg/g).
[e] Contained 0.12 μg/g CNCH.
[f] Both contained TNCH (0.19–0.25 μg/g).
[g] This series of seven *moffitti* shot by hunters at Umatilla; all others found dead.
[h] Two contained lindane (0.10–0.25 μg/g).
[i] Contained 0.12 μg/g TNCH.

ington indicated little or no exposure to heptachlor. HE residues in goose eggs from Three-Mile Island in 1979 were higher than in all other areas except Umatilla; however, all residues were below the critical level. Reasons for the differences in exposure of geese to heptachlor at certain collection areas were not clear-cut. Heptachlor seed treatments were almost certainly used to some extent in fields near

several of the areas, especially Three-Mile Island, which is only 24 km downstream from the major nesting areas at Umatilla; however, no detailed data were available on use of treated seed or goose-feeding patterns in those areas.

Ingested heptachlor is readily metabolized to HE, which is lipid soluble and readily stored in body fat (Cummings et al. 1967, Burrage and Saha 1972, de Vos et al. 1972). Most mortality of Canada geese occurs in late winter and early spring when stresses from reproduction, weather, food shortage, and migration (*taverneri* and *parvipes*) result in mobilization of fat reserves containing HE (Ecobichon and Saschenbrecker 1969, Findlay and deFreitas 1971). The bird is then susceptible to stress interacting with the epoxide and if sufficient residues are mobilized, adverse sublethal effects involving reproduction, behavior, or other aspects may occur and mobilization may prove lethal to some birds (deFreitas et al. 1969, Stickel and Rhodes 1970, Van Velzen et al. 1972).

A large segment of the avifauna in the region was exposed to heptachlor either by consumption of treated seed or secondarily by eating contaminated prey. Representatives of at least eight species died from this chemical (L. J. Blus et al., unpubl. data; Henny et al. 1984). Mortality and reduced reproductive success of American kestrels (*Falco sparverius*) were documented (Henny et al. 1983). Kestrels were more sensitive to heptachlor than Canada geese; reproductive impairment commenced when residues in eggs exceeded 1.5 μg/g compared with 10 μg/g in geese. In female Japanese quail (*Coturnix japonica*) that were given daily oral doses of 0.5–1 mg of heptachlor for 18–32 days, the fecundity rate and embryonic mortality were not affected. However, 50% of young died within 1 week of hatching in the group receiving the greatest exposure; mean HE residues in eggs from this group ranged from 14 to 17 μg/g (Grolleau and Froux 1973). In a related study, female gray partridges (*Perdix perdix*) that were given daily oral doses of 0.5–1.0 mg of heptachlor for 18 days laid eggs that contained 2–4 μg/g of HE and 3–7 μg/g, respectively. Hatching success was not affected in either treatment group, and survival of healthy chicks up to 30 days was 3% greater than controls in the low-treatment group and 13% less in the high-treatment group (Havet 1971).

The mode of action of heptachlor in depressing reproductive success of Canada geese is not known; it may be related to embryotoxicity or adverse effects on adults that result in nest desertion as a result of mortality or behavioral aberrations. Most unsuccessful nests at Umatilla seemed deserted. Desertion of Canada goose nests usually occurs early in the nesting cycle, particularly during egg-laying; desertion late in incubation as occurred at Umatilla is unusual (Hanson and Eberhardt 1971, Cooper 1978). The percentage of abandoned eggs at Umatilla increased from 12% in 1974 to 45% in 1975 (McCabe 1979). This apparently coincided with the increased use of heptachlor in the area, but initiation of use and rate of changeover to heptachlor are not precisely known. By 1978, seed wheat intended for north-central Oregon was routinely treated with heptachlor. Heptachlor was also the predominant seed treatment for wireworm control in Washington in 1978.

Long-term declines in northern bobwhite quail (*Colinus virginianus*) populations in Georgia (Rosene 1965) were associated with applications of granular heptachlor at 0.6 or 2.2 kg/ha. Short-term abrupt declines in populations of several passerines in Mississippi were noted shortly after heptachlor was applied at 0.3, 0.6, or 2.2 kg/ha (Ferguson 1964).

Lindane

The general absence of lindane in tissues and eggs of Canada geese at Umatilla in 1980 and 1981 and in other areas where lindane seed treatment was employed indicates that either geese were not ingesting the treated seed or that lindane was not retained to any extent in tissues in the unaltered form. After ingestion, lindane is rapidly metabolized to water-soluble chlorophenols and chlorobenzenes that are readily excreted. Its half-life in tissues and eggs of birds is much lower than other organochlorine insecticides (Cummings et al. 1966, 1967; Maďarič and Fellegiová 1977; Kan and Jonker-Den Rooyen 1978). Other desirable qualities of lindane are that it is degraded rapidly after exposure to field conditions (Copeland and Chadwick 1979), and it is generally less toxic and more repellant than other organochlorine seed treatments (Carnaghan and Blaxland 1957, Turtle et al. 1963). Although lindane is less bioaccumulative than HE, low levels of this insecticide were found in eggs, tissues, and young of ring-necked pheasants (Burrage and Saha 1972) and domestic turkeys (Stadelman et al. 1965) that ingested lindane-treated seed. Although French and Jefferies (1968) speculated that γ-HCH (=lindane) treated grain is as acceptable to wild birds as untreated grain, this is not borne out in other studies where domestic pigeons (Turtle et al. 1963) and ring-necked pheasants (Burrage and Saha 1972) were moderately to extremely repelled by treated seed. Lindane is odorless and tasteless (Ulmann 1972), whereas technical lindane contains the γ isomer (99%) in addition to contaminants including related isomers ($\leq$0.05% each) and other materials (Copeland and Chadwick 1979) that are probably responsible for any repellancy to birds (Burrage and Saha 1972). We made no analyses of lindane-treated seed; however, the repellant, more persistent isomers were not detected in tissues or eggs of the geese at Umatilla.

French and Jefferies (1968) concluded that there are practical difficulties in demonstrating mortality of wild birds from γ-HCH because of postmortem breakdown of lindane residues in livers of homing pigeons administered a capsule dose of lindane. Although there is undoubtedly postmortem breakdown of lindane, residues should still be detected in tissues of birds dying from this material, particularly if they are found soon after death and then frozen. The brain is usually preferred in deriving lethal diagnostic residues of organochlorines in birds (Stickel et al. 1966), but such data are unavailable for γ-HCH. Analyses of granivorous birds found dead in the field in Great Britain in 1961 indicated high residues of dieldrin and HE, but only one-third contained γ-HCH residues in small amounts (Turtle et al. 1963). The toxicity of lindane to birds was demonstrated when nearly half of a flock of domestic pigeons died several days after receiving a diet that was inadvertently contaminated with lindane-treated seed; however, the level of contamination (2,100 μg/g) was very high (Blakely 1982). French and Jefferies (1968) suspected, but did not demonstrate, that lindane-treated seed was hazardous to wild birds. To date, lindane has not been implicated in any problem with populations in the field. As in our study, changes in seed treatment from hazardous compounds such as aldrin, dieldrin, or heptachlor to lindane have ameliorated problems related to mortality and sublethal effects associated with seed-treatment chemicals (Carnaghan and Blaxland 1957, Turtle et al. 1963, Burrage and Saha 1972).

Results of experiments with domestic chickens to elucidate the effects of dietary

lindane on reproductive success are somewhat contradictory. No obvious reproductive problems were noted in birds presented diets containing 0.1, 1.0, and 10.0 μg/g of lindane for 2 months (Ware and Naber 1961). In a duplication of that study, reductions of about 10% in hatchability, 8–18% in eggshell thickness, and 20% in egg production occurred at each dietary level (Sauter and Steele 1972). A dietary level of about 100 μg/g of encapsulated lindane did not result in any reproductive problems until the birds were on dosage for several weeks (Whitehead et al. 1972). The only adverse effect noted was a 20% decrease in egg production when birds were given the diet for 6 weeks. A mixture of low dietary levels (0.05–1 μg/g) of each of seven organochlorines including α-, β-, and γ-HCH resulted in no observed reproductive problems in chickens (Kan and Tuinstra 1976). Lindane seed treatment apparently had no effect on reproduction of geese in our study; this conclusion is based on the absence of residues of lindane and other HCH isomers in eggs and tissues of geese and the general improvement in reproductive success that coincided with the changeover from heptachlor to lindane seed treatment.

PCNB and HCB

Contamination of PCNB is probably the primary source of HCB residues in eggs and tissues of Canada geese in north-central Oregon. In other areas, HCB residues originate from its use as a fungicidal seed treatment. Apparently, a small amount of HCB originates from breakdown of lindane (Engst et al. 1977), but in our study, HCB residues declined in eggs of geese at Umatilla after the use of lindane-treated seed was initiated in the restricted zone.

PCNB is not bioaccumulative, but HCB is lipid soluble and accumulative (Reed et al. 1977, Dunn et al. 1979). Residues of PCNB and HCB posed no identifiable threat to the geese; this conclusion is based on experimental evidence (Vos et al. 1971, Reed et al. 1977, Dunn et al. 1979) in conjunction with absence of PCNB residues and the infrequent occurrence of low residues of HCB in tissues and eggs.

The decline in HCB residues in goose eggs at Umatilla was apparently due to a change in fungicide from PCNB to Vitavax® (carboxin) within the past several years in north-central Oregon (R. L. Warner, pers. commun.). The potential for transforming lindane to HCB is low and occurs only in certain microorganisms (Engst et al. 1977); this metabolic pathway apparently represents no hazard to higher organisms such as the Canada goose.

The decline in the breeding population of *moffitti* at Umatilla seemed related primarily to widespread use of heptachlor, and the subsequent recovery was related to the changeover to lindane. The treated seed was apparently the sole source of heptachlor to the geese; and, when it was no longer available, residues of HE markedly declined in tissues and eggs of Canada geese. The elevated residues in brains of several *moffitti*, *taverneri*, and *parvipes* in the spring of 1980 and the heptachlor-induced mortality of two *moffitti* in the spring of 1981 indicated that some geese were still exposed to heptachlor-treated seed. Extensive observations in autumn 1978 revealed that large numbers of resident and overwintering Canada geese occasionally flew 30 km or more from the Columbia River to feed in dryland wheat fields.

The food chain of geese is relatively simple and consists largely of vegetative material. Because plants ordinarily have little potential for translocation of organochlorines into their tissues, hazards of these

chemicals to geese ordinarily result from external application to the foliage or seeds. Several organochlorine seed treatments also resulted in wildlife mortality in other areas. From 1973 to 1977, dieldrin seed treatments were considered responsible for 69 incidents involving wildlife mortality in Great Britain; a large number of pink-footed geese (*Anser brachyrhynchus*) found dead in Scotland contained high residues of dieldrin in their tissues (Stanley and Bunyan 1979). Mortality of snow geese (*Chen caerulescens*) and a number of other waterfowl occurred on the Texas Gulf Coast because of ingestion of rice seed treated with aldrin (Flickinger and King 1972).

Canada goose populations are generally flourishing in all parts of their range (Bellrose 1976). Die-offs from pollutants including organochlorines, organophosphates (Zinkl et al. 1978, White et al. 1982), and lead shot (Trainer and Hunt 1965) are usually of short duration and generally have had little impact on population stability. However, the study at Umatilla demonstrates that goose populations are vulnerable to certain types of pollutants when high concentrations are applied directly to their food resource.

LITERATURE CITED

BELLROSE, F. C. 1976. Ducks, geese and swans of North America. Stackpole Books, Harrisburg, Pa. 543pp.

BLAKELY, B. R. 1982. Lindane toxicity in pigeons. Can. Vet. J. 23:267–268.

BLUS, L. J., AND C. J. HENNY. 1980. Canada geese incubate eggs laid in previous years. West. Birds 11:112.

———, ———, D. J. LENHART, AND E. CROMARTIE. 1979. Effects of heptachlor-treated cereal grains on Canada geese in the Columbia Basin. Pages 105–116 *in* R. L. Jarvis and J. C. Bartonek, eds. Management and biology of Pacific Flyway geese: a symposium. Oregon State Univ. Book Stores, Inc., Corvallis.

BURRAGE, R. H., AND J. G. SAHA. 1967. Insecticide residues in spring wheat plants grown in the field from seed treated with aldrin or heptachlor. Can. J. Plant Sci. 47:114–115.

———, AND ———. 1972. Insecticide residues in pheasants after being fed on wheat seed treated with heptachlor and ^{14}C-lindane. J. Econ. Entomol. 65:1013–1017.

CARNAGHAN, R. B. A., AND J. D. BLAXLAND. 1957. The toxic effect of certain seed-dressings on wild and game birds. Vet. Rec. 69:324–325.

COOPER, J. A. 1978. The history and breeding biology of the Canada geese of Marshy Point, Manitoba. Wildl. Monogr. 61. 87pp.

COPELAND, M. F., AND R. W. CHADWICK. 1979. Bioisomerization of lindane in rats. J. Environ. Pathol. Toxicol. 2:737–749.

CROMARTIE, E., ET AL. 1975. Residues of organochlorine pesticides and polychlorinated biphenyls and autopsy data for bald eagles, 1971–72. Pestic. Monit. J. 9:11–14.

CUMMINGS, J. G., M. EIDELMAN, V. TURNER, D. REED, K. T. ZEE, AND R. E. COOK. 1967. Residues in poultry tissues from low level feeding of five chlorinated hydrocarbon insecticides to hens. J. Assoc. Off. Anal. Chem. 50:418–425.

———, K. T. ZEE, V. TURNER, F. QUINN, AND R. E. COOK. 1966. Residues in eggs from low level feeding of five chlorinated hydrocarbon insecticides to hens. J. Assoc. Off. Anal. Chem. 49: 354–364.

DEFREITAS, A. S. W., J. S. HART, AND H. V. MORLEY. 1969. Chronic cold exposure and DDT toxicity. Pages 361–366 *in* M. W. Miller and G. G. Berg, eds. Chemical fallout: current research on persistent pesticides. Charles C Thomas, Springfield, Ill.

DE VOS, R. H., J. BOUWMAN, AND A. B. ENGEL. 1972. Residues of organochlorine pesticides in broilers from feed fortified with known levels of these compounds. Pestic. Sci. 3:421–432.

DUNN, J. S., P. B. BUSH, N. H. BOOTH, R. L. FARRELL, D. M. THOMASON, AND D. D. GOETSCH. 1979. Effect of pentachloronitrobenzene upon egg production, hatchability, and residue accumulation in the tissues of white leghorn hens. Toxicol. Appl. Pharmacol. 48:425–433.

ECOBICHON, D. J., AND P. W. SASCHENBRECKER. 1969. The redistribution of stored DDT in cockerels under the influence of food deprivation. Toxicol. Appl. Pharmacol. 15:420–432.

ENGST, R., R. M. MACHOLZ, AND M. KUJAWA. 1977. Recent state of lindane metabolism. Residue Rev. 68:59–90.

FERGUSON, D. E. 1964. Some ecological effects of heptachlor on birds. J. Wildl. Manage. 28:158–163.

FINDLAY, G. M., AND A. S. W. DEFREITAS. 1971. DDT movement from adipocyte to muscle cell during lipid utilization. Nature 229:63–65.

FLICKINGER, E. L., AND K. A. KING. 1972. Some effects of aldrin-treated rice on Gulf Coast wildlife. J. Wildl. Manage. 36:706–727.

FRENCH, M. C., AND D. J. JEFFERIES. 1968. Disappearance of γ BHC from avian liver after death. Nature 219: 164–166.

FUCHS, P., J. ROOTH, AND R. H. DE VOS. 1972. Residue levels of persistent chemicals in the Netherlands in the period from 1965–1971. TNO-Nieuws 27:532–541.

GROLLEAU, G., AND Y. FROUX. 1973. Effet de l'heptachlore sur la reproduction de la caille *Coturnix coturnix japonica*. Ann. Zool.-Ecol. Anim. 5:261–270.

HANSON, W. C., AND L. L. EBERHARDT. 1971. A Columbia River Canada goose population, 1950–1970. Wildl. Monogr. 28. 61pp.

HAVET, P. 1971. Effets de l'heptachlore sur la reproduction de la perdix rouge *Alectoris rufa* L. et de la perdix grise *Perdix perdix* L. Trans. Int. Union of Game Biol. 10:175–183.

HENNY, C. J., L. J. BLUS, AND T. E. KAISER. 1984. Heptachlor seed treatment contaminates hawks, owl, and eagles of Columbia Basin, Oregon. Raptor Res.:In Press.

———, ———, AND C. J. STAFFORD. 1983. Effects of heptachlor on American kestrels in the Columbia Basin, Oregon. J. Wildl. Manage. 47: 1080–1087.

KAISER, T. E., ET AL. 1980. Organochlorine pesticides, PCB, and PBB residues and necropsy data for bald eagles from 29 states—1975–77. Pestic. Monit. J. 13:145–149.

KAN, C. A., AND J. C. JONKER-DEN ROOYEN. 1978. Accumulation and depletion of some organochlorine pesticides in high-producing laying hens. J. Agric. Food Chem. 26:935–940.

———, AND L. G. M. TH. TUINSTRA. 1976. Effect on performance of a low level mixture of organochlorine insecticides in broiler breeder feed. J. Agric. Food Chem. 24:772–775.

KRAMER, C. Y. 1956. Extension of multiple range tests to group means with unequal numbers of replications. Biometrics 12:307–310.

KUEBLER, R. R., AND H. SMITH, JR. 1976. Statistics: a beginning. John Wiley & Sons, New York, N.Y. 320pp.

MCCABE, T. R. 1979. Productivity and nesting habitat of Great Basin Canada geese, Umatilla, Oregon. Pages 117–129 *in* R. L. Jarvis and J. C. Bartonek, eds. Management and biology of Pacific Flyway geese: a symposium. Oregon State Univ. Book Stores, Inc., Corvallis.

MAĎARIČ, AND M. FELLEGIOVÁ. 1977. Distribution of ^{14}C-lindane in Japanese quail using autoradiography of whole body sections. J. Environ. Sci. Health B12:245–250.

REED, D. L., P. B. BUSH, N. H. BOOTH, J. T. KIKER, D. C. GOETSCH, AND R. L. FARRELL. 1977. Tissue residues from feeding pentachloronitrobenzene to broiler chickens. Toxicol. Appl. Pharmacol. 42:433–441.

ROSENE, W., JR. 1965. Effects of field applications of heptachlor on bobwhite quail and other wild animals. J. Wildl. Manage. 29:554–580.

SAUTER, E. A., AND E. E. STEELE. 1972. The effect of low level pesticide feeding on the fertility and hatchability of chicken eggs. Poult. Sci. 51:71–76.

STADELMAN, W. J., B. J. LISKA, B. E. LANGLOIS, G. C. MOSTERT, AND A. R. STEMP. 1965. Persistence of chlorinated hydrocarbon insecticide residues in chicken tissues and eggs. Poult. Sci. 44:435–437.

STANLEY, P. I., AND P. J. BUNYAN. 1979. Hazards to wintering geese and other wildlife from the use of dieldrin, chlorfenvinphos and carbophenothion as wheat seed treatments. Proc. R. Soc. London B. 205:31–45.

STICKEL, L. F., AND L. I. RHODES. 1970. The thin eggshell problem. Pages 31–35 *in* J. S. Gillett, ed. The biological impact of pesticides in the environment. Oregon State Univ. Press, Corvallis.

———, W. H. STICKEL, AND R. CHRISTENSEN. 1966. Residues of DDT in brains and bodies of birds that died on dosage and in survivors. Science 151:1549–1551.

———, ———, R. D. MCARTHUR, AND D. L. HUGHES. 1979. Chlordane in birds: a study of lethal residues and loss rates. Proc. Inter-Am. Conf. Toxicol. Occup. Med. 10:387–396.

TRAINER, D. O., AND R. A. HUNT. 1965. Lead poisoning of waterfowl in Wisconsin. J. Wildl. Manage. 29:95–103.

TURTLE, E. E., ET AL. 1963. The effects on birds of certain chlorinated insecticides used as seed dressings. J. Sci. Food Agric. 14:567–577.

ULMANN, E., editor. 1972. Lindane/monograph of an insecticide. Verlag K. Schillinger, Freiburg, West Germany. 384pp.

VAN VELZEN, A. C., W. B. STILES, AND L. F. STICKEL. 1972. Lethal mobilization of DDT by cowbirds. J. Wildl. Manage. 36:733–739.

VOS, J. G., H. L. VAN DER MAAS, A. MUSCH, AND E. RAM. 1971. Toxicity of hexachlorobenzene in Japanese quail with special reference to porphyria, liver damage, reproduction, and tissue residues. Toxicol. Appl. Pharmacol. 18:944–957.

WARE, G. W., AND E. C. NABER. 1961. Lindane in eggs and chicken tissues. J. Econ. Entomol. 54: 675–677.

WHITE, D. H., C. A. MITCHELL, L. D. WYNN, E. L. FLICKINGER, AND E. J. KOLBE. 1982. Organophosphate insecticide poisoning of Canada geese in the Texas Panhandle. J. Field Ornithol. 53: 22–27.

WHITEHEAD, C. C., A. G. DOWNING, AND R. J. PETTIGREW. 1972. The effects of lindane on laying hens. Br. Poult. Sci. 13:293–299.

ZINKL, J. G., J. RATHERT, AND R. R. HUDSON. 1978. Diazinon poisoning in wild Canada geese. J. Wildl. Manage. 42:406–408.

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