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Toxicity of Lead Shot to Wild Black Ducks and Mallards Fed Natural Foods

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Poisoning from ingested lead shot is a major problem affecting waterfowl in North America. The effects of lead ingestion on waterfowl are difficult to assess because the severity of lead toxicity is affected by differential susceptibility of species, the amount and size of shot ingested, and the diet consumed after lead ingestion (Bellrose 1981). Lead poisoning in waterfowl has been studied by several investigators including Irby et al. 1967, Longcore et al. 1974 a, b, Clemens et al. 1975, and Finley & Dieter 1978. However, most studies have used pen-raised birds that were fed corn, which enhances lead poisoning, or duck pellets which alleviate the effect of lead (Jordan 1968). Both the influence of the diet on lead toxicity and differential mortality for pen-raised and wild waterfowl have been documented (Bellrose 1959, Jordan 1968). However, the toxicity of lead to captive wild ducks fed a variety of foods simulating a natural diet has not been investigated. This study was designed to determine the toxicity of ingested lead shot to wild black ducks (Anas rubripes) and mallards (Anas platyrhynchos) fed natural foods normally consumed in the fall in New England (Martin & Uhler 1939, Cronan & Halla 1968). The objectives were to document mortality, weight loss, lead levels in the wing bones and liver, and shot retention for ducks dosed with various amounts of lead shot.

MATERIALS AND METHODS

Wild black ducks and mallards were trapped in Connecticut and Massachusetts in March, 1980. Ducks were randomly assigned to 1 of 4 groups of 10 birds (see Table 1 for numbers of each species). Each group was held in separate outdoor wire pens (8.5m long, 3.7m wide, and 2m high). A channeled stream (1.2m wide,

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0.3m deep) flowed through the width of the pens and provided drinking water and a swimming area. Sand and quartz grit was available from the stream.

Ducks were initially maintained on commercial duck pellets fed *ad libitum* for 4 weeks, then gradually switched to natural foods. On the natural diet, ducks were provided a 3:1 mixture of millet (*Setaria italica*): buckwheat (*Fagopyrum esculentum*) *ad libitum* at all times. Duckweed (*Lemna minor*) and eelgrass (*Zostera marina*) were provided *ad libitum* 3 days/week. Ducks were fed 4 fish per duck [mummichogs (*Fundulus heteroclitus*), killifish (*Fundulus* spp.), sheepshead minnows (*Cyprinodon variegatus*), or silversides (*Menidia* spp.)] and 4 sand shrimp per duck (*Crangon septemspinosa*) 1 day/week. Four blue mussels (*Mytilus edulis*) per duck were provided 2 days/week, except on 3 occasions when 2 crabs [lady crabs (*Ovalipes ocellatus*) or green crabs (*Carcinus maenas*)] per duck were substituted. Snails (*Physa* spp.) were associated with the duckweed and fed to the ducks in small quantities. Foods were provided in bowls or placed in the stream.

After ducks had been fed only natural foods for 4 weeks, lead shot was placed in the gizzards through a plastic tube. Groups were dosed with 0 (controls), 2, or 5 No. 6 shot ($\bar{x}$ weight=0.127g, N=20) on 20 May, 1980. Another group (5 shot repeated dose) received 1 shot on this date and then was dosed with 1 shot every third or fourth day for 2 weeks.

After dosage, ducks were weighed and fluoroscoped twice weekly to monitor weight change and shot retention. Observations were made daily for clinical symptoms of lead poisoning (i.e. green diarrhea, lethargy, ataxia, muscular paralysis). At the end of the 35 day experiment, survivors were sacrificed and gizzards, wings, and livers were removed. Ducks dying during the experiment were handled in the same manner. Gizzards were examined for shot, and wings and livers were sent to Raltech Scientific Services, Inc., Madison, Wisconsin, for lead analysis. Lead concentrations were determined by atomic absorption spectrophotometry and recorded in parts per million (ppm) on a wet weight basis. In this paper, a symptomatic duck is defined as a duck that survived the experiment but displayed at least one of the clinical symptoms of lead poisoning previously described and/or consistently lost weight for $\geq$2 weeks.

RESULTS AND DISCUSSION

Weight change, mortality, shot retention rates, and tissue lead levels were similar between sexes. Therefore, sexes were pooled to facilitate this presentation.

Mortality rates were similar between mallards and black ducks although a slightly higher percentage of black ducks died and were symptomatic (Table 1). Comparing mortality among experimental birds is the most direct assessment of lead toxicity. However, comparisons of weight change are also useful because weight loss is characteristic of lead poisoned ducks (Jordan & Bellrose 1951). Analysis of covariance indicated no significant difference in the rate of weight loss (gm day^{-1}) between mallards and black ducks for the control ($F=0.02$, $p<0.5$), 2 shot ($F=0.07$, $p<0.5$), and 5 shot groups ($F=3.72$, $p<0.5$). However, in the 5 shot repeated dose, black ducks had a greater rate of weight loss than mallards ($F=25.56$, $p<0.001$)(Fig. 1). Weight loss was compared only until the first death of a duck in dosed groups, because weights recorded after the removal of a dead bird would be biased upward.

Species comparisons show that wing and liver lead levels were generally higher in mallards than in black ducks (Tables 2 and 3). A difference in background levels in experimental birds may explain the consistently higher lead levels of mallards. Mean wing bone and liver lead levels for hunter-killed birds collected in Connecticut were significantly higher among mallards than among black ducks (wings: 27.5 ppm $\pm$ 8.09 SE, N=130 and 14.3 $\pm$ 1.71 SE, N=172, respectively, $p<0.0005$; livers: 1.3 $\pm$ 0.16 SE, N=158 and 0.8 $\pm$ 0.9 SE, N=216 respectively, $p<0.005$, Wilcoxon Rank Sum Test) (G. G. Chasko, unpublished data). The higher tissue lead levels of mallards indicates that mallards are somehow exposed to higher levels of lead than black ducks, perhaps due to a higher incidence of ingested shot. Gizzard analysis of hunter-killed birds in Connecticut showed a higher percentage of mallards with ingested shot than black ducks (13.5%, N=171 and 8.3%, N=228, respectively, $X^2=2.20$, $p<0.10$) (G. G. Chasko, unpublished data).

Shot retention rates among asymptomatic, symptomatic, and dead ducks were similar for mallards and black ducks (Table 4).

Weight loss and mortality generally increased with higher dosage (Fig. 1, Table 1). When dose groups were divided into asymptomatic, symptomatic, and dead ducks, a distinct pattern emerged. Regardless of dose, asymptomatic ducks of both species lost a similar amount of weight as the controls or even gained weight. Symptomatic birds lost about 20% of their initial weight and dead birds lost from 30% to 50% (Table 1).

Table 1. Numbers and weight change (%)[a] of asymptomatic, symptomatic, and dying birds.

Species, dose group (# males, females)	Asymptomatic N(%)	Asymptomatic $\bar{x}$ % Wt. Change	Symptomatic N(%)	Symptomatic $\bar{x}$ % Wt. Change	Dead N(%)	Dead $\bar{x}$ % Wt. Change	Group Total N	Group Total $\bar{x}$ % Wt. Change
Black Ducks								
Controls (3 males, 2 females)	5(100)	-6.3	0		0		5	- 6.3
2 Shot (3 males, 1 female)	3(75)	+4.3			1(25)	-30.2	4	- 4.3
5 Shot (3 males, 1 female)	2(50)	+2.6	0		2(50)	-31.3	4	-14.4
5 Shot - repeated dose (4 males, 1 female)	1(17)	+1.3	2(40)	-18.9	2(40)	-50.0	5	-27.3
Mallards								
Controls (2 males, 3 females)	5(100)	-0.9	0		0		5	- 0.9
2 Shot (3 males, 3 females)	5(83)	+4.4	1(17)	-20.0	0		6	+ 0.4
5 Shot (2 males, 4 females)	4(67)	-3.0	0		2(33)	-49.0	6	-17.8
5 Shot - repeated dose (2 males, 3 females)	3(60)	+4.4	0		2(40)	-37.7	5	-12.4
Total $\bar{x}$ % weight change (N)	+0.48 (28)		-19.3 (3)		-37.3 (9)			

[a]Reported as % change from weight at dosage to weight at the end of the experiment or at death.

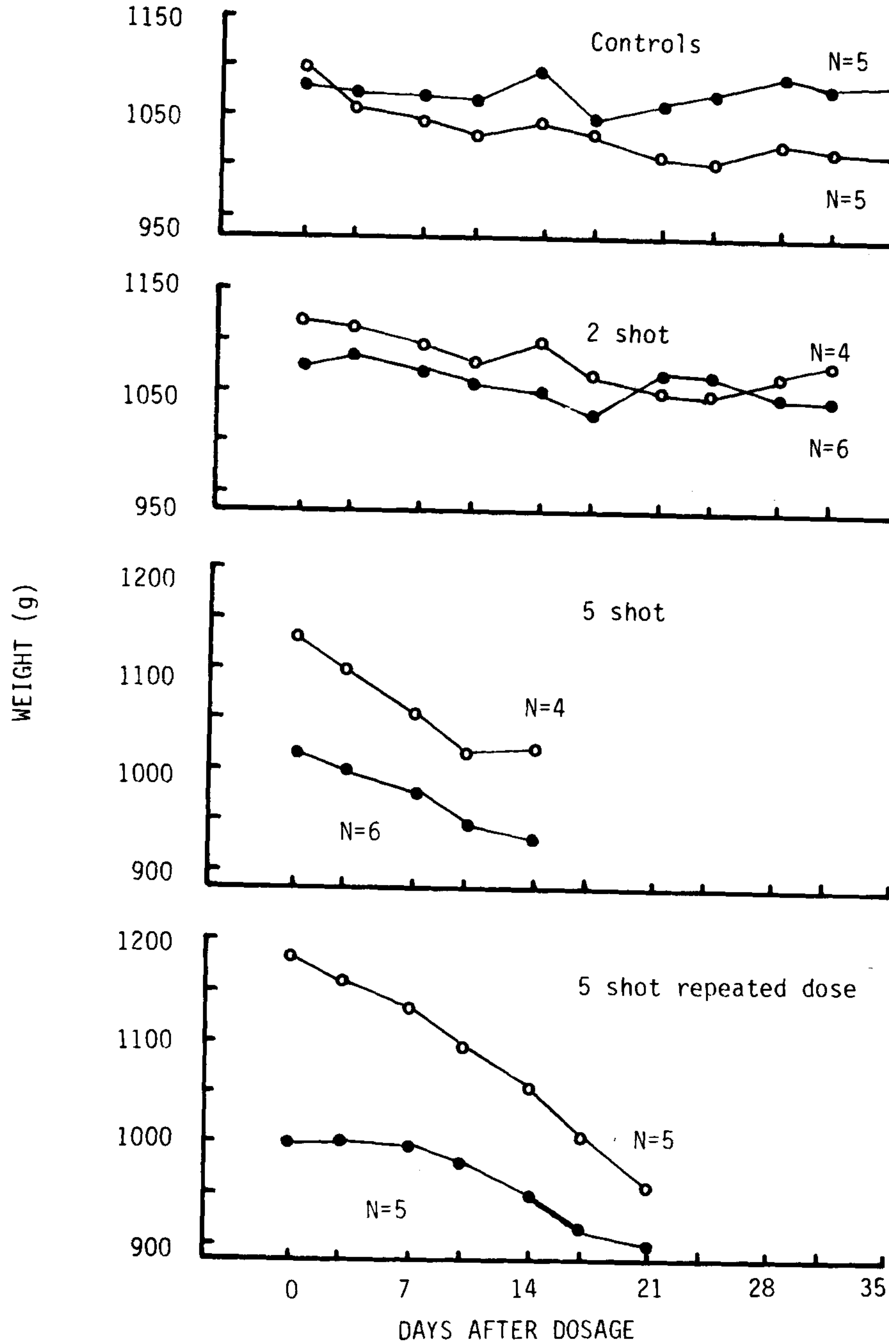

Fig. 1. Mean weights of black ducks (o——o) and mallards (●——●) (measured until the first mortality was recorded in each group).

Table 2. Wing bone lead concentrations (ppm) for asymptomatic, symptomatic, and dead birds. Sample sizes are in parentheses.

	Lead Concentrations, $\bar{x}$ ppm ± SE			
Species and Dose Group	Asymptomatic	Symptomatic	Dead	Group Total
Black Ducks				
Controls	8.5 ± 8.14 (5)			8.5 ± 8.14 (5)
2 Shot	12.9 ± 2.06 (3)		78.9 (1)	29.4 ± 16.56 (4)
5 Shot	21.8 ± 9.00 (2)		75.4 ± 48.6 (2)	48.6 ± 25.43 (4)
5 Shot - repeated dose	32.1 (1)	38.8 ± 0.10 (2)	104.7 ± 6.75 (2)	63.8 ± 18.54 (5)
Mallards				
Controls	17.3 ± 4.70 (5)			17.3 ± 4.70 (5)
2 Shot	29.6 ± 5.25 (5)	35.6 (1)		30.6 ± 4.40 (6)
5 Shot	33.0 ± 6.72 (4)		100.0 ± 8.00 (2)	55.3 ± 14.90 (6)
5 Shot - repeated dose	50.6 ± 15.61 (3)		123.8 ± 6.75 (2)	79.9 ± 20.00 (5)

Table 3. Liver lead concentrations (ppm) for asymptomatic, symptomatic, and dead birds. Sample sizes are in parentheses.

	Lead Concentrations, $\bar{x}$ ppm ± SE			
Species and Dose Group	Asymptomatic	Symptomatic	Dead	Group Total
Black Ducks				
Controls	0.1 ± 0.01 (5)			0.1 ± 0.01 (5)
2 Shot	0.6 ± 0.09 (3)		20.9 (1)	5.6 ± 5.09 (4)
5 Shot	0.4 ± 0.95 (2)		28.5 ± 10.33 (2)	14.5 ± 9.16 (4)
5 Shot - repeated dose	8.0 (1)	5.1 ± 0.40 (2)	36.0 ± 3.98 (2)	18.0 ± 7.46 (5)
Mallards				
Controls	0.2 ± 0.06 (5)			0.2 ± 0.06 (5)
2 Shot	4.6 ± 1.14 (5)	5.1 (1)		4.7 ± 0.93 (6)
5 Shot	5.1 ± 1.60 (4)		54.0 ± 0.05 (2)	21.4 ± 10.50 (6)
5 Shot - repeated dose	18.6 ± 9.38 (3)		58.8 ± 2.40 (2)	34.7 ± 11.12 (5)

Table 4. Number and percent (in parentheses) of lead shot remaining in gizzards of dosed birds.

Species, dose group, Condition of ducks	N	Day after dosage 0*	3*	7*	10*	14*	17	21	24	28	31
Black ducks											
2 shot-asymptomatic	3	6 (100)	6 (100)	5 (83)	1 (17)	0					
-symptomatic	1	2 (100)	2 (100)	2 (100)	2 (100)	1 (50)	0				
5 shot-asymptomatic	2	10 (100)	8 (80)	3 (30)	2 (20)	1 (10)	1 (10)	0			
-dead	2	10 (100)	10 (100)	10 (100)	10 (100)	5 (100)[b]	1 (20)	0			
5 shot repeated dose-asymptomatic	1	1 (100)	2 (100)	2 (67)	2 (50)	2 (40)	1 (20)	0			
-symptomatic	2	2 (100)	3 (75)	5 (83)	6 (75)	6 (60)	6 (60)	0			
-dead	2	2 (100)	4 (100)	6 (100)	8 (100)	10 (100)	10 (100)	10 (100)	10 (100)	10 (100)	0[c]
Mallards											
2 shot-asymptomatic	5	10 (100)	9 (90)	7 (70)	2 (20)	0					
-symptomatic	1	2 (100)	2 (100)	2 (100)	1 (50)	1 (50)	0				
5 shot-asymptomatic	4	20 (100)	15 (75)	9 (45)	5 (20)	1 (5)	0				
-dead	2	10 (100)	10 (100)	10 (100)	3[b] (60)	3[b] (60)	0				
5 shot repeated dose-asymptomatic	3	3 (100)	5 (83)	8 (89)	9 (75)	7 (44)	5 (33)	1 (7)	1 (7)	1 (7)	0
-dead	2	2 (100)	3 (75)	5 (83)	7 (88)	8 (80)	8 (80)	8 (80)	3[b] (60)	3[b] (60)	0

*birds in the 5 shot repeated dose group received 1 shot on each of these days. [a]N=2, [b]N=1, [c]N=0. As birds in these groups died they were removed from further calculations.

Mortality for black ducks dosed acutely with 2 and 5 shot was 25% (1 of 4) and 50% (2 of 4), respectively (Table 1). In a previous experiment using natural foods and conducted under similar conditions, no mortality was recorded for black ducks (N=6) dosed with 1 No. 6 lead shot (although 3 ducks did exhibit green diarrhea), and mortalilty for an 8 shot dose was 83% (5 of 6) (G. G. Chasko, unpublished data). Although these mortality estimates were determined from very small samples, the data do suggest that mortality of black ducks ingesting shot in an acute manner begins with 2 shot, increases with higher ingestion, and is nearly 100% for birds ingesting 8 shot. Mallards probably exhibit a similar pattern of dose related mortality, although they may be more tolerant of lead poisoning than black ducks (Table 1).

Our mortality estimates are most comparable to those of Jordan (1968) and Bellrose (1959). Jordan (1968) documented winter mortality of penned wild mallard drakes dosed with 1 No. 6 shot and fed various diets for 28 days. He recorded the following mortalities: 60% on a corn diet, 60% on a mixed grain diet (including corn), 10% on a small grain diet of tame rice and smartweed (*Polygonum pensylvanicum*) and no mortality on a diet of mixed grains and coontail (*Ceratophyllum demersum*). Mortality on all diets, except coontail and mixed grains was greater than in our study, where a wide variety of natural foods was used, including considerable amounts of animal foods.

In Bellrose's (1959) study, wild mallards were captured, dosed with lead shot, banded, and returned to the wild. Estimates of mortality under field conditions (i.e. free ranging wild ducks consuming a natural diet) were made from band return data and other information. He estimated mortality rates for wild mallards at 9% for 1 shot, 23% for 2 shot, and 43% for 5 shot. His mortality rates were similar to our estimates for black ducks except that we recorded no mortality for 1 shot ingestion.

The physical form and nutritional components of the diet are important factors influencing lead toxicity (Bellrose 1959, Jordan 1968). Aquatic vegetation, animal foods, and high levels of dietary calcium are effective in minimizing lead toxicity (Thompsett 1938, Jordan 1968). This could explain why Jordan's ducks fed coontail, an aquatic plant high in calcium exhibited no mortality. In our study, aquatic vegetation (duckweed and eelgrass), and animal foods constituted a substantial portion of the diet. Duckweed is high in calcium (Jordan 1969), and we assume that the animal foods used (fish, crabs, mussels) were also. Thus, the diet used in our experiments may be responsible for the relatively low mortality we recorded.

Mean lead levels in wings and livers increased with dosage (Tables 2 and 3). However, lead concentrations in all groups varied substantially, precluding determination of diagnostic tissue lead levels for each dose. Low precision of wing lead levels was not suprising because high variability is characteristic of wing lead residues among wild populations (Stendell et al. 1979).

Bone lead levels among waterfowl dying from lead poisoning are often $\geq$100 ppm (Longcore et al. 1974a). Our results are consistent with these findings (Table 2). Regardless of dose, lead concentrations were highest among dead ducks (most >100 ppm). Also, White & Stendell (1977) indicated that wing bone lead levels >20 ppm probably reflect previous exposure to a high amount of lead, most likely as shot. Our results support their findings except for asymptomatic black ducks in the 2 shot group (Table 2).

In this experiment, liver lead levels among dead birds (Table 3) were similar to those reported by other investigators (Bates et al. 1968, Andrews et al. unpublished data). Longcore et al. (1974a) suggested that liver lead levels between 6-20 ppm should be indicative of acute recent exposure to lead. In our study, only 1 of 3 symptomatic birds had levels >6 ppm, and mean levels of asymptomatic acutely dosed birds were <6.0 ppm (Table 3). Interpreting liver lead levels for experimentally dosed birds is difficult because liver lead concentrations will decrease with increasing time after dosage (Andrews et al. unpublished data, Chasko, unpublished data). Therefore, lead levels at the end of our experiment were probably lower than levels at dosage. A comparison of asymptomatic birds in the 5 shot (acute dose) and 5 shot repeated dose group supports this contention. Liver lead levels for the 5 shot group, measured 35 days after dosage, are lower than levels for the 5 shot repeated dose group, which received their last shot 21 days prior to the end of the experiment.

All acutely dosed birds had voided or completely eroded all ingested shot within 21 days after dosage. Birds in the 5 shot repeated dose group eliminated all shot within 17 days after completion of dosage. Our data indicates that lead retention time varies considerably among individual birds. Mortality was directly related to the length of lead retention. In all comparisons, retention was shortest for asymptomatic birds, intermediate for symptomatic birds, and longest for dead birds (Table 4).

Most studies of lead toxicity have used ducks dosed in an acute manner. However, it is likely that some ducks will ingest shot repeatedly as they are exposed to lead in different locations throughout their migratory route and wintering areas. Straub (1913) indicated that lead poisoning is caused by an accumulation of injuries from the passage of lead through the tissues and not by storage in tissues. Hanzlik (1923) found that tissue injury in pigeons did not increase in proportion to the quantity of deposited lead shot but was dependent on the concentrations of soluble lead in those tissues. This suggests that a quantity of lead ingested repeatedly may be more toxic than the same amount ingested acutely. In our study, black ducks and mallards in the 5 shot repeated dose group had higher wing and liver lead levels than the 5 shot group (acute dose) (Tables 2 and 3). Mortality was similar between groups for both species and weight loss was similar between groups for mallards (Table 1). However, in the 5 shot repeated dose group, black ducks had a higher percentage of weight loss and more symptomatic birds than in the 5 shot group (acute dose) (Table 1). Further research on repeated ingestion of lead shot is warranted.

Our data indicate that susceptibility to lead poisoning varies even in closely related species such as black ducks and mallards. Also, this study suggests that ducks feeding on a variety of plant and animal foods are less susceptible to lead poisoning than ducks feeding primarily on cereal grains.

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