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Authors: Sileo, L., and Fefer, S. I.

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PAINT CHIP POISONING OF LAYSAN ALBATROSS AT MIDWAY ATOLL

L. Sileo[1] and S. I. Fefer[2]

[1] National Wildlife Health Center, U.S. Fish and Wildlife Service, 6006 Schroeder Road, Madison, Wisconsin 53711, USA
[2] Office of Refuges and Wildlife Resources, U.S. Fish and Wildlife Service, P.O. Box 50167, 300 Ala Moana Boulevard, Honolulu, Hawaii 96850, USA

ABSTRACT: Epizootic mortality occurred in Laysan albatross (*Diomedea immutabilis*) fledgings at Midway Atoll in 1983. Heavy metal toxicity from ingestion of weathered paint chips was one of the causes. Sick albatrosses were unable to retract their wings, causing a "droop-wing" appearance. Five normal and 12 droop-winged fledglings were captured, killed, and examined. Paint chips found in the proventriculus of the affected fledglings contained up to 144,000 ppm lead. Blood, liver, and kidney concentrations of lead in affected birds were higher than in normal fledglings, and acid-fast intranuclear inclusion bodies were present in the kidneys. Degenerative lesions were present in the myelin of some brachial nerves. Weathered paint samples collected from 12 buildings contained up to 247,250 ppm lead and 101 ppm mercury. Lead poisoning was diagnosed in 10 of the droop-winged albatrosses and was one of the causes of morbidity. Mercury toxicosis and plastic impaction were other possible causes.

Key words: *Diomedea immutabilis*, Laysan albatross, sea-bird mortality, droop-wing, lead-poisoning, weathered paint.

INTRODUCTION

Midway Atoll is located approximately 1,851 km northwest of Honolulu (28°13'N latitude, 177°22'W longitude). The atoll's fringing reef is roughly circular in shape and 10.5 km in average diameter. The reef and lagoon cover 10,282 ha and enclose two main islands, Sand (453 ha) and Eastern (135 ha) (Fig. 1). The islands are heavily vegetated primarily with introduced plants such as *Casuarina litorea*, *Verbesina encelioides*, *Bidens alba*, and *Pluchea* spp.

Midway Atoll is presently the site of a U.S. Naval Air facility. Housing and schools for military dependents were built in 1957, and the station's population reached 3,000 in the late 1960's. In 1983, the total population on Midway Islands was less than 400. There were hundreds of buildings and other man-made structures including roads and runways on the islands. Many of these structures were abandoned and in various stages of disrepair. More than half a million seabirds of 15 species nest on the Midway Islands (Fefer et al., 1984). The Laysan albatross is the most abundant species with approximately 200,000 breeding pairs, constituting the largest colony of this species in the world. Laysan albatross nests are located every few meters over most natural areas of the islands. The Laysan albatrosses return to Midway in November and lay eggs in late November and early December. Hatching occurs in late January through early February and the chicks fledge in June or July. The chicks are altricial and sedentary, remaining near the nest for months. The adults feed the chicks by regurgitation about once a day for about 6 mo. The adults feed primarily on squid (Ommastrephidae) and flying fish (Exocoetidae) eggs on the open ocean (Harrison et al., 1983).

During the 1960's chick losses were uniformly distributed from hatching through fledging and ranged from 3 to 17% of the eggs laid (Fisher, 1975). Fefer et al. (1984) estimated 150,000 to 200,000 pairs of breeding Laysan albatrosses at Midway and a loss of 17% of 200,000 eggs would be 34,000 albatrosses, approximately 5,700 each month of the 6 mo nest period. In 1982, approximately 20,000 5- to 6-mo-old albatross chicks were found dead. This is twice the expected mortality rate and is

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considered unusually high mortality for fledgings (Hawaiian Island National Wildlife Refuge, Honolulu, Hawaii, unpubl. data). In 1982, six carcasses were examined and lead poisoning, from ingested paint chips, was diagnosed (National Wildlife Health Center, Madison, Wisconsin, unpubl. data). Several carcasses contained large quantities of ingested plastic in their upper gastrointestinal tracts. Plastic and other "ingested undigestibles" were previously implicated in Laysan albatross mortality (Kenyon and Kridler, 1969; Pettit et al., 1981).

In June 1983, epizootic mortality at Midway was reported again by personnel of Hawaiian Island National Wildlife Refuge, Honolulu, Hawaii. The objective of the present study was to collect necropsy samples from droop-wing and normal albatross fledglings, blood from healthy adults, and paint samples from buildings to determine if either lead from weathered paint or ingested plastic were causes of mortality.

METHODS

Five normal fledgling and 12 "droop-wing" albatrosses were captured by hand at Midway during July 1983. Collection sites were plotted on a map of the island. Blood was collected without anticoagulant by jugular venipuncture. Blood smears were made and stained with Camco Quik Stain (American Scientific Products, McGaw Park, Illinois 60085, USA).[3] Packed cell volume was determined by the microhematocrit method with heparinized capillary tubes. Whole blood was placed in 2 ml Vacutainer (Becton-Dickinson, Rutherford, New Jersey 07070, USA)[3] tubes and frozen for later lead analysis. The same blood sampling procedures were used for 19 adult albatrosses also captured by hand at sites where fledgling morbidity seemed especially high. After blood sampling, the adult birds were released, but the fledglings were euthanized with T-61 (American Hoechst Corporation, Somerville, New Jersey 08876, USA)[3] and dissected at the study site.

[3] Reference to commercial products and company name does not imply endorsement by the Federal government.

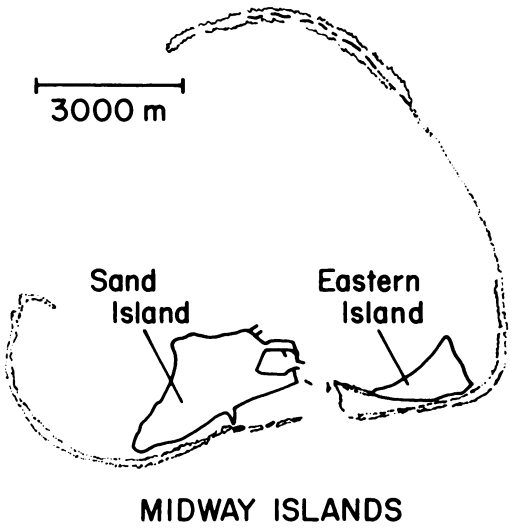


FIGURE 1. Outline map of Midway Islands.

Weight, a subjective evaluation of the condition of the bird based on the quantity of adipose in normal storage depots, sex, and a description of all gross morphologic lesions noted during the dissections were recorded. Plastic and other buoyant indigestibles from the proventriculus and gizzard were placed in a nylon mesh pouch and immersed in a graduated cylinder of water for volume determination. The liver and left kidney were weighed and portions of the liver, kidney, spleen, skeletal muscle, proventriculus, brachial plexus, and representative portions of all tissues containing gross morphological lesions were fixed in 10% buffered formalin and sealed in plastic bags for shipment to the National Wildlife Health Center. Liver, kidney, and tibia samples were frozen for toxicological analysis. Paint chips from the proventricular contents were collected also for toxicological analysis. Weathered paint samples were taken from 12 buildings on Sand Island.

Lead concentrations were determined by atomic absorption spectrophotometry (Williams, 1984). Because of suspected laboratory analytical errors, all the data from the first toxicological analyses of liver, kidney, and tibia samples were omitted from this report. The data that were included were from subsequent repeated tests of frozen samples that remained after the completion of the first tests. Formalin-fixed tissues were processed by routine histological procedures and stained with hematoxylin and eosin, acid-fast or luxol-fast blue. Kidney sections stained by the acid-fast method were examined for inclusion bodies at 800× for 10

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FIGURE 2. "Droopy" albatross suffering from lead poisoning. This bird had paint chips in its stomach (lead content, 38,000 ppm) and high lead concentration in its liver (59 ppm dry weight).

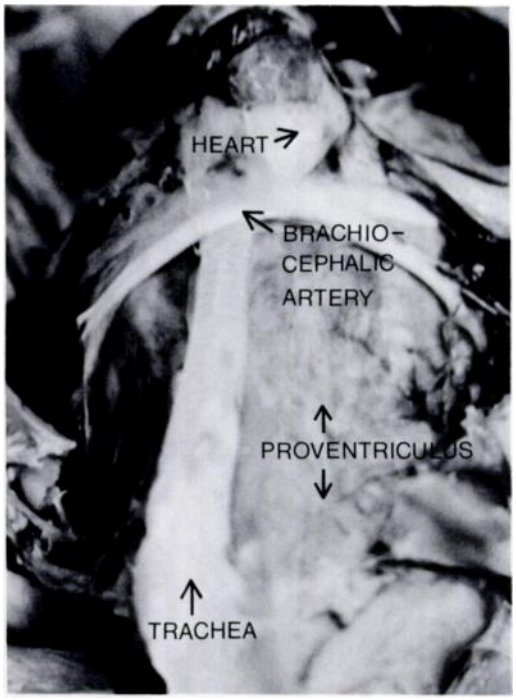


FIGURE 3. Albatross proventriculus massively distended by ingested plastic.

min. Blood smears were examined for parasites. Polychromatic erythrocytes were counted to determine their ratio per 1,000 red cells counted. Organ weights were compared by the student's *t*-test with pooled variances.

RESULTS

Droopy albatross chicks were unable to retract their wings (Fig. 2), but were alert and had normally coordinated movements. Adult albatrosses were not affected. We did not survey the entire island for droop-wing fledglings, but they seemed to occur more frequently in the vicinity of old wooden buildings with peeling paint. Our diagnosis of lead poisoning in 10 droopy birds was based on two or more of the following: paint chips in the proventriculus; lead concentrations in blood, liver, or kidney that were higher than in the normal birds; and/or acid-fast intranuclear inclusion bodies in the proximal convoluted tubules (Table 1). Inclusion bodies were sparse in all but two of the birds (Nos. 32 and 58). An unequivocal diagnosis of lead poisoning was not possible for two of the droopy albatrosses. One of these (No. 33) had ingested paint chips, but had comparatively low tissue concentrations of lead and no renal inclusion bodies. The other (No. 57) had neither ingested paint nor renal inclusion bodies; toxicological data were not available for this bird.

The birds were in excellent flesh with no evidence of organ atrophy, anemia, excessive bile production, diarrhea, or urate retention. The pectoralis minor muscle seemed unusually pale in two of the lead-poisoned birds. Droopy albatrosses had smaller livers ($\bar{x} = 39.2$ g $\pm$ 4.10) and larger kidneys ($\bar{x} = 8.5$ g $\pm$ 0.48) than normal birds ($\bar{x} = 49.3$ g $\pm$ 4.31 and 7.9 g $\pm$ 0.61, respectively), but the differences were not statistically significant (liver, $P < 0.6$; kid-

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TABLE 1. Necropsy and toxicological data from normal and "droop-winged" Laysan albatross fledglings euthanized at Midway Atoll.

Bird number	Paint chips in PV[a]	Lead concentrations				Renal inclusion bodies	Hematocrit (%)	Polychromatic RBC's/1,000 RBC's	Plastic in PV (ml[e])
		PV paint (ppm dw[b])	Blood (ppm ww[c])	Liver (ppm dw)	Kidney (ppm dw)				
Normal									
28	–[d]		<0.05	6.0	ND[f]	–	35	14	33
29	–		0.10	5.2	ND	ND	33	100	ND
30	–		<0.05	1.6	<1.0	–	41	28	10
82	–		0.25	1.9	ND	–	36	118	165
84	–		<0.05	1.2	ND	–	35	44	45
Lead-poisoned "droop-wings"									
35	+	2,000	0.23	44	ND	+	34	76	50
36	+	322	0.13	57	ND	+	37	57	trace
34	–		1.43	ND	ND	ND	36	23	0
55	+	3,930	0.24	6	44	+	40	30	20
56	+	144,000	1.20	100	ND	+	40	19	trace
58	+	38,000	0.40	59	ND	+	37	107	175
59	+	4,290	0.96	ND	ND	+	42	113	trace
60	+	177	0.49	33	ND	+	40	41	1
61	–		0.03	17	ND	+	26	47	1
32	+	11,700	4.80	110	ND	+	39	58	0
Other "droop-wings"									
57	–		<0.05	ND	ND	–	36	119	70
33	+	38	0.09	8	15	–	ND	19	1

[a] Proventriculus.
[b] Dry weight.
[c] Wet weight.
[d] Absence (–) or presence (+).
[e] ND = not determined.

ney, $P < 0.8$). The volume of proventricular plastic was not associated with either droop-wing or lead poisoning (Table 1). The proventriculus of one albatross (No. 58) was massively distended with plastic (Fig. 3). Histologically, there were severe chronic inflammatory changes in the muscularis and mucosal lamina propria of the organ. This was apparently a result of the severe mechanical distension and compression of the tissues. Three other birds also had chronic inflammatory lesions in their proventriculi or gizzards. Fourteen of the fledglings had scars on the epidermis of their beaks; these scars occurred in both normal and droopy birds.

Only a few microscopic lesions suggestive of lead poisoning were noted in the fledglings. Severe myocardial and arterial fibrinoid necrosis occurred in one, mild nephrosis in two, and mild Kupffer-cell hemosiderosis occurred in three of the other lead-poisoned fledglings. Demyelination was noted in the brachial nerves of two droopy and one normal albatross.

One of the lead-poisoned birds had a low hematocrit (26%), but mean hematocrits in lead-poisoned and normal groups were similar ($P < 0.8$). Marked variation in polychromatic erythrocyte density precluded comparison between the groups. Both high and low counts occurred in lead-poisoned birds.

Many of the abandoned buildings on Sand Island were peeling large quantities of weathered paint. Lead concentrations

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in 19 paint samples removed from 12 of these buildings or from the ground immediately adjacent to the buildings varied from 1,500 ppm to 247,250 ppm dry weight ($\bar{x}$ = 84,316 ± 16,183 ppm). Mercury concentrations in the same samples varied from 6 to 101 ppm ($\bar{x}$ = 39.9 ± 5.8 ppm).

One adult albatross had a blood lead concentration of 0.2 ppm wet weight and another had 0.1 ppm. The remaining 17 had concentrations below 0.05 ppm. Hematocrits of the adult birds varied from 21 to 42% ($\bar{x}$ = 33%).

DISCUSSION

Longcore et al. (1974) states that lead levels exceeding a range of 6–20 ppm wet weight in kidney or liver of mallards (*Anas platyrhynchos*) indicate acute exposure to lead. Most of the droopy albatrosses had both tissue concentrations of lead greater than these values, and renal inclusion bodies justifying a diagnosis of lead toxicosis. The differences in the lead concentrations between normal and droop-winged birds were great. Weathered paint chips were the apparent source of the lead. Adult albatrosses feed at sea and probably did not ingest weathered paint chips from the islands. The lead concentrations in the blood of all the adult albatrosses were normal. This is indirect evidence that the source of the lead was not in the regurgitated diet fed to the chicks, but rather that the source was on the island. Albatross fledglings mobilize stored body fat and begin to explore away from their nest sites on an instinctive quest for the beaches and the open sea (Fisher and Fisher, 1969). They seemingly became hungry, restless, curious, and explorative. Weathered paint chips on the ground may provide dangerous visual cues to these neophytes with poorly developed feeding behavior and little feeding experience. Younger nestlings who tenaciously remain at the nest site would not be exposed to paint chips, unless the nest was adjacent to a building. Few of the lesions usually associated with fulminant lead poisoning in waterfowl and raptors, such as emaciation, dilated gallbladder, proventricular impaction, bile-stained viscera, hepatocellular atrophy and necrosis, Kupffer-cell hemosiderosis, and nephrosis (Reiser and Temple, 1981; Wobeser, 1981), were present in the sick albatrosses. Euthanasia probably interrupted toxicosis before development of these characteristic lesions. "Wing-drop" is an effect of lead toxicosis in waterfowl (Wetmore, 1919). Wing-drop is possibly more pronounced in albatross fledglings because of their large, heavy wings. Generalized toxic asthenia may have caused the droop-wing. Lead may also have interferred with heme synthesis, and thus myoglobin formation, causing impaired pectoral muscle development. Neurologic damage may also have been involved because both lead and mercury cause peripheral neuropathies in birds (Pass et al., 1975; Hunter and Wobeser, 1980).

The paint contained considerable amounts of mercury, and mercury may have been present in the natural marine food chain. Ohlendorf and Harrison (1986) reported high mercury concentrations in red-footed booby (*Sula sula*) eggs from Midway. They discussed the influence of ocean currents and volcanism on mercury concentrations in seabirds. Unfortunately, the mercury content of the albatross tissues was not determined.

The role of ingested plastic in the etiology of the droop-wing syndrome was not determined. Severe proventricular impactions occur in waterfowl poisoned by lead shot and the plastic impactions were possibly homologous, secondary lesions in the lead-poisoned albatrosses. The plastic was the direct cause of severe lesions in one of the birds and may have caused the minor proventricular ulcers noted in others. Fry et al. (1987) report the prevalence of plastic ingestion in this flock. Ingested plastic could cause potentially extensive morbid-

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ity in albatross nestlings and should be investigated further.

The scars on the face and beaks of the birds probably were healed pox lesions. An avian pox epizootic in the Midway flock the previous March and April caused extensive morbidity and some mortality in juvenile albatross (National Wildlife Health Center files, unpubl. data). There was no indication of pox related mortality in July.

Razing of problem wooden buildings would mitigate the weathered paint problem. Buildings of reinforced concrete are a more difficult problem. Removal of the paint by sandblasting or incineration (flame-thrower?) might be feasible. Fencing or cover plantings to prevent fledgling access are also management possibilities.

ACKNOWLEDGMENTS

G. S. Grant, North Carolina Museum of Natural History, first diagnosed the paint chip lead-poisoning problem on Midway in 1981. R. K. Stroud confirmed the diagnosis at the National Wildlife Health Center in 1982. We gratefully acknowledge the cooperation of the U.S. Naval Air Facility at Midway; the assistance of M. B. Naughton during the field investigations; D. W. Zoromski of the Wisconsin Central Animal Health Laboratory, Madison, Wisconsin; and numerous staff members of the National Wildlife Health Center, Madison, Wisconsin, who conducted the laboratory tests.

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