AN INVESTIGATION INTO THE EFFECTS OF LEAD POISONING ON BALD EAGLES AND OTHER RAPTORS:

FINAL REPORT

State: Minnesota

Project Number: Endangered Species Program Study 100A-100B

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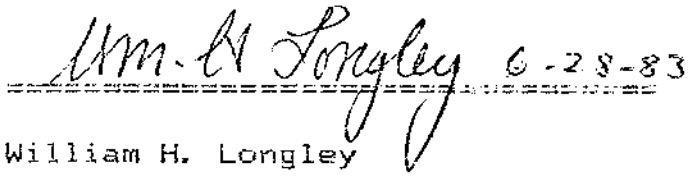
Principal Investigator: Patrick T. Redig

Co-Investigators: Gary E. Duke
Samuel Schwartz
Ellen Lawler

SIGNATURES:

Patrick T. Redig DVM, PhD
Assistant Professor
University of Minnesota



William H. Longley
Federal Aid Coordinator
Minnesota Department of
Natural Resources

2) Description of the Problem. Lead poisoning occurring as a result of the ingestion of lead shotgun pellets embedded in the flesh of prey species as well as the ingestion of contaminated flesh of animals that have died from lead poisoning has gained recognition as a discreet mortality causing factor among Bald Eagles (Haeliaaetus leukocephalus) wintering in the vicinity of large waterfowl concentrations (Jacobson et al., 1977; Patee and Hennes, 1983) and has been shown to affect other species of raptors and scavenging birds (Locke et al., 1969; Redig et al., 1980). Lead has been demonstrated as the likely cause of death in increasingly numerous instances (Jacobson et al., 1977; Locke et al., 1969;). Patee and Hennes (1983) cited recently obtained post mortem evidence from over 600 Bald Eagles and concluded that lead poisoning ranked fourth behind shooting, impact injuries, and electrocution as a mortality causing facter. The question of the effect of chronic low level and sub-clinical exposure to lead on physiological and behavioral functions of eagles remains unanswered. Evidence for the occurrence of chronic and regular exposure to lead has been demonstrated through the finding of large numbers of spent shotgun pellets in the castings collected from underneath bald eagle roosts in the vicinity of wintering waterfowl concentrations. Platt (1976) found 71 % of the castings from a roost in Utah to contain spent shot while Dunstan (1974) recovered spent shot in 50-60 % of the castings they examined from midwestern roost sites. Thus ingestion of lead is not immediately and always fatal, but the regular daily ingestion and egestion provides the means for the intake of small amounts of lead on a regular basis, the effects of which are affected by the kinetics of absorption and excretion. To the extent that the former exceeds the latter, the effects are cumulative. The effects of low levels of lead include neurological dysfunction, behavioral and learning

aberrations, anemia, and increased susceptibility to diseases and other mortality causing factors (Reiser and Temple, 1980). The extent to which Bald Eagles have been so affected are presently unknown. Therefore we perceived the need to further assess the morbidity of lead poisoning among wild eagles and investigate the effects of sub-lethal plasma concentrations on hematological, serum chemical, and immunological parameters of Bald Eagles and other species of raptors.

3) Review of Prior Research. Several investigations have been undertaken in raptors to study rates of absorption and tissue deposition of orally administered lead (Patee et al., 1981), the effects on porphyrin metabolism (Reiser and Temple, 1981) and serum enzyme activity (Hoffman et al., 1981; Meister and Koster, 1981). In all of these studies, sufficient doses of lead were given to produce clinical signs of toxicity as well as death. The question of whether raptors continually exposed to lead in sub-lethal doses are experiencing any reduced physiological functions remains unanswered. One study (Stendall, 1980) involved the feeding of shotgun pellets to kestrels on an irregular basis over a 60 day period produced no overt clinical signs. Post mortem analysis of the livers showed approximately a 5-fold increase in lead concentration compared to unexposed controls. No other parameters were measured in this study.

Among parameters that may be affected by lead, porphobilinogen synthase (PBG-s), an enzyme in the hemoglobin synthetic pathway, has been demonstrated to have notable and unique sensitivity to lead in a variety of mammalian and avian species (Finley et al., 1976; Hoffman et al., 1981; Meisters and Kosters, 1981). Zn-bound protoporphyrin, a product formed by an alternate pathway when PBG-s is blocked by lead, was found to be significantly increased in raptors chronically

poisoned with lead acetate (Reiser and Temple, 1981). Immunosuppression has been demonstrated in rats and mice exposed to various concentrations of lead acetate in their drinking water (Blakely and Archer, 1981). Immune functions assays in other than domesticated avian species have not been perfected, much less the assessment of the affects of lead on such functions.

4) Objectives. The following objectives were addressed in this study:

a. Refinement of immune functions assays (lymphocyte blastogenesis and antibody response to sheep red blood cells) for use in raptors.

b. Experimental evaluation of the effects of chronic sub-lethal, sub-clinical exposure to lead acetate on hematological, serum chemical, and immunological function in Red-tailed hawks (Buteo jamaicensis).

c. Determination in wild Bald Eagles admitted to the Raptor Research and Rehabilitation Program at the University of Minnesota of the plasma concentrations of lead, Zn-bound protoporphyrin, porphobilinogen synthase enzyme activity, hemoglobin concentration, hematological, and serum chemical parameters in order to assess the morbidity and physiological significance of elevated lead plasma concentrations in these birds

5) Approaches utilized. The following procedures were undertaken:

a. The optimal experimental parameters for conducting the whole blood lymphocyte stimulation assay were developed in three species of raptor, Bald Eagles, Red-tailed Hawks, and Great-horned owls. Parameters such as mitogen concentration, isotope concentration, incubation temperature and time, and antibiotic selection in the culture media were examined in checkerboard fashion to select the optimum parameters.

b. Twelve permanently crippled Red-tailed hawks were divided into two groups of six, a control group and an experimental group. The experimental group was given lead acetate in daily doses of 1.5 mg/kg by oral gavage; the control group received similar volumes of sodium acetate. These treatments were conducted for 24 days. Blood samples were collected one week before the initiation of the lead treatments, on the day of the first lead treatment, and at weekly intervals thereafter for 6 weeks, bimonthly for the next month, and at monthly intervals for the next 3 months. Hematology, lymphocyte stimulation, PBG-s activity, concentration of Zinc Protoporphyrin (ZPP), and total white cell counts were determined. At the

end of the experiment, the birds were killed and examined. At two points in the experiment following the cessation of lead dosing, 0.1 ml of a 50% suspension of washed sheep red blood cells was administered to each of the birds. One, two, and three weeks later blood samples were withdrawn and the plasma assayed for antibodies against sheep red blood cells by the hemagglutination methods described by Witlin (1971).

c. Blood samples were taken from incoming injured Bald Eagles at the time of admission and at approximately two week intervals throughout the course of their convalescence. The blood was assayed for lead concentration in two different laboratories. PBG-s activity, ZPP concentration, packed cel volume, hemoglobin concentration, and a battery of serum chemistry parameters were also determined. Correlations were drawn between the values for these parameters and lead concentration and between lead concentration, nature and severity of their injury, and the final outcom of the rehabilitation effort. In the 1980-81 season 42 samples were taken from 12 eagles and in 1981-82, 91 samples were taken from 20 different eagles, with as many as 8 sampling intervals being obtained from a single bird. The data is incomplete owing to circumstances beyond our control including breakage of tubes and reagent contamination in the laboratory conducting the assays (see letters attached at the end). Rigorous statistical analysis of the data was not undertaken since the samples were derived from heterogenous group of birds of varying origin, sex, age, and extenuating clinical circumstances that may or may not have been related t lead poisoning.

6. Findings.

a. Optimization of Immune Function Assays

The purpose of this research was to optimize the various parameters involved in mitogen-induced whole blood lymphocyte stimulation assays for Bald Eagles, Red-tailed Hawks, and Great-horned Owls. Blood samples were drawn from healthy, but in some cases, permanently crippled birds of prey and cultured by standard techniques. Pre- and post-labeling incubations times, blood dilution, concentrations of phytohemagglutinin and concanavalin A were tested for their effects on the stimulation index. An antibiotic combination of gentamicin and amphotericin B yielded very low stimulation indices with lymphocytes from Bald Eagles, but not with lymphocytes from Great-horned Owls or Red-tailed Hawks. Penicillin and streptomycin at a final concentration of 10 ug/ml in each well caused no such depression. Lymphocytes from all 3 species yielded maximal

responses with a 48 hour pre-label incubation period and a 12-to-16 hour post incubation period at 41C and a 1:20 blood dilution. Optimal mitogen concentrations for lymphocytes from Bald Eagles, Red-tails and Great-horned Owls were 25 ug/well, 25 ug/well, and 10 ug/well phytohemagglutinin, respectively, and 2.5 ug/well, 10 ug/well, and 10 ug/well concanavalin A, respectively. Differences in stimuation index were not seen between tritiated thymidine or 125-iodinated-thymidine. The optimal concentration of tritiated thymidine was in the range of 0.06 to 0.125 uCi/well.

A completed manuscript which provides further details on this technique and which has been accepted for publication by the American Journal of Veterinary Research is available for review.

b. The effects of chronic exposure to sub-lethal concentrations of lead acetate on porphyrin synthesis and immune function in Red-tailed Hawks.

The effects of 24 days of dosing with sub-lethal amounts of lead acetate on porphyrin metabolism and immune function in Red-tailed Hawks was investigated. Neither phytohemagglutinin or concanavalin A (T-cell responses) nor humoral antibody response (B-cell) to the injection of sheep red blood cells was demonstrably affected. Plasma lead concentrations were not measured, however, free and Zn-bound protoporphyrins were greatly elevated by the seventh day and there was a concurrent depression of activity of the enzyme, porphobilinogen synthase. The latter was decreased to 20% of normal response (1250 nM PBG/ml RBC/hour to about 240 nM PBG/ml RBC/hour) by the 20th day of dosing. Hematocrits and hemoglobin concentrations were not significantly altered during this time period, although, had the administration of lead continued, it is likely they would have begun to decline owing to the great

depression of PBG-s activity. Following the cessation of the administration of lead acetate, free prophyrin levels returned to control levels by the end of one week, Zn-bound protoporphyrin slowly decreased to control values over thirty days, and PBG-s slowly recovered to pre-dosing levels of activity in five weeks. The hawks themselves remained clinically asymptomatic throughout the duration of this experiment, despite severe alterations in their hemoglobin synthesizing enzyme system. Particular note is made of the extended period of time required for these systems to return to normal levels of function following the cessation of administration of this very small amount of lead acetate.

A manuscript of this experiment has been completed for submission for publication and is available upon request.

c. Determination of the concentration and significance of lead residues in the plasma of wild Bald Eagles.

A comparison of the values for lead concentration reported by the two laboratories for the 1981-82 data is presented in table 1. There was not a consistant variation in the way one laboratory reported the results compared to the other, hence a considerable degree of judgement had to be employed by the investigators in determining which values were most likely to be correct in cases where there was great disparity. All of the data collected are displayed in tables 2-A and 2-B for years 1980-81 and 1981-1982 respectively. The most notable feature of these data is the time course whereby lead concentrations were reduced over the course of time during hospitilization and the other parameters returned to normal. For further data analysis, only those values obtained at the time the birds were admitted were considered. The results of such analysis are presented in tables 3A and 3B for each of the years in

ascending order of lead concentration. Where significant discrepancies existed in the reports between the two laboratories, the lowest reported lead concentration was utilized except where simultaneously or subsequently collected data indicated a higher value may be more correct. These results were broken down into the following categories, based on lead concentration:

1. Less than 0.2 ppm -- Background or normal category

2. 0.201 to 0.6 ppm -- evidence of chronic, subclinical exposure. Values in this range appear to decrease rapidly upon cessation of exposure and there don't appear to be any lasting effects.

3. 0.601 to 1.0 ppm -- evidence of chronic, clinical exposure. The birds are showing clinical signs and require chelation therapy to expeditiously reduce plasma concentrations of lead along with other supportive care. Recovery is to be expected.

4. Greater than 1.0 ppm -- evidence for serious exposure, probably of an acute nature. At the lower end of this range the birds will recover over a long period of time provided they are given chelation therapy. Above 5.0 ppm death is the expected outcome even if there is an initial response to therapy.

These categories differ slightly from those proposed by Patee and Hennes (1983), which consisted of 3 levels (less than 0.1, 0.1 to 1.0, and greater than 1.0 ppm). The addition of our category 3 was necessary to accomodate the clinical observations associated with this group of birds.

Tables 4A and 4B contain the raw data when broken down into the four categories delineated above. These data are further summarized in table 5. Variations seen in PBG and ZPP correlated well with lead concentration. As lead increased, PBG decreased and ZPP increased. As shown above in the Red-tail experiment, there is a time lag between the onset of the exposure to lead and the change in the value of these two parameters. Hence, greatly elevated values for ZPP are indicative of more chronic exposure to lead and similarly extreme decreases in PBG are indicative of longer term exposure. These trends were most evident in the Group 3 birds (table 5) where the PBG values were the lowest

(range 0-79) and the ZPP values were the highest (range 94-448). Thus alterations of these magnitudes in these two parameters may be the most indicative of long term lower level exposure to lead. In contrast, the Group 4 birds (table 5) had only moderate alterations in these parameters despite the fact that all were showing signs of severe lead poisoning. Apparently the acuteness of the exposure resulted in the development of clinical signs before PBG and ZPP were significantly altered. Only 1 of the 4 Group 4 birds recovered from lead poisoning.

Whereas lead was responsible outright for the death of 3 eagles in this study, it was a clinically compromising factor in many more cases. In table 6A and 6B, the admission data is grouped into 1 of 4 clinical categories according to the condition of the bird as follows:

1. Normal birds, either received as healthy fledglings or convalesced birds about ready to be released.

2. Birds with minor injuires, simple fresh fractures uncomplicated by serious infection or emaciation.

3. Birds with complicated and/or open repairable fractures, exhibiting weight loss of no more than 25 %, having no significant depression and a good appetite.

4. Birds with severe traumatic injury or illness, exhibiting depression, weight loss, and anorexia.

The data from table 6A and 6B are summarized in table 7. A large number of the most severely injured eagles in clinical categories 3 and 4 (table 7) had lead residues in the range of 0.2 to 0.6 ppm. Of the 5 birds with lead residues in ths range between these two categories, 3 were trap victims, one was hit by a car, and one was shot. If we accept the 0.1 ppm lower cut-off of Pattee and Hennes, then virtually all of the injured birds had significantly elevated lead residues at the time of admission (table 6A and 6B). PBG-s depression in those cases where no lead values are available may be interpreted as indicative of a

significant plasma lead burden. Thus a conclusion may be drawn that suggests widespread lead poisoning in the eagle population reduces the foraging efficiency of the birds and renders them more susceptible to serious injury causing modes of food acquisition such as scavenging trap baits and dead carcasses from along roads.

CONCLUSIONS

1. Significantly elevated plasma lead residues are present in the majority of eagles involved in serious injuries and in fact may play a role in the circumstances leading to such injury.

2. Lead residues above 1.0 ppm do occur in the eagle population and are associated with clinical lead poisoning. Many of these affected birds can be expected to die or be partially crippled, even with treatment, due to permanent damage to organ and enzyme systems.

3. Where lead residues are moderately elevated, PBG-s is significantly decreased and ZPP is significantly increased, such an affected bird has had long-term, repeated exposure to lead.

4. Where lead residues are approaching 1.0 ppm or greater, the exposure to lead has probably been acute unless PBG-s and ZPP are significantly altered.

5. Significant disparity in the values for lead concentration exist between reputable laboratories, hence, other parameters should also be measured to accurately assess the lead status of individual birds.

6. The immune system is more refractory to the effects of lead than are the hemoglobin synthesizing mechanisms.

7. Physiological recovery from even very low exposure to lead requires 4 - 5 weeks and lags far behind the outward appearances of clinical recovery.

Table 1. COMPARISON OF DATA REPORTED BY TWO LABORATORIES FOR LEAD RESIDUES IN BALD EAGLE BLOOD

SPECIES	CASE #	DATE OF SAMPLE	[LEAD] (ppm)	LABORATORY (Lab1)	LABORATORY (Lab2)	COMMENT
BE	H-257	11-20-81	0.32		x	
	-----	"" ""	0.449	x		Lab1=Lab2
	-----	"" ""	0.239*	x		Note duplic. samples
BE	H-275	10-20-81	0.21		x	
	-----	"" ""	0.446	x		Lab1=Lab2
GE	H-280	11-23-81	0.28		x	
	-----	"" ""	-----	x		Sample contaminated
	-----	12-16-82	neg		x	
BE	H-297	10-29-81	0.15		x	
	-----	"" ""	0.203	x		Lab1=Lab2
	-----	"" ""	-----	x		Repli. contaminated
BE	H-305	11-02-81	0.50		x	
	-----	"" ""	0.205	x		Lab1 much less than Lab2
	-----	"" ""	----	x		One repl. contaminated
	-----	11-18-81	0.34		x	
	-----	"" ""	0.554	x		Lab1 greater than Lab2
	-----	"" ""	-----	x		One repl. contaminated
	-----	12-16-81	0.22		x	
	-----	"" ""	-----	x		Sample contaminated
	-----	12-31-81	-----	x		Sample contaminated
	-----	01-18-82	0.250	x		
	-----	02-16-82	-----	x		Sample contaminated
GE	H-307	11-02-81	1.06		x	
	-----	"" ""	0.048	x		Lab1 much less than Lab2
		"" ""	-----	x		One repl. contaminated
	-----	11-16-81	0.10		x	
	-----	"" ""	----	x		Sample contaminated
	----	12-15-81	0.40		x	
	-----	01-04-82	0.19		x	
	-----	"" ""	-----	x		Sample contaminated
	-----	01-21-82	0.032	x		
	-----	02-03-82	-----	x		Sample contaminated

BE	H-320	11-05-81	1.79		x	
	-----	"" ""	0.175	x		Lab1 much less than Lab2
	-----	"" ""	-----	x		Repli. contaminated
	-----	11-19-81	0.54		x	
	-----	"" ""	-----	x		Sample contaminated
BE	H-327	11-06-81	0.35		x	
	-----	"" ""	0.420	x		Lab1=Lab2
	-----	11-18-81	0.13		x	
	-----	"" ""	-----	x		Sample contaminated
BE	H-329	11-11-81	0.43		x	
	-----	"" ""	0.356	x		Lab1=Lab2
	-----	"" ""	-----	x		Repli. contaminated
	-----	11-23-81	0.24		x	
	-----	12-30-81	0.081		x	
	-----	12-30-81	0.438	x		
	-----	01-14-82	neg		x	
	-----	"" ""	-----	x		Sample contaminated
BE	H-332	11-11-81	0.16		x	
	-----	"" ""	0.187	x		
	-----	"" ""	-----	x		Repli. contaminated
	-----	12-31-82	-----	x		Sample contaminated
	-----	01-04-82	-----	x		Sample contaminated
	-----	01-27-82	0.269	x		
BE	H-341	11-16-81	0.40		x	
	-----	"" ""	0.049	x		Lab1 much less than Lab2
	-----	"" ""	-----	x		Repli. contaminated
BE	H-345	11-16-81	0.37		x	
	-----	"" ""	0.260	x		
BE	H-350	11-18-81	0.78		x	
	-----	"" ""	1.186	x		Lab1 greater thanLab2
	-----	"" ""	-----	x		Repli. contaminated
	-----	12-15-81	0.30		x	
	-----	"" ""	0.46		x	
	-----	"" ""	-----	x		Sample contaminated
	-----	01-14-82	-----	x		Sample contaminated
	-----	01-27-82	-----	x		Sample contaminated
BE	H-354	11-18-81	0.60		x	
	-----	"" ""	0.076	x		Lab1Lab2
	-----	"" ""	-----	x		Repli. contaminated
	-----	01-06-82	0.103		x	
	-----	"" ""	0.098	x		
	-----	01-21-82	0.201		x	
	-----	"" ""	0.117	x		
	-----	02-04-82	0.049	x		
	-----	02-16-82	-----	x		Sample contaminated
BE	H-356	11-19-81	0.40		x	
	-----	11-19-81	0.120	x		Lab1 less than Lab2
	-----	"" ""	-----	x		Repli. contaminated

	-----	12-31-81	0.331		x	
	-----	"" ""	-----	x		Sample contaminated
	-----	01-13-81	0.063	x		
	-----	02-11-82	broken	x		
BE	H-357	11-20-81	lost			
	-----	12-04-81	lost			
	-----	12-30-81	0.30		x	
	-----	"" ""	0.421	x		Lab1=Lab2
	-----	01-14-82	0.165	x		
	-----	02-11-82	-----	x		Sample contaminated
BE	H-358	11-24-81	0.11		x	
	-----	01-05-82	0.046	x		
	-----	01-06-82	0.054	x		Nearly replic. samples
	-----	01-28-82	0.114	x		
BE	H-372	12-04-81	0.35		x	
BE*	H-387	12-16-81	1.44		x	
	-----	"" ""	4.69	x		Lab1 much greater than Lab2
	-----	01-05-82	1.981	x		
	-----	01-20-82	0.450		x	
	-----	"" ""	0.296	x		Probably same sample
	-----	02-04-82	0.109		x	
	-----	"" ""	0.269	x		Probably same sample
BE*	H-382	19-09-81	15.0		x	Dying of Pb at admiss.
BE*	H-391	12-31-81	0.35		x	
	-----	12-31-81	2.061	x		
	----	01-14-82	0.447	x		
	-----	01-27-82	0.316	x		
BE	H-393	12-17-81	lost		x	
	-----	01-06-82	0.247		x	
	-----	"" ""	0.116	x		
	-----	01-21-82	0.228		x	
	-----	"" ""	0.302	x		
	-----	02-04-82	0.117	x		
	-----	02-16-82	3.244	x		Suspicious value
BE	H-400	12-21-81	0.26		x	
	-----	01-06-82	-----	x		Sample contaminated
BE	H-401	12-24-81	0.41		x	
BE	I-24	02-09-82	0.228	x		
	-----	03-24-82	0.168	x		

* indicates eagles admitted with signs of lead poisoning.

Table 2A. Raw data for 1980-81*

1	2	3	4	5	6	7	8	9	10	11	12
00001	G-74	BE	4	05/30/80	0	0.000	0	0	0.00	0	ds
00002	G-82	BE	4	06/10/80	0	0.000	0	0	0.00	0	dd
00003	G-206	BE	1	12/30/80	10	0.053	0	0	0.00	0	rl
00004	G-228	BE	1	12/30/80	8	0.203	510	0	0.00	68	rl
00005	G-234	BE	2	11/04/80	0	0.000	0	35	0.00	49	scp
00006	G-234	BE	2	11/04/80	3	0.047	668	0	0.00	64	scp
00007	G-234	BE	1	11/04/80	16	0.000	770	42	0.00	43	hcp
00008	G-234	BE	1	11/04/80	17	0.000	1025	0	0.00	40	hcp
00009	G-236	BE	2	11/05/80	0	0.000	685	39	0.00	38	erl
00010	G-236	BE	1	11/5/80	7	0.004	89	0	0.00	105	erl
00011	G-236	BE	1	11/05/80	11	0.004	0	45	0.00	161	arl
00012	G-246	BE	4	11/11/80	4	0.000	0	0	0.00	0	dd
00013	G-246	BE	4	11/11/80	4	0.000	0	0	0.00	0	dd
00014	G-263	BE	3	11/20/80	0	0.000	748	41	0.00	56	evl
00015	G-263	BE	3	11/20/80	4	0.047	0	40	0.00	254	erl
00016	G-263	BE	3	11/20/80	4	0.047	760	40	0.00	60	erl
00017	G-263	BE	2	11/20/80	7	0.000	324	38	0.00	94	erl
00018	G-263	BE	1	11/20/80	10	0.000	1139	42	0.00	77	erl
00019	G-263	BE	1	11/20/80	11	0.000	1281	40	0.00	69	arl
00020	G-267	BE	2	11/19/80	0	0.660	79	45	0.00	448	erl
00021	G-267	BE	2	11/19/80	4	0.475	88	0	0.00	40	erl
00022	G-267	BE	1	11/19/80	8	0.001	182	44	0.00	56	erl
00023	G-267	BE	1	11/19/80	9	0.001	0	45	0.00	50	arl
00024	G-272	BE	1	11/21/80	0	0.001	0	47	0.00	63	hcp
00025	G-272	BE	1	11/21/80	5	0.001	622	0	0.00	89	hcp
00026	G-272	BE	1	11/21/80	11	0.001	0	47	0.00	60	hcp
00027	G-272	BE	1	11/21/80	12	0.001	350	0	0.00	47	hcp
00028	G-278	BE	4	11/25/80	0	0.230	353	0	0.00	243	dd
00029	G-297	BE	1	12/08/80	6	0.135	181	44	0.00	56	arl
00030	G-301	BE	4	12/10/80	0	0.070	0	33	0.00	120	eds
00031	G-301	BE	4	12/10/80	8	0.000	697	36	0.00	58	eds
00032	G-302	BE	4	12/15/80	0	0.000	629	45	0.00	161	scp
00033	G-302	BE	4	12/15/80	5	0.000	0	33	0.00	83	scp
00034	G-302	BE	4	12/15/80	8	0.000	1352	29	0.00	109	scp
00035	G-302	BE	4	12/15/80	9	0.000	616	29	0.00	121	scp
00036	G-314	BE	2			0.000	0	0	0.00	0	erl

00037	H-8	BE	4	01/12/81	0	2.900	197	18	0.00	76	idd
00038	H-9	BE	3	01/20/81	3	0.056	656	41	0.00	65	edd
00039	H-9	BE	2	01/20/81	4	0.000	816	0	0.00	43	edd
00040	H-81	BE	2	05/12/81	0	0.000	753	50	0.00	42	arl
00041	H-127	BE	1	06/26/81	0	0.000	1458	32	0.00	38	arl
00042	H-128	BE	1	06/26/81	0	0.000	1029	31	0.00	6	arl

*1=record number, 2=case number, 3=species, 4=clinical category, 5=admission date, 6=clinical interval (weeks in hospital), 7=lead concentration, ppm, 8=PBG-s activity (nMPBG formed/ml of RBC's/hour, 9=hematocrit (%), 10=hemoglobin concentration (gm/100ml), 11=Zn-bound protoporphyrin (ug/dl), 12=fate of the bird (dd=died, ds=destroyed, rl=released, cp=crippled, i=immediately, e=eventually, a=at time of, s=sick, h=healthy.

Table 2B. Raw data for 1981-82.

1	2	3	4	5	6	7	8	9	10	11	12
00001	H-275	BE	4	10/20/81	2	0.446	757	40	12.4	64	ds
00002	H-280	GE	2	10/22/81	0	0.000	1430	30	9.5	64	erl
00003	H-280	GE	2	10/22/81	1	0.000	1430	30	9.5	52	erl
00004	H-280	GE	2	10/22/81	2	0.000	1564	39	13.1	33	erl
00005	H-280	GE	1	10/22/81	4	0.280	1523	38	13.3	33	erl
00006	H-280	GE	1	10/22/81	7	0.000	2048	39	13.0	32	arl
00007	H-305	BE	2	10/30/81	0	0.500	384	42	12.6	36	erl
00008	H-305	BE	2	10/30/81	2	0.400	776	43	15.3	43	erl
00009	H-305	BE	2	10/30/81	4	0.000	596	42	13.7	39	erl
00010	H-305	BE	2	10/30/81	6	0.220	1044	38	11.9	31	erl
00011	H-305	BE	2	10/30/81	8	0.000	1070	38	12.6	56	erl
00012	H-305	BE	2	10/30/81	10	0.000	1078	36	12.1	42	erl
00013	H-305	BE	2	10/30/81	12	0.000	1427	37	12.3	66	erl
00014	H-305	BE	2	10/30/82	14	0.000	998	42	14.0	52	erl
00015	H-307	GE	2	10/30/81	0	0.040	1994	31	10.4	90	erl
00016	H-307	GE	2	10/30/81	2	0.100	2054	39	12.8	120	erl
00017	H-307	GE	2	10/30/81	4	0.000	2093	42	14.6	80	erl
00018	H-307	GE	2	10/30/81	6	0.400	2218	45	15.0	51	erl
00019	H-307	GE	2	10/30/81	10	0.000	2333	40	12.7	32	erl
00020	H-307	GE	2	10/30/81	12	0.000	2596	44	15.4	47	erl
00021	H-307	GE	1	10/30/81	16	0.000	1929	49	16.1	59	erl
00022	H-320	BE	3	11/05/81	0	0.175	757	45	15.7	32	erl
00023	H-320	BE	2	11/05/81	2	0.540	1196	44	14.3	29	arl
00024	H-327	BE	3	11/06/81	0	0.350	756	39	13.1	42	edd
00025	H-327	BE	3	11/06/81	2	0.130	1347	38	12.6	40	edd
00026	H-327	BE	3	11/06/81	6	0.000	1428	36	11.4	55	dd
00027	H-329	BE	2	11/06/81	1	0.360	459	25	0.0	69	hcp
00028	H-329	BE	2	11/06/81	3	0.240	898	41	15.7	40	hcp
00029	H-329	BE	2	11/06/81	3	0.000	392	38	12.3	48	hcp
00030	H-329	BE	2	11/06/81	8	0.081	1157	48	15.4	67	hcp
00031	H-329	BE	2	11/06/81	9	0.001	1208	41	13.8	42	hcp
00032	H-332	BE	2	11/09/81	0	0.170	1382	42	14.5	43	erl
00033	H-332	BE	2	11/09/81	2	0.001	1546	40	13.8	44	erl
00034	H-332	BE	2	11/09/81	4	0.000	1501	39	12.8	49	erl
00035	H-332	BE	2	11/09/81	7	0.000	831	42	14.2	43	erl
00036	H-332	BE	1	11/09/81	10	0.269	1296	41	13.7	34	arl
00037	H-341	BE	4	11/13/81	0	0.049	455	44	9.6	32	idd
00038	H-345	BE	2	11/15/81	0	0.260	0	0	0.0	0	erl
00039	H-350	BE	3	11/17/81	0	0.780	0	46	17.5	94	erl
00040	H-350	BE	3	11/17/81	4	0.300	1288	41	13.2	10	erl
00041	H-350	BE	3	11/17/81	6	0.000	60	45	14.8	53	erl

00042	H-350	BE	2	11/17/81	8	0.000	879	40	12.6	63	erl
00043	H-350	BE	1	11/17/81	10	0.000	1018	40	14.1	70	arl
00044	H-354	BE	3	11/18/81	0	0.076	755	55	15.0	54	edd
00045	H-354	BE	3	11/18/81	2	0.000	989	38	12.0	29	edd
00046	H-354	BE	3	11/18/81	6	0.098	1002	41	13.6	41	edd
00047	H-354	BE	3	11/18/81	8	0.117	959	40	13.0	44	edd
00048	H-354	BE	3	11/18/81	10	0.000	1075	33	11.9	65	edd
00049	H-354	BE	3	11/18/81	12	0.000	794	39	12.6	70	edd
00050	H-356	BE	3	11/19/81	0	0.120	1160	40	13.7	54	erl
00051	H-356	BE	3	11/19/81	3	0.000	2025	29	9.6	46	erl
00052	H-356	BE	3	11/19/81	6	0.331	62	26	7.8	59	erl
00053	H-356	BE	3	11/19/81	8	0.063	516	31	9.7	76	erl
00054	H-356	BE	0	11/19/81	0	0.000	0	0	0.0	0	
00055	H-356	BE	3	11/19/81	10	0.000	1892	28	9.2	138	erl
00056	H-356	BE	3	11/19/81	12	0.000	1661	31	9.1	160	erl
00057	H-356	BE	3	11/19/81	14	0.000	1614	25	7.3	125	erl
00058	H-357	BE	3	11/20/81	0	0.000	1308	38	15.8	56	edd
00059	H-357	BE	3	11/20/81	2	0.000	1078	36	11.4	63	edd
00060	H-357	BE	3	11/20/81	5	0.300	24	38	11.5	68	edd
00061	H-357	BE	3	11/20/81	7	0.165	249	35	11.0	77	edd
00062	H-357	BE	3	11/20/81	11	0.000	861	35	11.0	94	edd
00063	H-357	BE	0		0	0.000	0	0	0.0	0	
00064	H-358	BE	3	11/24/81	0	0.110	1304	40	12.4	36	erl
00065	H-358	BE	3	11/24/81	2	0.000	1476	39	12.7	27	erl
00066	H-358	BE	3	11/24/81	6	0.054	1579	43	14.2	34	erl
00067	H-358	BE	1	11/24/81	9	0.000	1582	44	15.0	38	arl
00068	H-358	BE	1	11/24/81	9	0.000	1130	33	11.0	90	arl
00069	H-372	BE	4	12/03/81	0	0.350	1239	39	10.3	0	idd
00070	H-372	BE	4	12/03/81	1	0.000	1609	38	11.2	33	idd
00071	H-382	BE	4	12/09/81	0	5.000	281	26	8.0	77	idd
00072	H-387	BE	3	12/14/81	0	1.440	187	45	14.7	73	hcp
00073	H-387	BE	3	12/14/81	3	1.981	85	30	9.2	89	hcp
00074	H-387	BE	3	12/14/81	5	0.296	282	36	11.4	82	hcp
00075	H-387	BE	3	12/14/81	7	0.109	460	37	12.8	48	hcp
00076	H-387	BE	3	12/14/81	10	0.000	441	37	11.8	63	hcp
00077	H-391	BE	3	12/16/81	0	2.061	144	22	6.6	215	edd
00078	H-391	BE	3	12/16/81	1	0.000	852	23	5.6	150	edd
00079	H-391	BE	3	12/16/81	2	0.350	144	48	11.9	47	edd
00080	H-391	BE	3	12/16/81	4	0.447	144	46	13.6	56	edd
00081	H-393	BE	3	12/16/81	6	0.316	356	40	11.7	40	edd
00082	H-393	BE	4	12/17/81	0	0.000	472	56	18.6	31	edd
00083	H-393	BE	4	12/17/81	3	0.116	233	26	8.6	79	edd
00084	H-393	BE	3	12/17/81	5	0.228	942	30	9.6	69	edd
00085	H-393	BE	3	12/16/81	7	0.117	850	32	10.5	61	edd
00086	H-393	BE	3	12/16/81	9	3.244	771	31	10.1	71	edd
00087	H-400	BE	4	12/21/81	0	0.260	859	48	15.6	36	eds

00088	H-400	BE	4	12/21/81	3	0.000	1753	29	9.4	62	eds
00089	H-401	BE	4	12/23/81	0	0.410	557	33	11.0	40	ids
00090	I-24	BE	2	02/08/82	0	0.228	255	31	11.0	134	erl
00091	I-24	BE	1	02/08/82	4	0.168	569	42	13.3	79	arl

*See table 2A for column headings.

Table 3A. Admission data for 1980-81 indexed on lead concentration.

CASE NO	SPEC.	CAT	ADMISSION DATE	CLINICAL INTERVAL	LEAD (ppm)	PBG (nM/hr /ml RBC)	PCV (%)	HGB (gm/dl)	ZPP (ug/dl)	FATE
G-74	BE	4	05/30/80	0	0.000	0	0	0.00	0	ds
G-82	BE	4	06/10/80	0	0.000	0	0	0.00	0	dd
G-234	BE	2	11/04/80	0	0.000	0	35	0.00	49	scp
G-236	BE	2	11/05/80	0	0.000	685	39	0.00	38	erl
G-263	BE	3	11/20/80	0	0.000	748	41	0.00	56	erl
G-302	BE	4	12/15/80	0	0.000	629	45	0.00	161	scp
H-81	BE	2	05/12/81	0	0.000	753	50	0.00	42	arl
H-127	BE	1	06/26/81	0	0.000	1458	32	0.00	38	arl
H-128	BE	1	06/26/81	0	0.000	1029	31	0.00	6	arl
G-272	BE	1	11/21/80	0	0.001	0	47	0.00	63	hcp
G-301	BE	4	12/10/80	0	0.070	0	33	0.00	120	eds
G-278	BE	4	11/25/80	0	0.230	353	0	0.00	243	dd
G-267	BE	2	11/19/80	0	0.660	79	45	0.00	448	erl
H-8	BE	4	01/12/81	0	2.900	197	18	0.00	76	idd

See table 1A for explanation of FATE codes.

Table 3B. Admission data for 1981-82 indexed on lead concentration.

CASE NO	SPEC.	CAT	ADMISSION DATE	CLINICAL INTERVAL	LEAD (ppm)	PBG (nM/hr /mlRBC)	PCV (%)	HGB (gm/dl)	ZPP (ug/dl)	FATE
H-280	GE	2	10/22/81	0	0.000	1430	30	9.5	64	erl
H-357	BE	3	11/20/81	0	0.000	1308	38	15.8	56	edd
H-393	BE	4	12/17/81	0	0.000	472	56	18.6	31	edd
H-307	GE	2	10/30/81	0	0.040	1994	31	10.4	90	erl
H-341	BE	4	11/13/81	0	0.049	455	44	9.6	32	idd
H-354	BE	3	11/18/81	0	0.076	755	55	15.0	54	edd
H-358	BE	3	11/24/81	0	0.110	1304	40	12.4	36	erl
H-356	BE	3	11/19/81	0	0.120	1160	40	13.7	54	erl
H-332	BE	2	11/09/81	0	0.170	1382	42	14.5	43	erl
H-320	BE	3	11/05/81	0	0.175	757	45	15.7	32	erl
I-24	BE	2	02/08/82	0	0.228	255	31	11.0	134	erl
H-345	BE	2	11/15/81	0	0.260	0	0	0.0	0	erl
H-400	BE	4	12/21/81	0	0.260	859	48	15.6	36	eds
H-327	BE	3	11/06/81	0	0.350	756	39	13.1	42	edd
H-372	BE	4	12/03/81	0	0.350	1239	39	10.3	0	idd
H-401	BE	4	12/23/81	0	0.410	557	33	11.0	40	ids
H-305	BE	2	10/30/81	0	0.500	384	42	12.6	36	erl
H-350	BE	3	11/17/81	0	0.780	0	46	17.5	94	erl
H-387	BE	3	12/14/81	0	1.440	187	45	14.7	73	erl
H-391	BE	3	12/16/81	0	2.061	144	22	6.6	215	edd
H-382	BE	4	12/09/81	0	5.000	281	26	8.0	77	idd

See table 1A for explanation of FATE codes

Table 4A. 1980-81 values sorted by lead concentration.

Lead values less than 0.20 ppm

CASE NO	SPEC.	CAT	ADMISSION DATE	CLINICAL INTERVAL	LEAD (ppm)	PBG (nM/hr /ml RBC)	PCV (%)	HGB* (gm/dl)	ZPP (ug/dl)	FATE
G-74	BE	4	05/30/80	0	0.000#	0	0	0.00	0	ds
G-82	BE	4	06/10/80	0	0.000	0	0	0.00	0	dd
G-234	BE	2	11/04/80	0	0.000	0	35	0.00	49	scp
G-236	BE	2	11/05/80	0	0.000	685	39	0.00	38	erl
G-263	BE	3	11/20/80	0	0.000	748	41	0.00	56	evl
G-302	BE	4	12/15/80	0	0.000	629	45	0.00	161	scp
H-81	BE	2	05/12/81	0	0.000	753	50	0.00	42	arl
H-127	BE	1	06/26/81	0	0.000	1458	32	0.00	38	arl
H-128	BE	1	06/26/81	0	0.000	1029	31	0.00	6	arl
G-272	BE	1	11/21/80	0	0.001	0	47	0.00	63	hcp
Lead values between 0.201 and 0.6 ppm										
G-278	BE	4	11/25/80	0	0.230	353	0	0.00	243	dd
Lead values between 0.601 and 1.0 ppm.										
G-267	BE	2	11/19/80	0	0.660	79	45	0.00	448	erl
Lead values greater than 1.0 ppm.										
H-8	BE	4	01/12/81	0	2.90	197	18	0.00	76	idd

*No hemoglobin determinations were made on these samples.
#A value of 0.000 means no sample was taken; 0.001 means a sample was taken, but there were no detectable residues.
-See table 1A for explanation of FATE codes

Table 4B. 1981-82 admission values sorted on lead concentrations.

Lead values less than 0.20 ppm

CASE NO	SPEC.	CAT	ADMISSION DATE	CLINICAL INTERVAL	LEAD (ppm)	PBG (nM/hr /mlRBC)	PCV (%)	HGB (gm/dl)	ZPP (ug/dl)	FATE
H-280	GE	2	10/22/81	0	0.000	1430	30	9.5	64	erl
H-357	BE	3	11/20/81	0	0.000	1308	38	15.8	56	edd
H-393	BE	4	12/17/81	0	0.000	472	56	18.6	31	edd
H-307	GE	2	10/30/81	0	0.040	1994	31	10.4	90	erl
H-341	BE	4	11/13/81	0	0.049	455	44	9.6	32	idd
H-354	BE	3	11/18/81	0	0.076	755	55	15.0	54	edd
H-358	BE	3	11/24/81	0	0.110	1304	40	12.4	36	erl
H-356	BE	3	11/19/81	0	0.120	1160	40	13.7	54	erl
H-332	BE	2	11/09/81	0	0.170	1382	42	14.5	43	erl
H-320	BE	3	11/05/81	0	0.175	757	45	15.7	32	erl
Lead values between 0.201 and 0.60 ppm										
I-24	BE	2	02/08/82	0	0.228	255	31	11.0	134	erl
H-345	BE	2	11/15/81	0	0.260	0	0	0.0	0	erl
H-400	BE	4	12/21/81	0	0.260	859	48	15.6	36	eds
H-327	BE	3	11/06/81	0	0.350	756	39	13.1	42	edd
H-372	BE	4	12/03/81	0	0.350	1239	39	10.3	0	idd
H-401	BE	4	12/23/81	0	0.410	557	33	11.0	40	ids
H-305	BE	2	10/30/81	0	0.500	384	42	12.6	36	erl
Lead values between 0.601 and 1.0 ppm										
H-350	BE	3	11/17/81	0	0.780	0	46	17.5	94	erl
Lead values greater than 1.0 ppm										
H-387	BE	3	12/14/81	0	1.440	187	45	14.7	73	erl
H-391	BE	3	12/16/81	0	2.061	144	22	6.6	215	edd
H-382	BE	4	12/09/81	0	15.000	281	26	8.0	77	idd

See table 1A for explanation of FATE codes

Table 5. Summary of admission data sorted by lead concentration interval for both years of the study. Data expressed as means plus or minus S.E.

GROUP*	LEAD CONC. (ppm)	PBG-s (nM-PBG/ ml-RBC/hr)	PCV (%)	HGB (gm/dl)	ZPP (ug/dl)
1	(n=11) 0.10 (0.02)	(n=16) 1020 (108) range: 455-1994	(n=19) 41 (1.8) range: 31-56	(n=10) 13.5 (1) range: 9.5-18.6	(n=19) 56 (8) range: 31-161
2	(n=8) 0.32 (0.03) range:	(n=7) 629 (131) range: 255-1239	(n=7) 39 (2.5) range: 31-48	(n=6) 12.3 (0.8) range: 10.3-15.6	(n=7) 88.5 (34) range: 36-243
3	(n=2) 0.72 (0.06)	(n=2) 39.5 (39.5) range: 0-79	(n=2) 46 (.05) range: 45-46	(n=1) 17.5	(n=2) 271 (177) range: 94-448
4	(n=4) 5.35 (3.23)	(n=4) 202 (29) range: 1.0-15.0	(n=4) 27.75 (6) range: 144-281	(n=3) 9.8 (2.5) range: 18-45	(n=4) 110 (35) range: 73-215

*Group 1 had less than 0.2 ppm lead, group 2 was between 0.201 and 0.6 ppm lead, group 3 was between 0.601 and 1.0 ppm lead, and group 4 was greater than 1.01 ppm lead

Table 6A. 1980-81 Admissions sorted by clinical category.

Clinical category 1

CASE NO	SPEC.	CAT	ADMISSION DATE	CLINICAL INTERVAL	LEAD (ppm)	PBG (nM/hr /ml RBC)	PCV (%)	HGB (gm/dl)	ZPP (ug/dl)	FATE
H-127	BE	1	06/26/81	0	0.000	1458	32	0.00	38	arl
H-128	BE	1	06/26/81	0	0.000	1029	31	0.00	6	arl
G-272	BE	1	11/21/80	0	0.001	0	47	0.00	63	hcp

Clinical category 2

CASE NO	SPEC.	CAT	ADMISSION DATE	CLINICAL INTERVAL	LEAD (ppm)	PBG (nM/hr /ml RBC)	PCV (%)	HGB (gm/dl)	ZPP (ug/dl)	FATE
G-234	BE	2	11/04/80	0	0.000	0	35	0.00	49	scp
G-236	BE	2	11/05/80	0	0.000	685	39	0.00	38	erl
H-81	BE	2	05/12/81	0	0.000	753	50	0.00	42	arl
G-267	BE	2	11/19/80	0	0.660	79	45	0.00	448	erl

Clinical category 3

CASE NO	SPEC.	CAT	ADMISSION DATE	CLINICAL INTERVAL	LEAD (ppm)	PBG (nM/hr /ml RBC)	PCV (%)	HGB (gm/dl)	ZPP (ug/dl)	FATE
G-263	BE	3	11/20/80	0	0.000	748	41	0.00	56	erl

Clinical category 4

CASE NO	SPEC.	CAT	ADMISSION DATE	CLINICAL INTERVAL	LEAD (ppm)	PBG (nM/hr /ml RBC)	PCV (%)	HGB (gm/dl)	ZPP (ug/dl)	FATE
G-74	BE	4	05/30/80	0	0.000	0	0	0.00	0	ds
G-82	BE	4	06/10/80	0	0.000	0	0	0.00	0	dd
G-302	BE	4	12/15/80	0	0.000	629	45	0.00	161	scp
G-301	BE	4	12/10/80	0	0.070	0	33	0.00	120	eds
G-278	BE	4	11/25/80	0	0.230	353	0	0.00	243	dd
H-8	BE	4	01/12/81	0	2.900	197	18	0.00	76	idd

See table 1A for explanation of FATE codes

Table 6B. 1981-82 Admissions sorted by clinical category.

Clinical category 1

CASE NO	SPEC.	CAT	ADMISSION DATE	CLINICAL INTERVAL	LEAD (ppm)	PBG (nM/hr /mlRBC)	PCV (%)	HGB (gm/dl)	ZPP (ug/dl)	FATE
No entries										

Clinical category 2

H-280	GE	2	10/22/81	0	0.000	1430	30	9.5	64	erl
H-307	GE	2	10/30/81	0	0.040	1994	31	10.4	90	erl
H-332	BE	2	11/09/81	0	0.170	1382	42	14.5	43	erl
I-24	BE	2	02/08/82	0	0.228	255	31	11.0	134	erl
H-345	BE	2	11/15/81	0	0.260	0	0	0.0	0	erl
H-305	BE	2	10/30/81	0	0.500	384	42	12.6	36	erl

Clinical category 3

H-357	BE	3	11/20/81	0	0.000	1308	38	15.8	56	edd
H-354	BE	3	11/18/81	0	0.076	755	55	15.0	54	edd
H-358	BE	3	11/24/81	0	0.110	1304	40	12.4	36	erl
H-356	BE	3	11/19/81	0	0.120	1160	40	13.7	54	erl
H-320	BE	3	11/05/81	0	0.175	757	45	15.7	32	erl
H-327	BE	3	11/06/81	0	0.350	756	39	13.1	42	edd
H-350	BE	3	11/17/81	0	0.780	0	46	17.5	94	erl
H-387	BE	3	12/14/81	0	1.440	187	45	14.7	73	erl
H-391	BE	3	12/16/81	0	2.061	144	22	6.6	215	edd

Clinical category 4

H-393	BE	4	12/17/81	0	0.000	472	56	18.6	31	edd
H-341	BE	4	11/13/81	0	0.049	455	44	9.6	32	idd
H-400	BE	4	12/21/81	0	0.260	859	48	15.6	36	eds
H-372	BE	4	12/03/81	0	0.350	1239	39	10.3	0	idd
H-401	BE	4	12/23/81	0	0.410	557	33	11.0	40	ids
H-382	BE	4	12/09/81	0	5.000	281	26	8.0	77	idd

See table 1A for explanation of FATE codes

Table 7. Summary data sorted by clinical category at time of admission for both years of the study. Data expressed as mean plus or minus S.E.

Cat.*	Lead Conc. (ppm)	No. in each Pb interval a	b	c	d"	PBG-s (nM-PBG/ ml-RBC/hr)	PCV (%)	HGB (gm/dl)	ZPP (ug/dl)
1	(n=1) 0.001	1	0	0	0	(n=2) 1243 (214) range: 1029-1458	(n=3) 37 (5) range: 31-47#	n.d.	(n=3) 36 (16.5) range:
2	(n=6) 0.31 (0.09) range: 0.040-0.660	0	3	1	0	(n=8) 1001 (243) range: 79-1994	(n=9) 38 (2.4) range: 30-50	(n=5) 11.6 (0.9) range: 9.5-14.5	(n=9) 104 (44) range: 36-448%
3	(n=8) 0.64 (0.15) range: 0.076-2.06	0	1	1	2	(n=10) 712 (150) range: 0-1308	(n=9) 41 (2.6) range: 22-55	(n=6) 13.8 (1) range: 6.6-17.5	(n=9) 71.2 (17) range: 32-215
4	(n=8) 2.4 (1.8) range: 0.049-15.0	0	4	0	2	(n=9) 560 (107) range: 197-1239	(n=9) 38 (3.9) range: 18-56	(n=6) 12.2 (1.6) range: 8.0-18.6	(n=9) 91 (24) range: 32-243

* see text for definition of clinical categories
" correspond to same lead intervals as used in table 5.
contains values for unfledged eaglets which have a normal pcv of 25-30 %
includes 2 high values from golden egles and 1 low value from a very healthy bald eagle with a lead concentration of 0.660 ppm.
% same as previous comment

Note: PCV's are difficult to interpret because of the effects of dehydration in clinically ill birds.

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DNR INFORMATION
(612) 296-6157

File No.

September 30, 1981

Dr. Patrick Redig
Raptor Rehabilitation Center
University of Minnesota
St. Paul, MN

Dear Dr. Redig:

I have enclosed the results of my analysis for total lead residues in the 18 eagle blood samples that you delivered to this laboratory on 21 August 1981.

The samples were defrosted, homogenized in a Virtis blender, diluted with a 1% Triton X-100 solution and analyzed directly by flameless atomic absorption spectrophotometry as modified by Hindenberger et al. (1981. Atomic Spectroscopy. Vol. 2, No. 1, pp. 1-7). I can supply the specific conditions of analysis upon request.

The results of the analysis are expressed as microgram of lead per milliliter of whole blood (ppm).

If you have any questions concerning this analysis please feel free to call.

Sincerely,

Ron Lawrenz, Aquatic Biologist/
A.A. Analysis
MDNR Chemistry Laboratory
Carlos Avery Game Farm
Forest Lake, MN 55025

RL:blt
cc: Carrol Henderson
Bill Longley

Total Lead Residues in Eagle
Blood Samples Delivered on 21 August 1981

Sample Number	Lead Concentration µ gm/ml (ppm)
CL-82-1	.455
CL-82-2	.305
DR-8-1	.320
DR-8-2	.255
DR-14	.190
DR-22	.235
DR-30-1	.285
DR-30-2	.240
DR-31-1	.450
DR-31-2	.150
DR-39	.210
DR-77-1	.215
DR-77-2	.255
H-54	.850
H-126	.190
H-127	.165
H-128	.305
H-181	.300

STATE OF
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BOX 25, CENTENNIAL OFFICE BUILDING • ST. PAUL, MINNESOTA • 55155

DNR INFORMATION
(612) 296-6157

FILE NO.

April 12, 1983

Dr. Patrick Redig
Raptor Rehabilitation Center
University of Minnesota
St. Paul, MN 55108

Dear Dr. Redig,

We are finally able to send you some reportable results from our lead analysis of the avian blood samples you sent last summer. By way of explanation for the big lapse of time between when you gave us the samples and now I must tell you that Ron Lawrenz our former metals specialist, vacated his position in the laboratory for a better opportunity within the department. It took some time to get a replacement through the state system and even more time to recoup the lost skills and experience to run your samples. All this left us with quite a backlog of work through and some false starts on outstanding work.

To date we have been able to generate results for 21 samples, all of which are tabulated below.

Bird No.	Sample Date	Lead, (µg/gm blood)
H 257	11-20-81	0.449 ± 0.049
	11-20-81	0.239 ± 0.006
H 275	10-20-81	0.446 ± 0.042
H 305	11- 2-81	0.205 ± 0.008
	11-18-81	0.554 ± 0.016
H 307	11- 2-81	0.048 ± 0.004
H 320	11- 5-81	0.175 ± 0.029
H 327	11- 6-81	0.420 ± 0.043
H 329	11-11-81	0.356 ± 0.011
H 332	11-11-81	0.187 ± 0.005
H 341	11-16-81	0.049 ± 0.003
H 345	11-16-81	0.260 ± 0.014
H 356	11-19-81	0.120 ± 0.013
	1-13-82	0.053 ± 0.004
H 357	3-14-82	0.165 ± 0.011
H 358	1- 6-82	0.054 ± 0.003
H 387	12-16-81	4.688 ± 0.182
	2-22-82	0.297 ± 0.005
H 393	4- 6-82	0.116 ± 0.007
I 24	2- 9-82	0.223 ± 0.027
	3- 4-82	0.168 ± 0.009

Our method of analysis was similar to that used for your earlier work with minor variations. That is we used a flameless furnace technique to insure adequate detection limits. Our modification was that we ran our wet ash "in situ", that is, in the original blood collection tube so as to avoid contamination through unneeded handling or transfer operations.

A minimum of 3 replicate instrument readings were made from each wet ash digest to average out the minor experimental variations that seem to be inevitable.

Even though we were able to get some instrument readings above the high background, we regard our results to be highly questionable.

There were 31 samples that fell into this category. The identity of these are as follows:

Contaminated Samples

Bird No.	Blood Sample (by date)
H 280	11-23-81
H 297	10-29-81
H 300 Barred Owl	2-11-82
H 305	11-2-81, 11-18-81, 12-31-80, 12-16-81, 2-16-82
H 307	11-2-81, 11-16-81, 1-4-82, 2-3-82
H 320	11-19-81, 11-5-81
H 327	11-18-81
H 329	11-11-81, 1-4-82
H 332	12-31-80, 1-4-82, 11-11-81
H 341	10-16-81
H 350	11-18-81, 12-15-81, 1-14-82, 1-27-82
H 354	11-18-81, 2-16-82
H 356	11-19-81, 12-31-81
H 357	2-11-82
H 400	1-6-82

I am sure that you will find the table of results self explanatory and since the samples were blind to us I don't think I should try to make any comment on their meaning.

I am sorry to report that we had a mishap with the first 31 samples we tried to analyze. We found that our "ultrex" nitric acid had unexpectantly become contaminated with lead and this left us with unmanageable background levels.

You will note that the samples are not run in order, but were randomly picked as we happened across them in the storage box. There is a balance of 24 samples left to finish. These are now in various stages of progress but should be finished up in 2 to 3 weeks.

There will be no charge for the analysis since the contract has lapsed and we acknowledge fault for the lost samples.

If you have any questions concerning this correspondence or about the work involved, please contact me on 464-5200.

Sincerely yours,

Robert Glazer

Robert Glazer, Supervisor
Chemistry Laboratory
Carlos Avery Game Farm
5463 Broadway
Forest Lake, MN 55025

RG:blt

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DEPARTMENT OF NATURAL RESOURCES
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DNR INFORMATION
(612) 296-6157

FILE NO.

June 2, 1983

Patrick Redig
[illegible]tor Rehabilitation Center
[illegible]iversity of Minnesot[illegible]
St. Paul, MN 55108

Dear Dr. Redig,

Listed below are the results for the balance of the eagle blood/lead determinations.

Eagle Blood Pb Levels (22 samples)

Eagle ID No.	Date Sampled	Pb µg/g blood (ppm) Corrected for blank
H 297	10-29-81	0.203 ± 0.012
H 305	1-18-82	0.250 ± 0.018
H 307 (GE)	1-21-82	0.032 ± 0.001
H 329	12-30-81	0.438 ± 0.025
H 332	1-27-82	0.269 ± 0.020
H 350	11-18-81	1.186 ± 0.039
H 354	11-18-81	0.076 ± 0.008
	1- 6-82	0.098 ± 0.007
	1-21-82	0.117 ± 0.019
	2- 4-82	0.049 ± 0.007
H 357	12-30-81	0.421 ± 0.027
H 358	1- 5-82	0.046 ± 0.007
	1-28-82	0.114 ± 0.005
H 387	1- 5-82	1.981 ± 0.161
	1-20-82	0.296 ± 0.011
	2- 4-82	0.269 ± 0.017
H 391	12-31-81	2.061 ± 0.111
	1-14-82	0.447 ± 0.018
	1-27-82	0.316 ± 0.028
H 393	1-21-82	0.302 ± 0.031
	2- 4-82	0.117 ± 0.008
	2-16-82	3.244 ± 0.241

Worst case precision: H 354 (1-21-82) ± 16.60%

18 of 22 better than ± 10% precision

H 356 (2-11-82) and H 308 (1-27-82) - sample tubes broken - not enough material remaining to obtain reliable analysis.

Our methods and conditions were the same as described for the earlier reported samples of the group.

The plus or minus quantity after each result in the table is our calculated standard deviation for that particular sample.

As before we have no feeling the correctness of our results as far as your project is concerned because the samples were blind to us. However we are confident of our procedure and trust that what we have reported fits the situation.

There will be no billing for this work for the reasons stated in my letter to you of April 12, 1983.

If you have any questions concerning the included table of results, etc., please feel free to call me on 464-5200.

Sincerely,

Robert L. Glazer

Robert L. Glazer
Chemistry Lab Supervisor
Carlos Avery Research Center
5463 Broadway
Forest Lake, MN 55025

RLG:blt