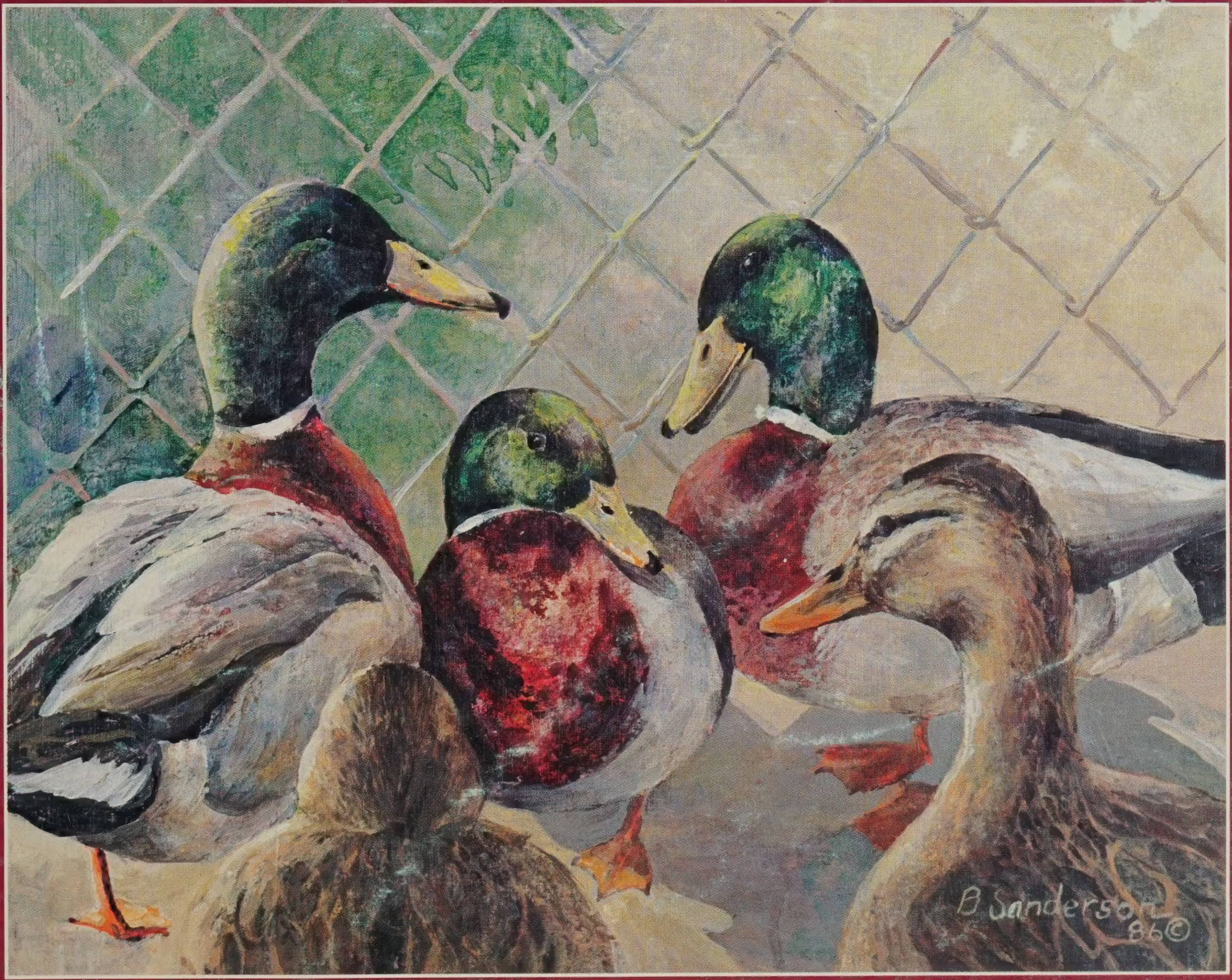
A Review of the Problem of

Lead Poisoning in Waterfowl

Glen C. Sanderson
Frank C. Bellrose



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Lead Poisoning in Waterfowl

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Abstract

Waterfowl die from ingesting lead shotgun pellets deposited on the bottoms of lakes and marshes and in fields. In most instances, they die after ingesting 1 or 2 pellets, their bodies wasting away over a period of several weeks, losing from 30 to 50 percent of normal weight. No other disease produces such a consistent chronic weight loss. Less frequently, a large number of shot are ingested, an acute form of lead poisoning results, and the bird dies in good weight. More definitive diagnoses of lead toxicosis have been made from levels of lead in wing bones, in blood, and in the liver and other organs.

Because of the widespread distribution of lead shot from the northern breeding grounds to the southern wintering grounds, it is available fall through spring to waterfowl feeding on areas that have been hunted. As a result, mortality accrues on a day-to-day basis. These losses, however, are usually overlooked because predators quickly dispose of moribund birds. Studies in Missouri and Texas, for example, revealed that predators rapidly removed waterfowl carcasses placed by biologists in wetland habitats. Moreover, dead ducks in natural settings are diffficult to find, and freshly planted carcasses in marsh vegetation were largely overlooked by searchers employed to find them. Only when massive die-offs of waterfowl occur in a limited area do losses from lead poisoning attract public attention. Such die-offs are the result of unusually high rates of shot ingestion; however, nutrition and low temperatures may be ancillary causes. Under these conditions, waterfowl die in numbers that exceed the ability of predators to consume them and to keep the environment tidy. Most die-offs from lead toxicosis occur after the hunting season—in winter and early spring.

The potential impact of lead poisoning on waterfowl populations has been ascertained from diverse sources of information. Almost 200,000 gizzards from more than 16 species of waterfowl in a number of geographical regions have been examined for lead shot during hunting seasons in fall and early winter. Scores of experiments with penned wild and game-farm waterfowl have been conducted by numerous investigators to evaluate the effects of shot dose, nutrition, age, and sex and to study the physiological manifestations of lead toxicosis. A pertinent finding of nutrition studies was that protein, calcium, and phosphorus play an important role in determining the lethality of lead.

Species of waterfowl vary in their proclivity to ingest shot and, because of differing food habits, in their susceptibility to ingested lead. Lead toxicosis poses the greatest threat to mallards, followed in lessening degrees by black ducks, mottled ducks, pintails, canvasbacks, redheads, and ring-necked ducks. The potential for lead poisoning in other duck species is low. At times swans and geese become victims in numbers sufficiently large to cause concern.

The use of steel shot as a substitute for lead shot in waterfowl hunting is the only currently feasible solution to the problem of lead poisoning. Steel shot is less dense than lead shot but produces a tighter pattern and shorter shot string. The lower density of steel shot can be compensated for by increasing shot size and velocity, thus delivering similar levels of energy to the target. No significant differences in crippling rates were found in all but 3 of 15 tests comparing the effects of steel and lead shot. In only 1 of those 3 tests did steel shot cripple more ducks than the lead shot being tested. Although not significantly different, rates of ducks lost from crippling wounds with steel shot were 5.3 percent lower in a Michigan study, 4.9 percent lower to 7.3 percent higher in a Missouri study, and 14.3 percent higher in a Louisiana study. The marshes hunted in Missouri and Michigan, however, are more typical of the vast majority of hunting habitats than the marsh hunted in Louisiana.

Crippling losses to waterfowl populations from steel shot are less harmful than crippling losses plus lead toxicosis from lead shot. Several related points merit consideration. Lead poisoning causes important losses to the most abundant species of waterfowl. The sublethal effects of lead poisoning are recognized but have not been quantified. Except for a brief period in spring, lead may affect females more adversely than males. Finally, seasonal differences in the time of losses are important. A cripple lost during the hunting season has less impact on the breeding population than a lead-poisoned duck lost during the winter or spring.

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Introduction

Although lead poisoning in waterfowl has generated controversy in recent years—primarily from the questioning of its extent by hunters opposed to steel shot—the problem has been recognized for more than 100 years. In 1874, two groups of ducks killed at Stephenson Lake, Galveston, Texas, were confiscated as unfit for human consumption, presumably as a result of lead poisoning. Lead-poisoned ducks were also reported on nearby Lake Surprise and in North Carolina at Currituck Sound (Grinnell 1894, 1901; Hough 1894; Phillips and Lincoln 1930).

Little or no controversy regarding Bellrose's (1959) definitive study on the incidence and effects of lead poisoning in wild waterfowl occurred at the time it was published. Much controversy, however, developed approximately 20 years later when the U.S. Fish and Wildlife Service (1974) proposed to require the use of steel shot for hunting waterfowl in selected areas. In his 1959 study, Bellrose stated, "At the present time, lead poisoning losses do not appear to be of sufficient magnitude to warrant such drastic regulations as, for example, prohibition of the use of lead shot in waterfowl hunting. Should lead poisoning become a more serious menace to waterfowl populations, iron shot provides a possible means of overcoming it. Because of the increasing numbers of waterfowl hunters and the increasing incidence of lead poisoning, as well as because of the suffering that results among waterfowl seriously afflicted with the malady, the search for the best possible solution to the lead poisoning problem should be continued" (p. 286). Sixteen years later, however, Bellrose (1975:167) commented on his earlier statements, "Why has my view on this problem changed? The principal reason is that our waterfowl populations have declined. Like all of our disappearing natural resources they are relatively more valuable today than they were then."

With the alarming decline in waterfowl nesting habitats in recent years, many biologists and wildlife managers believe that all reasonable steps that benefit waterfowl should be taken. As a result, the U.S. Fish and Wildlife Service (1974) proposed to require the use of steel shot for hunting waterfowl in certain areas of the United States. Most wildlife biologists and managers, the professional society representing them (The Wildlife Society 1984), and the National Wildlife Federation (1978), among other organizations, supported the use of steel shot for hunting waterfowl. Well-organized and widespread opposition to steel shot surfaced immediately (e.g., National Rifle Association 1978; Arnett 1985). Recently, the National Wildlife Federation (1985b) has provided an excellent summary of the misunderstandings on which most of the opposition to steel shot has been based.

Because professionals and the general public have been inadequately informed about the problem of lead poisoning in waterfowl and because of misconceptions about the effectiveness of steel shot, we have undertaken a comprehensive review of these subjects. Our purposes here are three: (1) to provide an up-to-date summary of the effects of lead poisoning in waterfowl, (2) to summarize and briefly discuss the main issues that have led to differences of opinion regarding the magnitude of the problem, and (3) to review the differences to be found from the use of steel rather than lead shot. We have prepared this report with the expectation that biologists, wildlife managers and administrators, legislators, the general public, and especially waterfowl hunters will find the information helpful in understanding an extremely complex problem.

Sources of Lead Poisoning

Ingested Lead Shot

Laboratory and field studies alike indicate that waterfowl ingest lead shot during feeding. Although ingestion of grit is not an important factor in the ingestion of shot, it does affect the erosion rate of lead after the shot is in the gizzard. Species that feed most actively on the bottom of shot-laden areas have the highest rate of shot ingestion (Bellrose 1959). Moreover, when traps were set over areas previously subjected to intensive hunting, captured ducks (fluoroscoped live) had an appreciably higher rate of shot ingestion than ducks from other locations. For example, 7.3 percent of 3,900 blue-winged teal (*Anas discors*) captured in September on areas previously hunted had swallowed shot pellets (Bellrose 1959:256). Nowhere else in the United States was the incidence of shot that high for this species. Similarly, wood ducks (*Aix sponsa*) have a low ingestion rate of shot in many areas of the nation (1.6 percent), but 9.4 percent of 941 birds trapped in areas extensively hunted had shot in their gizzards. Feeding on bait in the soft bottom mud in these duck traps was so intense that over a period of several weeks 1 foot of fine silt was removed as the ducks worked deeper and deeper to obtain the grain that had been used as bait. They also ingested lead shot in the process, apparently as food.

Because lead poisoning results from ingested lead pellets, the occurrence of lead shot in waterfowl gizzards provides information on the degree of exposure. Although die-offs show that waterfowl continue to ingest shot after the hunting season, large numbers of waterfowl gizzards cannot be obtained easily except during the hunting season. Until recently, therefore, much of what was known about shot ingestion was derived from the examination of waterfowl gizzards collected from hunters during the fall and early winter. Bellrose (1951) found that 3-4 percent of the ducks livetrapped and examined by fluoroscopy before the hunting season had shot in their gizzards. The numbers increased steadily until December when 12 percent of the gizzards contained lead.

Bellrose (1959:262-263) analyzed 39,610 gizzards collected from waterfowl in habitats scattered across the United States and in British Columbia. An average of 6.6 percent of those gizzards contained one or more ingested shot. His findings also showed that lead pellets occurred in gizzards from waterfowl in all regions, although the incidence of ingested lead varied by region and species. Such generalized occurrence is understandable because the wetlands bring waterfowl and hunters together. Because wetlands are so widely distributed, the incidence of ingested lead is not confined to focal points of infection as is the case in diseases caused by pathogens.

Subsequent studies indicate that the shot ingestion rates reported by Bellrose in 1959 were low. Montalbano and Hines (1978) evaluated three techniques for determining the presence of shot in waterfowl gizzards: manual examination, X-rays of intact gizzards, and X-rays of gizzard contents. They obtained contents of 300 lead-free gizzards, seeded them at varying rates with varying numbers of sanded and partially eroded shot removed from other gizzards, and asked 3 cooperators to examine the gizzards manually for lead shot and then to examine X-rays of the gizzard contents. They found that manual examination missed 24 percent of the ingested shot and X-rays of intact gizzards missed 28 percent. Manual examination missed small, eroded pellets camouflaged among seeds and grit; X-rays of intact gizzards sometimes failed to detect small shot obscured by grit, food, and the muscular wall of the gizzard. Sporre and Blevins (1981:19) compared manual and X-ray examinations of 998 waterfowl gizzards in Indiana. Manually, they found ingested shot in 6.7 percent of the gizzards; by X-ray they found shot in 10.4 percent. In an Illinois study, Anderson and Havera (1985:29) concluded that manual examination underestimates ingestion of shot pellets by 20-25 percent.

A compilation of data from studies that reported shot ingestion rates from 1973 through 1984 shows that 8.9 percent of 171,697 duck gizzards analyzed contained shot pellets (Table 1). Because these data are more recent and from a larger sample than data obtained earlier, we employ them in subsequent calculations. Because lead-poisoned birds at all levels of ingested shot are about 1.65 times more likely to be bagged by hunters than healthy ducks (Bellrose 1959: Table 20), we calculate that the population actually contained closer to 5.4 percent lead-poisoned birds at any one time during the fall—a reasonable reduction from 8.9 percent because of the inflated bag rate of poisoned ducks. However, a much larger percentage of all ducks actually consume lead shot in any given year—perhaps as much as one-third of the continental population. This estimate is reached as follows:

(1) If a duck that eats a lead shot does not die, the pellet will disappear from the gizzard in about 20 days, either because it has passed through the gastrointestinal tract or because it has eroded to an undetectable particle in the gizzard. (Even when the pellet passes through the bird's digestive system, ill effects may show up in the form of poorer physical condition or the subsequent death of the bird, or both.) When we find that 5.4 percent of the population sampled at a particular time has ingested lead shot, we imply that those ducks swallowed lead shot *within the preceding 20 days*. In the next 20 days, therefore, a comparable number of ducks will consume shot, and so on.

(2) At the most conservative estimate, waterfowl spend about 150 days (November 1 through March 30) in migration and on the wintering grounds. These areas are subject to the heaviest hunting pressure and unquestionably contain higher densities of lead shot than the breeding grounds. (Ducks pick up lead shot on northern breeding grounds (Elder 1950), but because of the paucity of data we have omitted those areas from our estimate.)

Table 1.—Ingestion rates of lead and steel shot pellets in the more important species of game ducks as recorded in various states of the four flyways, 1973-1984.

			Mallard		Mottled/ Black Duck		Wood Duck		Gadwall		Wigeon		Pintail		Green-winged Teal	
Flyway/State	Years	Investigator	No.[a]	%[b]	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
Atlantic																
Florida	1973-78	Baker & Thompson 1979	50	32.0	452[c]	29.0	—	—	87	1.1	968	1.4	1,134	25.6	447	1.3
Maine	1976-80	Longcore et al. 1982	164	3.0	506[d]	6.9	9	0.0	2	0.0	9	0.0	42	2.4	397	0.3
Maryland		Scanlon et al. 1980	144	18.8	105[d]	21.9	14	0.0	17	0.0	16	0.0	10	10.0	17	0.0
New York	1977-82	Moser 1983	8,154	12.1	3,450[d]	8.7	1,204	1.6	295	1.0	581	1.0	224	6.7	803	1.2
Florida	1976-84	Thul 1985	90	16.7	202[c]	14.9	242	4.5	68	1.5	171	4.1	114	12.3	277	3.3
Subtotal & Mean			8,602	12.3	4,061	8.8	1,469	1.9	469	1.1	1,745	1.5	1,524	21.1	1,941	1.9
Mississippi																
Arkansas	1977-79	Sullivan 1980	4,445	6.7	9[d]	0.0	88	2.3	65	4.6	13	7.7	11	9.1	207[e]	0.0
Michigan	1977-79	Nelson & Johnson 1980	6,025	8.6	664[d]	9.6	468	4.0	76	5.3	284	2.4	273	4.8	808	1.5
Ohio	1977-79	Bednarik & Shieldcastle 1980	2,073	6.8	271[d]	5.9	556	0.9	286	0.7	250	0.8	622	5.3	500	0.2
Indiana	1977-80	Sporre & Blevins 1981	1,809	9.8	188[d]	9.0	—	—	—	—	—	—	99	12.1	—	—
Louisiana	1974-81	Smith 1981	6,834	15.2	611[c]	26.4	378	2.6	422	1.4	182	2.2	3,956	16.6	555	1.8
Missouri	1978-81	Humburg & Babcock 1982	14,638	6.0	2[d]	0.0	32	0.0	100	2.0	141	0.7	472	5.7	262	0.0
Illinois	1979-82	Anderson 1982	9,574	6.4	—	—	—	—	—	—	—	—	—	—	—	—
Subtotal & Mean			45,448	8.1	1,134	8.6	1,522	2.4	949	1.8	870	1.7	5,433	13.7	2,332	1.0
Central																
Kansas	1973-74	Funk 1974	407	4.2	—	—	—	—	—	—	—	—	171	4.7	—	—
N. Dakota	1973,77,78	Johnson 1985	746	0.8	—	—	—	—	—	—	—	—	57	1.8	—	—
S. Dakota	1973, 83	Funk 1974; Fowler & Simpson 1984	1,080	2.3	—	—	—	—	—	—	—	—	46	2.2	—	—
Nebraska	1973-83	Funk 1974; Gabig 1984	4,643	1.1	—	—	—	—	181	0.0	216	0.0	258	0.8	197	0.5
Oklahoma	1979-84	Due 1985	2,811	2.0	—	—	92	2.2	248	0.4	139	1.4	83	2.4	288	0.0
Texas	1981-83	TX Parks & Wildl. Dept. 1982, 1983	1,405	12.0	1,347[c]	29.5	73	2.7	569	0.7	518	1.0	2,633	14.3	858	0.9
Subtotal & Mean			11,092	2.6	—	—	165	2.4	998	0.5	873	0.8	3,248	12.0	1,343	0.7
Pacific																
Nevada	1974-77	Barngrover 1977	1,388	9.1	—	—	—	—	—	—	—	—	1,460	8.4	412	0.2
California	1974-80	Moore & King 1980	9,271	7.8	—	—	—	—	83	3.6	499	1.6	18,386	8.8	1,372[e]	0.1
Montana	1976-81	Childress 1985	2,467	2.4	—	—	—	—	1,140	0.4	510	0.8	687	1.9	363	0.0
Oregon	1974-83	Vendshus n.d.	3,212	21.3	—	—	—	—	—	—	—	—	2,981	25.2	—	—
Subtotal & Mean			16,338	9.7	—	—	—	—	1,223	0.7	1,009	1.2	23,514	10.6	2,147	1.4
Total & Mean			*81,480*	*8.1*	*5,195[d]* *2,612[c]*	*8.8[d]* *27.5[c]*	*3,156*	*2.2*	*4,160*	*0.9*	*4,497*	*1.3*	*33,719*	*11.7*	*7,763*	*0.1*

Grand total: 171,697 ducks examined; 8.9% of the gizzards had ≥1 shot.

[a]Number of gizzards examined. [b]Percentage of gizzards with ≥1 shot. [c]Mottled duck. [d]Black duck. [e]Teal, largely green-winged.

Table 1.—continued.

			Blue-winged Teal		Shoveler		Canvasback		Redhead		Ring-necked Duck		Greater and Lesser Scaup	
Flyway/State	Years	Investigator	No.[a]	%[b]	No.	%	No.	%	No.	%	No.	%	No.	%
Atlantic														
Florida	1973-78	Baker & Thompson 1979	1,025	1.7	308	0.3	234	65.4	178	29.8	3,455	24.0	1,078	7.6
Maine	1976-80	Longcore et al. 1982	111	1.8	—	—	—	—	—	—	5	20.0	1	0.0
Maryland		Scanlon et al. 1980	—	—	4	2.5	—	—	—	—	—	—	9	0.0
New York	1977-82	Moser 1983	343	0.3	—	—	209	6.2	199	6.0	107	7.5	592	7.9
Florida	1976-84	Thul 1985	1,605	3.1	519	1.3	33	9.1	25	12.0	5,436	15.9	1,072	16.9
Subtotal & Mean			3,084	2.3	831	1.0	476	35.5	402	16.9	9,003	20.0	2,752	11.3
Mississippi														
Arkansas	1977-79	Sullivan 1980	—	—	5	0.0	4	0.0	2	0.0	22	4.5	10	0.0
Michigan	1977-79	Nelson & Johnson 1980	285	1.0	28	7.1	8	12.5	51	17.6	364	18.4	248	4.0
Ohio	1977-79	Bednarik & Shieldcastle 1980	833	0.6	361	0.6	—	—	29	10.3	83	7.2	114	14.9
Indiana	1977-80	Sporre & Blevins 1981	—	—	—	—	—	—	—	—	—	—	—	—
Louisiana	1974-81	Smith 1981	2,251	6.4	155	1.3	13	7.7	14	28.6	205	14.1	523	26.8
Missouri	1978-81	Humburg & Babcock 1982	59	0.0	67	3.0	8	12.5	34	0.0	438	13.0	92	4.3
Illinois	1979-82	Anderson 1982	—	—	—	—	—	—	18	0.0	5	20.0	323	2.5
Subtotal & Mean			3,428	4.4	295	1.6	29	10.3	148	10.8	1,117	14.3	1,310	13.7
Central														
Kansas	1973-74	Funk 1974	—	—	—	—	—	—	—	—	—	—	86	14.0
N. Dakota	1973,77,78	Johnson 1985	—	—	—	—	—	—	—	—	—	—	27	0.0
S. Dakota	1973, 83	Funk 1974; Fowler & Simpson 1984	—	—	—	—	—	—	—	—	—	—	69	1.4
Nebraska	1973-83	Funk 1974; Gabig 1984	193	0.5	—	—	—	—	—	—	—	—	78	1.3
Oklahoma	1979-84	Due 1985	10	0.0	33	3.0	6	0.0	33	9.1	45	2.2	76	6.6
Texas	1981-83	TX Parks & Wildl. Dept. 1982, 1983	325	2.8	198	4.5	39	12.8	299	22.4	404	24.8	820	23.4
Subtotal & Mean			528	1.9	231	4.3	45	11.1	332	21.1	449	22.5	1,156	18.3
Pacific														
Nevada	1974-77	Barngrover 1977	—	—	29	0.0	349	17.5	509	17.7	—	—	—	—
California	1974-80	Moore & King 1980	—	—	723	1.4	—	—	—	—	—	—	—	—
Montana	1976-81	Childress 1985	592	2.4	550	0.2	141	9.2	99	4.0	19	0.0	505	3.2
Oregon	1974-83	Vendshus n.d.	—	—	—	—	—	—	—	—	—	—	—	—
Subtotal & Mean			592	2.4	1,302	0.8	490	15.1	608	15.5	19	0.0	505	3.2
Total & Mean			*7,632*	*3.2*	*2,624*	*1.3*	*1,040*	*24.1*	*1,490*	*16.6*	*10,588*	*19.5*	*5,723*	*12.5*

Grand total: 171,697 ducks examined; 8.9% of the gizzards had ≥1 shot.

[a]Number of gizzards examined. [b]Percentage of gizzards with ≥1 shot.

(3) This 150-day period is equal to 7.5 intervals of 20 days each. Multiplying 5.4 percent by 7.5, we find that as much as 40 percent of the waterfowl population ingests lead shot during a single season of exposure. Because some ducks ingest lead shot, survive, and ingest shot again, this estimate for individuals affected may be high; however, the omission of data on the ingestion of lead shot on breeding grounds and the probable higher rate of ingestion after the hunting season seem to make the 40 percent ingestion rate conservative. In addition, some ducks die of lead poisoning before they are shot and therefore are not included among the 40 percent, a figure that is based on the corrected incidence of shot in gizzards of ducks bagged by hunters.

Waterfowl that ingest 1 or 2 shot more often live than die, and waterfowl tend to pick up only a small number of pellets at any one time: 65 percent take one shot, 15 percent two shot, 7 percent three shot, 3 percent four shot, 2 percent five shot, 1 percent six shot, and 7 percent seven or more shot (Bellrose 1959:260). A compilation of reports on the incidence of shot ingestion by mallards during the 1970s and early 1980s provided findings similar to those of Bellrose. Of 1,211 mallard (*Anas platyrhynchos*) gizzards containing shot pellets, 63.0 percent contained one pellet, and 13.6 percent contained 2 pellets (Table 2).

Because of varying habitats and feeding habits, waterfowl species differ greatly in their rate of shot ingestion. From 1938 through 1953, the bay diving ducks—canvasback (*Aythya valisineria*), lesser scaup (*Aythya affinis*), redhead (*Aythya americana*), and ring-necked duck (*Aythya collaris*)—had the highest rate of shot ingestion among waterfowl (12-14 percent of the gizzards analyzed); mallard, black duck (*Anas rubripes*), and pintail (*Anas acuta*) had an intermediate rate of shot ingestion (7-9 percent); green-winged teal (*Anas crecca carolinensis*), shoveler (*Anas clypeata*), wood duck, gadwall (*Anas strepera*), blue-winged teal, and wigeon (*Anas americana*) had the lowest rates of shot ingestion (1-3 percent) (Fig. 1).

The percentage of waterfowl that ingest shot depends upon hunting pressure and other variables. Because these factors vary from region to region and from year to year, so does the percentage. Bellrose (1959: Table 12) found that from 1938 through 1953 a relatively small proportion of ducks from the Dakotas, Nebraska, Colorado, and Missouri ingested shot (2-3 percent of the sample); however, an exceptionally high percentage of ducks from Indiana, Louisiana, Texas, and British Columbia ingested shot (11-21 percent). Between 5 and 9 percent of ducks from other states ingested shot. The relative proportions of mallards and black ducks and all other ducks ingesting shot in the four flyways are given in Figure 2.

Since Bellrose's studies on the rate of shot ingestion, numerous studies during the 1970s and early 1980s have provided much more information on this subject (Tables 1, 2, 3). Higher percentages of most species of ducks and all species of geese ingested shot during 1973-84 (Tables 1, 3) than during 1938-1953 (Bellrose 1959:260). As was true for Bellrose's study, ducks may readily be categorized according to their propensity to ingest shot (Table 1): the bay diving ducks and the mottled duck (*Anas fulvigula*) have the highest propensity (12 to 28 percent of the sample); mallard, black duck, and pintail have moderate rates (8 to 12 percent); and wood duck, teals, wigeon, gadwall, and shoveler have the lowest rates (1 to 3 percent). Shot ingestion among Canada geese (*Branta*

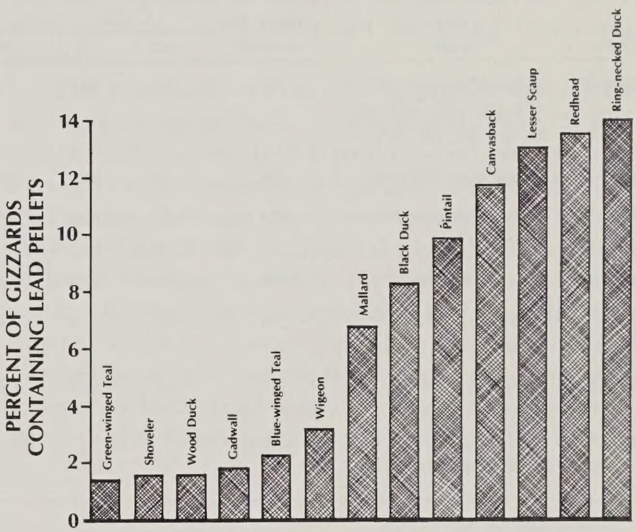

Figure 1.—Percentage of gizzards with varying numbers of ingested lead shot in 35,220 gizzards of various duck species collected at many locations in North America, 1938-1953. Data from Bellrose (1959:260).

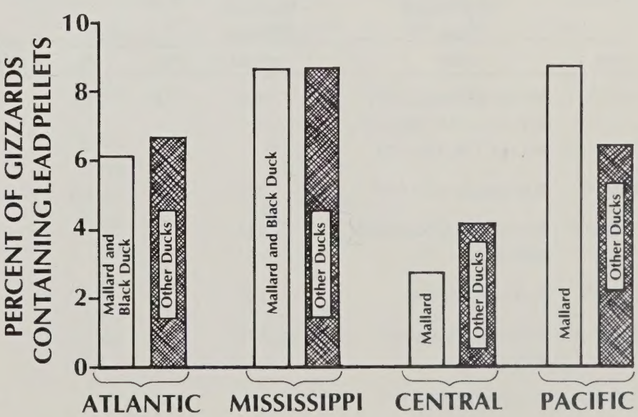

Figure 2.—Regional comparison by flyway of the percentage of mallards and black ducks ingesting lead pellets versus the percentage of other ducks, 1938-1953. Data from Bellrose (1959:262-263).

canadensis) varied by state from 1.9 to 44.1 percent; variation among snow geese (*Anser caerulescens*) was from 0.0 to 4.2 percent (two states). The rate was 4.3 percent among 494 white-fronted geese (*Anser albifrons frontalis*) from Texas (Table 3).

With few exceptions, states with extensive wintering populations of waterfowl had the highest rates: Florida, Louisiana, and Texas. Waterfowl in Oregon showed an excessive rate, but all the data were from Sauvie Island, an area noted for high rates of lead shot ingestion due to hard bottoms. Other areas where shot ingestion was exceptionally high were Merritt Island, Florida; Catahoula Lake, Louisiana; and Murphree Wildlife Area, Texas.

Although some localities within the broad confines of the flyways have low rates of shot ingestion, others have extremely high rates. Except for the Great Plains region of the United States, appreciable numbers of waterfowl ingest shot. Mudge (1983:340) reported the incidence of shot ingestion among 2,453 British wildfowl of 18 species (ducks, geese, swans, and coots) shot, 1979-81, and in 230 wildfowl of 21 species (ducks, geese, and swans) found dead during the same period. The percentage of shot birds with ingested

Table 2.—Incidence of various levels of ingested shot in gizzards of mallards.

Years	Investigator and State	Number Gizzards w/lead	1 shot		2 shot		3 shot		4 shot		5 shot		6 shot	
			No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
1974-75	White & Stendell 1977 (CA, CO, MA, MO, MT, NJ, NY, OK, OR, UT)	122	73	59.8	17	13.9	12	9.8	5	4.1	2	1.6	3	2.5
1974-77	Barngrover 1977 (NV)	126	76	60.3	17	13.5	6	4.8	9	7.1	1	0.8	3	2.4
1977-79	Bednarik & Shieldcastle 1980 (OH)	114	83	72.8	14	12.3	6	5.4	3	2.6	1	0.9	2	1.8
1977-79	Sullivan 1980 (AR)	299	237	79.3	36	12.0	9	3.0	5	1.7[a]	—	—	—	—
1979	Anderson & Brewer 1980 (IL)	279	202	72.4	41	14.7	17	6.1	7	2.5	3	1.1	1	0.4
1978-80	Smith 1979, 1980 (LA)	271	92	33.9	40	14.7[b]	—	—	—	—	—	—	—	—
Total number and average percent		*1,211*	*763*	*63.0*	*165*	*13.6*	*50*	*5.3*	*29*	*3.1*	*7*	*1.1*	*9*	*1.4*

[a]Excludes Arkansas data ≥5 shot.
[b]Excludes Louisiana data ≥3 shot.

Table 2.—continued.

Years	Investigator and State	Number Gizzards w/lead	7 shot		8 shot		9 shot		10 shot		>10 shot	
			No.	%	No.	%	No.	%	No.	%	No.	%
1974-75	White & Stendell 1977 (CA, CO, MA, MO, MT, NJ, NY, OK, OR, UT)	122	2	1.6	0	—	2	1.6	1	0.8	5	4.1
1974-77	Barngrover 1977 (NV)	126	2	1.6	2	1.6	1	0.8	3	2.4	6	4.8
1977-79	Bednarik & Shieldcastle 1980 (OH)	114	0	—	2	1.8	0	—	0	—	3	2.6
1977-79	Sullivan 1980 (AR)	299	—	—	—	—	—	—	—	—	—	—
1979	Anderson & Brewer 1980 (IL)	279	3	1.1	1	0.4	0	—	0	—	4	1.4
1978-80	Smith 1979, 1980 (LA)	271	—	—	—	—	—	—	—	—	—	—
Total number and average percent		*1,211*	*7*	*1.1*	*5*	*0.8*	*3*	*0.5*	*4*	*0.6*	*18*	*2.8*

lead pellets ranged from zero for nine species to 4.2 for mallards, 6.7 for goldeneye (*Bucephala clangula*), 7.1 for greylag goose (*Anser anser*), 10.9 for pochard (*Aythya ferina*), 11.7 for tufted duck (*Aytha fuligula*), and 11.8 for gadwall. Of 35 mallards found dead, 17.1 percent had lead pellets in their gizzards. In a comparison of regional variation, Mudge (p.360) found that 6.0 percent of mallards shot at inland sites had ingested pellets; only 2.6 percent of those shot on coastal areas had ingested pellets.

Most investigators agree that spent shot from the guns of hunters is the main source of lead that poisons wild waterfowl. Bellrose (1959) reported that the number of spent lead pellets found on 24 areas in 7 states and provinces ranged from 0 to 291,579 per ha, an average of 69,847 per ha (0 to 118,083 per acre, an average of 28,277 per acre). Mudge (1984:299) reported densities of lead shot in the top 15 cm of wetland soils at 22 sites in 9 areas of Britain. No pellets were found at 3 sites, but at the others densities varied from 2.04 pellets per m^2 to 30.0 per m^2 (20,388 to 300,000 pellets per ha).

In addition to the prevalence of lead shot, several other factors influence the ingestion of shot by waterfowl. These include feeding habits, firmness of the lake or marsh bottom, depth of water, size of pellets, ice cover, and season (Bellrose 1959). In spring, high water often decreases the availability of lead pellets to ducks. Little carry-over of lead shot available to ducks from one season to the next has been found in feeding areas with soft bottoms.

Sources Other Than Ingested Shot

Waterfowl do have access to other sources of lead, but these are relatively insignificant compared with the availability of lead shot. Lead in mine wastes, for example, has caused the death of ducks, geese, and swans (Phillips and Lincoln 1930; MacLennan 1954; Chupp and Dalke 1964). Gelston and Stuht (1975) successfully treated a mute swan (*Cygnus olor*) for lead poisoning after the bird had swallowed lead fishing sinkers. Bagley and Locke (1967) found that all birds they examined had been exposed to sublethal amounts of lead in their food. These investigators studied 28 species of birds, including 17 species of waterfowl, and generally found low levels of lead in livers (2.0-26.0 ppm, wet weight), a finding that indicates chronic but low exposure to lead. As far as could be determined, these birds were not suffering from lead poisoning.

The Winchester Group (1974) argued that lead in the wing bones of ducks as reported by the U.S. Fish and Wildlife Service (1974) could come from low-level exposure to environmental lead and not from ingested lead pellets. Because scientists from the U.S. Fish and Wildlife Service had analyzed the bones of immature mallards that had had only a few months in which to accumulate lead, they assumed that when two distributions of lead were found, the higher level represented ingested lead shot and the lower other environmental sources of lead. Lead levels in wing bones from mallards in the Mississippi, Central, and Pacific flyways

Table 3.—The rate of shot ingestion (lead and steel) by geese in various states of the four flyways.

			Canada Geese		Snow Geese		White-fronted Geese		Unidentified Geese	
Flyway/State	Years	Investigator	No.[a]	%[b]	No.	%	No.	%	No.	%
Atlantic										
Maryland	Unknown	Scanlon et al. 1980	161	44.1	—	—	—	—	—	—
Mississippi										
Michigan	1977-79	Nelson & Johnson 1980	2,507	3.9	—	—	—	—	—	—
Ohio	1977-79	Bednarik & Shieldcastle 1980	1,828	2.0	—	—	—	—	—	—
Indiana	1977-80	Sporre & Blevins 1981	141	6.4	—	—	—	—	—	—
Missouri	1978-81	Humburg & Babcock 1982	2,330	5.5	—	—	—	—	—	—
Illinois	1979-81	Anderson 1982	724	5.4	—	—	—	—	—	—
Central										
S. Dakota	1980-83	Fowler & Simpson 1984	3,943	4.1	—	—	—	—	—	—
Texas	1981-83	Texas Parks & Wildl. Dept. 1982, 1983	298	2.3	1,181	4.2	494	4.3	9	22.2
Pacific										
California	1974-80	Moore & King 1980	—	—	—	—	—	—	1,380	5.7
Nevada[c]	1974-77	Barngrover 1977	269	1.9	104	0.0	—	—	—	—

[a]Number of gizzards examined.
[b]Percentage of gizzards with ≥1 shot.
[c]There were 39 tundra swans examined from Nevada and 5.1 percent contained ingested shot.

fitted this assumption, as did levels in birds from Virginia and North Carolina in the Atlantic Flyway. Bones of birds from Massachusetts, Maryland, New Jersey, and Georgia, however, fitted one simple exponential probability distribution. Lead from sources other than shot could have contributed appreciable amounts of lead to the wing bones of mallards from these four states, but no source other than lead pellets was identified to explain the high levels found. Thus, the U.S. Fish and Wildlife Service (1976) concluded that lead pellets are the primary source of lead available to wild waterfowl.

Niethammer et al. (1985) found low levels of lead (geometric means of 0.07-0.55 ppm, wet weight) in the livers of green-backed herons (*Butorides striatus*) collected downriver from lead mines in Missouri. They found somewhat higher levels (0.23-2.39 ppm, wet weight) of lead in the carcasses of northern rough-winged swallows (*Riparia riparia*) from a colony nesting in a mine tailings pile containing lead concentrations from 2,360 to 26,600 ppm dry weight. Presumably the respiratory and dermal exposure to lead-contaminated dust explained the higher lead levels in the swallows. Niethammer and his colleagues did not report clinical symptoms of lead poisoning in either the herons or the swallows.

Additional examples of birds that have acquired lead from sources other than shot include laughing gulls (*Larus atricilla*) collected near Galveston, Texas (Munoz and Gesell 1976). Adults and prefledglings had lead concentrations ranging from 0 to 16 u/g of wet weight in liver, brain, heart, and skeletal tissues. No significant difference between gulls in the nest and adults was found, an observation that suggests that the young birds had reached an equilibrium regarding lead before they left the nest. No lead toxicosis was reported in these birds. Siegfried et al. (1972) found a significant difference in the mean content of lead in laughing doves (*Streptopelia senegalensis*) collected in the city of Cape Town, South Africa, and those collected in a rural area 50 km away. They concluded that the difference resulted from the higher level of lead in the city atmosphere. Ohi et al. (1974) found a significant difference in the lead content of femurs from pigeons collected from a farm house in a rural area and those collected from a crowded temple in downtown Tokyo. Higher levels in the city birds were attributed to atmospheric lead from automobile exhaust. No mention was made of clinical symptoms or mortality from lead poisoning in either the Cape Town or the Tokyo study.

Several reports of zoo animals suffering from lead poisoning, which in some cases was fatal, are available. Bazell (1971:130), for example, studied animals in the Staten Island Zoo and observed that although "some of the lead in the animals' bodies may have come from paint in their cages, the major source appears to be atmospheric contamination." Unlike these zoo animals, wild waterfowl are seldom exposed to high levels of atmospheric lead for extended periods.

We are concerned about the levels of lead pollution in the atmosphere, soils, water, and plants of the world; however, we found no evidence of extensive mortality from lead poisoning in wild animals other than lead poisoning in waterfowl as a result of ingesting lead pellets.

Diagnosis of Lead Poisoning

Symptoms

The clinical symptoms of lead poisoning in waterfowl have often been described, and a thorough summary is found in Forbes and Sanderson (1978: 255-256). Some of the earlier studies (Grinnell 1901; Phillips and Lincoln 1930) describe lead-poisoned ducks, geese, and swans (*Cygnus* spp.) as unable to fly, as sick often with little loss of body weight, as having a rattling in the throat, as so weak as to be easily captured, and as occasionally dribbling a yellowish fluid from the bill—which is held open much of the time. Remains of lead pellets are often found in the gizzards of lead-poisoned waterfowl. The inner lining of the gizzard is dark, soft, decayed, easily eroded, inflamed, corroded, and incomplete. Often the bird cannot fly (and later cannot walk) because of progressive paralysis of the muscles of the wings and legs. On land the tips of the primaries drag the ground and on water the wings float loosely on the surface. The proventriculus is often distended, thin and watery green-stained feces are common, and the voice of geese is often changed.

Like some of the earlier studies, several subsequent studies (Quortrup and Shillinger 1941; Jordan and Bellrose 1950; Rosen and Bankowski 1960) did not stress the loss of body weight that is characteristic of chronic lead poisoning in waterfowl. Bellrose (1964) reported that waterfowl starving because of lead poisoning weighed about 50% of normal, but Trainer and Hunt (1965) found no correlation between the body weights of swans with more than 100 lead pellets in their gizzards and swans with 10 or fewer pellets.

Generally, captive waterfowl that die of *chronic* lead poisoning lose from 40 to 60% of their body weight before death; they also lose a greater percentage of body weight during mild weather than during cold weather. On the other hand, captive waterfowl that die of *acute* lead poisoning may lose relatively little weight before death. W.L. Anderson (1975) also found a direct correlation between body weight and number of lead pellets in gizzards of wild ducks that had died of acute lead poisoning. Sanderson and Irwin (1976: Table 16) reported that 8 of 20 male game-farm mallards on a diet of corn and dosed with five No. 4 lead pellets on 1 July died of acute lead poisoning an average of 7.6 days later after losing 20.5 percent of their body weight. The 12 remaining ducks died of chronic lead poisoning an average of 20.7 days after dosing and had lost 47.6 percent of their body weight. Waterfowl in the wild, we assume, would follow patterns similar to those of captive waterfowl.

Reports vary concerning the effects of lead poisoning on the appetites of waterfowl, and the topic merits further investigation. Early workers (Phillips and Lincoln 1930; Shillinger and Cottam 1937; Quortrup and Shillinger 1941; Adler 1942) indicated that lead-poisoned waterfowl showed no decrease and sometimes showed an increase in appetite. Beer and Stanley (1965) found that many birds that had died of lead poisoning had eaten shortly before death. Other investigators, however, have reported that lead poisoning causes a loss of appetite and that a decreased intake of food is one of the earliest external symptoms in lead-poisoned birds. Jordan and Bellrose (1951) found that captive mallards not dosed with lead but fed only the amount of food eaten by paired ducks dosed with lead showed weight loss and other symptoms that were similar to those shown by the lead-poisoned ducks. Irby et al. (1967) reported that ducks decreased their consumption of corn for 1-3 weeks after dosing with lead. During the second half of the experiment (the second month), however, the surviving dosed ducks ate as much or more corn than did their controls. Irwin et al. (1974) found that adult game-farm mallards dosed with lead and fed corn had reduced appetites but that dosed ducks fed an "adequate" diet (among other components, the adequate diet contained 19.2 percent protein compared with 8.8 percent protein in corn) showed no loss of appetite. Sanderson and Irwin (1976:63) did not measure food consumption in their studies; however, they noted decreased consumption of corn in ducks dosed with lead. In some instances, they found that shortly before ducks died of lead poisoning, their appetite for corn returned.

Quortrup and Shillinger (1941) reported that distention of the proventriculus and esophagus occurs because food cannot pass the gizzard. In one group of 70 captive mallards, Sanderson and Irwin (1976:63, Table 62) found that only 1 bird of 17 that died of lead poisoning from 4 to 8 days after dosing with lead had food in the digestive tract; none of the 17 showed impaction. In the same group of 70 ducks, among those that died from 9 through 39 days after dosing, 7 (10.0 percent) had corn in the esophagus and 2 (2.9 percent) had the esophagus impacted with corn; 26 (37.1 percent) had corn in the proventriculus and 7 (10.0 percent) had the proventriculus impacted with corn; 53 (75.7 percent) had corn present in the gizzard and 1 (1.4 percent) had the gizzard impacted with corn.

Diagnostic Techniques and Their Results

In spite of several clinical symptoms for lead poisoning in waterfowl, researchers cannot always be certain that an individual bird has died of or is suffering from lead poisoning. Many symptoms are seen only at necropsy. Recently, however, several diagnostic techniques, some of which can be used with live birds, have been described.

Bone analysis. Although no individual relationship has been found between the amount of lead shot in a gizzard and lead residues in wing bones, a significant correlation between the two occurs on a population basis (White and Stendell 1977:472). The amount of lead in gizzards of mallards and pintails was closely related to the amount of lead in their wing bones as reported by the U.S. Fish and Wildlife Service (1978) and by W.L. Anderson (1975) for lesser scaups at Rice Lake, Illinois. Lead appears almost immediately in the wing bones after lead shot are ingested by birds. Thus, ducks that have expelled eroded pellets from their digestive tracts show an absence of gizzard lead but retain lead residue in their wing bones.

Wing bones (radii-ulnae) collected at random from 4,190 ducks during the National Waterfowl Wing Survey in 1972 and 1973 were analyzed for lead by the WARF Institute of Madison, Wisconsin, for the U.S. Fish and Wildlife Service (Stendell et al. 1979). Bones of adults contained concentrations of lead about twice as high as concentrations found in juveniles. Lead levels were highest in the Atlantic Flyway, at moderate levels in the Mississippi and Pacific flyways, and lowest in the Central Flyway.

A bimodal distribution of lead was found in most of the immature mallards with wing bones containing lead; the higher levels were believed to be the result of shot ingestion. About one-third of the wings analyzed from immature mallards had high levels of lead: 37.5 percent in the Mississippi Flyway, 36.6 percent in the Pacific Flyway, and 21.2 percent in the Central Flyway (Stendell et al. 1979). Moreover, these birds were only a few months old and had been exposed to heavily hunted areas for 4 months at most. Elevated lead levels ranged from 8.5 to 82.3 ppm, with a mean of 24.4 ± 17.3, and were found in samples from various states.

When penned year-old mallards were dosed with one No. 4 lead pellet, concentrations of lead in the wing bones of laying hens proved to be more than 4 times higher than levels found in the wing bones of nonlaying hens (Finley and Dieter 1978). Apparently the mobilization of calcium for egg laying increased the absorption of lead from the blood stream. In a similar but earlier experiment, Finley et al. (1976) found high levels of lead deposition in skeletons of laying hens; these levels may have resulted from calcium mobilization from bones during eggshell formation. According to Stendell et al. (1979), the lead content of bones is similar in males and females outside of the breeding season.

Blood analysis. Blood samples have been used to determine the extent of lead toxicosis in waterfowl populations. The level of blood lead considered toxic but sublethal is 0.5 ppm, and the U.S. Fish and Wildlife Service recently established ⩾ 0.2 ppm as a level above background levels. As demonstrated by Dieter (1979) in an examination of blood from 400 canvasbacks from Chesapeake Bay and the Upper Mississippi River, 1974-1978, symptoms of lead toxicity began to appear at 0.2 ppm. At levels of 0.5 ppm and higher (average 0.58 ppm), 12 percent of the canvasbacks showed significant depression of delta-aminolevulinic acid dehydratase (ALAD) enzyme activity. A reduction of ALAD enzyme activity in the brain causes cerebellar damage (Dieter and Finley 1979). Biochemical lesions in the brain precede such external symptoms of lead poisoning as wing droop and vent staining.

The level of protoporphyrin (PP) in the blood has also been used to determine levels of lead toxicosis in waterfowl. Roscoe et al. (1979) found that PP levels exceeding 40 ppm indicate lead poisoning; at 500 ppm an impairment of motor functions occurs. Motor functions of the nervous system correlate and control muscular activity. PP is important because it is a precursor to hemoglobin and because PP increases in the blood of lead-poisoned waterfowl, thereby indirectly indicating the amount of lead in the blood.

Anderson and Havera (1985) evaluated three methods of determining lead poisoning in Illinois waterfowl: (1) lead in blood, (2) PP in blood, and (3) ingested lead pellets in gizzards. They concluded that the lead level in blood was the most sensitive indicator of toxicosis, the PP level was less so, and the presence of ingested lead pellets was least likely to indicate the degree of exposure to lead. They found that 8.1 percent of the blood samples from 1,135 mallard in 4 areas in Illinois had lead levels of ⩾0.5 ppm. Of blood samples from 864 Canada geese at 3 locations, 6.5 percent had ⩾0.5 ppm lead levels. Fifteen (5.7 percent) of the blood samples from 264 canvasbacks collected in March from the Keokuk Pool on the Mississippi River had ⩾0.5 ppm lead.

Analysis of the liver and other organs. A number of studies have used the analysis of heavy metals in the livers of waterfowl as a measure of exposure to lead. Adrian and Stevens (1979) emphasized the importance of using liver samples that are oven-dried to a constant weight; wet weights were found to produce sizeable errors.

In dead and moribund lesser scaup collected at Rice Lake, Illinois, W.L. Anderson (1975:267) found means of 47 and 43 ppm (wet weight) lead in livers of males and females, respectively; 62 and 77 ppm (wet weight) in kidneys; and 34 and 55 ppm (wet weight) in wing bones. He reported a high correlation between lead in the livers and lead in the wing bones of female scaup.

An analysis of Canada geese, victims of lead poisoning in eastern Colorado, disclosed an average lead level of 102 ppm in livers, 125 ppm in kidneys, and 41 ppm (dry weight) in wing bones (Szymczak and Adrian 1978: 301). A high correlation between the lead concentration in the liver and the concentration in the kidneys of the same specimens was reported.

Scanlon et al. (1980) examined waterfowl taken by Maryland hunters and compared the number of birds with ⩾10 ppm lead in their livers (dry weight) and the presence or absence of ingested shot. They found that 28.8 percent of the waterfowl with shot in the gizzards had ⩾ 10 ppm lead in their livers but that 16.2 percent of the birds without lead in their gizzards had equally high levels of lead in their livers. Of 613 specimens representing 14 species, 18.8 percent had liver lead of ⩾ 10 ppm.

At Catahoula Lake and Lacassine National Wildlife Refuge, Louisiana, 1,110 dead and incapacitated waterfowl were collected for liver analysis and shot ingestion (Shealy et al. 1982). A level of 6.0 ppm wet weight or 20.0 ppm dry weight was used to indicate lead toxicosis. Of the entire sample, 74.8 percent had liver lead at those levels or higher. Lead toxicosis, as determined by levels of lead in livers, was distributed among species as follows: pintails, 82.2 percent; mallards, 80.0 percent; snow geese, 77.2 percent; white-fronted geese, 68.6 percent; and canvasbacks, 52.4 percent. The ingestion of lead shot among species was comparable: 75.0 percent of the pintails, 68.3 percent of the mallards, 76.9 percent of the snow geese, 71.0 percent of the white-fronted geese, and 60.9 percent of the canvasbacks contained ingested lead pellets. The average number of pellets ingested for pintails was 3.9; for mallards, 4.2; for snow geese, 2.0; and for white-fronted geese, 5.4 (Zwank et al. 1985).

The effect of lead poisoning on the size of certain internal organs may differ according to species and the stage of toxicosis and its nature—acute or chronic. Several investigators (Coburn et al. 1951; Jordan and Bellrose 1951; Locke and Bagley 1967; and Bates et al. 1968) found smaller-than-average livers, kidneys, hearts, and spleens in waterfowl suffering from lead poisoning. In contrast, Chupp and Dalke (1964) reported large livers in swans that died from lead poisoning from mine wastes in Idaho. Adler (1944) also reported enlarged kidneys, spleens, and livers in 4 wild lead-poisoned Canada geese in Wisconsin.

Sanderson and Irwin (1976) agreed that lead toxicosis results in a reduction of liver size. They also pointed out that the effect of lead on the liver of ducks is confounded "by the effects of seasons and their differing influences on the total rate of food consumption and on the relative rates of food consumption by the sexes, the average postdosing survival time, diet, and the lead-induced results of anorexia" (p. 30A). They also found that "the mean weights of livers of most dosed ducks were heavier than livers of undosed controls." They had no explanation for the heavy livers in dosed ducks, but they also found (p. 62) that the mean weights of livers, spleens, and testes of lead-dosed ducks that survived to the end of the experiment were significantly heavier than the mean weights of these organs for all lead-dosed ducks that died during the experiment.

Other techniques. Locke et al. (1966) found that acid-fast inclusion bodies in the proximal convoluted tubules of the kidneys can be used as presumptive evidence of lead poisoning in mallards, but this technique does not work for Canada geese (Locke et al. 1967).

Barrett and Karstad (1971) reported that erythrocytes from lead-poisoned Canada geese and mallards subjected to blue-ultraviolet light showed red fluorescence. This quick and simple technique can be used on live birds and is as reliable as some of the more conventional techniques.

One of the common characteristics of lead-poisoned waterfowl is severe anemia (Beer and Stanley 1965). The main sources of this anemia are probably the production of defective red cells and the impaired release of red cells (Bates et al. 1968). Hemosiderosis commonly occurs in kidneys, livers, and spleens of lead-poisoned waterfowl.

Calcium versenate (Ca EDTA) injected intraveously is diagnostic for heavy metals. If a live, lead-poisoned bird is injected with Ca EDTA, the symptoms do not reappear for about 48 hours (Rosen and Bankowski 1960). Several intraperitoneal injections of Ca EDTA in a solution of 6.6 percent cause lead-poisoned ducks to regain their appetites and to recover (Wobeser 1969).

For a discussion of several other methods of diagnosing lead poisoning in waterfowl, see Forbes and Sanderson (1978:256-260).

Susceptibility to Lead Poisoning

Susceptibility by Species

The susceptibility of a waterfowl species to lead poisoning depends on its tendency to ingest lead shot and its food habits relating to the intake of protein, calcium, and phosphorus. Variability among species in shot ingestion has been established through the analysis of more than 200,000 gizzards collected from waterfowl across the United States. Food habits are more difficult to evaluate.

Necropsies of waterfowl sent to the National Wildlife Health Laboratory, Madison, Wisconsin, 1939-1984, help us to understand the susceptibility of various species (Table 4). Obviously, the carcasses of large species, such as swans and geese, are more noticeable than those of smaller species, and people tend to attach more importance to reporting them. The data in Table 4, therefore, are undoubtedly biased toward large species, particularly swans and geese. In addition, the data were recorded by number of lead poisoning episodes that were investigated, not by total number of birds involved. Even with these limitations, mallards were involved in 25.7 percent of the episodes in which lead was determined to be the cause of death. The pintail, involved in 8.7 percent of the episodes, was the second most important duck species. Redhead, canvasback, and lesser scaup were the next most important species. Other duck species were reported less frequently.

On the basis of tendency to ingest shot, level of lead in wing bones, and food habits, we attempted to rate the susceptibility of species to lead toxicosis. All data point to the mallard as highly vulnerable. This susceptibility results from its moderately high tendency to ingest shot (8 percent of the sample; Fig. 1, Table 1) and from its food habits. More than any other duck, mallards feed extensively upon cereal grains, followed by weed seeds; only in restricted areas do they make use of aquatic plants, animal life, or both (Bellrose 1976:242, 243). Consequently, in most areas of the United States, mallard diets are high in carbohydrates and low in protein.

Black ducks ingest lead at about the same rate as mallards. On coastal marshes their diet is higher in invertebrates and aquatic plants than is the diet of mallards (Bellrose 1976:260, 261). In the Midwest, black ducks feed extensively on waste corn along with mallards. Perhaps because of their high protein intake in the Northeast, the proportion of black duck wing bones with lead levels over 20 ppm was lower than that of mallards (Stendell et al. 1979 and Table 5). In Maine, however, the shot ingestion rate of black ducks was higher than that of mallards (Longcore et al. 1982).

Mottled ducks have the highest rate of shot ingestion of any species (Table 1). The percentage of wing bones in juvenile mottled ducks with lead over 20 ppm is three times as high as the percentage in juvenile mallards; the percentage of wing bones with lead over 20 ppm in adult mottled ducks, however, is only slightly more than twice as high as the percentage in adult mallards (Table 5). The food of mottled ducks in fall, based on an analysis of 1,105 gizzards by Stutzenbaker (1984: Table 11), is composed of 30 percent spikerush (*Eleocharis cellulosa*). This and other green vegetation consumed by mottled ducks provide a high level of protein to complement their 15 percent use of rice, which is high in carbohydrates. The protein level of their diet probably mitigates against the catastrophic losses that might otherwise occur in this species.

Pintails have a shot ingestion rate slightly higher than that of mallards (Fig. 1, Table 1). Because of a more favorable protein diet, especially in California, their losses from lead poisoning probably are not proportionally as large as those of mallards even though shot ingestion rates for pintails are average in California. Only about half as many pintails from the Pacific Flyway as mallards in that flyway had lead concentrations in their wing bones greater than 20 ppm (Table 5). The large losses of pintails from lead toxicosis at Catahoula Lake, Louisiana, however, confirm that this species can be highly susceptible (Bellrose 1959: Table 1; Wills and Glasgow 1964; Zwank et al. 1985). Among the lead-poisoned dead ducks picked up in California, the pintail has been the most numerous, a finding to be expected because pintails comprise

Table 4.—Principal species of waterfowl in lead poisoning episodes investigated by the National Wildlife Health Laboratory, Madison, Wisconsin, 1939-1984.[a]

	FLYWAY									
	Atlantic		Mississippi		Central		Pacific		Total	
	No.[b]	%	No.	%	No.	%	No.	%	No.	%
Episodes	47	10.8	230	53.0	59	13.6	98	22.6	434	100.0
Species										
Mallard	9[c]	17.0[d]	81	29.6	28	34.6	23	16.4	141	25.7
Canada Goose	22	41.5	101	36.9	9	11.1	9	6.4	141	25.7
Tundra Swan	10	18.9	32	11.7	1	1.2	12	8.6	55	10.0
Snow Goose	—	—	17	6.2	8	9.9	28	20.0	53	9.6
Pintail	—	—	8	2.9	10	12.3	30	21.4	48	8.7
Waterfowl	—	—	1	0.4	7	8.6	7	5.0	15	2.7
Redhead	2	3.8	6	2.2	1	1.2	2	1.4	11	2.0
Lesser Scaup	2	3.8	5	1.8	3	3.7	1	0.7	11	2.0
Canvasback	2	3.8	6	2.2	2	2.5	—	—	10	1.8
Trumpeter Swan	—	—	—	—	4	4.9	5	3.6	9	1.6
Goldeneye	—	—	4	1.4	—	—	2	2.8	8	1.4
Green-winged Teal	—	—	3	1.1	2	2.5	3	2.1	8	1.4
Wigeon	1	1.9	—	—	2	2.5	4	2.8	7	1.3
White-fronted Goose	—	—	1	0.4	1	1.2	5	3.6	7	1.3
Black Duck	4	7.5	2	0.7	—	—	—	—	6	1.1
Ross' Goose	—	—	—	—	—	—	3	2.1	3	0.5
Ring-necked Duck	—	—	1	0.4	2	2.5	—	—	3	0.5
Greater Scaup	—	—	1	0.4	—	—	1	0.7	2	0.4
Ducks	—	—	1	0.4	—	—	1	0.7	2	0.4
Ruddy Duck	—	—	1	0.4	—	—	—	—	1	0.2
Mute Swan	1	1.9	—	—	—	—	—	—	1	0.2
Gadwall	—	—	—	—	1	1.2	—	—	1	0.2
Blue-winged Teal	—	—	1	0.4	—	—	—	—	1	0.2
Bufflehead	—	—	—	—	—	—	1	0.7	1	0.2
Shoveler	—	—	1	0.4	—	—	—	—	1	0.2
Wood Duck	—	—	1	0.4	—	—	—	—	1	0.2
Mixed Geese	—	—	—	—	—	—	1	0.7	1	0.2
Mottled Duck	—	—	—	—	1	1.2	—	—	1	0.2
Total[e]	*53*	*100.0*	*274*	*100.0*	*81*	*100.0*	*140*	*100.0*	*549*	*100.0*

[a]Dr. Milton B. Friend, personal communication, 17 April 1985.

[b]Number of episodes investigated in each flyway.

[c]Number of episodes in a flyway (i.e., Atlantic Flyway) in which a given species (i.e., mallards) was a "principal species."

[d]Mallards, for example, comprised 17.0 percent of the groups (not numbers) of the "principal species" of waterfowl involved in lead poisoning episodes in the Atlantic Flyway.

[e]Because some episodes involved more than one species, the total for each flyway is larger than its number of episodes.

almost half (44 percent) of the fall and winter duck population in that area.

The remaining dabbling ducks (wood duck, gadwall, wigeon, green-winged teal, blue-winged teal, and shoveler) are involved in relatively fewer episodes of lead poisoning (Table 4), but their losses are difficult to estimate. Shot ingestion rates are low for these species (Fig. 1, Table 1), and no analyses of lead levels in wing bones, blood, or liver have been undertaken.

Lead ingestion rates in the bay diving ducks are appreciably higher than those in mallards and pintails (Fig. 1, Table 1). A large proportion of the wing bones from canvasbacks and redheads had lead levels over 20 ppm (Table 5). Although the aquatic plants and animals in the diets of canvasbacks and redheads (Bellrose 1976:312-313, 324) would tend to reduce toxicity to levels below those suffered by mallards, the large proportion of their wing bones with lead concentrations of over 20 ppm suggests that the beneficial aspects of their diet are overwhelmed by the large amount of lead these birds ingest. Less than 1 percent of the wing bones of the lesser scaup, however, which also has a high rate of shot ingestion (Fig. 1, Table 1), had lead levels over 20 ppm. Its extensive diet of mollusca, a rich source of protein and calcium (Bellrose 1976:354), may inhibit the absorption of lead into the blood. Ring-necked ducks also ingest large amounts of lead (Fig. 1, Table 1) and have diets primarily composed of vegetable matter (Bellrose 1976: 334). Thus, we anticipate that the incidence of lead poisoning among ring-necked ducks should be comparable to that of redheads. On the basis of lead shot ingestion and levels of lead in wing bones, we estimate that losses of canvasbacks, redheads, and ring-necked ducks are on the same order of magnitude in their populations as is the case in mallards.

Table 5.—Percentage of several duck species with lead >20.0 ppm in their wing bones (from Stendell et al. 1979).

Species	Flyway	Age: Immature	Age: Adult	Combined Mean
Mallard	A	17.0	38.5	27.8
	M	14.0	16.4	15.2
	C	4.9	5.3	5.1
	P	13.2	19.1	16.2
Mean		12.3	19.8	16.1
Black Duck	A	9.6	8.9	9.3
Mottled	A,M,C	43.4	43.5	43.4
Pintail	C	4.1	12.3	8.2
	P	7.1	11.3	9.2
Canvasback	M,C,P	7.3	26.8	17.1
Redhead	C,P	20.0	35.3	27.7
Lesser Scaup	A,M,C,P	0.4	1.0	0.7

Tundra (*Cygnus columbianus*) and trumpeter swans (*C. buccinator*) and Canada and snow geese collected from a sizable number of lead poisoning episodes were sent to the National Health Laboratory (Table 4); the rates of shot ingestion by Canada and snow geese (Table 3) are generally lower than those of mallards. The diet of Canada geese focuses on grains, which are high in carbohydrates, and green forage of cereal grains, pasture grasses, and legumes, which are all high in protein (Bellrose 1976:164). Like Canada geese, snow geese have increasingly deserted feeding on marsh plants to feed on agricultural crops (Bellrose 1976:123).

As long as geese utilize appreciable amounts of green forage, the amount of lead they normally ingest seldom leads to death. However, when green forage is unavailable or in short supply (often because of weather), lead poisoning may become an important factor in mortality. During the late winter of 1977, for example, an estimated 3,500 Canada geese died in southern Illinois from the effects of lead poisoning (Illinois Department of Conservation 1977). From late January through early April 1974, an estimated 925 Canada geese were victims of lead poisoning at Turk's Pond in southeastern Colorado (Szymczak and Adrian 1978). On the Missouri River in South Dakota during the winter of 1978-79, a minimum of 3,665 Canada geese and 1,091 ducks lost to lead poisoning were counted and as many as 8,000 Canada geese were estimated to have been lost (South Dakota press release from Chuck Post). A winter inventory on four areas with relatively deep water in east-central Wisconsin in December 1980 indicated the presence of 66,900 Canada geese (Amundson in press). Severe cold weather hit the area on 3 January 1981, and the water on the four areas froze. Many of the geese moved to the relatively shallow Lake Puckaway and to Grand River, where water was less than 2 feet deep and shot deposition was as high as 12,000 lead pellets per acre. Nearly 3,200 Canada geese dead from lead poisoning were picked up in and around Lake Puckaway from 14 January through March 1981. These birds amounted to more than 20 percent of the entire wintering flock of Canada geese in east-central Wisconsin. In January-February 1984 an estimated 431 waterfowl died from lead poisoning on the Suter National Wildlife Refuge, California. Of these, 346 (80.3 percent) were snow geese. Necropsy of sick and dead snow geese indicated lead poisoning in 79 percent of the immature birds and 21 percent of the adults. Avian cholera accounted for 11 percent of the immature and 38 percent of the adult snow geese and avian botulism for 0.4 percent of the immatures. Rice, moist soil plants, millet, and other foods for waterfowl are produced on the refuge. Of the lead-poisoned snow geese with shot in their gizzards, the gizzards of immature geese contained an average of 9.4 pellets; those of adults contained an average of 8.0 (U.S. Fish and Wildlife Service 1984).

Mitigating Effects of Diet

Over a span of 35 years, we and others have conducted dozens of experiments with penned ducks in an effort to evaluate the importance of lead shot, to screen potential substitutes for lead shot, to study the physiology of lead poisoning, and to relate our findings to the results of similar studies. We found that weight change between the control and experimental birds was one of the best criteria for assessing levels of lead toxicity. Mortality differences among the dosed and undosed groups provided a more tangible but less precise measurement of toxicosis. These studies reveal that many variables affect toxicity levels. The important variables that have been identified are type and amount of food consumed, amount of soil taken into the digestive tract, age, sex, and size of the bird, amount of lead shot ingested, and season.

Probably the single most important factor controlling the level of toxicity of ingested lead is the type of food consumed (Jordan and Bellrose 1951). In numerous experiments with various kinds of foods, the highest levels of lead toxicity occurred with a diet of corn and the lowest with commercial duck pellets that were high in protein. Of the aquatic plants tested, the green foliage of the following was found to be the most effective in suppressing the toxic effects of lead: coontail (*Ceratophyllum demersum*), sago pondweed (*Potamogeton pectinatus*), duckweed (*Spirodela polyrhiza*, *Lemna minor*), and chara (*Chara* sp.). Animal food in the form of quahog clam meat (*Mercenaria mercenaria*) and oyster shells fed to lesser scaups also suppressed toxic symptoms. On the other hand, in addition to corn, toxic effects increased when penned ducks were fed diets of weed seeds, wheat, rice, or other small grains (Fig. 3).

Foods most successful in alleviating lead toxicity were those high in protein. The crude protein content of several foods was determined by the Department of Animal Nutrition, University of Illinois at Urbana-Champaign: large seed smartweed (*Polygonum pensylvanicum*), 7.6 percent; corn, 9.3 percent; duck millet (*Echinochloa crusgalli*), 9.5 percent; coontail, 12.3 percent; mixed small grains, 13.5 percent; duckweed, 18.3 percent; and commercial duck pellets, 18.9 percent. Increasing protein in the diet of penned mallards by adding egg albumen to corn reduced weight loss by 47 percent and increased survival by 71 percent. Similar results were achieved by adding oyster shell, calcium carbonate, and phosphorus to corn (Jordan and Bellrose 1951). Godin (1967) and Longcore et al. (1974) demonstrated that oyster shell grit, high in calcium content, reduced lead toxicosis in mallards.

Comparisons of lead-dosed mallards on a high protein-calcium diet of turkey mash with lead-dosed mallards on a low protein-calcium diet of hen scratch indicated the importance of calcium and protein in mitigating the effects of ingested lead (Koranda et al. 1979). Although the function of protein could not be determined from the data in this study, its presence undoubtedly lowered body burdens from ingested lead and prevented lethality in ducks that had received 3-6 pellets. Koranda and his colleagues considered the value of a diet high in calcium and protein to be twofold: (1) it reduces the absorption of lead from the gastrointestinal tract, and (2) it lowers the general body burden of lead in the bird. Longcore et al. (1974:10) also concluded that lead storage in animal tissues is decreased by a high calcium diet and increased by a low calcium diet.

A comparison of lead erosion rates with lead excretion rates was made by Irwin (1977:287) for mallards on corn and corn-soybean diets. The lead excretion rate of ducks fed corn and soybeans was similar to the lead erosion rate, a finding that indicates that most of the lead was not absorbed into the blood stream but passed out through the gastrointestinal tract. Carcasses of ducks solely on corn diets had lead concentrations of less than 5 percent of the total dissolved lead, evidence that much of the apparent retention of lead was caused by an accumulation of lead in the gastrointestinal tract. Irwin concluded that no dietary component in the corn-soybean diet explained its antagonism to lead, but as the composition of nutrients added to the corn diet approached those in the corn-soybean diet, lead toxicosis was further abated.

The importance of diet in mitigating the effects of lead toxicosis is emphasized by Stendell et al. (1979:9), who concluded, "These studies show that the uptake of lead in mallard wing bones can be rapid, but that the diet—here corn vs. a commercial mash—has an important influence on rate of uptake of lead by bone."

Another example of low uptake of lead probably related to diet is shown in the low percentages of juvenile lesser scaup (0.4 percent) and adults (0.1 percent) with more than 20.0 ppm in their wing bones (Stendell et al. 1979:7). On the other hand, 20.0 percent of juvenile redheads and 35.3 percent of adults and 7.3 percent of juvenile canvasbacks and 26.8 percent of adults had levels higher than 20.0 ppm. All three species of these bay diving ducks have similar high rates of shot ingestion. Lesser scaups usually feed most extensively on animal life, followed by canvasbacks utilizing more plant material, and redheads feeding still more heavily upon vegetative matter (Bellrose 1976:312, 313, 324, 354). A correlation between the frequency of high levels of lead in the wing bones of these three species of bay diving ducks and their food habits appears to exist. The large losses of lesser scaup from lead poisoning at Rice Lake, Illinois, during the spring of 1972 appear to have occurred because the birds fed largely upon smartweed seeds rather than on mollusca (W.L. Anderson 1975; Bellrose, personal observation).

Much but not all reduction of lead toxicosis in waterfowl by diets high in protein and certain minerals appears to take place in the digestive tract. In addition, protein and minerals may reduce absorption of lead into the blood stream. If lead were absorbed into the blood stream, higher lead levels in the wing bones of the lesser scaups analyzed by Stendell et al. (1979) would be anticipated. Calcium has long been known to reduce the toxicity to aquatic organisms of some metal ions (cations). According to Skidmore (1964:233), this reduced toxicity may occur because calcium antagonizes heavy metal (lead) ions through reduction in the permeability of cell membranes and thereby reduces the speed by which lead penetrates tissues.

Findings by Sanderson and Irwin (1976:57), however, indicate that the effects of diet on lead poisoning may go beyond the digestive tract. They held game-farm mallards in individual pens and dosed each duck with five No. 4 lead shot. Diets were corn, corn plus 10 g of soil daily, duck pellets, and duck pellets plus 10 g of soil daily. The daily erosion rate of lead in the gizzard and the daily excretion rate of lead in the feces were measured for each bird. Ducks on corn alone eroded the least lead on a daily and total basis, excreted the least lead on a daily and total basis, and

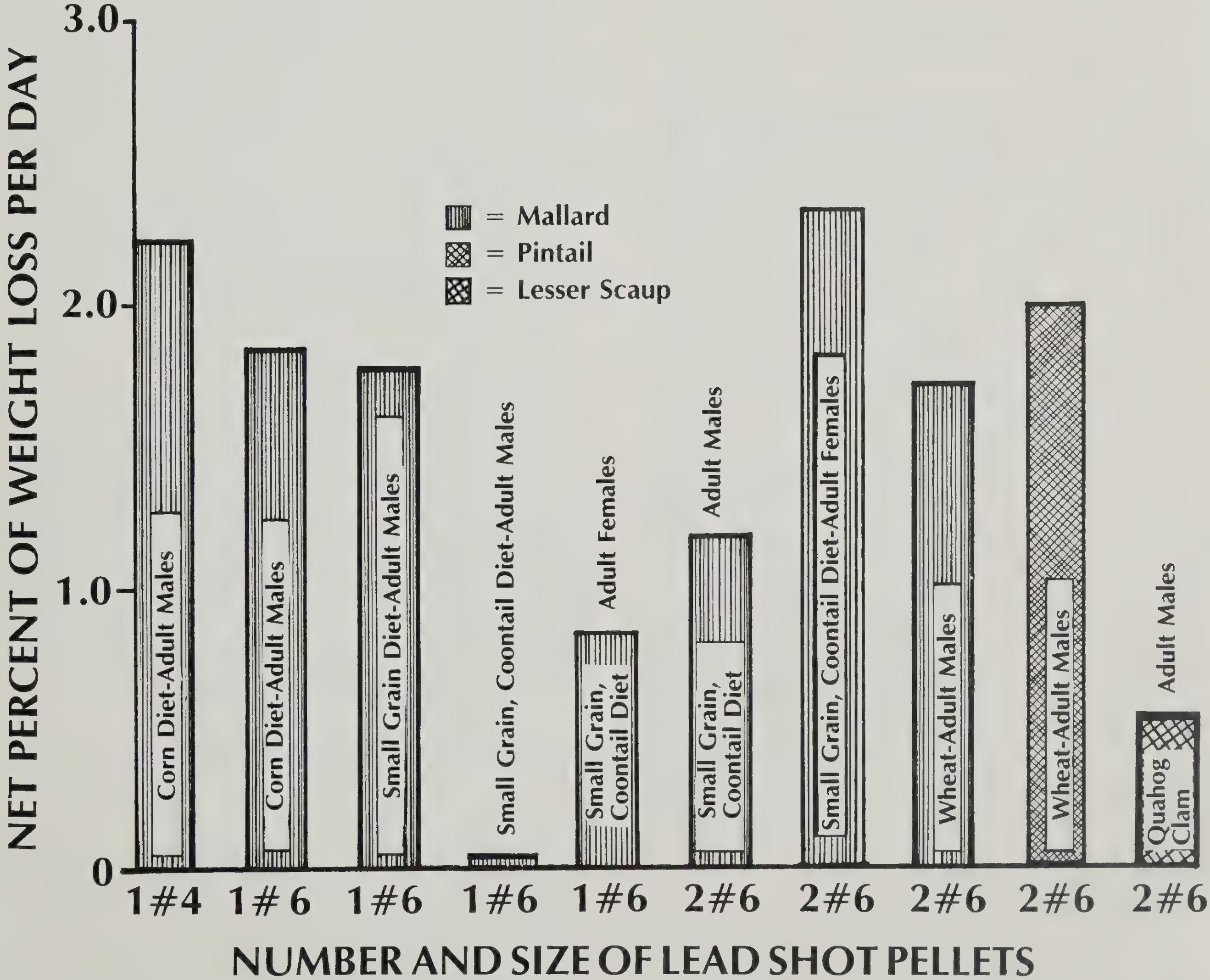

Figure 3.—Average daily net percentage body weight loss among ducks on various diets and dosed with one or two No. 4 or No. 6 lead shot. Data from Illinois Natural History Survey files.

retained the least lead on a total basis; these ducks, however, were most severely affected by lead poisoning. Ducks on corn alone retained more lead on a daily basis than ducks on corn plus soil and ducks on a pellet diet; ducks on pellets and soil retained only slightly more lead on a daily basis than ducks on corn. Ducks on pellets and soil had the highest total and daily erosion rates of lead, the second highest total and daily excretion rates of lead, and retained the most lead on a daily and total basis; these ducks, however, showed the least adverse effects of lead poisoning. Tests were not run for lead residues in the ducks, but a reasonable assumption is that more lead entered the bloodstreams of ducks on pellets and soil than entered the bloodstreams of ducks on a corn diet with no soil. In this study, diet appears to provide protection from lead even after it was absorbed from the digestive tract; diet also seemed to account for a higher excretion rate of lead.

Differences in Susceptibility Attributable to Sex

Experiments with male and female domestic and wild mallards demonstrated differences between the sexes in the manifestations of lead poisoning. Females were affected to a greater extent than males, except during the spring. The increased resistance of females to lead poisoning in spring during the prebreeding and breeding periods appears to be related to a high metabolic rate and to the mobilization of energy resources for egg-laying (Finley and Dieter 1978). In addition, spring was the only season in which food consumption by females exceeded that of males (Jordan and Bellrose 1951:21).

According to White and Stendell (1977:474), the frequency of shot ingestion by males and females among mallards and black ducks was similar. Among pintails, males ingested shot at a significantly higher rate than females. White and Stendell also reported that both sexes of these three species had similar proportions of lead (over 20 ppm) in their wing bones. Among five species of trapped ducks, Bellrose (1959:256) found that a larger percentage of female (16.0%) than male (9.8%) mallards ingested shot. Little difference was found, however, between the sexes in pintail, blue-winged teal, and wood duck (8.9% for males and 7.4% for females); more lesser scaup males (9.0%) ingested shot than did females (4.5%).

Differences in Susceptibility Attributable to Age

Several experiments compared survival and weight loss between adult and immature ducks. Up to about 7 months of age, lead had less effect on younger birds under laboratory conditions. After late December little difference was found between age groups. We surmise that because lead salts follow the same pathways in the blood stream as calcium, a high proportion of the lead was deposited in the skeletons of the maturing young mallards. This deposition would remove circulating lead from the blood stream and help to reduce lead toxicosis in vital organs. As the skeleton (particularly the sternum) of the young ducks became increasingly ossified, less lead, we postulate, was deposited in bone structures. As a result, after about 7 months higher levels were found in the blood. Nevertheless, lead is deposited in wing bones almost immediately after exposure in both juveniles and adults (Stendell et al. 1979:9).

At Catahoula Lake, Louisiana, Shealy and his colleagues. (1982:43) found that lead ingestion occurred more frequently in adults than in juveniles in both mallards and pintails. Lead content of wing bones examined by Stendell et al. (1979:6,7) showed slightly higher concentrations of lead in adults and a higher proportion of adults with ≥ 20 ppm lead among mallards, mottled ducks, pintails, canvasbacks, redheads, and lesser scaups. Only juvenile black ducks had slightly higher levels of lead in their wing bones than the adults of their species. These data suggest that juvenile ducks of these species ingest fewer shot than adults or that the ossifying sternum of juveniles takes up a greater share of blood lead. The latter assumption seems more likely.

Differences in Susceptibility Attributable to Size

As might be expected, the larger the waterfowl, the less effect a given amount of ingested lead has. Under controlled experiments, Canada geese (*Branta canadensis interior*) showed the least effects of lead toxicosis followed in ascending order by mallards, pintails, and blue-winged teal. All tests were not made at the same time or with the same foods and shot doses, but studies pairing mallards with each of the other species provided a basis for comparison.

Mortality from Lead Poisoning

Overlooked Losses

Dead ducks are seldom noticed in the marsh, and most hunters are unaware of the extensive losses of waterfowl caused by lead poisoning. Nevertheless, banding data indicate that approximately one-fourth of all ducks alive in September die from *natural causes* within the year—slightly more than are killed by hunters. The fall population of game ducks is usually around 90,000,000, although it declined to about 62,000,000 in 1985. With a natural mortality rate of 22.2 percent, a minimum of 14,000,000 to 20,000,000 of these ducks can be expected to die from natural causes each year.

The most convincing data on this aspect of waterfowl population dynamics stem from a study of 134,000 bands recovered from mallards by D.R. Anderson (1975). His analysis showed that adult male mallards suffered an annual mortality of 37 percent; adult females, 44 percent; and immatures, 50 percent. Natural losses accounted for 45 percent of the annual mortality of adult males, for 58 percent of that of adult females, and for 52 percent of that of immatures; hunting was responsible for the remainder of the losses. Most other species of ducks have slightly higher annual mortality rates than those of mallards, and, with the exception of the wood duck, natural losses account for an even higher percentage of the total mortality in these other species (Bellrose 1976).

One might wonder why the death of so many ducks goes largely unnoticed. Our observations indicate that when a duck becomes seriously ill, it leaves its flock and seeks dense cover out of water in marshes and along the shores of lakes. There it becomes a potential meal for a mink, raccoon, fox, coyote, eagle, hawk, owl, crow, gull, or any of a host of other predators.

A rapid disappearance of mallard and Canada and snow goose carcasses was reported by Humburg et al. (1983). At the Squaw Creek National Wildlife Refuge (NWR) in northwestern Missouri, 90 carcasses disappeared at the following rates: 9.4 percent after 1 day, 12.3 percent after 2 days, 36.7 percent after 3 days, and 62.2 percent at the end of 4 days. In central Missouri at the Swan Lake NWR, 62 carcasses were depredated at the following daily cumulative rates: 43.5, 67.7, 79.0, and 82.3 percent. In Texas coastal marshes, 47 carcasses disappeared at the following cumulative rates: 32 percent in less than 1 day, 47 in 2 days, 62 in 3 days, and 89 by the eighth day (Stutzenbaker et al. 1983).

Zwank et al. (1985) removed 1,072 sick, dead, and dying lead-poisoned waterfowl from Catahoula Lake, Louisiana, from 13 October 1980 through 31 January 1981. No reports of waterfowl die-offs were received during the time these collections were made, but removal of the waterfowl might have made reports of die-offs less probable. No die-off, however, had been reported during the 1979-1980 season on this same area when Smith (1980) found levels of ingested lead similar to those reported by Zwank and his colleagues. They concluded, "This magnitude of mortality without a corresponding reported die-off supports Bellrose's (1976) contention that the most important aspect of lead toxicosis mortality may not be the recorded massive die-offs, but the day-to-day losses" (p. 23).

As long as the numbers of ill and dead ducks do not exceed the ability of predators and scavengers to consume them, few carcasses are left as evidence. When mortality reaches greater proportions, however, carcasses become evident. Because lead poisoning generally results in the wasting away of flight muscles, victims are often unable to fly and are sometimes immobilized for a week or two before death, circumstances that make them easy prey. In diseases such as botulism and duck virus enteritis, birds succumb more quickly than they do to lead poisoning. Diseased carcasses therefore are more likely to be seen than lead-poisoned ones because predators and scavengers fail to keep pace with the death rate.

Unless waterfowl losses are so extensive that they focus attention on a particular area, they are often overlooked, especially by the public. Humburg et al. (1983:254) found that one-fourth of the waterfowl carcasses "planted" in quadrats on Swan Lake Refuge, Missouri, were not located when the areas were searched. Texas biologists (Stutzenbaker et al. 1983) experienced an even greater surprise. They planted 100 waterfowl carcasses on a 40.5-ha (100-acre) area, 10 carcasses in each of 5 cover types and 50 carcasses randomly tossed atop vegetation. Within 30 minutes after placement, 8 searchers were able to locate only 6 birds, all of which had been placed on top of vegetation. None of the carcasses placed in cover, where a sick or crippled duck might be expected to hide, was found. Because scavengers and predators probably did not remove all of the 94 unfound ducks within 30 minutes after they had been planted, these results also demonstrate the difficulty of finding waterfowl carcasses hidden in vegetation.

Bellrose's (1959: Table 31) data suggest that 58.5 percent of the male mallards in the Mississippi Flyway with one or more lead pellets in their gizzards at a given time will die of lead poisoning during that year. Of individual male mallards with one or more lead pellets in their gizzards at a given time, only 7.8 percent (58.5 percent ÷ 7.5, the number of 20-day intervals in the 150 days ducks typically spend in migration and on wintering grounds) will die of lead poisoning at that time. The remaining 50.7 percent of the deaths from lead poisoning will come from ducks that have not yet ingested shot or from the 92.2 percent that had shot in their gizzards but will not die until they ingest shot a second, third, fourth, fifth, sixth, or seventh time during the year.

As an approximation, however, and until better data are available, we can calculate the estimated mortality from lead poisoning of an assumed population of 50,000 male mallards on a given wintering or migration area in the following manner:

50,000 male mallards x 6.80 percent that have one or more lead pellets in their gizzards (Bellrose 1959) = 3,400 x 58.5 percent = 1,989 male mallards that will die of lead poisoning on the area.

If the area were searched each day for 150 days (the calculated time ducks spend in migration and on wintering grounds) and if 10 percent of the male mallards that died of lead poisoning were found—in a Texas study (Stutzenbaker et al. 1983), 8 searchers found only 6 percent of 100 dead ducks planted in a 40.5-ha area—searchers should find 1.3 male mallards per day dead of lead poisoning (1,989 male mallards dead of lead poisoning x 10.0 percent found = 199 ÷ 150 days = 1.3). Thus, we should not be surprised that "routine" losses from lead poisoning go largely unnoticed.

In contrast to lead poisoning, the self-perpetuating nature of duck virus enteritis, other contagious diseases, and botulism causes victims to spread the disease or toxin among healthy waterfowl. Local epizootics are then prone to develop. Even so, outbreaks are often far advanced before they are noticed. During the past several years, the highest known losses from diseases in the United States other than lead poisoning are botulism, 150,000 (1970); fowl cholera, 70,000 (1956-66) up to 100,000 (1974-75); and duck virus enteritis, 40,000 (1973) (U.S. Fish and Wildlife Service 1976). More recently, 122,555 waterfowl died from disease in California and Nevada. Of these, 78 percent died of botulism, 21 percent of avian cholera, and 1 percent from other diseases (memo from C.T. Osuzi, disease biologist, U.S. Fish and Wildlife Service, Klamath Basin Refuges, Tule Lake, California, to refuge biologists, U.S. Fish and Wildlife Service, 31 May 1984). Such epizootics among waterfowl have been sporadic both in time and place. Obviously, known losses from disease are but a tiny fraction of the nonhunting mortality of ducks.

Lead poisoning, however, is not as confined to particular times or places as are bacterial and viral diseases. Because of the widespread distribution of spent lead shot on the bottoms of lakes and marshes and on upland feeding grounds, the potential for lead poisoning is everywhere that ducks and geese are hunted.

Because lead-poisoned ducks are easier for hunters to bag than healthy ducks, man is frequently the agent that removes lead-poisoned ducks from the population. In a sense, the hunter fills the same role as other predators. The role of wild predators increases in importance after the hunting season when man is no longer removing a portion of the afflicted ducks.

In experiments in central Illinois, 1949-1951, trapped wild mallards were banded and released either as controls (not dosed with lead) or as experimental birds dosed with one, two, or four No. 6 lead shot (Bellrose 1959). During the following 25 days, hunters returned bands in the following proportions: 1.5 bands from birds dosed with one pellet, 1.9 bands from birds dosed with two pellets, and 2.1 bands from birds dosed with four pellets were returned for every band returned from nondosed control birds. This evidence indicates that mallards ill from lead poisoning were more readily taken by hunters than were healthy ducks. The temporal effect of increasing levels of shot on the availability of dosed ducks to hunters is shown in Figure 4. Many of the ducks shot were undoubtedly suitable for human consumption, but others in the late stages of lead poisoning were emaciated and probably were discarded. Many thin ducks that we have examined in hunters' bags showed the effects of lead poisoning. Relationships between number of lead pellets in the gizzard and body weight in male and female mallards found dead or moribund in Minnesota, Illinois, Arkansas, and Louisiana are shown in Table 6.

Weight loss as a result of lead poisoning has been demonstrated in other studies of experimental dosing. The daily percentage weight loss in captive game-farm mallards dosed with from zero to six No. 6 lead pellets is shown in Figure 5, based on unpublished data in the Illinois Natural History Survey (INHS) files. The average daily percentage loss of body weight among mallards dosed with one No. 4 or one or two No. 6 lead pellets on various diets is shown in Figure 3 (from unpublished data in the INHS files). All were adults, but differences between males and females were shown in some cases.

Losses attributed to crippling often conceal losses due to lead poisoning. We found that two-thirds of

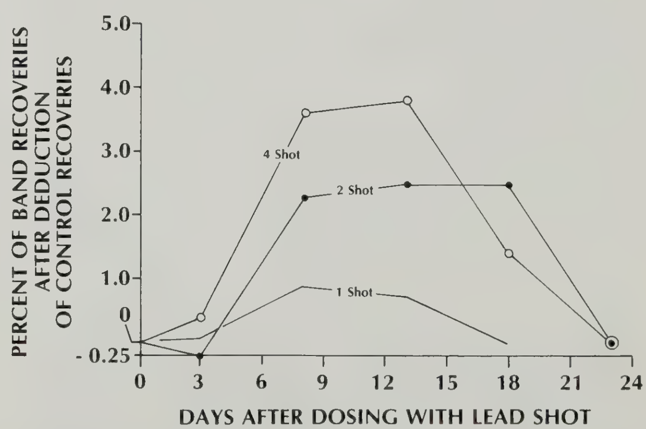

Figure 4.—Band recovery rates of adult and immature male mallards dosed with lead shot above the recovery rate of nondosed control birds. Data from Illinois Natural History Survey files.

the ducks we initially believed crippled proved instead to be dead or incapacitated from lead poisoning (Bellrose 1953:348). We also determined that lead-poisoned mallards weighed from 0.2 to 0.7 lb less than mallards that succumbed to shot wounds (Bellrose 1953). Most moribund ducks of light weight are lead-poisoned rather than victims of debilitating wounds. Similar findings were made in Missouri by Humburg et al. (1983:252), who reported that 83.5 percent of necropsied mallards died from lead poisoning; only 15.6 percent had died from gunshot wounds. In Louisiana, Zwank et al. (1985:25) calculated that ">7 times as many northern pintails, >5 times as many mallards, >7 times as many snow geese, and 3 times as many greater white-fronted geese died, or would have died, from lead toxicosis than died, or would have died, from wounding by hunters." Results were different in California, however, where 4,991 waterfowl carcasses were necropsied from 1977 through 1980 (Moore and King 1980). Of the 4,092 specimens for which cause of death was determined, 55 percent had died from avian cholera, 24 percent from crippling wounds, 13 percent from botulism, 6 percent from lead poisoning, and 2 percent from miscellaneous diseases.

Data from Duck Die-offs

Large-scale losses of waterfowl from lead poisoning are most evident after the hunting season, a time when few hunters or other people are in marshes and swamps. Lists of die-offs known to have been caused by lead poisoning were compiled by Bellrose (1959:240-241) and the Mississippi Flyway Council Planning Committee (Hawkins 1965). Most of these were noted in winter and early spring; only two were observed during the hunting season, both late in the season. As determined from carcasses collected periodically, Humburg et al. (1983:255) reported that mallard losses from lead poisoning in Missouri also appeared most frequently late in the hunting season and after the season had closed.

Losses from lead poisoning occur most frequently during the winter and spring for several reasons. Perhaps the most important is that hunting deters waterfowl from feeding in many areas until the close of the season. Hunters place their blinds on or near the best waterfowl feeding grounds, and spent lead, of course, is deposited most densely in the vicinity of these blinds. After the close of the hunting season and if freeze-up has not occurred, waterfowl are attracted to the abundant food still available near the blinds and consequently ingest spent lead shot while feeding, apparently mistaking shot for seeds, tubers, small mollusks, and other food items. (Waterfowl may ingest lead during the hunting season by feeding at night near blinds, but this ingestion probably results only in chronic losses until the birds are stressed by winter weather late in the season.) When large numbers of ducks gather to feed on grounds that were heavily hunted, the number ingesting shot may increase mortality to a level beyond the appetites of scavengers and predators, and the die-off then becomes noticeable. In addition, these predators had been helped during the hunting season by hunters

Table 6.—Average weights (g) in relationship to number of ingested lead shot of male and female mallards found dead or moribund during massive die-offs in Minnesota, Illinois, Arkansas, and Louisiana, 1938-1955 (unpublished data from Illinois Natural History Survey files).

Number of Lead Shot	Males			Females		
	No.	Wt.	s_x	No.	Wt.	s_x
0	48	900	154.0	43	767	83.7
1	106	838	60.1	87	748	69.1
2	71	860	51.1	48	767	101.3
3	60	841	117.0	41	785	87.0
4	43	868	108.2	21	767	45.1
5	30	889	92.7	14	842	82.9
6	28	852	37.8	13	757	46.8
7	9	847	87.8	6	771	70.2
8	11	866	82.3	3	771	90.7
9	10	816	122.8	4	794	45.3
10	9	892	130.2	1	726	—
>10	30	885	121.3	12	771	146.0
Total and average	*455*	*860*[a]	*89.3*	*293*	*768*[b]	*79.8*

[a]Average normal weight = 1,247 g = 31.0 percent loss.
[b]Average normal weight = 1,106 g = 30.6 percent loss.

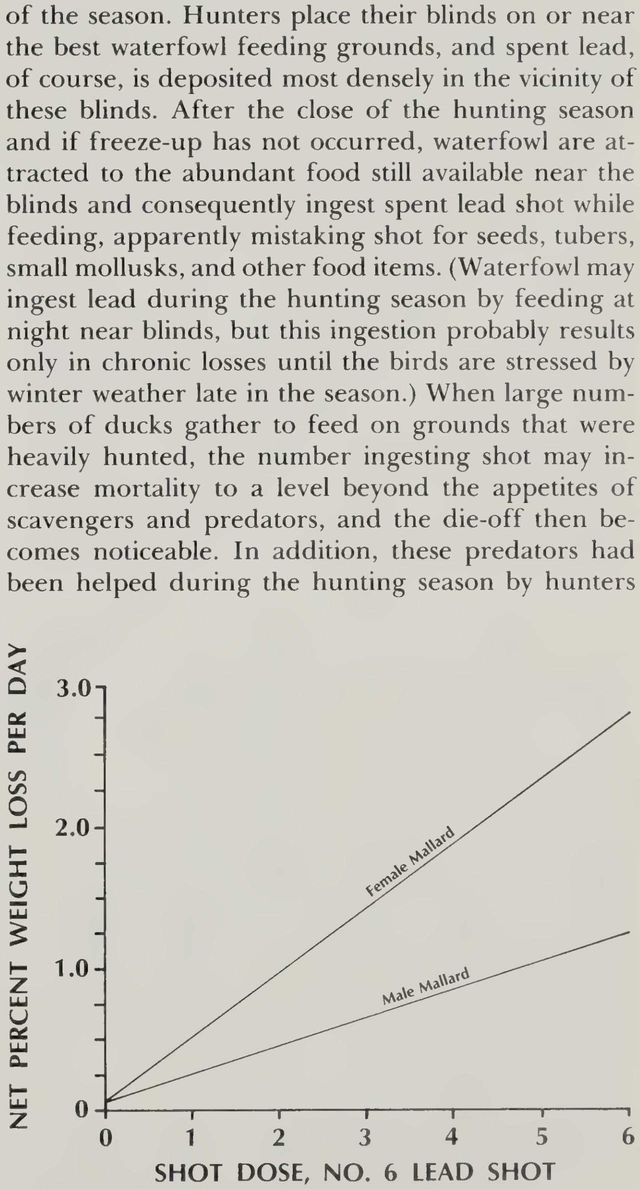

Figure 5.—Net percentage of daily body weight loss in game-farm mallards with increasing doses of No. 6 lead shot. Data from Illinois Natural History Survey files.

who killed and removed a sizable proportion of poisoned birds.

Although observed die-offs of waterfowl from lead poisoning represent a tragic loss, they represent only a small proportion of the actual loss. Knowledge of the magnitude of these obscure, usually overlooked, day-to-day losses comes from several sources: (1) the presence of ingested lead in waterfowl gizzards obtained from hunters during the fall and early winter, (2) the occurrence of lead in wing bones, (3) the level of lead in blood, (4) the level of lead in livers, and (5) the numerous experiments with penned or released ducks dosed with lead shot.

The shot levels found among mass die-offs of ducks from lead toxicosis provide ancillary information on the lethality of lead. Most dead or moribund male and female mallards in three regions of the Mississippi Flyway were found with three or fewer pellets (Table 7). From 4.4 to 18.1 percent of these birds contained no ingested pellets. In Illinois and Minnesota-South Dakota, no statistical differences were found between the percentages of males and females ingesting lead or between the number of pellets ingested by them (X^2 = 12.5, P = 0.67; X^2 = 11.4, P = 0.59). In Arkansas-Louisiana, however, mallard females died with significantly fewer pellets in their gizzards than did males (X^2 = 22.0, P = 0.98). A comparison of mallard males in the three flyway regions revealed no statistical difference in rates of ingested shot between birds found in Illinois and birds found in Minnesota-South Dakota (X^2 = 15.4, P = 0.83) but a significant difference between rates of ingested shot in birds found in Illinois and those found in Arkansas-Louisiana (X^2 = 69.5, P = 0.99). Rates of shot ingestion by male and female mallards in Arkansas-Louisiana were considerably higher than they were for male mallards in Illinois (data from Bellrose 1959: Table 2, 3).

These differing rates may be accounted for by differing food habits. Mallard diets in Arkansas-Louisiana were more beneficial than those in the upper Midwest, and a higher level of shot ingestion was consequently required to incur mortality in mallards in Arkansas-Louisiana than in the upper Midwest. Stated another way, lead-poisoned birds dropped out of the population more quickly in the upper Midwest than they did in Arkansas-Louisiana.

The weights of mallards that died in the Mississippi Flyway die-offs discussed above were remarkably similar by sex over the most common levels of pellet ingestion (one to four pellets) (Table 6). Many of these specimens were alive when picked up but incapable of flight. Both males and females were 31 percent below their average weights (Bellrose 1976:229).

These findings suggest that chronic rather than acute conditions usually prevail in lead poisoning die-offs. W.L. Anderson (1975) reported on certain acute effects of lead in lesser scaups. He observed a positive correlation between the number of ingested pellets and body weight, but these birds had ingested large levels of lead shot (44 percent had ingested more than 10 shot each). The lack of relationship between low numbers of ingested shot and body weight implies that once ducks are affected by lead the physiological results are similar among individual ducks no matter how many shot are in their gizzards.

Table 7.—Incidence of ingested lead shot pellets in male and female mallards found dead or moribund at die-offs from lead poisoning, in three regions of the Mississippi Flyway, 1938-1955 (from Bellrose 1959:Table 2, 3).

	Illinois				Minnesota-South Dakota				Arkansas-Louisiana			
	Males		Females		Males		Females		Males		Females	
Shot Level	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
0	37	11.9	32	18.1	21	13.4	11	11.5	8	4.4	14	8.6
1	90	29.0	57	32.2	53	33.8	42	43.8	9	5.0	18	11.1
2	49	15.8	30	16.9	30	19.1	20	20.8	35	19.3	23	14.2
3	36	11.6	19	10.7	20	12.7	10	10.4	24	13.3	34	21.0
4	18	5.8	7	4.0	10	6.4	6	6.3	25	13.8	18	11.1
5	23	7.4	5	2.8	4	2.5	3	3.1	18	9.9	19	11.7
6	9	2.9	5	2.8	8	5.1	2	2.1	16	8.8	12	7.4
7	5	1.6	1	0.6	2	1.3	0	0.0	8	4.4	10	6.2
8	7	2.3	2	1.1	1	0.6	1	1.0	9	5.0	2	1.2
9	4	1.3	1	0.6	0	0.0	1	1.0	8	4.4	5	3.1
10	6	1.9	1	0.6	3	1.9	0	0.0	6	3.3	2	1.2
>10	26	8.4	17	9.6	5	3.2	0	0.0	15	8.3	5	3.1
Total	*310*	*99.9*	*177*	*100.0*	*157*	*100.0*	96	*100.0*	*181*	*99.9*	*162*	*99.9*

In chronic lead poisoning, gizzard activity is reduced and the afflicted bird literally starves to death, dying at a weight approaching that caused by starvation. Conversely, birds succumbing to acute lead poisoning have massive destruction of blood and other tissues and die at much higher weight levels.

Data from Laboratory and Field Studies

The incidence of lead pellets found in the gizzards of waterfowl and the high levels of lead present in their wing bones, blood, and livers provide ample evidence of the magnitude of exposure to lead. Other data are needed to tell us how many waterfowl die from this exposure. Initially, we thought that experiments with penned waterfowl would provide data on the relationship between amount of lead ingested and mortality; however, varying kinds and amounts of food consumed affected the toxicity of lead so significantly that results were tenuous. Furthermore, a wild duck's diet cannot be precisely duplicated in the laboratory, and ducks in the wild eat larger quantities of food to maintain their weight than do captive birds. In addition, experiments with penned birds cannot reveal at what point a predator might take a duck ill from lead poisoning or under what circumstances stress from lead poisoning might increase a bird's susceptibility to other mortal diseases. Finally, wild mallards in captivity and game-farm mallards in captivity react differently to diet and to dosed lead. Wild mallards, no doubt, are under greater stress in captivity than are game-farm mallards.

Field experiments, on the other hand, provide meaningful data on the relationships between ingested lead and mortality in wild ducks. Such an experiment involves trapping large numbers of wild ducks of one species, fluoroscoping and weighing them to select healthy birds, dosing a portion with lead shot, banding all of them, and returning the birds to the wild— all within a few hours.

Bellrose (1959) conducted such a study. All trap sites were located within 0.8 km (0.5 mi) of the Havana Field Station of the Illinois Natural History Survey on the Chautauqua National Wildlife Refuge so that trapped birds could be speedily handled. During three consecutive autumns, beginning in October 1949, a total of 6,099 mallards were trapped; 4,307 were used in the experiment. These birds were divided into four groups: undosed birds (controls), birds dosed with one No. 6 lead shot, birds dosed with two No. 6 shot, and birds dosed with four No. 6 shot. To increase the number of band recoveries in 1950 and 1951, a $2.00 Reward Band was attached in addition to the standard band. This tactic increased the recovery rate 2.2 times (Bellrose 1955).

In this experiment, band recoveries from hunters provided the data for assessing the mortality of the several groups of mallards. We found a higher rate of band recoveries from dosed wild mallards during the first 10 months after banding than from the undosed controls (Fig. 6). These higher recoveries began to appear 8 days after dosing (Fig. 4). As previously noted, lead-poisoned birds are more likely to be killed by hunters. Moreover, reduced rates of band recoveries among dosed ducks during the hunting season 1 year after banding suggest that additional nonhunting mortality may have occurred in the interim. Clearly, adult male mallards dosed with one, two, or four No. 6 lead shot suffered greater mortality from lead poisoning than did juvenile males during the year of banding (Bellrose 1959:274). Differences in band recovery rates between dosed and undosed birds during the second season were highly significant for adult males ($X^2 = 18.72$, $P < 0.005$) and for juveniles ($X^2 = 16.02$, $P < 0.005$). As in the experiments with penned mallards, juveniles in the wild were not as susceptible to lead toxicity during the fall as were adults. However, this difference in susceptibility appears to cease late in December; thereafter juveniles ingesting lead suffered losses similar to those of adults.

Band data on female mallards were not of the same quality as data for males. Nevertheless, the data suggest that females suffer greater mortality during the fall and early winter than do males. This pattern agrees with the data from our laboratory experiments.

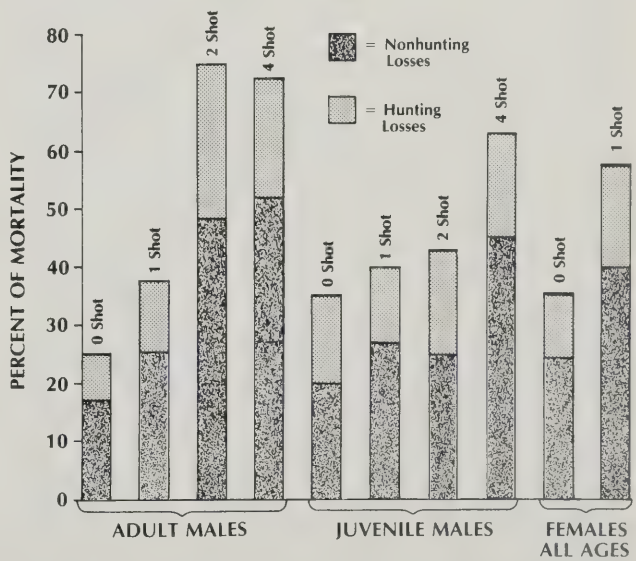

Figure 6.—Percentage of the total 4-year mortality after banding that occurred during the first 10 months after wild mallards dosed with zero, one, two, or four No. 6 lead shot were released. (Data from Bellrose 1959, Table 27; data for females adjusted by information in Bellrose 1955).

As previously pointed out, however, females become less susceptible than males to lead toxicosis late in the spring.

A similar dosing experiment was conducted in California between 22 January and 23 March 1979 by the U.S. Fish and Wildlife Service and the California Department of Fish and Game (Deuel 1985). Slightly over 12,000 pintails were trapped and banded; 6,109 were dosed with two No. 5 lead shot, and all ducks were released. Bands subsequently recovered revealed no significant differences in survival between dosed and nondosed birds. The presence of lead shot had no apparent effect upon subsequent survival.

To understand the difference in results between experiments with free-flying wild mallards in Illinois and free-flying wild pintails in California is to understand the difference in food habits between the two species in the respective regions. Numerous laboratory experiments (Jordan and Bellrose 1951; Longcore et al. 1974; Irwin 1977; Koranda et al. 1979) have shown that duck diets high in protein, calcium, and phosphorus help to alleviate lead toxicosis.

Diets of pintails in three regions of California (Pederson and Pederson 1983; Euliss 1984; Deuel 1985) have a high protein level, particularly in late winter and early spring, the period of the experimental dosing study. Pintails were found to consume appreciable quantities of invertebrates; midge (chironomid) larvae were especially important and most prevalent in late winter and early spring diets. The crude protein of the most important invertebrates ranged from 46 to 76 percent (Deuel 1985: Table 7). Swamp timothy (*Helechloa schoenoides*), an important food for pintails in the Central Valley of California, has a crude protein content of 13.9 percent, high for a plant species (Deuel 1985). We believe that the high protein diet of pintails in late winter and early spring in California reduced mortality from lead at the level tested (two No. 5 pellets). Laboratory studies indicate that as shot levels increase they increasingly overwhelm the beneficial effect of diet. According to Pederson and Pederson (1983), Euliss (1984), and Deuel (1985), pintails feed most extensively on seeds of moist soil plants during the fall, a period when invertebrate consumption is at a seasonal low.

The diet of the mallards in the Illinois experiment was composed of corn, seeds of moist soil plants, and coontail, a minor item high in protein (Anderson 1959). Overall, this diet was high in carbohydrates but low in protein. The difference between the protein intake of mallards in Illinois and pintails in California appears responsible for the difference in mortality rates from lead poisoning.

Had the California experiments been conducted during the fall when the pintails feed more extensively on rice, barley, and weed seeds, results might have been different. Stendell et al. (1979:6) reported that 12.5 percent of the wing bones of adult pintails in California and 8.9 percent of those of juveniles had >20.0 ppm lead. These findings establish that significant amounts of lead entered the bodies of pintails during the hunting season, the period in which wing bones were collected for analysis.

Several reports of waterfowl, including pintails, dying of lead poisoning in California have been made. Moore and King (1980) conducted intensive waterfowl mortality surveys during 1979-1980 on Delevan National Wildlife Refuge and Grizzly Island Wildlife Area, California. They collected 779 waterfowl of which 340 were necropsied; 36 (10.6 percent) were diagnosed as lead poisoning mortalities. Pintails were also involved in 21.4 percent of the lead poisoning episodes in the Pacific Flyway as determined by the National Wildlife Health Laboratory (Table 4). Stendell et al. (1979) reported that 9.2 percent of wing bones from pintails from the Pacific Flyway contained >20.0 ppm lead (Table 5).

Practices to Reduce Lead Toxicosis

Manipulation of the Habitat

Several ways of manipulating the habitat to reduce lead poisoning in waterfowl have been considered. Osmer (1940) suggested distributing gravel to be used as grit by waterfowl. In winter, the gravel could be placed on the ice. Beer and Stanley (1965) reported that when grit is limited, lead pellets are more likely to be ingested and are retained longer than when grit is plentiful. Trost (1981:70) also surmised from his study of grit passage that ducks deprived of grit retain shot longer than ducks with access to grit. Although they presented no supporting evidence, Osmer (1940), Rosen and Bankowski (1960), and Beer and Stanley (1965) suggested that excess grit moves through the gastrointestinal tract of birds quickly and carries eroded lead pellets with it. Sanderson and Irwin (1976:39, Table 38) noted a significantly higher expulsion rate of lead pellets from game-farm mallards with access to soil 1 to 7 days after dosing as compared with ducks on a wire floor. The diet for both groups was shelled corn. From 7 to 21 days after dosing, ducks with access to soil continued to pass shot more quickly than did ducks on wire, but the differences were not significant. Balanced against the expulsion rate, however, was the increased erosion rate of the ingested shot among ducks with access to soil. The daily erosion rate of lead from ingested pellets was significantly higher 1-7, 8-14, and 15-21 days after dosing in ducks with access to soil than it was in ducks on wire. The presence of soil or grit, or both, results in a higher daily exposure to eroded lead in ducks before the pellets are expelled or entirely eroded. Thus, the total effect of readily available grit on mortality from ingested lead is unclear. In individual ducks that expel ingested lead shot quickly, perhaps as a result of a readily available supply of grit, the grit may be beneficial. In ducks that do not quickly expel ingested lead shot but erode it faster because of abundant grit in the gizzard, the grit may prove deleterious (Sanderson and Irwin 1976:39).

Jordan and Bellrose (1951) reported modest success in reducing lead ingestion by using scare devices after the hunting season to drive waterfowl from heavily shot areas. Chupp and Dalke (1964) reported that attempts to drive swans from an area in Idaho polluted by lead from mine wastes were generally unsuccessful.

Losses of waterfowl to lead poisoning have been reduced in some cases by lowering water levels in feeding grounds after the hunting season so that the ducks will leave. Bishop (1972-1973) reported that water levels have sometimes been kept low in Iowa in the spring and sections of the area disked to make lead shot less available to feeding waterfowl when the area was reflooded. When ducks are discouraged from using waterfowl habitat except in hunting season, however, the value of that habitat is largely lost to waterfowl. Further, much waterfowl habitat cannot be drained, flooded, or disked between the time of lead deposition in the fall and the time ducks migrate north the following spring. Disking is not possible in areas that cannot be drained, in green tree reservoirs, and in various other habitats.

Szymczak and Adrian (1978:305) found that lead pellets on the surface of farmland near goose hunting pits in Colorado were most numerous in an alfalfa field that had not been plowed for several years. They also observed that corn and winter wheat fields on which there had been heavy shooting had comparatively high densities of pellets. Turk's Pond, the rest area for the geese in this study, did not contain significant densities of lead pellets. Szymczak and Adrian concluded that the large-scale mortality of Canada geese that had occurred in this area resulted from lead pellets ingested from agricultural fields. Fredrickson et al. (1977) found that shot pellets were redistributed by cultivation: 45 percent of the total were in the upper 5 cm (2 inches) of cultivated soil samples; 66 percent, however, were found in the upper 5 cm of uncultivated soil samples. Esslinger (1979) reported an 86-percent reduction in the number of lead pellets in the top 2.5 cm (1 inch) of soil under normal farming operations. Thus, the availability of lead to birds, including waterfowl, on heavily shot upland fields may be reduced, but not eliminated, by plowing and disking.

Jordan and Bellrose (1950, 1951) suggested management practices that encourage the growth of submerged, leafy aquatic plants for duck food because these plants provide more protection against lead poisoning than other types of natural plant foods. This practice, however, is not feasible for much of the waterfowl habitat in Illinois and elsewhere because siltation has decreased the water depth in some areas and reduced the penetration of sunlight in others so that only in limited areas can submerged aquatic plants be supported. In addition, most large-scale impoundments do not offer suitable habitats for aquatic plants.

Few success stories can be cited in which management practices have significantly reduced lead poisoning. As long as lead shot continues to be used for hunting waterfowl, it seems prudent to keep the boundaries of refuge areas constant year after year so that rest areas will remain free of lead shot. On many state and federal waterfowl refuges, however, areas opened and closed to hunting are rotated, a practice that spreads the availability of shot to feeding waterfowl.

The Case for Steel Shot

The only management practice, other than closing the waterfowl hunting season, that will eliminate waterfowl deaths caused by ingested shot is the substitution of nontoxic shot for lead shot. At present, the only proven nontoxic shot available is steel. Sanderson and Irwin (1976) reported that five No. 4 steel shot ingested by captive game-farm mallards caused no significant changes in body weights compared with nondosed control birds, 1, 3, and 6 weeks after dosing. Packed cell volume and hemoglobin concentrations in the blood of these birds also remained at comparable levels.

Losses due to crippling. As early as 1978 Roster (1978[b]:26) argued the case for steel shot: "Although steel shot can bag ducks as well as lead shot can, the belief persists that steel shot will cripple more waterfowl and damage shotguns. This belief stems from ignorance of the results of tests to investigate gunbarrel damage as well as from ignorance of the ballistic properties of steel shot. Ballistically, steel shot can be loaded to perform as well as lead shot in bagging waterfowl out to seventy yards. Steel shot retains its shape better than lead shot does, and compensations can be made for its lighter weight, enabling it to retain energy as well as lead shot." Nevertheless, waterfowl hunters continue to voice objections to steel shot. A primary objection is based on the belief that a greater number of ducks are crippled (mortally wounded and unretrieved) by steel shot than are poisoned and crippled by lead shot (National Wildlife Federation 1985[b]). In an Ohio survey, 45 percent of the hunters cited this reason for their opposition to steel shot (Smith and Townsend 1981). In Colorado, hunters of Canada geese feared that crippling losses would increase if steel shot were used (Szymczak 1978). Waterfowl hunters in California also identified crippling as their principal objection to steel shot (Leach 1980).

No single uncontroversial way to present data on the crippling of waterfowl has been devised. Methods commonly used include birds lost per shot fired, per bird bagged, per hunter party per day, per blind per day, and per man-day of hunting. Birds lost per bird bagged would be an appropriate method if all hunters obtained their bag limit each trip, a condition that is rarely the case. Hebert et al. (1984:395) did not analyze cripples per bird bagged in their Louisiana study because of instances in which ducks were crippled but none was bagged. The remaining three methods have reasonably uniform bases; however, the number of hunters per party, the number of hunters per blind, and the hours hunted per man-day are all subject to variation. We chose a method based on birds lost per 100 shots fired because data from all field tests that have been conducted could be handled in that fashion. We also would have presented crippling losses as birds lost per hunting party per day, per blind per day (blind-day), per man-day of hunting, or other expressions of hunting effort if these figures could have been calculated for all studies cited in Table 8; this was not the case. The only calculations possible for all five studies were birds lost per shot fired and birds lost per bird bagged; we chose the former method for the reasons cited above.

The results of several intensive field-shooting experiments that compare the effectiveness of lead and steel loads are shown in Table 8. These data indicate crippling losses in ducks and geese under actual shooting conditions in the field. No statistically significant differences were found among the three duck studies in cripples (birds lost) per shot fired for steel and lead shot. The smallest differences between lead and steel loads occurred in Missouri (Humburg et al. 1982) and in Michigan (Mikula et al. 1977), and the greatest difference was found in Louisiana (Hebert et al. 1984).

Viewing these crippling losses within the context of the national bag of ducks and recent crippling

Table 8.—Intensively monitored field-shooting experiments comparing lead and steel loads for waterfowl hunting.

Area	Investigator	Shot Load	Birds Bagged/ 100 Shots	Shots/ Bird Bagged	Birds Lost/100 Shots: Number	Birds Lost/100 Shots: % ± steel compared with lead
Union County Illinois	Anderson & Sanderson 1979[a]	#2 lead	19.5	5.1	7.0	—
		#1 steel	17.9	5.6	5.4	-22.8
		BB steel	20.7	4.8	5.7	-18.6
Tule Lake California	Smith & Roster 1979[b]	lead	16.8	6.0	7.6	—
		steel	15.6	6.4	8.1	+6.6
Shiawasse Michigan	Mikula et al. 1977[c]	#4 lead	23.9	4.2	3.8	—
		#4 steel	18.6	5.4	3.6	-5.3
Schell-Osage Missouri	Humburg et al. 1982[c]	#4 lead (buffered)	17.7	5.6	4.2	—
		#4 lead	19.5	5.1	4.1	—
		#4 steel	17.4	5.7	4.4	+7.3[d]
		#2 steel	17.8	5.6	3.9	-4.9[d]
Lacassine Louisiana	Hebert et al. 1984[c]	#6 lead	15.0	6.7	4.2	—
		#4 steel	10.6	9.4	4.8	+14.3

[a]Moderately large interior Canada geese.
[b]Smaller Canada geese.
[c]Ducks
[d]Compared with #4 unbuffered lead.

losses, however, provides a broader perspective. The national bag and crippling losses of ducks (data based largely on the use of lead shot) has averaged 12,810,600 ducks bagged per year and 2,729,000 ducks crippled (21.3 percent of the bag) for 1974-1983 (compiled from Carney et al. 1976, 1978, 1979, 1980, 1981, 1982, 1983, 1984; Schroeder et al. 1975; Sorensen et al. 1977). We can use these figures to estimate the magnitude of change that might be expected if steel shot were used for hunting ducks. Our estimates are based on the assumption that hunters will fire the same number of steel shot shells as they have of lead, an assumption that may not be valid because Anderson (1979) found that hunters fired more steel than lead shot shells. In addition, data in Table 8 suggest that hunters will bag fewer ducks with steel shot if the same number of shots are fired. In spite of these limitations, the data in Table 8 suggest that little or no difference between crippling losses with steel and lead shot would be found nationwide.

Based upon the data reported in the three duck shooting studies, the largest decrease in crippling losses that might be anticipated if steel shot were used is 5.3 percent (comparison of No. 4 lead with No. 4 steel in Michigan, Table 8). Data from the Missouri study comparing No. 4 steel with No. 4 lead loads suggest than an increase of crippled ducks (7.3 percent) would occur with steel. The largest increase in crippling losses from steel was found in the Louisiana study—14.3 percent.

Data from three comparable studies, therefore, suggest that the use of steel shot could produce changes in crippling losses that range from a relatively minor decrease in the number of crippled ducks to a relatively minor increase. Where within this range might we anticipate that losses from steel loads would occur if the use of steel shot were implemented across the nation?

On the basis of shots fired per duck bagged, Louisiana hunters fired more shots than hunters elsewhere, whether they used lead or steel (Table 8). Indeed, the number of lead shots fired per duck bagged in the Louisiana experiment (6.7) is above the national average of 6 (U.S. Fish and Wildlife Service 1976). Part of this difference may be aim error and part may be due to the greater difficulty of retrieving downed ducks in the high, dense marsh vegetation surrounding the shooting sites at Lacassine NWR (Hebert et al. 1984:392). Only a small proportion of wetlands in the United States possesses such vegetation. Michigan's Shiawassee River State Game Area (Mikula et al. 1972:443) and Missouri's Schell-Osage Wildlife Management Area consist of flooded timber and marsh. About 30 percent of Shiawassee also contains flooded cropland. Although no single typical shooting marsh exists, Shiawassee and Schell-Osage are more nearly representative of hunting habitats in the United States than is the Louisiana site. National estimates based on the increase (14.3 percent) of crippling losses shown for steel shot in the Louisiana study might well prove high, and estimates based on the more modest increase in crippling losses—or the decrease in crippling losses in Michigan and Missouri—might be more accurate.

The two field-shooting experiments with geese shown in Table 8 involved the moderately large interior Canada goose in Illinois (Anderson and Sanderson 1979) and smaller geese in Tule Lake, California (Smith and Roster 1979). Neither study reported significant differences between crippling rates for lead and steel shot. Extrapolating data from these two studies, we find that No. 1 steel shot would reduce crippling losses in large geese by 22.8 percent, and BB steel shot would reduce crippling losses by 18.6 percent. With midsized and smaller geese, steel shot would increase the current crippling loss by 6.6 percent. For all geese, steel shot would seem to reduce crippling losses slightly over the losses that might be expected with lead shot.

Again, viewing these losses against the national bag of geese and against recent crippling losses gives a clearer picture of what is at stake. Between 1974-1983, the national bag of geese averaged 1,757,000 per year (data based largely on the use of lead shot) with a crippling loss of 247,000 or 14.1 percent of the bag (compiled from Carney et al. 1976, 1978, 1979, 1980, 1981, 1982, 1983, 1984; Schroeder et al. 1975; and Sorensen et al. 1977). Of the total bag, about 930,000 geese are the size of interior Canadas and about 823,000 are smaller.

None of the tests reported here reveals how steel shot performs in the hands of expert hunters who know they are shooting steel and alter their gunning habits accordingly. If such tests were made, we expect that steel would perform significantly better in comparison with lead than it has in the "blind" tests reported here.

In the United States (1) average annual crippling losses for ducks, coots, geese, and all waterfowl species combined were lower after steel shot implementation (1979-1984) than before implementation (1971-1975); (2) the lowest crippling losses occurred in recent years (1980-1984) when both steel shot and lead shot were used; (3) the highest crippling losses took place in the earlier years (1971-1974) when only lead shot was used; (4) crippling losses have not increased with the increase in use of steel shot in recent years; and (5) the decrease in crippling losses is a long-term trend that began before the implementation of steel shot (Table 9, Figs. 7, 8).

Table 9.—Mean number of waterfowl crippled (knocked down but not retrieved) per 100 birds retrieved in the United States before, during, and after the implementation of nontoxic (steel) shot for waterfowl hunting. Data from U.S. Fish & Wildlife Service, Office of Migratory Bird Management, Administrative Reports, 1973-1985.

Species	Before (1971-1975)	During (1976-1978)	After (1979-1984)
Ducks	21.6	20.0	19.5
Coots	29.0	28.2	27.1
Geese	14.6	14.9	14.0
All species	*21.4*	*19.9*	*19.1*

Because of the long-term downward trend in crippling losses, a simple cause-and-effect relationship cannot be claimed between steel shot and the reduction in crippling losses in recent years. However, the data clearly demonstrate that the use of steel shot has not resulted in an increase in crippling losses in the national waterfowl population. If an effect is present, it is positive—that is, steel shot contributed to a reduction in crippling losses.

Effect of range. Early shooting experiments with soft steel and lead loads indicated that steel shot was deficient in killing power at ranges of 45.7 m (50 yards) and greater (Bellrose 1959: Table 29; Andrews and Longcore 1969: Fig. 1). More recent field tests with lead and improved steel shot, however, show little difference in killing power between the two loads at long ranges. Anderson and Sanderson (1979: Table 5) found steel shot as effective or better than lead for killing interior Canada geese at ranges >45.7 m (50 yards). Hunters shooting small-to-midsize geese at Tule Lake, California, crippled fewer geese at ranges >45.7 m (50 yards) with steel loads than with lead (Smith and Roster 1979:7). Mikula et al. (1977: Table 4) reported that at ranges >42.1 m (46 yards), hunters failed to retrieve 23 percent of the ducks hit with steel and 32 percent of those hit with lead. Humburg et al. (1982:124) concluded that bagging, crippling, and missing rates for ducks were similar for steel and lead loads at ranges of ≥36.6 m (40 yards). Although Hebert et al. (1984:394) found differences in lead and steel shot loads, distance was not a factor in the comparative effectiveness of the two loads for shooting ducks when all distances were combined. Steel loads, however, produced more cripples per shot and per blind-day than lead at ≤32 m (35 yards) but fewer cripples per shot and per blind-day than lead at >32 m (35 yards).

Gun damage. Almost half the hunters contacted in an Ohio opinion survey taken in 1978 believed that steel shot would damage their guns; 40 percent thought that steel shot would expand the choke and 36 percent thought it would scour the barrel (Smith and Townsend 1981:6). Although the likelihood of excessive gun barrel damage was disproved long ago, the notion lingers. Nearly a decade ago, the three major arms and ammunition companies stated that steel shot causes no significant reduction in the life of most modern full-choke shotguns (U.S. Fish and Wildlife Service 1976). Roster (1978[a]:6) received no reports or claims of barrel damage after 18,000 rounds of steel shot had been fired. The plastic shot cups prevent steel shot from eroding the barrel. Some choke expansion may occur in full-choked, thin-walled, soft steel shotguns, some Brownings of early vintage, and shotguns with sharp-angled or swadged full choke (Roster 1978[a]). Magnum lead loads also cause chokes to expand. When there is a minor choke expansion in modern guns, it is largely cosmetic. We find no evidence that steel loads adversely affect modern gun barrels, and barrel damage, therefore, is not a valid reason for refusing to use steel shot.

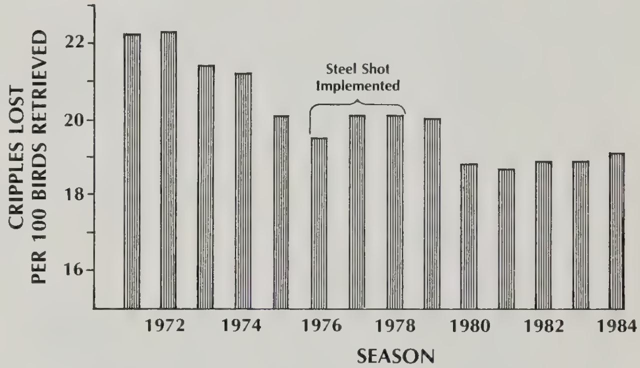

Figure 7.—Crippling rates of waterfowl (ducks, geese, and coots) in the United States, hunting seasons 1971-1984. Data from U.S. Fish and Wildlife Service, Office of Migratory Bird Management, Administrative Reports, 1973-1985.

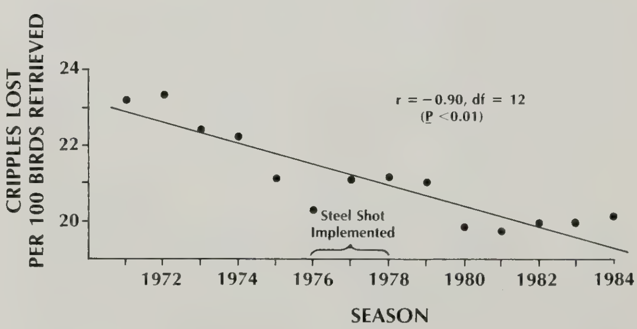

Figure 8.—Linear correlation between crippling rates of waterfowl (ducks, geese, and coots) in the United States and year, hunting seasons 1971-1984. Data from U.S. Fish and Wildlife Service, Office of Migratory Bird Management, Administrative Reports, 1973-1985.

Cost of shells. The spread in price between steel and lead shot is a deterrent to the use of steel by many hunters (National Wildlife Federation 1985[b]), but it is a reason more often cited in private than in public. The difference in cost seems to be not so much in the manufacturing of shot as in its retail markup.

One manufacturer's suggested retail prices for selected lead and steel shotgun shells are shown in Table 10 along with percentage comparisons of the costs of loads and shot sizes that are roughly equivalent. Three-inch 20-gauge lead and steel loads are priced essentially the same, but steel 2¾-inch 20-gauge shells are 27.2 percent more expensive than their lead counterpart. Twelve-gauge steel loads are priced from 7.3 to 25.2 percent higher than approximately equivalent lead loads. Steel loads for 10-gauge shells are listed at 9.3 percent less than comparable lead loads. In practice, however, prices at stores may show larger differences because dealers commonly mark down lead loads and seldom reduce the price of steel loads. Some shooters reload for economy (although some derive pleasure from the mere act of reloading), and components for reloading steel shot are now readily available from at least one reliable source.

Shells, however, make up a minor part of the overall expense of waterfowl hunting. The average duck hunter kills six ducks with 36 shots each year (U.S. Fish and Wildlife Service 1976). At the price differentials noted above, the average duck hunter would spend about $4.50 more on steel than on lead shot shells per hunting season. If a 10-gauge gun were used, a saving of $4.86 would accrue. Since the mid-1930s, waterfowl hunters have responsibly supported funding for wetlands through federal and state duck stamp programs and through contributions to Ducks Unlimited. We conclude, therefore, that the slightly higher cost of steel loads should not be a deterrent to their use, particularly in view of the dwindling populations of ducks and the keen interest of waterfowlers in perpetuating their sport. Furthermore, as the production of steel shot shells increases, costs and consequently prices should decline.

Ballistics. Lead and steel loads differ ballistically. Surprising to many ballisticians, however, steel shot has been found to possess a quality of form retention that makes for a better pattern and a shorter shot string than soft lead. Brister (1976:296,300) pointed out that lead shot pellets become more deformed from impact among the pellets as they pass down the gun barrel than do steel pellets. Steel, which is harder than regular lead shot, resists deformation from pellet impact and, therefore, leaves the barrel in a more nearly spherical form. In addition, steel pellets are more nearly round and are more uniform than lead pellets before they are fired. Because of the larger proportion

Table 10.—Percentage difference in suggested retail prices (effective 2 January 1985) for selected lead and steel shotgun shells.[a]

Gauge	Lead or Steel (L or S)	Shell Length (in)	Oz Shot	Shot Sizes	Suggested Retail Price	% Difference In Cost: Steel vs Lead
20	L	2¾	1	4-5-6-7½-8-9	$11.00	—
20	S	2¾	¾	4-6	14.00	+27.2
20[b]	L	2¾	1⅛	4-6	14.60	—
20	L	2¾	1⅛	4-6-7½	13.30	—
20	L	3	1¼	2-4-6-7½	15.05	—
20	S	3	1	4	15.00	−0.3
12	L	2¾	1¼	BB-2-4-5-6-7½-8-9	12.50	—
12	S	2¾	1⅛	2-4-6	15.65	+25.2
12[c]	L	2¾	1½	BB-2-4-5-6	16.25	—
12[b]	L	2¾	1½	BB-2-4-6	18.45	—
12	S	2¾	1¼	BB-1-2-4	17.10	−7.3
12[d]	L	3	1⅞	BB-2-4	19.40	—
12	S	3	1⅜	BB-1-2-4	20.95	+8.0
12[b,d]	L	3	1⅞	BB-2-4-6	20.80	—
10	L	3½	2	BB-2-4	29.00	—
10	S	3½	1⅝	BB-2	26.30	−9.3
10[b]	L	3½	2¼	BB-2-4	31.10	—

[a]Data from William F. Stevens, Manager, Conservation Activities, Federal Cartridge Corporation.

[b]Federal Premium brand ammunition.

[c]High-power magnum.

[d]Anderson (1980) reported that duck hunters on public areas in Illinois in 1979 who used 12-gauge, 3-inch lead shot shells relied on 1⅞ oz shot 60 percent of the time and 1⅝ oz shot 33 percent of the time.

of steel pellets that remain in spherical form, the steel charge is more compact and has fewer empty spaces and "flyers" in its pattern than is the case for the softer lead shot.

To overcome the deficiency of soft lead, ammunition manufacturers have increased the antimony content and reduced shot-column interstices by the addition of a filler, which buffers the collisions between pellets during their passage through the gun barrel. Buffered lead shot loads are comparable in patterning to steel loads, but in most cases cost more than comparable steel loads (Table 10).

Steel is lighter than lead, but the consequent downrange energy loss can be compensated for by using a steel pellet one or two sizes larger than that used in lead and by increasing muzzle velocity. Because of the greater velocity and the greater retention of form, however, many hunters have learned that steel shot in the same sizes as their favorite lead loads perform satisfactorily.

More steel than lead pellets occur in a given weight. Roster (1978[a]) found similar numbers of shot pellets in the following paired charges: 1⅛ oz steel-1½ oz lead, 1¼ oz steel-1¾ oz lead, 1⅜ oz steel-1⅞ oz lead, 1½ oz steel-2⅛ oz lead, and 1⅝ oz steel-2¼ oz lead.

Evidence suggests that because of the tighter pattern of both steel and buffered lead loads, the ability to aim in relation to choke may well have a bearing on bag/cripple results. This suggestion may be supported by the results of shooting tests at the Schell-Osage Wildlife Management Area, Missouri, where No. 4 lead performed better than No. 4 buffered lead (Table 8). Both the shorter shot string and the tighter pattern of steel contribute to more hits on a target or to a "clean" miss. These factors may explain the generally superior performance of steel shot over lead for hunting Canada geese. Because of the tighter patterns of steel and buffered lead loads, a modified or improved cylinder choke is recommended rather than a full choke.

Discussion

Most of the waterfowl that die from lead poisoning do so after the hunting season. The history of mass die-offs (Bellrose 1959: Table 1) as well as the chronology of specimens sent to the National Wildlife Health Laboratory (Table 11) substantiate this conclusion. Without the activities of hunters to drive them from the feed beds and with a steadily diminishing food

Table 11.—Temporal distribution of waterfowl dead of lead poisoning and received at the National Wildlife Health Laboratory, Madison, Wisconsin, 1975-79.[a]

Species or Group	Jan No.	Jan %	Feb No.	Feb %	Mar No.	Mar %	Apr No.	Apr %	May No.	May %	Jun No.	Jul No.	Jul %
Blue and													
Snow Goose	5	5.4	8	8.7	9	8.7	1	1.1	1	1.1	—	—	—
Diving Ducks	4	7.7	1	1.9	8	15.4	15	28.8	2	3.8	0	4	7.7
Puddle Ducks	143	33.5	32	7.5	73	17.1	7	1.6	0	—	0	0	—
Canada Goose	160	27.5	161	27.7	73	12.6	1	0.2	0	—	0	0	—
Whistling Swan	2	1.6	121	93.4	1	0.8	2	1.6	3	2.3	0	0	—
Mallard	106	44.2	14	5.8	25	10.4	4	1.7	0	—	0	0	—
Total	*420*	*27.6*	*337*	*22.2*	*189*	*12.4*	*30*	*2.0*	*6*	*0.4*	*0*	*4*	*0.3*

[a]Dr. Milton B. Friend, personal communication, 17 April 1985.

Table 11.—continued.

Species or Group	Aug No.	Aug %	Sep No.	Sep %	Oct No.	Oct %	Nov No.	Nov %	Dec No.	Dec %	Total
Blue and											
Snow Goose	—	—	—	—	—	—	29	31.5	39	42.4	92
Diving Ducks	12	23.1	2	3.8	2	3.8	1	1.9	1	1.9	52
Puddle Ducks	7	1.6	8	1.9	13	3.0	14	3.3	130	30.4	427
Canada Goose	0	—	1	0.2	62	10.7	88	15.1	35	5.9	581
Whistling Swan	0	—	0	—	0	—	0	—	0	—	129
Mallard	2	0.8	7	2.9	8	3.3	7	2.9	67	27.9	240
Total	*21*	*1.4*	*18*	*1.2*	*85*	*5.6*	*139*	*9.1*	*272*	*17.9*	*1,521*

supply, waterfowl are attracted back to shotover areas, where spent shot is abundant. Waterfowl undoubtedly ingest more shot after than during the hunting season. As a result of this seasonal variation in ingestion rates, calculations based on gizzards from ducks shot by hunters underestimate the incidence of shot ingestion. Moreover, lead-poisoned birds are more likely to be killed by hunters because these birds are in an already weakened condition. After the hunting season, such birds would, in all probability, fall victim to predators other than man, but in either case their deaths are at least indirectly a result of lead poisoning.

The proclivity of a given waterfowl species to ingest shot pellets is based on its feeding habits and habitats. Some species consistently ingest more pellets than others. After a shot has been ingested, however, many factors determine its lethality. The current diet of the bird is the single most important factor: the intake of protein, calcium, and phosphorus reduce lead toxicosis. The volume of food consumed and its rate of passage through the gastrointestinal tract help to prevent the absorption of intestinal lead. Similarly, the volume of grit ingested and its rate of passage play a role in the elimination of lead.

These variables are compounded by those introduced by the deposition and withdrawal of lead from the skeletal system. The more active the calcium metabolism of the bird, the more active the deposition or transportation of lead, or both. Consequently, breeding females and young ducks may have a greater resistance to lead poisoning.

In addition to the outright lethality of lead, its sublethal effects have only recently become known. Dieter and Finley (1979) found that biochemical lesions in the brains of mallards dosed with one No. 4 lead shot pellet occurred earlier than did such external evidence of the disease as wing droop and vent staining.

In establishing the role that steel shot might play in the welfare of waterfowl populations, we need to evaluate further the differential effect of lead toxicosis on the sexes and the effect on population dynamics of seasonal losses.

Two hypotheses have been advanced regarding the effect of gender on lead poisoning in ducks. In the first, lead has no differential effect on males and females. In the second, lead has a greater effect upon females. Evidence supporting the first hypothesis comes from White and Stendell (1977:474), who found no significant difference between lead levels in male and female wing bones of mallards, black ducks, and pintails. Support for the assumption that females suffer more from the effects of lead than males is found in the higher mortality rate (approximately 25 percent higher) of banded female mallards dosed with one shot compared with similarly dosed males (Fig. 6). Moreover, weight loss among penned game-farm female mallards dosed with lead was greater than that among drakes (Fig. 5). In a series of experiments with penned mallards, Jordan and Bellrose (1951:21) found a mortality rate twice as high among hens as drakes except during spring. Only during a brief period from late February through March were mallard females less susceptible to lead than males (Bellrose 1959:276).

As previously noted, losses due to crippling occur throughout the hunting season, but losses from lead poisoning are most extensive after hunting ceases. Of the many die-offs of waterfowl cited by Bellrose (1959: Table 1), only two occurred late in the hunting season; the others were reported still later in the winter and spring. Moreover, the chronology of lead-poisoned specimens received at the National Wildlife Health Laboratory, Madison, Wisconsin, from throughout the nation documents that losses largely occur from December through February (Table 11). An examination of waterfowl population dynamics suggests that the late winter, early spring lead poisoning losses have a more important influence on potential production than do the crippling losses that occur during the hunting season. Birds lost from one factor, hunting, for example, are replaced by birds that survived the hunting season and continue to resist natural causes of death because more habitat niches were made available to them. The earlier in the fall that mortality occurs, the better the opportunity for the remaining birds to survive and breed. The nearer to the breeding season that a bird survives, the more likely it will achieve breeding status. Because most losses from lead poisoning occur just prior to the breeding season, they affect the breeding population more severely than an equal number of crippling losses during the previous fall.

The potential impact of lead poisoning on fall duck populations is related to the relative abundance of the most vulnerable species (Table 12): mallard, black duck, mottled duck, pintail, canvasback, redhead, and ring-necked duck. Together, these species make up 43 percent of the continental game duck population and, ironically, are the species that have been of most concern to conservationists in the past decade.

We believe more is to be gained by the judicious use of steel shot than by the continued blanket shooting of lead shot. Although we recognize that the use of steel shot would have limited impact in some habitats and on certain species of waterfowl, its use would be of considerable benefit in many habitats and on several species. In addition, we recognize that in some areas botulism, fowl cholera, and duck virus enteritus cause extensive waterfowl mortality; however, merely because large numbers of ducks in some areas die from other diseases does not justify ignoring losses due to lead. Deaths due to lead poisoning, unlike deaths caused by other diseases, can be eliminated by regulation. If other diseases could be eliminated by regulations, we would urge their implementation.

Table 12.—Estimated fall duck population and relative abundance of the principal species in recent years based on breeding populations and number of young equal to number of adults.

Species	Fall Population 1970-79[a]	Percent
Mallard	21,200,000	21.3
Black Duck	2,800,000	2.8
Mottled Duck[b]	200,000	0.2
Pintail	14,000,000	14.1
Wood Duck	6,400,000	6.4
Wigeon	7,000,000	7.0
Gadwall	4,000,000	4.0
Green-winged Teal	6,300,000	6.3
Blue-winged Teal	12,300,000	12.4
Shoveler	4,400,000	4.4
Canvasback	1,200,000	1.2
Redhead	1,700,000	1.7
Ring-necked Duck	1,900,000	1.9
Lesser Scaup	16,000,000	16.1
Total	*99,400,000*	*99.8*

[a]Continental estimate of breeding ducks from draft copy of North American waterfowl management plan, November 1985 (Canadian Wildlife Service and U.S. Fish and Wildlife Service: Table 1).

[b]Data for the mottled duck from U.S. Fish and Wildlife Service winter inventories, 1980-1985, × 2.

The partial changeover from lead to steel that has occurred has already had a measurable impact on lead poisoning. Although the use of steel shot has been limited in time and place, a surprisingly large proportion of analyzed gizzards show steel replacing lead in shot ingestion (Table 13). Thus, the regulated use of steel shot would appear to have the potential for promptly reducing mortality due to lead poisoning.

Although the extent of opposition to steel shot by waterfowl hunters has not been documented, strong and well-organized opposition continues (Arnett 1985:7; Kendzie 1985). Recently, Feierabend (1985) summarized the legal challenges to nontoxic (steel) shot regulations and found that all decisions, including a recent case in Federal Court (National Wildlife Federation 1985[a]), have gone in favor of steel shot. On the other hand, as late as the 1981 waterfowl hunting season, 53.9 percent of Illinois waterfowl hunters replying to a questionnaire had never used steel shot for hunting waterfowl; however, 47.5 percent of these hunters believed that lead poisoning was a problem "on some, many, or all areas" and 51.9 percent responded that they would voluntarily use steel shot in some areas if asked to do so by the Illinois Department of Conservation (Anderson 1983:1).

Although disagreement continues regarding the extent of lead poisoning in waterfowl, most biologists, wildlife managers and administrators, and waterfowl hunters agree that appreciable mortality results. With the continuing decline in quality and quantity of nest habitat for waterfowl and the consequent declines in continental waterfowl populations (the length of the

Table 13.—Comparative ingestion of lead and steel shot by waterfowl in states and other regions, 1974-1984.

Flyway/State	Years	Investigator	Lead No.	Lead %	Steel No.	Steel %	Both No.	Both %	% with lead only[a]
Atlantic	No record								
Mississippi									
Michigan	1977-79	Nelson & Johnson 1980	416	3.4	416	3.4	14	0.1	49.2
Ohio	1977-79	Bednarik & Shieldcastle 1980	230	2.7	61	0.7	—[b]	—	79.0
Indiana	1977-80	Sporre & Blevins 1981	150	6.7	50	2.2	15	0.7	69.8
Missouri	1977-81	Humburg & Babcock 1982	719	3.8	319	1.7	56	0.3	65.7
Illinois	1979-82	Anderson 1982	525	5.1	108	1.0	23	1.2	80.0
Central									
Texas	1981-83	Texas Parks & Wildl. Dept. 1982, 1983	753	6.1	395	3.2	288	2.3	52.4
Pacific									
Oregon[c]	1974-83	Vendshus n.d.	127	16.9	52	6.9	55	7.3	54.3
California	1979-80	Moore & King 1980	726	9.0	34	0.4	—[b]	—	95.5
United States	1974-75	White & Stendell 1977	244	8.9	74	2.7	15	0.5	73.3

[a]Percent of gizzards with lead pellets only.

[b]Not classified.

[c]Sauvie Island only.

1985 waterfowl hunting seasons were reduced by the U.S. Fish and Wildlife Service in response to lower populations), a conservative approach to the problem of lead poisoning in waterfowl seems prudent. It sometimes seems as if advocates of steel shot are being asked to demonstrate that steel is "better" than lead before its use is acceptable. Instead, we should focus on the effects of the use of lead and steel shot on ducks and geese—the mortality rate from lead poisoning and crippling by lead shot versus the mortality rate from crippling by steel shot.

References Cited

Adler, F.E.W. 1942. The problem of lead poisoning in waterfowl. Wisconsin Conservation Bulletin 7(9):5-7.

______. 1944. Chemical analyses of organs from lead-poisoned Canada geese. Journal of Wildlife Management 8(1):83-85.

Adrian, W.J., and M.L. Stevens. 1979. Wet versus dry weights for heavy metal toxicity determinations in duck liver. Journal of Wildlife Disease 15:125-126.

Amundson, T. Lead poisoning of Canada geese in east-central Wisconsin: a case study. *In* Lead Poisoning in Wild Waterfowl: A Workshop. 3-4 March 1984. Wichita, Kans. International Association Fish and Wildlife Agencies and the Cooperative Lead Poisoning Control Information Program. (in press)

Anderson, D.R. 1975. Population ecology of the mallard. V. Temporal and geographic estimates of survival, recovery, and harvest rates. U.S. Fish and Wildlife Service Resource Publication 125. 110 p.

Anderson, H.G. 1959. Food habits of migratory ducks in Illinois. Illinois Natural History Survey Bulletin 27(4):289-344.

Anderson, W.L. 1975. Lead poisoning in waterfowl at Rice Lake, Illinois. Journal of Wildlife Management 39(2):264-270.

______. 1979. Hunting efficiency and crippling losses of waterfowl on public areas in Illinois: 1978 versus pre-steel shot years. Illinois Department of Conservation. 8 p. + 9 tables.

______. 1980. Characteristics of shotgun shells used for hunting ducks in Illinois. Illinois Department of Conservation. 8 p.

______. 1982. Lead poisoning in Illinois waterfowl, 1979 through January 1982: ingested shot, blood chemistry, and die-offs. Illinois Department of Conservation Periodic Report 35.

______. 1983. Illinois waterfowl harvest, hunter activity, and attitudes toward shooting hours, lead poisoning, and steel shot in 1981. Illinois Department of Conservation Periodic Report 39.

______, and G.C. Sanderson. 1979. Effectiveness of steel shot in 3-inch, 12-gauge shells for hunting Canada geese. Wildlife Society Bulletin 7(4):213-220.

______, and P.A. Brewer. 1980. Incidence of ingested shotgun pellets in Illinois mallards as determined by radiology: 1979 season. Illinois Department of Conservation. 15 p.

______, and S.P. Havera. 1985. Blood lead, protoporphyrin, and ingested shot for detecting lead poisoning in waterfowl. Wildlife Society Bulletin 13(1):26-31.

Andrews, R., and J.R. Longcore. 1969. The killing efficiency of soft iron shot. North American Wildlife and Natural Resources Conference Transactions 34:337-345.

Arnett, G.R. 1985. Sincerely, GRA. American Rifleman 133:7.

Bagley, G.E., and L.N. Locke. 1967. The occurrence of lead in tissues of wild birds. Bulletin of Environmental Contamination and Toxicology 2(5):297-305.

Baker, J.L., and R.L. Thompson. 1979. Shot ingestion by waterfowl on national wildlife refuges in Florida. Southeastern Association of Fish and Wildlife Agencies Annual Conference Proceedings 32:256-262.

Barngrover, L. 1977. Evaluation of Nevada's waterfowl for ingested lead shot: completion report. Federal Aid Project W-48-8, R XV:4. Nevada Department of Fish and Game. Reno, Nev. 17 p.

Barrett, M.W., and L.H. Karstad. 1971. A fluorescent erythrocyte test for lead poisoning in waterfowl. Journal of Wildlife Management 35(1):109-119.

Bates, F.Y., D.M. Barnes, and J.M. Higbee. 1968. Lead toxicosis in mallard ducks. Wildlife Disease Association Bulletin 4(4):116-125.

Bazell, R.J. 1971. Lead poisoning: zoo animals may be the first victims. Science 173(3992):130-131.

Bednarik, K.E., and M. Shieldcastle. 1980. Analysis of a three-year (1977-1979) shot ingestion study in Ohio waterfowl. Ohio Department of Natural Resources Wildlife Inservice Note 435. 27 p.

Beer, J.V., and P. Stanley. 1965. Lead poisoning in the Slimbridge wildfowl collection. The Wildfowl Trust Annual Report 16:30-34.

Bellrose, F.C. 1951. Effects of ingested lead shot upon waterfowl populations. North American Wildlife Conference Transactions 16:125-133.

______. 1953. A preliminary evaluation of cripple losses in waterfowl. North American Wildlife Conference Transactions 18:337-360.

______. 1955. A comparison of recoveries from reward and standard bands. Journal of Wildlife Management 19(1):71-75.

______. 1959. Lead poisoning as a mortality factor in waterfowl populations. Illinois Natural History Survey Bulletin 27(3):235-288.

______. 1964. Spent shot and lead poisoning. Pages 479-485 *in* J.P. Linduska, ed., Waterfowl tomorrow. U.S. Department of the Interior, Bureau of Sport Fisheries and Wildlife, Fish and Wildlife Service, U.S. Government Printing Office, Washington, D.C. 770 p.

______. 1975. Impact of ingested lead pellets on waterfowl. Pages 163-167 *in* First International Waterfowl Symposium. 4-6 February 1975. St. Louis, Mo. Ducks Unlimited. 224 p.

______. 1976. Ducks, geese and swans of North America. Stackpole Books, Harrisburg, Pa. 544 p.

Bishop, R.A. 1972-1973. Waterfowl lead poisoning studies at Forney's Lake, Ia. Iowa Academy of Science Proceedings 79:12-13.

Brister, B. 1976. Shot gunning: the art and the science. Winchester Press, New York. 321 p.

Carney, S.M., M.F. Sorenson, and L.D. Schroeder. 1976. Waterfowl harvest and hunter activity in the United States during the 1975 hunting season. Administrative Report, 6 July 1976. U.S. Fish and Wildlife Service. Laurel, Md. 27 p.

______, ______, and E.M. Martin. 1978. Waterfowl harvest and hunter activity in the United States during the 1977 hunting season. Administrative Report, 9 June 1978. U.S. Fish and Wildlife Service. Laurel, Md. 27 p.

______, ______, and ______. 1979. Waterfowl harvest and hunter activity in the United States during the 1978 hunting season. Administrative Report, 21 June 1979. U.S. Fish and Wildlife Service. Laurel, Md. 27 p.

______, ______, and ______. 1980. Waterfowl harvest and hunter activity in the United States during the 1979 hunting season. Administrative Report, 8 July 1980. U.S. Fish and Wildlife Service. Laurel, Md. 27 p.

______, ______, and ______. 1981. Waterfowl harvest and hunter activity in the United States during the 1980 hunting season. Administrative Report, 14 July 1981. U.S. Fish and Wildlife Service. Laurel, Md. 27 p.

______, ______, and ______. 1982. Waterfowl harvest and hunter activity in the United States during the 1981 hunting season. Administrative Report, 8 June 1982. U.S. Fish and Wildlife Service. Laurel, Md. 27 p.

______, ______, and ______. 1983. Waterfowl harvest and hunter activity in the United States during the 1982 hunting season. Administrative Report, 21 June 1983. U.S. Fish and Wildlife Service. Laurel, Md. 27 p.

______, ______, and ______. 1984. Waterfowl harvest and hunter activity in the United States during the 1983 hunting season. Administrative Report, 27 June 1984. U.S. Fish and Wildlife Service. Laurel, Md. 27 p.

Childress, D. 1985. Unpublished Reports. Montana Department Fish, Wildlife, and Parks. Helena, Mont.

Chupp, N.R., and P.D. Dalke. 1964. Waterfowl mortality in the Coeur D'Alene River Valley, Ida. Journal of Wildlife Management 28(4):692-702.

Coburn, D.R., D.W. Metzler, and R. Treichler. 1951. A study of absorption and retention of lead in wild waterfowl in relation to clinical evidence of lead poisoning. Journal of Wildlife Management 15(2):186-192.

Deuel, B. 1985. Environmental lead dosing of northern pintails in California. California Fish and Game 7:125-128.

Dieter, M.P. 1979. Blood delta-aminolevulinic acid dehydratase (ALAD) to monitor lead contamination in canvasback ducks (*Aythya valisineria*). Pages 177-191 *in* Animals as monitors of environmental pollutants. National Academy of Science, Washington, D.C. 421 p.

______, and M.T. Finley. 1979. Delta-aminolevulinic acid dehydratase enzyme activity in blood, brain, and liver of lead-dosed ducks. Environmental Research 19:127-135.

Due, L. 1985. Unpublished Table. Oklahoma Department Wildlife Conservation, Oklahoma City, Okla.

Elder, W.H. 1950. Measurement of hunting pressure in waterfowl by means of X-ray. North American Wildlife Conference Transactions 15:490-503.

Esslinger, C.G. 1979. Continued availability of lead shot on a public goose hunting area in southern Illinois. M.A. Thesis. Southern Illinois University, Carbondale, Ill. 36 p.

Euliss, N.H., Jr. 1984. The feeding ecology of pintail and green-winged teal wintering on Kern National Wildlife Refuge. M.S. Thesis, Humboldt State University, Arcata, Calif. 188 p.

Feierabend, J.S. 1985. Legal challenges to nontoxic (steel) shot. Southeastern Association of Fish and Wildlife Agencies Annual Conference Proceedings. (in press)

Finley, M.T., and M.P. Dieter. 1978. Influence of laying on lead accumulation in bone of mallard ducks. Journal of Toxicology and Environmental Health 4:123-129.

______, M.P. Dieter, and L.N. Locke. 1976. Lead in tissues of mallard ducks dosed with two types of lead shot. Bulletin of Environmental Contamination and Toxicology 16(3):261-269.

Forbes, R.M., and G.C. Sanderson. 1978. Lead toxicity in domestic animals and wildlife. Pages 225-277 *in* J.O Nriagu, ed., The biogeochemistry of lead in the environment. Part B: biological effects. Elsevier/ North Holland Biomedical Press, Amsterdam, New York, and Oxford. 397 p.

Fowler, R., and G. Simpson. 1984. Occurrence of lead and steel shot in the gizzards of Canada geese and mallards in the nontoxic shot zone of South Dakota in fall and winter, 1983. South Dakota Department Game, Fish, and Parks, Wildlife Division, Game Report 84-2. Pierre, S. Dak. 5 p.

Fredrickson, L.H., T.S. Baskett, G.K. Brakhage, and V.C. Cravens. 1977. Evaluating cultivation near duck blinds to reduce lead poisoning hazard. Journal of Wildlife Management 41(4):624-631.

Funk, H.D. 1974. Lead shot investigations. Gizzard collections. Mallard, scaup, pintails. 1973-74. Presented at Central Flyway Technical Section Meeting, 14 March 1974. 9 p.

Gabig, J. 1984. Ingestion frequency of shot found in waterfowl gizzards collected during the 1983 season in Nebraska–includes a summary of years 1976-83. Federal Aid P-R Project W-15-R-C5. Nebraska Game and Parks Commission, Lincoln, Nebr. 6 p.

Gelston, W.L., and J. Stuht. 1975. Successful treatment of a mute swan for lead poisoning. Jack-Pine Warbler 53(4):156-158.

Godin, A.J. 1967. Test of grit types in alleviating lead poisoning in mallards. U.S. Fish and Wildlife Service Special Scientific Report. Wildlife 107. 9 p.

Grinnell, G.B. 1894. Lead poisoning. Forest and Stream 42(6):117-118.

______. 1901. American duck shooting. Forest and Stream Publishing Company, New York. 627 p.

Hawkins, A.S. 1965. The lead poisoning problem in the four flyways. Pages 21-60 *in* Wasted Waterfowl. Mississippi Flyway Council Planning Committee Report. 72 p.

Hebert, C.E., V.L. Wright, P.J. Zwank, J.D. Newsom, and R.L. Kasul. 1984. Hunter performance using steel and lead loads for hunting ducks in coastal Louisiana. Journal of Wildlife Management 48(2):388-398.

Hough, E. 1894. Lead-poisoned ducks. Forest and Stream 42(6):117.

Humburg, D.D., and K.M. Babcock. 1982. Lead poisoning and lead/steel shot: Missouri studies and a historical perspective. Missouri Department of Conservation Technical Report Terrestrial Series 10. Jefferson City, Mo. 23 p.

______, D. Graber, S. Sheriff, and T. Miller. 1983. Estimating autumn-spring waterfowl nonhunting mortality in north Missouri. North American Wildlife and Natural Resources Conference Transactions 48:241-256.

______, S.L. Sheriff, P.H. Geissler, and T. Roster. 1982. Shotshell and shooter effectiveness: lead vs. steel shot for duck hunting. Wildlife Society Bulletin 10(2):121-126.

Illinois Department of Conservation. 1977. Goose die-off ends at state refuges. Outdoor Highlights 5(5):5.

Irby, H.D., L.N. Locke, and G.E. Bagley. 1967. Relative toxicity of lead and selected substitute shot types to game-farm mallards. Journal of Wildlife Management 31(2):253-257.

Irwin, J.C. 1977. The influence of diet on the pathogenesis of lead poisoning in waterfowl. Ph.D. Dissertation. University of Guelph, Guelph, Ontario. 384 p.

______, L.H. Karstad, and R.G. Thomson. 1974. Progress report on lead poisoning in waterfowl, March 1973 to March 1974. Department of Pathology, University of Guelph, Guelph, Ontario. 66 p.

Johnson, M.A. 1985. The incidence of lead shot in mallard, pintail and scaup gizzards. Progress Report Project No. W-67-R-14, 1973. North Dakota Game and Fish Department. Bismarck, N.. Dak. 1 p.

Jordan, J.S., and F.C. Bellrose. 1950. Shot alloys and lead poisoning in waterfowl. North American Wildlife Conference Transactions 15:155-170.

______, and ______. 1951. Lead poisoning in wild waterfowl. Illinois Natural History Survey Biological Notes 26. 27 p.

Kendzie, A. 1985. Sportsmen seek answer to lead shot/steel shot controversy. National Rifle Association Institute for Legislative Action, Washington, D.C. 2 p.

Koranda, J., K. Moore, M. Stuart, and C. Conrado. 1979. Dietary effects of lead uptake and trace element distribution in mallard ducks dosed with lead shot. Lawrence Livermore Laboratory Report. 39 p.

Leach, H.R. 1980. Hunter acceptance of steel shot, Delevan National Wildlife Refuge and Grizzly Island Wildlife Area, 1979-80. California Department of Fish and Game. Unpublished memorandum. 2 p. + appendix.

Locke, L.N., and G.E. Bagley. 1967. Case report. Coccidiosis and lead poisoning in Canada geese. Chesapeake Science 8(1):68-69.

______, ______, and H.D. Irby. 1966. Acid-fast intra-nuclear inclusion bodies in the kidneys of mallards fed lead shot. Wildlife Disease Association Bulletin 2(4):127-131.

______, ______, and L.T. Young. 1967. The ineffectiveness of acid-fast inclusions in diagnosis of lead poisoning in Canada geese. Wildlife Disease Association Bulletin 3(4):176.

Longcore, J.R., R. Andrews, L.N. Locke, G.E. Bagley, and L.T. Young. 1974. Toxicity of lead and proposed substitute shot to mallards. U.S. Fish and Wildlife Science, Special Scientific Report. Wildlife No. 183. 23 p.

______, P.O. Corr, and H.E. Spencer, Jr. 1982. Lead shot incidence in sediments and waterfowl gizzards from Merrymeeting Bay, Me. Wildlife Society Bulletin 10(1):3-10.

MacLennan, J.M. 1954. Pollution and waterfowl. First Ontario Industrial Waste Conference Proceedings.

Mikula, E.J., G.F. Martz, and C.L. Bennett, Jr. 1972. Field evaluation of three types of waterfowl hunting regulations. Journal of Wildlife Management 36(2):441-459.

______, ______, and L.A. Ryel. 1977. A comparison of lead and steel shot for waterfowl hunting. Wildlife Society Bulletin 5(1):3-8.

Montalbano, F., and T.C. Hines. 1978. An improved X-ray technique for investigating ingestion of lead by waterfowl. Southeastern Association of Fish and Wildlife Agencies Annual Conference Proceedings 32:364-368.

Moore, K.C., and J.T. King. 1980. Investigations of lead poisoning in waterfowl in California, October 1, 1979 to March 31, 1980. California Department Fish and Game, Resource Agency Wildlife Investigations Laboratory. 9 p.

Moser, J.D. 1983. The incidence of shot ingestion in New York waterfowl: results of a 1977-1982 gizzard collection study. Transactions Northeastern Section, The Wildlife Society 40:110-117.

Mudge, G.P. 1983. The incidence and significance of ingested lead pellet poisoning in British wildfowl. Biological Conservation 27(1983): 333-372.

______. 1984. Densities and settlement rates of spent shotgun pellets in British wetland soils. Environmental Pollution (Series B) 8 (1984): 299-318.

Munoz, R.V., Jr., and T.F. Gesell. 1976. Environmentally acquired lead in the laughing gull, *Larus atricilla*. Wildlife Disease Journal 12(2):139-142.

National Rifle Association. 1978. Interior steel shot data lacking. Reports from Washington 5(10):1, 4.

National Wildlife Federation. 1978. Steel shot program is necessary. Steel shot controversy goes to committee. Conservation Report No. 25:355, 379.

______. 1985[a]. In the United States District Court for the Eastern District of California. National Wildlife Federation, Plaintiff, vs. Donald Paul Hodel, Secretary of the Interior, et al., defendants.

______. 1985[b]. Myths and facts: Straight talk on lead poisoning and steel shot. National Wildlife Federation. 12 p.

Nelson, C., and M. Johnson. 1980. Lead-steel shot report: Michigan ingestion study (1976-1979). Michigan Department of Natural Resources, Wildlife Division Report 2852. Lansing, Mich. 5 p.

Niethammer, K.R., R.D. Atkinson, T.S. Baskett, and F.B. Samson. 1985. Metals in riparian wildlife of the lead mining district of southeastern Missouri. Archives of Environmental Contamination and Toxicology 14:213-223.

Ohi, G., H. Seki, K. Akiyama, and H. Yagyu. 1974. The pigeon, a sensor of lead pollution. Bulletin of Environmental Contamination and Toxicology 12(1):92-98.

Osmer, T.L.G. 1940. Lead shot: its danger to water-fowl. Science Monthly 50:455-459.

Pederson, G.B., and R.L. Pederson. 1983. Feeding ecology of pintails and mallards on Lower Klamath Marshes. U.S. Fish and Wildlife Service. Final Report. Contract No. 14-16-0001-79106. 89 p.

Phillips, J.C., and F.C. Lincoln. 1930. American waterfowl. Houghton Mifflin Company, Boston and New York. 312 p.

Quortrup, E.R., and J.E. Shillinger. 1941. 3,000 wild bird autopsies in western lake areas. American Veterinary Medical Association Journal 99(776):382-387.

Roscoe, D.E., S.W. Nielson, A.A. Lamola, and D. Zuckerman. 1979. A simple, quantitative test for erythrocytic protoporphyrin in lead-poisoned ducks. Wildlife Disease Journal 15(1):127-136.

Rosen, M., and R.A. Bankowski. 1960. A diagnostic technic and treatment for lead poisoning in swans. California Fish and Game 46(1):81-90.

Roster, T. 1978[a]. Steel shot: recent developments and gaining an understanding. Pages 221-237 *in* Western Section Wildlife Society Annual Meeting Transactions.

______. 1978[b]. Steel shot. Recent developments and gaining an understanding. Handloader Magazine (July-August):26-29, 44-48.

Sanderson, G.C., and J.C. Irwin. 1976. Effects of various combinations and numbers of lead:iron pellets dosed in wild-type captive mallards. Final Report. Contract No. 14-16-0008-914. U.S. Fish and Wildlife Service and Illinois Natural History Survey. 67 p. + 62 tables, 7 figs.

Scanlon, P.F., V.D. Stotts, R.G. Oderwald, T.J. Dietrick, and R.J. Kendall. 1980. Lead concentrations in livers of Maryland waterfowl with and without ingested lead shot present in gizzards. Bulletin of Environmental Contamination and Toxicology 25:855-860.

Schroeder, L.D., M.F. Sorensen, and S.M. Carney. 1975. Waterfowl harvest and hunter activity in the United States during the 1974 hunting season. Administrative Report, 7 July 1975. U.S. Fish and Wildlife Service. Laurel, Md. 28 p.

Shealy, P.M., J.D. Newsom, and P.J. Zwank. 1982. A lead toxicity study of waterfowl on Catahoula Lake and Lacassine National Wildlife Refuge. Submitted to Louisiana State University by Louisiana Cooperative Wildlife Research Unit. 77 p.

Shillinger, J.E., and C.C. Cottam. 1937. The importance of shot in waterfowl. North American Wildlife Conference Transactions 2:398-403.

Siegfried, W.R., P.G.H. Frost, E.P. Redelinghuys, and R.P. Van Der Merwe. 1972. Lead concentrations in the bones of city and country doves. South African Journal of Science 68(9):229-230.

Skidmore, J.F. 1964. Toxicity of zinc compounds to aquatic animals, with special reference to fish. Quarterly Review of Biology 39(3):227-247.

Smith, C.M. 1979. Lead shot ingestion of ducks colllected in Louisiana during the 1978-79 hunting season. Louisiana P-R Project W-29-26. 9 p.

______. 1980. Lead shot ingestion study of hunter bagged ducks in Louisiana during the 1979-1980 waterfowl season. Louisiana Department of Wildlife and Fisheries, Baton Rouge, La. 12 p.

______. 1981. Duck gizzard collection for examination for ingested lead shot: completion report. Federal Aid Project W-29, II:4

Louisiana Department of Wildlife and Fisheries, Baton Rouge, La. 12 p.

Smith, R.I., and T. Roster. 1979. Steel versus lead: results from the latest test. U.S. Fish and Wildlife Service Memorandum. 4 p.

______, and T.W. Townsend. 1981. Attitudes of Ohio hunters toward steel shot. Wildlife Society Bulletin 9(1):4-7.

Sorensen, M.F., S.M. Carney, and E.M. Martin. 1977. Waterfowl harvest and hunter activity in the United States during the 1976 hunting season. Administrative Report, 31 May 1977. U.S. Fish and Wildlife Service. Laurel, Md. 26 p.

Sporre, D.T., and W. Blevins. 1981. Incidence of lead and steel shot in Indiana waterfowl gizzards during three hunting seasons, 1977-78 through 1979-80. Pittman-Robertson Bulletin No. 12, Indiana Department of Natural Resources, Indianapolis, Ind. 12 p.

Stendell, R.C., R.I. Smith, K.P. Burnham, and R.E. Christensen. 1979. Exposure of waterfowl to lead: a nationwide survey of residues in wing bones of seven species, 1972-73. U.S. Fish and Wildlife Service Special Scientific Report. Wildlife No. 223. 12 p.

Stutzenbaker, C.D. 1984. The mottled duck: its life history, ecology, and management. Texas Parks and Wildlife Department. Second draft MS. Austin, Tex. 280 p.

______, K. Brown, and D. Lobpries. 1983. An assessment of the accuracy of documenting waterfowl die-offs in a Texas coastal marsh. Special Report. Federal Aid Project W-106-R, Texas Parks and Wildlife Department, Austin, Tex. 21 p.

Sullivan, J.D. 1980. Investigation of waterfowl lead shot ingestion in Arkansas. Arkansas Game and Fish Commission. Unpublished Report. 46 p.

Szymczak, M.R. 1978. Steel shot use on a goose hunting area in Colorado. Wildlife Society Bulletin 6(4):217-225.

______, and W.J. Adrian. 1978. Lead poisoning in Canada geese in southeast Colorado. Journal of Wildlife Management 42(2):299-306.

Texas Parks and Wildlife Department. 1982. 1981-82 Texas coastal waterfowl lead ingestion study. Unpublished Report. Austin, Tex. Pages 11-19.

______. 1983. 1982-83 analysis of Texas waterfowl gizzards for evidence of shotshell pellet ingestion. Unpublished Report. Austin, Tex. Pages 2-9.

Thul, J.E. 1985. Prevalence of ingested shot pellets in species of waterfowl collected in Florida during the hunting seasons of 1976-77 through 1983-84. Unpublished Report. 3 p.

Trainer, D.O., and R.A. Hunt. 1965. Lead poisoning of whistling swans in Wisconsin. Avian Diseases 9(2):252-264.

Trost, Robert E. 1981. Dynamics of grit selection and retention in captive mallards. Journal of Wildlife Management 45(1):64-73.

U.S. Fish and Wildlife Service. 1974. Proposed use of steel shot for hunting waterfowl in the United States. U.S. Department of the Interior, Draft Environmental Statement, DES 74-76. 79 p.

______. 1976. Final Environmental Statement: proposed use of steel shot for hunting waterfowl in the United States. U.S. Department of the Interior, U.S. Government Printing Office, Washington, D.C. 276 p.

______. 1978. Non-toxic shot regulations for waterfowl hunting, 1979-80. U.S. Department of the Interior, U.S. Fish and Wildlife Service. 10 p. + tables.

______. 1984. Investigation of waterfowl mortality at Suter National Wildlife Refuge, California. National Wildlife Health Laboratory Final Report, Lead Poisoning Monitoring Program. 11 p. + 3 figs.

Vendshus, S. [1984?]. Sauvie Island Wildlife Area. Lead shot report: 1983-84 waterfowl season. Unpublished Report. 12 p.

White, D.H., and R.C. Stendell. 1977. Waterfowl exposure to lead and steel shot on selected hunting areas. Journal of Wildlife Management 41(3):469-475.

Wildlife Society. 1984. Control of environmental contamination due to lead shot. Position Statement of The Wildlife Society. The Wildlifer 203:15.

Wills, D., and L.L. Glasgow. 1964. Lead shot on Catahoula Lake and its management implications. Southeastern Association of Fish and Game Commission Annual Conference Proceedings 18:90-105.

Winchester Group. 1974. A summary of comments on Department of Interior draft environmental statement DES 74-76: the proposed use of steel shot for waterfowl hunting in the United States. Olin Corporation, New Haven, Conn. 10 p.

Wobeser, G. 1969. Apparent favorable response of lead poisoning in a duck to treatment with a chelating agent. Wildlife Disease Association Bulletin 5(2):120.

Zwank, P.J., V.L. Wright, P.M. Shealy, and J.D. Newsom. 1985. Lead toxicosis in waterfowl on two major wintering areas in Louisiana. Wildlife Society Bulletin 13(1):17-26.

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