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CAUSES OF OWL MORTALITY IN HAWAII, 1992 TO 1994

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ABSTRACT: Eighty-one barn owls (*Tyto alba*) and five Hawaiian owls or pueo (*Asio flammeus sandwichensis*) from Kauai, Oahu, Lanai, Molokai, Maui and Hawaii (USA) were evaluated for cause of death, November 1992 through August 1994. The most common cause of death in barn owls was trauma (50%) followed by infectious disease (28%) and emaciation (22%). Most traumas apparently resulted from vehicular collisions. Trichomoniasis was the predominant infectious disease and appeared to be a significant cause of death in barn owls in Hawaii. Pasteurellosis and aspergillosis were encountered less commonly. No predisposing cause of emaciation was detected. Stomach contents from 28 barn owls contained mainly insects (64%) of the family Tetigoniidae and Gryllidae, and rodents (18%); the remainder had mixtures of rodents and insects or grass. Three pueo died from trauma and one each died from emaciation and pasteurellosis. We found no evidence of organochlorine, organophosphorus, or carbamate pesticides as causes of death in pueo or barn owls.

Key words: Barn owl, *Tyto alba*, short eared owl, *Asio flammeus*, pueo, disease, toxicology, pathology, food habits.

INTRODUCTION

Two species of owls, the endemic short eared owl or pueo (*Asio flammeus sandwichensis*) and barn owl (*Tyto alba*) exist in the six main Hawaiian Islands (USA). Barn owls were introduced originally onto the island of Hawaii from California (USA) in 1958 for rodent control (Thistle, 1959). Subsequent releases were made from Texas (USA) and California to Oahu and Kauai (Tomich, 1962).

Since the 1960s, there have been several reports of owl mortalities. Au and Swedberg (1966) described seven sick or weak barn owls of which three were traumatized. Tomich (1971) observed pueo hunting along roads on Hawaii and also noted dead owls which probably had collided with vehicles. Unusually numerous barn owl and pueo mortalities with weak and emaciated owls were noted by Telfer (1973, 1987). Gassman-Duvall and Telfer (1987) described weak owls exhibiting auditory and visual difficulties alongside roads. Gassman-Duvall (1988) reported emaciation and multiple organ hemorrhage in barn owls. Based on histopathologic examination of three owls, she hypothesized vitamin E insufficiency as a cause of the clinical signs and mortalities described by Telfer (1987). However, Gassman-Duvall (1988) provided little information on methods, species examined, source and collection of samples, and analysis of results. Based on examining 82 barn owls and two pueo, Aye et al. (1995) hypothesized that trauma due to vehicular collisions was the major cause of mortality in owls in Hawaii. Aye et al. (1995) attributed sick or weak owls to dehydration and emaciation secondary to trauma. However, their sample was biased towards animals shot as part of aircraft hazard control operations. No sick owls were examined, and samples were limited to birds from Oahu, and Lihue Airport, Kauai.

Our objective was to determine causes of owl mortality on the six main islands of Hawaii using gross, clinical and microscopic pathology, microbiology, and toxicology. We emphasized examining animals from all islands that were found dead or sick rather than shot. We judged that diagnostic findings from such birds would most likely contribute to our knowledge of death or illness in owls in Hawaii.

MATERIALS AND METHODS

From 6 November 1992 through 10 August 1994, owl carcasses were submitted to the Na-

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tional Wildlife Health Center (NWHC) Honolulu Field Station (HFS) by federal, state, and municipal agencies such as zoos. Submitters were requested to provide date and location of collection along with the specimen, and we encouraged submission of freshly dead (0 to 48 hr old) refrigerated animals. Animals that could not be shipped within 48 hr of death or collection were frozen and shipped later. In addition to carcasses, blood samples were obtained from two sick owls.

Necropsies included a systematic external and internal examination of all organs and stomach contents. Weights to the nearest 1 g were obtained using a 500 g spring scale. Owls were classified as male or female through examination of gonads. Adults (>1 yr) were differentiated from immature birds by a combination of feather wear (Marti, 1990), size of the bursa (>0.5 cm immature), degree of tracheal ossification (ossified, adult; cartilaginous, immature), talon flange development (Johnson, 1991), and gonadal size (testes > 10 × 5 mm, adult male; oviduct tortuous and dilated, adult female). Recognizable stomach contents were identified and insect remains were keyed to family (Borror et al., 1981).

Tissues from 48 barn owls and four pueo examined histologically included lung, trachea, kidneys, gonads, adrenals, liver, heart, pancreas, spleen, cerebrum, cerebellum, pharynx, esophagus, proventriculus, ventriculus, small and large intestines, ceca, bursa, skeletal muscle, thyroid, parathyroids, peripheral nerve, spinal cord, air sacs, bone, and skin. Tissues were fixed in 10% neutral buffered formalin, embedded in paraffin, cut into 5 μm sections, and stained with hematoxylin and eosin (H&E). We used the following special stains (Prophet et al., 1992) to further evaluate suspected lesions: Grocott's methenamine silver and periodic acid Schiff for fungi, Von-Kossa calcium for tissue mineralization, iron-ferric hemosiderin for iron deposits, and Gomori trichrome for trichomonads (Mesa et al., 1961). Fungi were identified microscopically as *Aspergillus* sp. based on characteristic morphology of fruiting bodies (Richards, 1991). All microscopy was done on an Olympus BH-2 compound microscope (Olympus, Lake Success, New York, USA).

Samples for bacteriology consisted of heart blood, liver or lung swabs from 12 barn owls and two pueo (Table 1). Swabs (Mini-tip Culturette, Becton Dickinson, Cockeysville, Maryland, USA) were streaked within 30 min of collection onto 5% sheep blood and McConkey agar (Difco Laboratories, Detroit, Michigan, USA) and incubated at 37 C at 5 to 15% CO_2 for 48 to 72 hr. Individual colonies were subcultured on trypticase soy agar (Becton Dick-

TABLE 1. Numbers of barn owls and pueos in each diagnostic category, test performed (bacteriology, histology, toxicology, parasitology), mean weight, standard deviation (SD), sample size (*n*), age, sex and seasonal distribution.[a]

Group	Species		Tests[b]				Weights			Age/sex[c]					Season[d]			
	Barn	Pueo	Bact.	Hist.	Tox.	Para.	Mean	SD	*n*	AM	AF	IM	IF	UNK	Spr.	Sum.	Fall	Wint.
Trauma	37	3	2	24	7	1	386	64	36	11	15	3	6	2	25	10	4	1
Infectious disease	21	1	6	13	7	10	310	46	21	10	5	3	3	0	5	8	7	2
Emaciation	16	1	5	14	8	3	284	26	16	5	3	4	4	0	6	9	0	2
Undetermined	7	0	1	1	0	0	NA[e]	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
Total	81	5	14	52	22	14	NA	NA	NA	26	23	10	13	2	36	27	11	5

[a] Weight, age, sex, and season data for barn owls only.
[b] Bact. = bacteriology; Hist. = histology; Tox. = toxicology; Para. = parasitology. Diagnostic tests are for barn owls and pueos.
[c] A = adult; I = immature; M = male; F = female; UNK = unknown.
[d] Spr. = spring; Sum. = summer; Wint. = winter.
[e] NA, not applicable.

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inson, Cockeysville, Maryland, USA) slants at the HFS and identified using API 20E and API NFP strips (Biomerieux Vitek, Hazelwood, Missouri, USA). Isolates of *Pasteurella multocida* were serotyped at NWHC laboratory, Madison, Wisconsin using methods of Heddleston et al. (1972).

Oral swabs from 12 barn owls and two pueo were cultured for flagellates at the HFS using Diamonds media or thioglycolate broth (Difco Laboratories) at 27 C and 37 C and examined for growth at 3 to 5-day intervals. Owls with oral lesions from which flagellates were cultured or observed microscopically on direct wet smear were diagnosed as having trichomoniasis (McDougald, 1991). In owls with oral lesions where trichomonads could not be visualized, microscopic pathology was used to rule out other causes of stomatitis such as avian pox (Tripathy, 1991), avitaminosis A (Austic and Scott, 1991), herpesvirus (Gerlach, 1994a), and candidiasis (Bauck, 1994). Such animals were classified as suspect trichomoniasis.

We obtained 1 cc of blood from the brachial vein of two owls using a sterile 3-cc syringe and 22 gauge 25 mm needle. Whole blood in ethylenediaminetetraacetate (EDTA) was processed for white cell and differential counts (Campbell, 1994) while total solids were obtained with a temperature-adjusted refractometer (Schuco, American Caduceus Industries, Carle Place, New York) using plasma (Campbell, 1994).

Birds were grouped into four categories according to the lesion judged to be the most significant contributor to cause of death. These included trauma, infectious disease, emaciation, and undetermined. Criteria for trauma included gross evidence of broken bones accompanied by hemorrhage, bruising of major muscle masses or the calvarium, or rupture and hemorrhage of major internal organs. Criteria for infectious disease included microscopic or microbiologic evidence of infectious agents. Criteria for emaciation included gross evidence of breast muscle atrophy, little to no body fat, gall bladder distended with thick bile, ventriculus empty or containing only grass or sticks, ventricular mucosa coated with dark mucus, and little to no intestinal contents. Additionally, emaciated animals had microscopic evidence of hepatocellular atrophy and hemosiderosis, splenic lymphoid depletion, and pancreatic atrophy. Owls unsuitable for examination or for which cause of death could not be identified were placed in the undetermined category. Euthanasia was by intravenous injection of 1 cc Terminal-III (Ampro Pharmaceutical, Arcadia, California) or cervical dislocation.

Because of insufficient data for each year, we examined temporal trends by pooling data on age, sex, and cause of mortality by season; spring (21 March to 20 June), summer (21 June 21 to 20 September), fall (21 September to 20 December) and winter (21 December to 20 March). Individual owls used for toxicologic investigations were selected based on availability of tissue and condition of carcass. For toxicology, livers, kidneys, and brains were stored separately at −20 C. To test for lethal exposure to organochlorines, livers and kidneys were analyzed by Hazelton Laboratories, Madison, Wisconsin, for organochlorine pollutant residues (OPR) (ppm wet weight) including hexachlorobenzene (HCB), total polychlorinated biphenyls (PCB), alpha, beta, gamma hexachlorocyclohexane (BHC), alpha and gamma chlordane, dieldrin, endrin, heptachlor epoxide, mirex, o,p′ -DDD, o,p′ -DDE, o,p′ -DDT, oxychlordane, p,p′ -DDD, p,p′ -DDE, p,p′ -DDT, toxaphene, and trans-nonachlor (U.S. Environmental Protection Agency, 1986). Brains were analyzed for acetylcholinesterase activity (μmoles of acetylthiocholine iodide hydrolyzed per min per g wet weight of whole brain tissue at 25 C) (Hill and Fleming, 1982) to test for lethal exposure to carbamates and organophosphorus pesticides.

We made statistical comparisons using Sigmastat software (Jandel, San Rafael, California) with one way analysis of variance (ANOVA) for parametric and Kruskall-Wallis ANOVA for non parametric procedures. Level of significance was 0.05. Pair-wise comparisons were made using the Student-Newman-Keuls test.

RESULTS

Eighty-one barn owls were submitted, including 31 from Maui, 24 from Oahu, 14 from Kauai, nine from Hawaii, two from Molokai, and one from Lanai. Most owls were adult males, followed by adult females, immature females, immature males and birds of unknown sex or age (Table 1). Three pueo came from Maui and one each from Oahu and Kauai. Two pueo were immature females and one each was an adult male, adult female and immature male. Five barn owls and one pueo were euthanized because of severe trauma (three barn owls), infectious disease (two barn owls), and emaciation (pueo). One, 67, and 13 barn owls were received in 1992, 1993, and 1994, respectively. All pueo were received in 1993. Twenty-eight (35%) carcasses including one pueo were submitted frozen.

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The primary cause of death in 74 barn owls and pueo was trauma, followed by infectious disease, and emaciation. Trauma was observed most often in the spring while emaciation and infectious disease predominated in summer (Table 1). Cause of death was undetermined in seven owls. Mean weight of barn owls in the trauma category was significantly ($P < 0.05$) greater than that of owls dying from infectious disease or emaciation (Table 1). Mean weight of emaciated barn owls was not significantly different than those dying from infectious disease. Of 28 identifiable barn owl stomach contents, 18 (64%) contained mainly insects of the family Tetigoniidae and Gryllidae, five (18%) contained mainly rodents, and the remaining five had mixtures of rodents and insects or grass.

Of 40 owls dying from trauma, 29, including the pueo, apparently collided with vehicles. Fractures were observed most often in the right tibiotarsus, skull, humeri, femurs, radius and ulna, synsacrum, keelbone, cervical spine, tarsometatarsi, and scapulohumeral joints. Hemorrhages were noted in the liver and abdominal cavity, lung, heart and thoracic cavity, skull and brain, major axial and epaxial muscle groups, and eyes in these birds. Microscopically, there were no lesions indicative of toxic or infectious disease except for two barn owls: one had a granulomatous airsacculitis due to *Aspergillus* sp. and another had diffuse necrotic stomatitis compatible with trichomoniasis. Four traumatized barn owls were also severely emaciated. Ten traumatized barn owls were shot as part of an airport hazard control operation on Kauai. Of these, one had medial hypertrophy of the pulmonary arteries of unknown origin, and no lesions were detected in the remaining nine. All shot animals were in good condition with well developed breast muscles, but little body fat. One barn owl electrocuted on a power pole on Hawaii had multiple burns and diffuse coagulation necrosis of skeletal muscle and liver.

Trichomonas gallinae killed 20 barn owls. Of these, 13 were confirmed by microscopic observation of the parasite from the oral lesion. Based on microscopic pathology, we ruled out other known causes of this type of stomatitis in the remaining seven owls thus leading to presumptive diagnosis of trichomoniasis. All owls with trichomoniasis presented with the classic diphtheritic lesion on the dorsal pharynx varying from a few yellow raised plaques to necrosis of the entire dorsal oral mucosa and underlying pharyngeal musculature. Three owls with oral necrosis had concurrent myiasis with unidentified dipteran larvae. Occasionally, infection with trichomoniasis appeared to extend systemically as evidenced by focal fibrinous plaques on the esophageal mucosa, foci of discoloration in the lung, and a pale swollen liver with rounded borders. One barn owl with trichomoniasis had a concurrent air sacculitis attributable to *Aspergillus* sp.

Pure cultures of *Pasteurella multocida* serotype 3 were isolated from one barn owl and one pueo. The barn owl from Hawaii was an emaciated adult male with an old traumatic foot lesion and a grossly swollen spleen with multiple white foci. Microscopically, there were multiple thrombi in the lung and multiple foci of necrosis associated with bacteria in the liver, spleen, myocardium, cerebrum, and adrenal. A pure culture of *Pasteurella multocida* was obtained from heart blood. The pueo from Kauai was a severely emaciated immature female with no gross or microscopic lesions. Antemortem hematology revealed total solids of 2 mg/dl, a hematocrit of 20% and a total white cell count of 2890 cells/μl. A pure culture of *Pasteurella multocida* was obtained from the lung.

Three emaciated barn owls had lesions indicative of other diseases. One had numerous coccidia in the lamina propria of the small intestines compatible with *Sarcocystis* sp. and judged to be incidental based on lack of associated pathology. Another had focal fibrosis of the myocardium of unknown origin. A third owl had a large,

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TABLE 2. Detectable organochlorine pollutant residues (ppm wet weight) in livers and kidneys of 18 barn owls and 4 pueos.

	Barn Owl		Pueo	
	Kidney	Liver	Kidney	Liver
PCB[a] total	0.49–1.2 (2)[b]	0.1–1.9 (10)	7.9 (1)	ND[c]
Dieldrin	ND	0.02 (1)	ND	ND
Heptachlor	0.1–2.1 (2)	0.04–3.3 (4)	ND	ND
Mirex	0.12–0.29 (2)	0.09–1.0 (3)	0.31 (1)	0.51 (1)
Oxychlordane	0.18–1.0 (3)	0.02–1.5 (9)	ND	ND
pp DDD	ND	0.19 (1)	ND	ND
pp DDE	0.13–31 (7)	0.03–21 (13)	0.83–3 (3)	1.4–9.7 (2)
Transnonachlor	0.12–0.28 (2)	0.02–0.25 (4)	ND	ND

[a] PCB, polychlorinated biphenyls.
[b] Range (number of samples over detection limit).
[c] ND, not detected.

bullous, thin walled, cavitated deformity of the sternum displacing the liver and heart dorsally; the cause was unknown. Based on hematology, one live emaciated barn owl found weak on Oahu had total solids of 2 mg/dl, a hematocrit of 40%, and a total white cell count of 2929/μl which subsequently increased to 4.9 mg/dl, 48%, and 20533/μl, respectively, after 3 wk of supportive care.

Detectable levels of organochlorines were noted for eight of 20 compounds analyzed (Table 2). Mean (±SD) cholinesterase activity (μmoles of acetylthiocholine iodide hydrolyzed per min per g wet weight of whole brain tissue at 25 C) in brains from 17 barn owls was 22.8 ± 2.9 while that of three pueo was 16.3 ± 0.6.

DISCUSSION

Although substantial, the number of carcasses submitted frozen did not negatively affect our ability to diagnose cause of death. Our findings of insects as principal food source for owls contrasted with those of Snetsinger et al. (1994). Examination of pellets by Snetsinger et al. (1994) versus stomach contents in our study would account for the difference. Owls probably assimilate insects more completely than bones, thus decreasing the likelihood of regurgitating recognizable insect remains in pellets. The assertion by Aye et al. (1995) that trauma was responsible for clinical signs of sick owls is only partially correct. We judged from both our euthanized and rehabilitated owls that disease and emaciation were responsible for clinical signs in at least half of ill or weak barn owls and pueo.

The high proportion of traumatized animals was consistent with findings by others in Hawaii (Tomich, 1962; Au and Swedberg, 1966; Tomich, 1971; Aye et al., 1995) and temperate areas (Newton et al., 1991; Cooper, 1993). Newton et al. (1991) noted that trauma diagnoses are probably overrepresented in necropsy surveys because owls with trauma are found more easily. Collision with vehicles are common elsewhere for barn owls (Cooper, 1993; Newton et al. 1991), short eared owls (Harding, 1986), screech owls (*Otus asio*) and saw-whet owls (*Aegolius acadilus*) (Loos and Kerlinger, 1993). Six traumatized barn owls had microscopic lesions in major organs that could have predisposed them to vehicle collisions; these include four emaciated owls, one with trichomoniasis, and one with aspergillosis. Our finding a predominance of traumatized owls in the spring was consistent with other studies (Telfer, 1987; Newton et al., 1991; Aye et al., 1995). This is evidence that owls probably are particularly active at this time of year. Mean weight of owls with trauma in our study was within range reported for

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traumatized owls but lower than that reported for shot owls (Aye et al. 1995).

The finding of infectious diseases in 28% of the owls was considerably higher than the prevalence found by others in Hawaii (Aye et al., 1995) or the European continent (Cooper, 1993; Newton et al., 1991). Trichomoniasis in 86% of the infected barn owls in Hawaii exceeds that of infected owls found by Schultz (1986) in California (56%). Barn owls in Hawaii appeared to be most susceptible to trichomoniasis in the summer and fall, and this disease was present on at least four islands (data not shown).

Owls with trichomoniasis had the classic oral lesion (Pokras et al., 1993) and one owl had lesions in the liver and lung compatible with disseminated trichomoniasis (Mesa et al., 1961). Trichomoniasis probably was responsible for the necrotic stomatitis seen in one owl by Aye et al. (1995), and our findings contradict their conclusion that disease plays a minor role in owl mortalities in Hawaii. Doves (*Geopelia* sp., *Zenaida* sp.) and pigeons (*Columbia* sp.) are considered reservoirs for trichomoniasis in other areas (Pokras et al., 1993), and trichomoniasis has been documented in the barred dove (*Geopelia striata*) in Hawaii (Kocan and Banko, 1974). Neither doves or pigeons have been noted as an avian food source of barn owls in Hawaii although forest birds are a food source (Tomich, 1971; Baker and Russell, 1980; Byrd and Telfer, 1980; Snetsinger et al., 1994). Van Riper and van Riper (1984) noted an apapane with trichomoniasis but did not elaborate as to how this diagnosis was confirmed. Thus, route of exposure of owls to trichomonads in Hawaii remains open to speculation.

Pasteurellosis in the barn owl was probably cat-induced trauma based on location of the foot lesion and serotype (Gerlach, 1994b). Pathogenesis of pasteurellosis in the pueo was less clear although emaciation, evidenced by pathology and antemortem hematology, may have predisposed it to bacterial infection. Aspergillosis in owls suffering from trauma or trichomoniasis would support Campbell's (1986) assertion that the fungus usually is an opportunistic pathogen.

Our percentage of emaciated owls was close to the findings of Newton et al. (1991) and Cooper (1993), but greater than those of Schultz (1986) and Aye et al. (1995). In Europe (Glue, 1973) and on the U.S. mainland (Marti and Wagner, 1985), emaciation in barn owls was attributed to winter exposure and lack of available prey. In Hawaii, the year-round, temperate weather would rule out exposure as a cause of death in owls. Rats, mice, and birds are abundant on all islands and comprise the predominant portion of the barn owl diet in Hawaii (Snetsinger et al., 1994). On the U.S. mainland and Europe, emaciation in owls occurs more frequently in immature birds (Glue, 1973; Marti and Wagner, 1985) in contrast to the equal prominence of adults and immatures in our sample.

The paucity of life history information for barn owls in Hawaii makes conclusions as to why they die from starvation speculative. Based on our pathological analyses, we found no predisposing causes of emaciation in most birds. Likewise, the return of hematologic values to normal limits (Smith and Bush, 1978) in a successfully rehabilitated emaciated barn owl is evidence that some birds suffer from simple uncomplicated emaciation. Possible reasons for this include low digestive efficiency of barn owls (Barton and Houston, 1993), lower amount of fat stores relative to other raptors (Honer, 1963), or inability to minimize energy expenditure when recovering from bouts of starvation (Handrich et al., 1993). Other reasons include lack of available prey, natural predator-prey cycles or intraspecific competition for prime foraging habitat.

Based on our data, there was no evidence for exposure to enough organochlorine pollutant residues, organophosphates, or carbamates to cause death. Although diagnosis of organochlorine poisoning in

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birds is ideally done through analysis of the brain (Stickel, 1973), insufficient tissue was available for this study. The detectable levels of organochlorines in livers and kidneys of barn owls in Hawaii (Table 2) were considered within background levels and much below levels in the brain (Porter, 1993) or liver (Newton et al., 1991) considered toxic for raptors. Brain cholinesterase activity in barn owls was considered within normal limits (Hill, 1988). Brain cholinesterase activity in pueo was consistent with that seen in healthy raptors (Hill, 1988). This, in conjunction with lack of exposure history, does not support the likelihood that organophosphorus or carbamate pesticides caused death in pueos or barn owls. Possible reasons for this include decreasing use of organochlorine pesticides in Hawaii or owls frequenting areas where exposure to pesticides is minimal.

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