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SIGNIFICANCE OF LEAD RESIDUES IN MALLARD TISSUES

by

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ABSTRACT

Tissues of adult, lead-dosed mallards that either died or were sacrificed were analyzed for lead. Lead levels in brains, tibiae, and breast muscle of ducks that died and in tibiae of ducks that were sacrificed increased significantly from dosage until death. Lead in the heart, lung, and blood from sacrificed ducks decreased significantly from dosage until death.

Lead concentrations in tissues from ducks in the two groups were not significantly different except for the liver, kidney, and lung. Average lead levels in the livers and kidneys of ducks that died were significantly higher than those in ducks that were sacrificed. The mean concentration of lead in the lungs of the ducks sacrificed was significantly higher than the mean level in the lungs of ducks that died.

Measurements of the lead concentrations in this study, when compared with lead levels reported in the literature for avian and non-avian species, showed that arbitrary diagnostic levels indicating lead poisoning could be set. In mallard ducks, lead levels exceeding 3 ppm in the brain, 6 to 20 ppm in the kidney or liver, or 10 ppm in clotted blood from the heart indicated acute exposure to lead.

INTRODUCTION

Each year thousands of ducks and geese die from lead poisoning after ingesting lead pellets that accumulate as the result of hunting in waterfowl feeding areas. Many waterfowl from these "die-offs" have been examined and the tissues chemically analyzed to help determine more exactly the relationship between lead ingestion and mortality. However, the tissues most useful in diagnosis and the concentrations of lead that are hazardous have not been clearly defined in experimental studies. The objective of this work was to determine if concentrations of lead in one or more tissues could be associated with mortality of mallard ducks (Anas platyrhynchos) and so serve as diagnostic criteria. The present paper reports the results of this experiment and also includes a review of the literature on experimental studies of lead poisoning in waterfowl and the occurrence of lead in field-collected waterfowl that died from ingesting lead shot.

PROCEDURES

One hundred 4-month-old drake mallards were force-fed one number 4 size lead buckshot (about 1.4 g) which had been preweighed to the nearest 0.001 g. All birds were weighed and banded before dosing. Ducks were held in groups of 20 in five 4.6 x 9.1 m gravel-based pens. Two additional ducks were placed in each of the five pens to serve as controls. Whole kernel corn was supplied ad libitum in sheltered feeders. Water was available in 946 liter fiber glass troughs.

Twenty experimental ducks from one randomly chosen pen were selected to provide samples of ducks that died of poisoning. The live ducks in the other four pens were sacrificed for comparisons with those that actually died.

Earlier studies of lead poisoning indicated that two or more ducks would die on the same day. We scheduled our sampling at 2-day intervals when we collected two dead ducks and two live ducks (which were sacrificed). In three instances, to fulfill the two-bird complement for dead ducks, we substituted a bird that died either the day before or day after the scheduled sampling day. Two of the 20 experimental ducks voided the shot, limiting analysis to 18 birds. Deaths began 4 days after dosing and continued for 20 days.

Sampling began the fourth day after dosing and continued until 18 ducks were killed. Each duck to be sacrificed was fluoroscoped first to ensure that it had actually retained the shot; only ducks retaining shot were used. All pens were checked daily for dead birds.

Ducks randomly selected for sacrifice were asphyxiated with carbon dioxide and all ducks were frozen until necropsy. At necropsy, the following organs and tissues were removed, weighed, and refrozen to await chemical analysis: brain, liver, kidney, tibia, gallbladder, breast muscle, lung, spleen, pancreas, heart, and blood clot from heart.

The ingested lead shot was recovered from each duck that was analyzed and the shot was weighed to determine the amount of lead which had eroded. Lead content of tissues was measured with a Perkin-Elmer[1/] atomic absorption spectrophotometer, Model 303. Instrument settings were essentially those recommended in "Analytical Methods for Atomic Absorption Spectrophotometry," Perkin-Elmer Corp., Norwalk, Connecticut (instructions with spectrophotometer). Thirty-six ducks were analyzed--18 that had died following lead dosage and 18 that had been sacrificed. Unless otherwise stated all parts per million (ppm) are on a wet-weight basis.

Concentrations of lead in organs of ducks from the two mortality groups were compared by the Student's t-test. Data for the kidneys and gallbladder of both groups and for pancreata of ducks that died were transformed by logarithms prior to statistical analysis because of an apparent log-normal distribution.

LEAD CONCENTRATIONS

General

Lead concentrations in the tissues of each lead-dosed duck that died or was sacrificed are presented in Appendix Tables A1 and A2. Concentrations of lead in the livers and kidneys of ducks that died averaged higher ($P<0.05$) than concentrations in the same organs of sacrificed ducks (Table 1). The mean concentration of lead in the lungs of ducks that died was lower than that in the sacrificed ducks ($P<0.05$). No other significant differences were found between average lead concentrations in tissues of the two mortality groups. The highest lead concentrations were in the pancreas (1,551 ppm) and kidney (408 ppm), of ducks that died (Table A1), and in the tibia (212 ppm) of sacrificed ducks (Table A2).

Lead concentrations in brains and breast muscle of the ducks that died increased significantly with the length of time between dosage and death, as did concentrations in the tibiae of birds from both groups (died and sacrificed, Table 1). Lead concentrations in the brain and breast muscle of sacrificed ducks also increased but not by significant amounts. Concentrations of lead in the heart, lungs, and blood clots of sacrificed ducks decreased significantly with time.

The average amount of lead eroded from the ingested shot was identical (0.627 g) for ducks in each mortality group. For the ducks that died, the concentrations of lead in tibiae and brain increased significantly with the increased amount of lead eroded from the shot, and the same was true for lead in the heart, blood, and liver of ducks that were sacrificed. These group differences may relate to the availability of lead or to the difference in lead storage and/or lead transport in different organs.

[1/] Use of trade names does not imply U.S. Government endorsement of commercial products.

Table 1. Lead levels (ppm, wet weight) in tissues of male mallards in relation to mortality and time until death.

Tissue	Died(D) or sacrificed(S)	Number of ducks	Arithmetic mean ± s.e. (ppm)	Range (ppm)	r^2[a]	Regression equation Residue level(y) on days(x)
Brain	D	18	4.6 ± 0.50	2.1 - 10.6	0.49[b]	$\hat{y}$ = 1.1811 + 0.2823 x
	S	18	3.9 ± 0.30	1.7 - 7.2	[d]	
Tibia	D	18	136.7 ± 8.0	87.0 - 209.0	0.28[c]	$\hat{y}$ = 95.3879 + 3.3973 x
	S	18	126.7 ± 9.2	57.0 - 212.0	0.32[b]	$\hat{y}$ = 60.6722 + 5.5041 x
Liver	D[b]	18	57.4 ± 3.4	32.0 - 83.0	[d]	
	S	18	44.1 ± 2.5	26.0 - 65.0	[d]	
Gallbladder	D[e]	18	24.9 ± 3.2	9.1 - 54.0	[d]	
	S[e]	18	21.8 ± 4.9	3.7 - 97.5	[d]	
Spleen	D	17	27.9 ± 3.9	12.0 - 69.0	[d]	
	S	17	27.0 ± 2.9	10.8 - 56.0	[d]	
Kidney	D[c/e]	17	175.7 ± 26.1	53.0 - 408.0	[d]	
	S[e]	18	104.3 ± 13.6	22.0 - 243.0	[d]	
Pancreas	D[e]	18	389.9 ± 98.8	45.0 -1551.0	[d]	
	S	18	212.9 ± 25.3	79.0 - 522.0	[d]	
Heart	D	18	2.5 ± 0.21	1.3 - 4.8	[d]	
	S	18	2.4 ± 0.25	0.4 - 4.6	0.62[b]	$\hat{y}$ = 4.3488 - 0.1591 x
Lung	D[c]	18	4.9 ± 0.36	2.5 - 8.2	[d]	
	S	18	6.8 ± 0.75	1.7 - 15.0	0.31[c]	$\hat{y}$ = 10.8750 - 0.3354 x
Blood	D	18	10.2 ± 1.23	2.7 - 20.0	[d]	
	S	18	10.6 ± 1.18	3.7 - 22.9	0.36[b]	$\hat{y}$ = 17.3777 - 0.5666 x
Breast muscle	D	18	1.4 ± 0.17	0.6 - 3.2	0.23[c]	$\hat{y}$ = 0.5793 + 0.0642 x
	S	18	1.4 ± 0.09	0.9 - 2.3	[d]	

[a] Coefficient of determination for residue with days till death; [b] $P<0.01$; [c] $P<0.05$; [d] Not significant;
[e] Data log transformed for analysis.

None of the two undosed control ducks died in any of the pens and none was analyzed for lead residues.

For the 11 tissues analyzed we could not determine a lead level in ducks that died that was clearly diagnostic of death when compared with the lead levels in ducks that were sacrificed. From outward signs some of the sacrificed ducks, especially late in the study, were obviously suffering from lead poisoning and would probably have succumbed. However, concentrations in certain tissues--the brain, liver, and kidneys--do appear to have value in indicating lethal lead toxicosis. In the text that follows, lead levels in the various tissues are discussed separately and compared with reports from the literature.

Brain

Lead concentrations in brains of ducks that died averaged 4.6 ppm, ranging from 2.1 to 10.6 ppm (Table 1) and increased with time (Fig. 1). There was no significant correlation between tissue residues and time for the sacrificed ducks. Lead levels in brains of lead-poisoned, field-collected or experimentally poisoned waterfowl all fall within this range: 3 to 6 ppm in Canada geese (Branta canadensis) (Locke and Bagley 1967a); 3 to 6 ppm in mallards (Bates et al. 1968); 5 ppm in black duck (Anas rubripes) (Locke and Bagley 1967b); and 3 ppm in Canada geese (Bagley et al. 1967). In studies of the effects of diet on lead poisoning in ducks (Andrews et al., unpublished data, Patuxent Wildlife Research Center), lead-dosed mallards that died on a whole-corn diet had concentrations of lead in the brain that averaged 4.3 ppm. In one study, the average concentration of lead in the brains of two lead-dosed mallards that died while maintained on a commercial duck mash diet was 4.5 ppm. Lead-dosed survivors on the same diet contained an average of 2.0 ppm of lead in the brain. Levels of lead in the brain exceeding 3 ppm probably could be considered to indicate an advanced state of lead intoxication.

In the present study brain weights of ducks that died decreased significantly ($P<0.05$) with time from dosage to death ($r^2 = 0.34$). Brain weights of sacrificed ducks did not decrease significantly with time from dosage to death. A significant difference was not demonstrated between average brain weights of the ducks in the two mortality groups (died and sacrificed, Table 2). The facts that lead poisoning induces a starvation syndrome and that fat in the brain is among the last of the deposits to be mobilized may explain why the brains of birds that died last weighed less. It appears that concentrations of lead in brains are not as definitive in diagnosing death as are residues of some of the organochlorine pesticides (Stickel, W. H. et al. 1969; Stickel, L. F., and W. H. Stickel 1969).

Tibia

Lead in bone tissue has long been associated with chronic exposure. The high lead levels in tibiae, therefore, do not necessarily indicate acute lead intoxication. For example, lead in the tibiae of five mallards fed commercial duck pellets averaged 41 ppm and ranged to 100 ppm (Andrews et al., unpublished

data, Patuxent Wildlife Research Center. Although the ducks were fed similar amounts of lead and exposed the same amount of time, they exhibited no outward signs of lead poisoning.

Table 2. Organ and tissue weights (wet) of lead-poisoned mallards.

Organ or tissue	Sacrificed (n=18)		Died (n=18)	
	Mean weight (grams)	s.e.	Mean weight (grams)	s.e.
Tibia	4.76	0.210	4.34	0.134
Brain	4.75	0.086	4.64	0.107
Lung	7.41	0.037	6.98	0.495
Spleen	0.321	0.041	0.347	0.048
Pancreas	1.212	0.096	1.097	0.160
Liver	19.62	1.209	18.15	1.47
Kidney	3.62	0.175	3.52	0.185
Gallbladder	1.72	0.185	1.63	0.197
Heart	8.76	0.453	7.89	0.464
Blood clot (heart)	2.49	0.281	1.72	0.194

Lead levels in the tibiae of ducks in both groups of the present study increased with time after dosage and followed the well documented accumulative behavior of lead in bone (Fig. 2). Lead concentrations in bone, therefore, may result from either acute high-level exposure or chronic low-level exposure. Hence the use of bone lead levels as a suitable criterion for determining acute lead poisoning is questionable, but its presence is evidence of exposure to lead.

The mean lead concentration (136.7 ppm) in the tibiae of ducks that died in our study (Table 1) was higher than the concentrations reported by other workers for field-collected lead-poisoned specimens: 67 to 98 ppm in Canada geese (Adler 1944); 40 to 50 ppm in whistling swan (Olor columbianus) (Chupp and Dalke 1964); and 12 to 102 ppm in Canada geese from two separate "die-offs" (Bagley et al. 1967). Coburn et al. (1951) reported residues of 203 to 400

ppm in the whole skeleton of mallards dosed with an aqueous solution of lead nitrate. Average lead concentrations ranged from 2 to 13 ppm in the tibiae of seven different species of waterfowl with no known history of lead exposure (Bagley and Locke 1967).

Liver

Concentrations of lead in the liver have been considered to be the most useful measurements for diagnosis of acute lead intoxication (Adler 1944; Coburn et al. 1951; Cook and Trainer 1966). Most of the mean lead levels reported by various investigators (Table 3) are below those of ducks that died in our study (32 to 83 ppm). These differences may have occurred because only ducks retaining the lead pellet fed to them were analyzed in our study. Exposure of waterfowl involved in "die-offs" is more variable because of differences both in the number of shot ingested and in the number of shot retained. Lead concentrations in the livers of ducks from both mortality groups were not statistically correlated with postdosage days until death (Fig. 3).

Lead levels that range between 6 to 20 ppm in the liver should be considered as an indication of recent, acute lead exposure and as being diagnostic of active lead intoxication. Background levels of lead averaged 0.5 to 1.5 ppm in the livers of 11 different species of waterfowl with no known history of lead exposure (Bagley and Locke 1967).

Gallbladder and Spleen

The gallbladders of the group that died and the sacrificed group averaged 24.9 and 21.8 ppm, respectively (Table 1). The average concentration of lead in the spleen for the group that died was 27.9 and 27.0 ppm for the group that was sacrificed. There was no association between lead concentration and time until death for either mortality group (Figs. 4 and 5). Adler (1944) reported lead concentrations ranging from 21 to 73 ppm in spleens of lead poisoned Canada geese, which is within the range we found.

Kidney

The average level of lead in kidneys of ducks that died (175.7 ppm) was significantly higher than that in kidneys of sacrificed ducks (104.3 ppm) (Table 1). It was also higher than values for waterfowl that died under field conditions: 4 to 99 ppm in mallards (Erne and Borg 1969); 12 to 57 ppm in Canada geese (Adler 1944); and 8 to 32 ppm in Canada geese (Bagley et al. 1967). Lead concentrations in the kidney were not correlated with time until death (Fig. 1).

Because of the kidney's essential role in excretion of body wastes and ingested toxic material, lead in kidney tissues may serve as an important indicator of lead poisoning. Allcroft (1950) concluded that, for calves, the amount of lead in the kidney cortex was of specific diagnostic value. In kidney tissue of our mallards a level of 20 ppm appeared to indicate a serious effect of lead.

Table 3. Lead concentrations in livers of lead poisoned waterfowl.

Species	Source	Amount lead (ppm wet wt)[a] Average	Range	Reference
Canada geese	"die-off"	18	9-27	Adler, 1944
Canada geese	"die-off"	12	1-20	Bagley, et al.,
(2 separate die-offs)		22	12-53	1967
Canada geese	expt'l.	±20	5-32	Cook & Trainer, 1966
Canada geese	expt'l.	19	8-42	Karstad, 1971
Canada geese	"die-off"	26	12-44	Locke, et al., 1967
Canada geese	"die-off"	21	10-45	Locke & Bagley, 1967a
Mallards	expt'l.	33	20-64	Bates, et al., 1968
Mallards	expt'l.	44	23-80	Coburn, et al., 1951
Mallards	expt'l.	12	8-14	Barrett & Karstad,
(2 separate groups)		40	11-61	1971
Mallards	expt'l.	51	16-76	Locke, et al., 1966
Whistling swan	"die-off"	28	18-37	Chupp & Dalke, 1964
Mallard		12	12	
Mute swan	"die-off"	--	1-70[b]	Erne & Borg, 1969
Mallard		--	5-45[b]	
Mallard	expt'l.			Andrews, et al.,
(died: corn diet)		39	8-66	(unpublished data,
(died: comm. mix pellet diet)		23	19-26	Patuxent Wildlife
(survived: comm. mix pellet diet)		1	1-3	Research Center)
Black duck	"die-off"	25	25	Locke & Bagley, 1967b

[a] Values rounded to nearest whole number.

[b] This range of values for more than half of the livers examined.

Pancreas

Lead concentrations in the pancreata of ducks in both groups were extremely high and variable, 45 to 1,551 ppm in ducks that died; and 79 to 522 ppm in ducks that were sacrificed (Table 1). The mean lead levels in the pancreata of ducks that died exceeded those in ducks that were sacrificed, but extreme variability precluded obtaining significant differences. There was no correlation between lead concentrations and days until death (Fig. 6).

Heart

Lead levels in heart tissue were almost identical in ducks of both groups, averaging 2.5 ppm for those that died and 2.4 ppm for those that were sacrificed (Table 1). Concentrations of lead in hearts of sacrificed ducks decreased with time until death ($P<0.01$) (Fig. 5) but not in those that died. Heart weights of ducks that were sacrificed decreased with time until death ($P<0.05$; $r^2 = 0.53$). An average lead level of 1.1 ppm has been reported in hearts of experimentally lead-poisoned Canada geese that died (Karstad 1971).

Lung

The mean amount of lead in the lungs of ducks that died was significantly ($P<0.05$) lower than that in the lungs of the sacrificed ducks (Table 1). Lead levels in lungs of sacrificed ducks decreased with time between dosage and death ($P<0.05$) but this was not true for those that died. Lead in lungs of lead-poisoned Canada geese that died ranged from 2 to 9 ppm (Adler 1944).

Blood Clot

Lead in clotted blood from hearts averaged 10.2 and 10.6 ppm in the groups that died and were sacrificed, respectively (Table 1). Barrett and Karstad (1971) reported blood levels of 8.6 to 13.9 ppm in experimentally lead-poisoned mallards. Lead levels in blood of our sacrificed ducks decreased with time until death ($P<0.01$) (Fig. 2) but those in ducks that died did not decrease. Lead levels in blood of humans have been considered important indicators of lead exposure (Cantarow and Trumper 1944). Lead in the amount of 0.06 mg or more per 100 ml whole blood (±0.006 ppm) has been considered diagnostic of lead poisoning in dogs (Zook et al. 1969).

Breast Muscle

The average level of lead in samples of breast muscles was 1.4 ppm in both groups (Table 1).

A level of 2 ppm was reported in the breast muscle of a lead-poisoned Canada goose that died (Locke and Bagley 1967a). Low levels of lead in the breast muscle may be explained by the finding of Aub et al. (1925), who reported that the formation of lactic acid in skeletal muscle facilitates the dissolution of lead phosphate and thereby renders its storage in high amounts impossible.

Organ and Tissue Weights

Significant differences were not demonstrated between organ weights of the ducks that died and those that were sacrificed. However, in 9 of 10 organs, the mean organ weight of the sacrificed ducks was numerically greater than the respective mean organ weight of the ducks that died (Table 2). The probability of 9 of 10 being heavier by chance alone is less than 0.011, or about 11 in 1,000. Average whole body weights of ducks that were sacrificed and those that died were one-third lower than their predosage average weights.

DISCUSSION

Factors Affecting Lead Deposition

The diagnosis of lead poisoning in wild waterfowl is commonly based on the presence of one or more of the following: ingested lead shot, bile staining of the gastrointestinal tract (particularly the gizzard), crop impaction, and elevated lead levels in tissues. A number of workers have stated that lead levels in the liver are more or less diagnostic of acute lead intoxication (Adler 1944; Coburn et al. 1951; Cook and Trainer 1966).

The occurrence of acid-fast intranuclear inclusion bodies in the proximal convoluted tubules of the kidneys has been shown to be presumptive evidence of lead intoxication in mallards (Locke et al. 1966; Bates et al. 1968). These inclusion bodies are a good indication of recent exposure to lead when present in ducks, but they occur only sporadically in lead-poisoned Canada geese (Locke et al. 1967).

The presence of fluorescent erythrocytes in blood samples subjected to blue-ultraviolet light also has been used as a test for lead poisoning in waterfowl (Barrett and Karstad 1971). The strength of the fluorescence decreased with time after exposure but remained diagnostic at death for most of the experimental mallards and Canada geese that died. The authors concluded, however, that the most reliable objective test for diagnosing lead poisoning in dead birds was chemical analysis of soft tissue.

The distribution and deposition of lead in intoxicated animals depends on many factors, primarily on the portal of entry (Clarke and Clarke 1967). Continuous oral doses of lead result in largest residues in bones, smaller amounts in liver and kidneys, and the lowest amounts in the heart, lung, muscle, and brain. Acute exposure, a rapid large dose of lead, results in highest amounts in liver and kidney.

The progressive decrease in lead levels of the heart, lung, and blood clot from dosage to sacrifice in our study may have been related to the circulatory function of these tissues. It seems probable that lead in circulation decreases as more lead is stored. Lead levels in tibiae, a known area of storage, increased with time until death. The increasing impairment

of normal body function as the degree of poisoning progressed may have lowered the amount of lead being absorbed from the gastrointestinal tract, thereby lowering lead levels in the blood of these tissues. We did not measure lead excretion rates in the ducks in this study.

It is also known that lead deposition and retention in rats is associated with the age of the animal (Shields et al. 1938). Young rats fed lead acetate retained more lead at a given dietary intake than mature rats. The influence of calcium and phosphorus on the behavior of lead is well documented (Shields and Mitchell 1941; Grant et al. 1938; Sobel et al. 1940). Generally, lead storage is increased by a low-calcium diet and decreased by a high-calcium diet.

Sobel et al. (1940) concluded that "lead deposition is directed by a system of its own, which is governed by the same laws as is calcium deposition but does not necessarily go in the same direction. The effect of calcium on deposition of lead is essentially competitive, because it tends to remove phosphorus available for lead deposition." Rats store lead chiefly in the bones, and the kidneys contain relatively high concentrations compared with the livers (Lederer and Bing 1940).

Certainly the low-calcium corn diet of our study was conducive to relatively high lead storage as evidenced by the generally high lead levels with respect to those reported in the literature or by the low lead levels in organs associated with the circulatory system.

The influence of dietary constituents on lead solubility also apparently affects lead absorption (Thompsett 1939; Hanzlik and Presho 1923). Vitamin D also appears important in deposition of lead in bones (Sobel et al. 1940). Carroll et al. (1970), in studies of the localization of lead in rat kidneys, showed that calcium and phosphorus are codeposited with lead in complexes, a result consistent with the hypothesis of the importance of lead-protein complexes in lead poisoning. Gage and Litchfield (1968), in studies of the lead content in blood, bile, urine, liver, kidney, and bone from lead-dosed rats, determined that the bile was the best index of lead absorption.

Straub (1913), when considering lead storage during his studies of lead poisoning in cats, established a theory explaining chronic toxicity. He explained that lead poisoning is due to accumulation of injuries from the continued passage of certain concentrations of lead through the tissues and not by lead storage in the tissues. Hanzlik (1923) supported Straub's conclusions with his studies of lead poisoning in pigeons: "....tissue injury, or poisoning, by lead is not increased in proportion to the mere quantity of deposited lead, but is dependent on the concentration of soluble lead in the tissues...." It appears that our mallard ducks may have similar reactions to lead, as no clearly defined lead levels in any tissue could be identified as being diagnostic of death.

Lead Concentrations in Nonwaterfowl Species

Lead concentrations reported for tissues of nonwaterfowl species are mostly consistent with our findings. Lead levels in a young calf dosed with 0.22 g/kg lead carbonate were as follows: liver, 27 ppm; kidney cortex, 260 ppm; spleen, 2.3 ppm; lung, 2.4 ppm; heart, 1.3 ppm; and brain, 1.4 ppm (Allcroft 1951). Osweiler et al. (1971) reported mean lead levels in sick cattle of 30 ppm in liver, 58 ppm in kidneys, and 0.78 ppm in blood. Normal lead levels in kidneys of cattle and sheep range from 0.3 to 1.5 ppm (Allcroft 1950). Fenstermacher et al. (1946) regarded 3 ppm of lead in cattle livers as not significant in terms of toxic effects, 5 ppm as suspicious, and 10 ppm or greater as positive evidence of lead poisoning. Chickens that had ingested lead pellets had 127 to 510 ppm (dry weight) of lead in the liver (Shifrine et al. 1964). Dogs with lead poisoning had lead concentrations of 5 to 50 ppm in the liver (Wilson and Lewis 1963). Clarke and Clarke (1967), referring to domestic animals, state that lead values higher than 25 ppm in kidney cortex and 10 ppm in livers are of definite diagnostic significance.

CONCLUSION

Although it is apparent that lead concentrations in tissues of higher vertebrates are quite variable in relation to dose and effects, approximations can be made of levels in certain tissues that indicate a recent, probably debilitating, exposure to lead. In mallard ducks, a lead level exceeding 3 ppm in the brain, a range of 6 to 20 ppm in the kidney or liver, or an amount of 10 ppm in the clotted blood from the heart indicate acute exposure to lead.

The final diagnosis of lead poisoning should be based on necropsy findings, case history, and appropriate histopathological tests in addition to chemical analysis of tissues.

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A P P E N D I X

Table A1. Lead levels (ppm) in tissues from mallards that died.

Day till death	Organ or tissue											Amount lead eroded(g)
	Kidney	Heart	Liver	Breast muscle	Lung	Gall-bladder	Pancreas	Blood	Spleen	Tibia	Brain	
5	94	1.8	32	0.7	6.3	18.3	222	20.2	13.6	109	2.1	0.200
5	128	2.4	61	0.9	5.2	46.0	208	4.4	33.0	100	2.6	0.347
6	53	1.6	43	0.9	2.8	10.3	117	14.0	12.0	87	2.5	0.709
6	169	3.5	48	1.0	5.0	42.0	90	13.0	16.0	115	3.3	0.314
8	206	2.7	58	0.9	6.6	27.0	185	9.2	15.0	94	4.7	0.272
8	299	3.5	63	1.1	5.2	54.0	72	14.0	14.0	167	4.7	0.641
10	80	1.9	54	0.6	2.5	16.0	119	9.1	20.0	102	4.5	0.169
10	131	3.1	54	1.1	4.0	38.0	697	18.0	38.0	191	6.0	0.755
11	205	2.3	67	3.2	5.3	33.0	594	9.0	60.0	139	2.6	0.670
12	148	3.0	83	1.6	5.7	16.5	289	6.7	30.0	152	3.8	0.384
14	69	1.3	43	0.7	3.5	9.1	77	5.4	15.2	150	5.7	1.043
14	109	3.0	46	0.9	4.0	20.0	302	8.1	26.0	111	3.1	0.700
16	129	1.9	72	2.3	4.2	15.4	300	6.5	36.0	134	3.7	0.786
17	408	4.8	68	1.6	7.6	24.0	1285	17.5	31.0	209	4.7	1.025
18	273	3.1	53	1.7	8.2	13.0	45	14.4	-	168	4.1	0.490
18	388	2.2	76	1.3	5.1	11.9	353	2.7	69.0	130	6.9	0.771
20	-	1.8	75	2.5	4.1	36.0	1551	9.7	27.0	145	7.5	0.680
21	98	1.7	37	1.5	3.9	18.3	512	3.3	18.0	158	10.6	1.332
mean	175.7	2.5	57.4	1.4	4.9	24.9	389.9	10.2	27.9	136.7	4.6	0.627
median	131.0	2.4	56.0	1.1	5.1	19.1	255.5	9.2	26.0	136.5	4.3	0.675

Table A2. Lead levels (ppm) in tissues from mallards that were sacrificed.

Day till death	Organ or tissue											Amount lead eroded(g)
	Kidney	Heart	Liver	Breast muscle	Lung	Gall-bladder	Pancreas	Blood	Spleen	Tibia	Brain	
4	181	3.2	52	1.2	7.8	3.7	129	13.9	15.8	93	4.7	0.341
4	107	4.5	55	1.7	11.9	23.2	224	22.9	10.8	82	3.5	0.266
6	49	2.8	44	1.0	5.9	19.0	275	8.5	18.0	109	4.3	0.343
6	112	4.6	52	1.5	10.4	16.7	209	16.0	29.0	57	2.9	0.309
8	142	3.7	51	1.1	7.3	39.0	212	6.3	41.0	111	4.1	0.636
8	59	2.6	50	1.1	5.9	30.0	91	15.0	-	121	3.2	0.470
10	78	2.7	65	0.9	15.0	18.0	229	14.0	43.0	137	2.8	0.709
10	79	2.0	37	1.6	7.5	28.0	131	11.0	35.0	125	3.0	0.788
12	104	2.6	39	1.1	5.1	6.8	92	7.0	28.0	123	1.7	0.672
12	22	2.5	32	1.0	3.3	15.5	109	4.6	21.0	71	4.1	0.661
14	77	1.8	39	1.1	8.4	16.0	79	10.4	32.0	142	3.5	0.817
14	142	2.2	46	2.3	8.0	16.5	522	16.0	20.8	155	3.7	0.622
16	113	0.4	27	1.5	5.7	10.0	242	7.5	13.6	145	3.7	1.133
16	55	1.6	35	1.5	4.7	17.0	249	12.0	23.0	114	3.9	0.586
18	36	1.5	26	0.9	1.7	7.3	282	5.2	18.3	186	2.9	1.031
18	190	1.2	55	1.6	5.0	15.6	312	9.1	21.0	168	4.1	0.254
20	89	2.2	36	1.5	5.7	12.4	162	7.3	33.0	212	7.2	0.646
20	243	1.8	52	1.8	4.0	97.5	284	3.7	56.0	130	6.5	1.011
mean	104.3	2.4	44.1	1.4	6.8	21.8	212.9	10.6	27.0	126.7	3.9	0.627
median	95.6	2.4	45.0	1.4	5.9	16.6	218.0	10.7	22.0	124.0	3.7	0.641

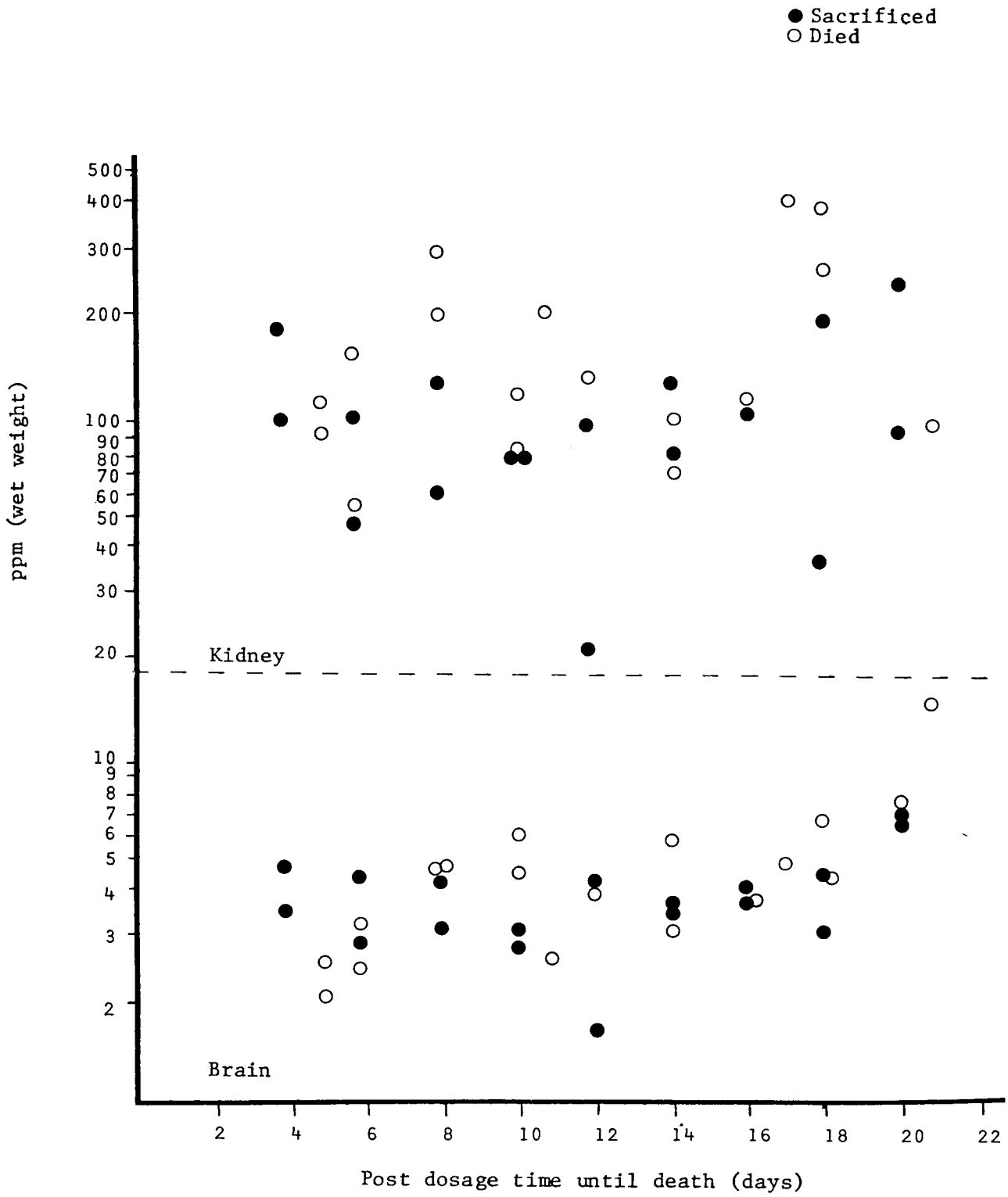

Fig. 1. Log concentrations of lead in brains and kidneys of lead-dosed game-farm mallards.

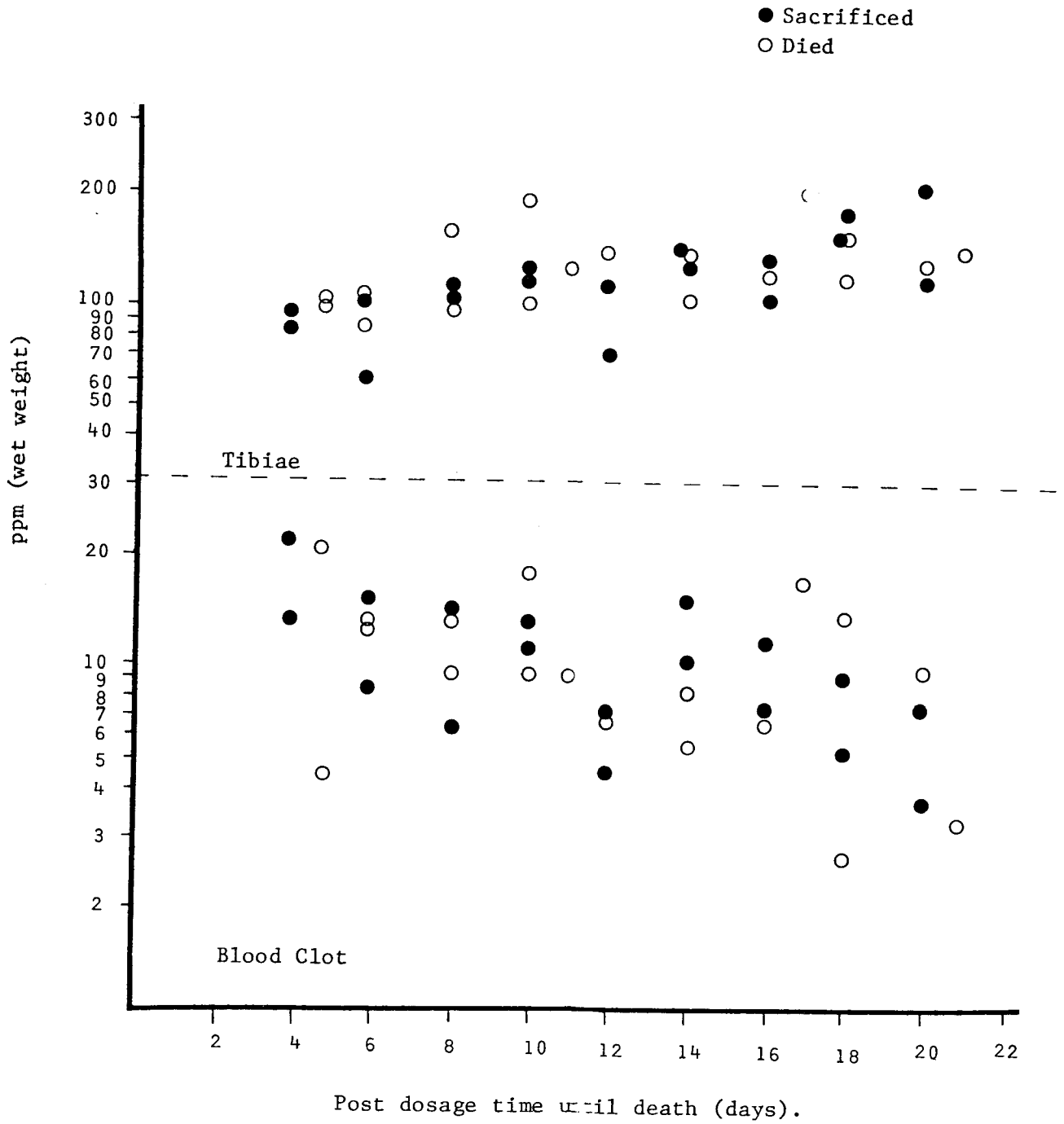

Fig. 2. Log concentrations of lead in clotted blood and tibiae of lead-dosed game-farm mallards.

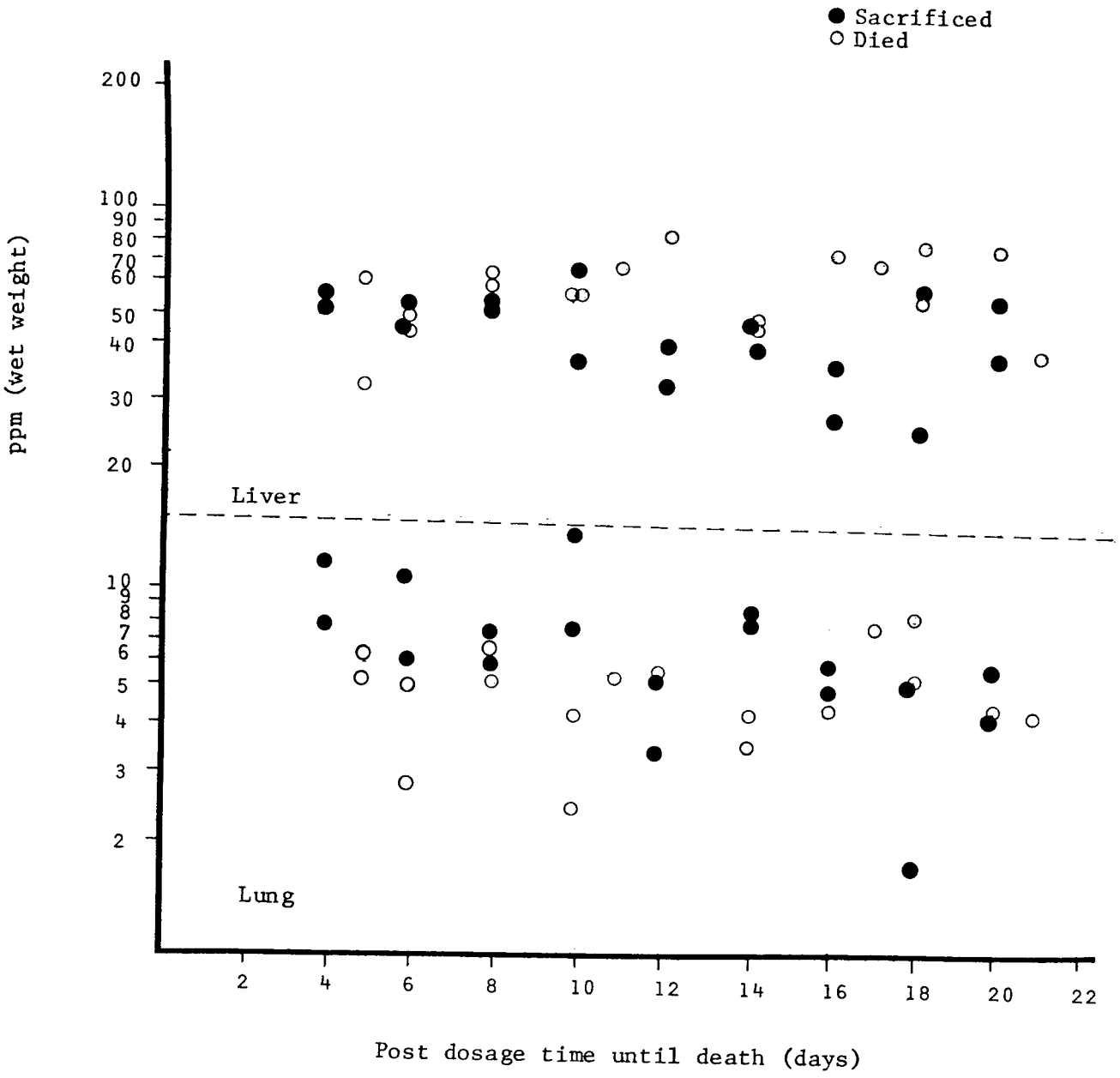

Fig. 3. Log concentrations of lead in lung and liver tissue of lead-dosed game-farm mallards.

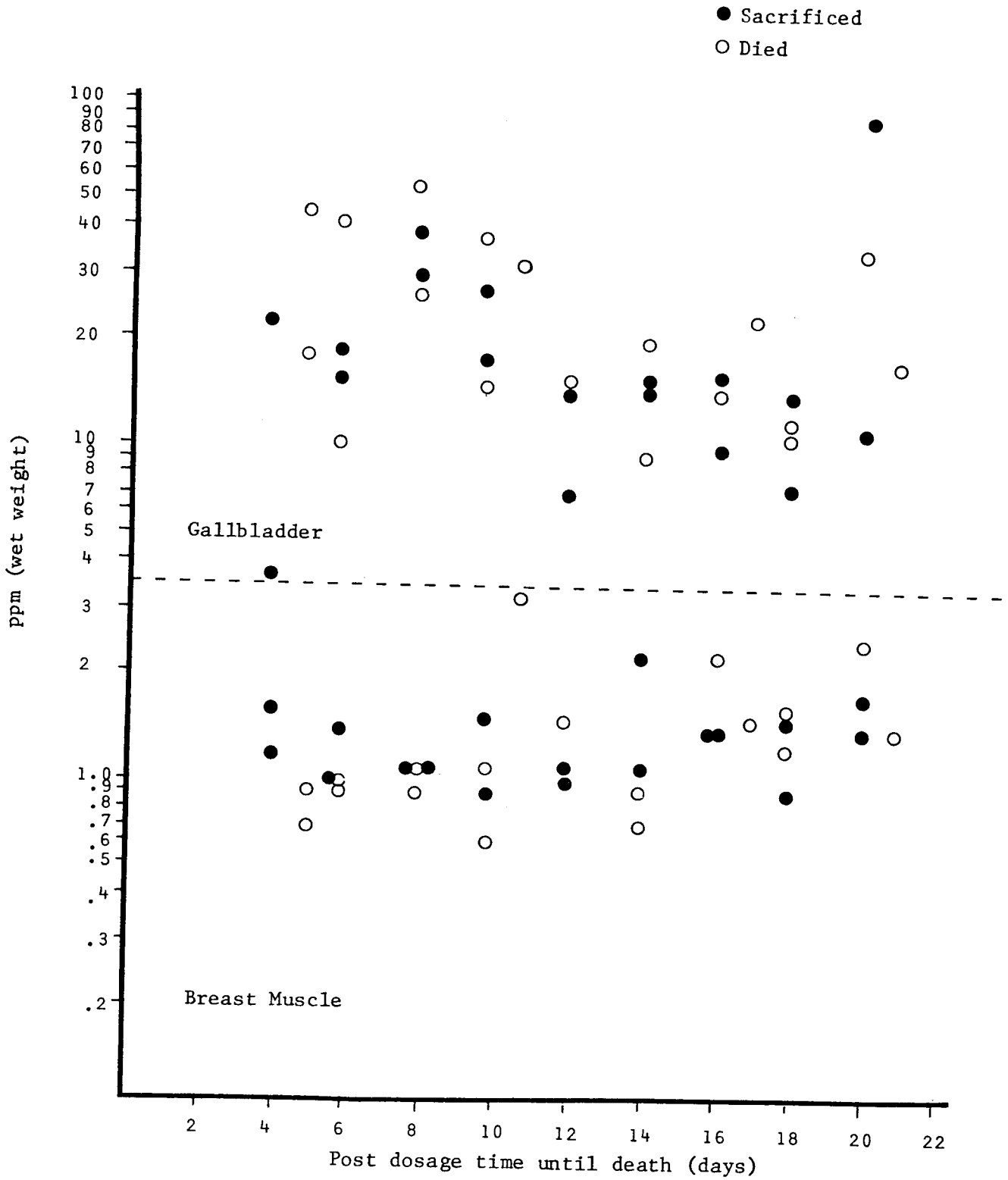

Fig. 4. Log concentrations of lead in breast muscle and gallbladder of lead-dosed game-farm mallards.

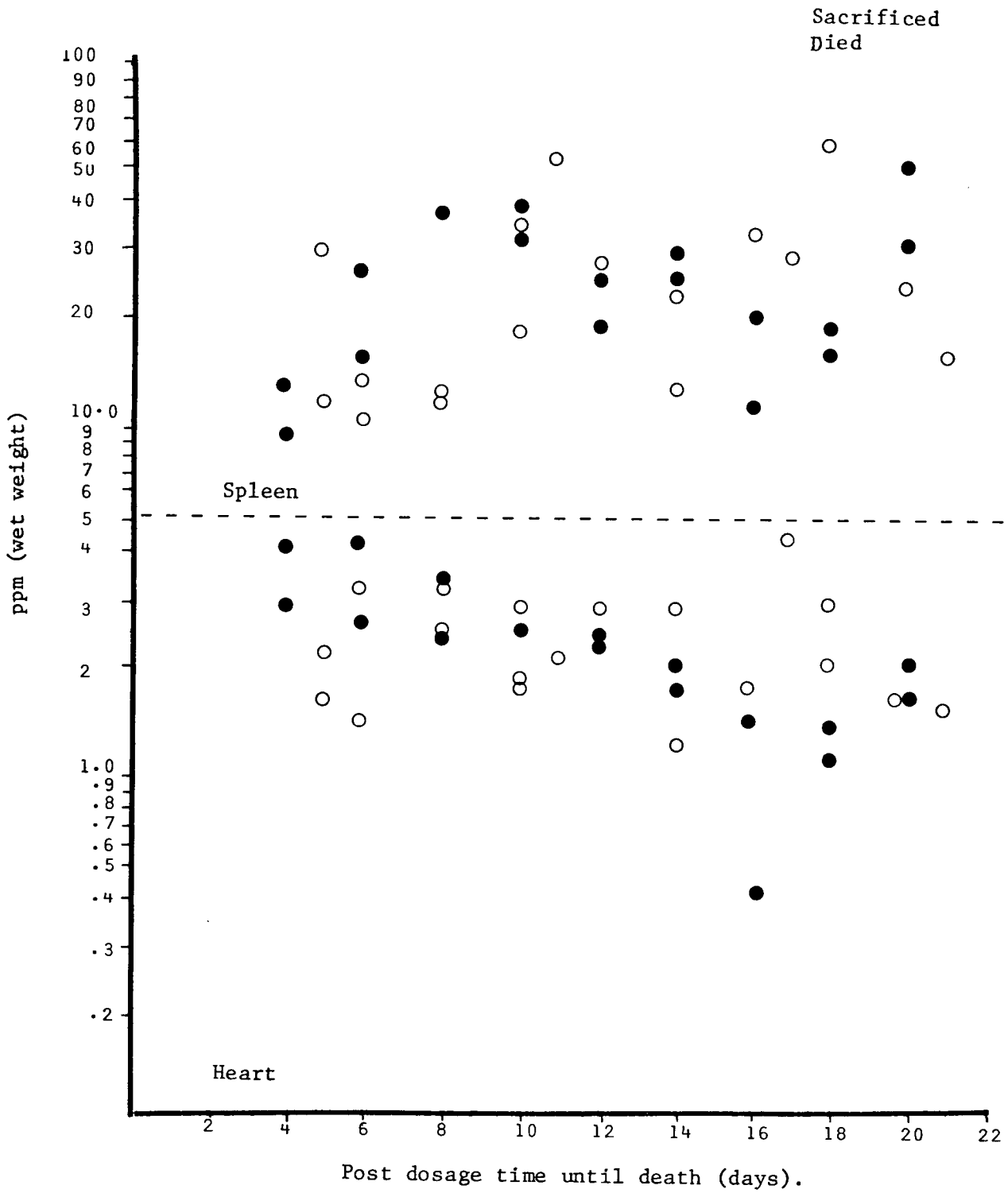

Fig. 5. Log concentrations of lead in heart and spleen of lead-dosed game-farm mallards.

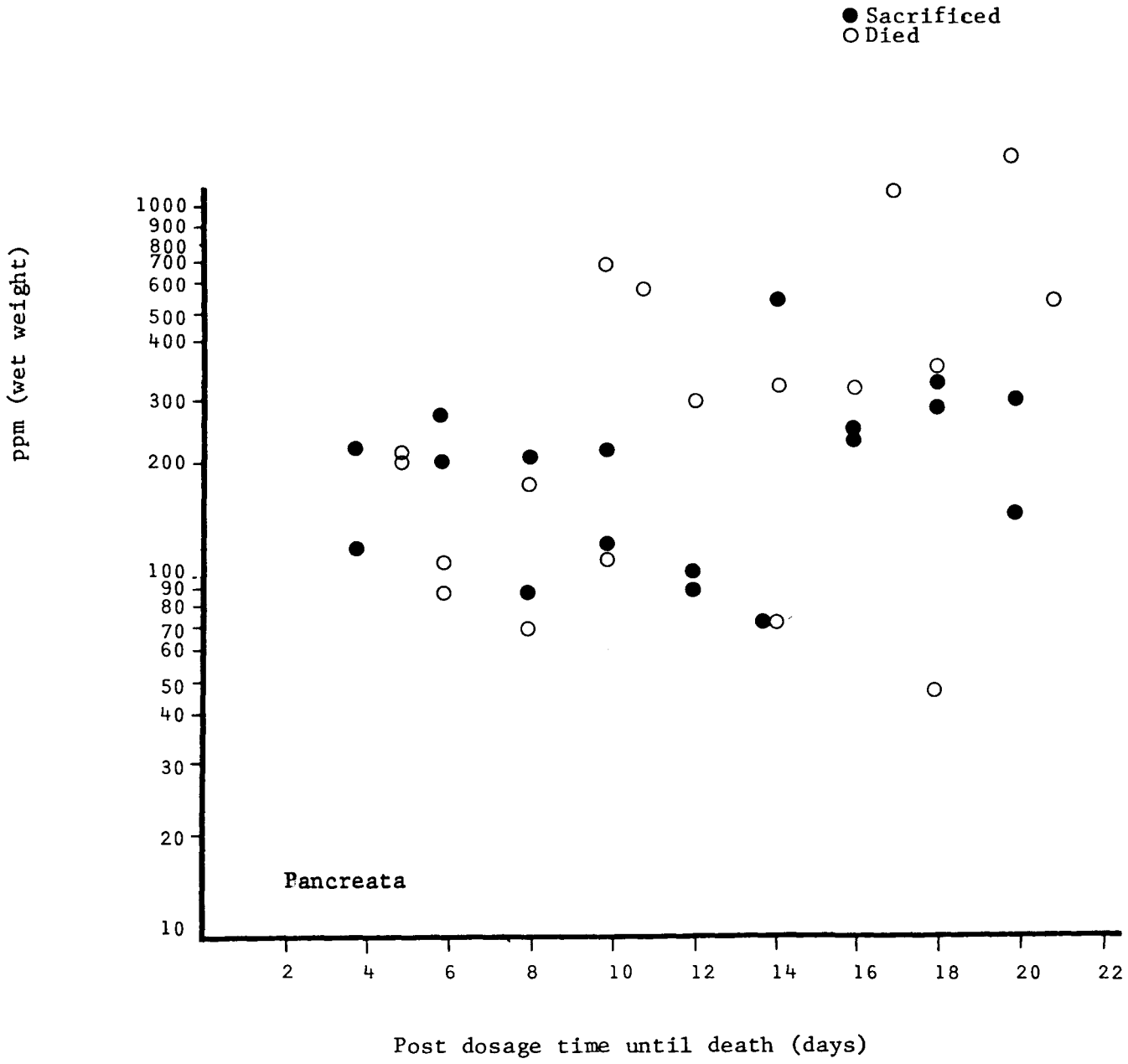

Fig. 6. Log concentrations of lead in the pancreata of lead-dosed game-farm mallards.

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