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## Probable Drowning of Tundra Swans on the Northern Coast of California

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Incidents of suspected drowning have been reported for ducks in North America (Wooten, 1954, *J. Wildl. Manage.* 18: 140–141; Denson, 1961, Waterfowl populations and a comparison of hunting methods on south Humboldt Bay, California in 1959 and 1960, M.S. Thesis, Humboldt State University, Arcata, California, pp. 108–109). There are only a few reports of drowning of swans in North America (Fleming, 1908, *Auk* 25: 306–309; Fleming, 1912, *Auk* 29: 445–448). This is a report of the probable drowning of tundra swans (*Cygnus columbianus*) on the northern coast of California.

On 7 January 1984, dead swans were reported at the mouth of the Eel River, Humboldt County, California. Aerial observations confirmed the presence of approximately 135 swan carcasses in the sand on the north and south spits of the river mouth. Approximately 750 live swans, in six groups, were seen grazing in nearby agricultural fields. Based on the U.S. Fish and Wildlife Service waterfowl survey of the Eel River delta on 3 January 1984, an estimated 14,200 ducks (composed of 11 species), 975 coots (*Fulica americana*) and 861 swans were present on the area. However, during the aerial observations of swan mortality over the Eel River and a surrounding 65 km<sup>2</sup> area, no dead birds other than the 135 swans were seen on the coast, in the river, or in adjacent fields.

On 10 January, 95 swan carcasses were collected from the north spit beach by staff of the U.S. Fish and Wildlife Service. Each

swan was identified as a tundra swan by Dr. Stanley Harris, Humboldt State University, Arcata, California.

Two swans were sent to the National Wildlife Health Laboratory, U.S.F.W.S., Madison, Wisconsin. Five additional swans were sent to the California Department of Fish and Game Field Station in Rancho Cordova, California. The remaining 88 swans were frozen. They were later thawed and necropsied at Humboldt State University between 14 January and 30 October 1984.

The sex of each swan was determined internally. Swans with gray plumage on the head and neck were identified as immature (under 2 yr old). Swans lacking gray plumage were considered adults.

Heart blood from 67 swans was tested for the presence of *Pasteurella multocida*. Brain Heart Infusion agar was inoculated with heart blood from each swan and incubated at 37 C for 48 hr. Suspect colonies were characterized by a Gram stain, and tested for motility, growth on MacConkey agar, production of catalase, cytochrome oxidase, urease, H<sub>2</sub>S, indole and gelatinase, ability to utilize citrate as the sole source of carbon, and capacity to metabolize glucose and lactose (MacFaddin, 1976, *Biochemical Tests for Identification of Medical Bacteria*, Williams and Wilkins Company, Baltimore, Maryland, 312 pp.).

The gizzard and its contents were visually examined for the presence of lesions, lead shot and parasites. A hydraulic gizzard contents separator (Brewer, 1981, *J. Wildl. Manage.* 45: 496–498) also was used to check for lead shot.

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After each carcass was thawed, both lungs were removed, blotted dry and weighed. To estimate the amount of fluid present, 53 individual lungs from 27 swans were drained on a sloping tray and weighed hourly, for 2 hr. Preliminary studies on 10 lungs indicated over 95% of the fluid in the lungs was drained by the end of 2 hr.

The exterior postmortem condition of the swans appeared similar. Sand was embedded in the plumage of all the swans, penetrating completely to the skin on many. Minor to extensive scavenging was observed on 34 (39%) of the 88 swans.

Forty-five (51%) of the 88 swans were males and 37 (42%) were females. We were unable to determine the sex of six swans due to scavenging. Forty-eight (55%) swans were adults; 39 (44%) were immature. The age of one swan was not determined due to scavenging.

The swans appeared to have been in excellent condition at the time of death. Except for a few immature swans, all had large subcutaneous fat deposits over the sternum and near the base of the tail, as well as fat distributed throughout the viscera and around the heart.

*Pasteurella multocida* was not isolated from the heart blood of any of the 67 swans tested, but a variety of other bacteria was observed. The bacteria from eight swans were further characterized; a bacterium tentatively identified as *Aeromonas* sp. was found in the heart blood of six swans. Gram positive cocci and unidentified Gram negative rods were also observed.

The two swans examined in the National Wildlife Health Laboratory had no lesions of avian cholera, botulism, or any other common waterfowl diseases. *Aeromonas hydrophila* was isolated from the livers and lungs of both swans; it was speculated that its presence was due to inhalation of contaminated water (Stroud, pers. comm.).

No evidence was found for the involve-

ment of organophosphates or carbamates. Tests were conducted on the livers of four swans by the California Department of Fish and Game Pesticide Laboratory for the following pesticides: Atrazine, Malathion, Diphenamid, Methidathione, DEF, Simazine, Pronamid, Carbaryl, Fenthion, Guthion, Parathion, and Methyl Parathion. There was no evidence for involvement of any of these pesticides (Littrell, pers. comm.). Likewise, tests for cholinesterase inhibition, an indication of carbamate or organophosphate poisoning, were conducted by the Patuxent Wildlife Research Center, U.S.F.W.S., Laurel, Maryland, and were negative (Stroud, pers. comm.).

Strychnine and fumarin are two pesticides licensed for use in Humboldt County. Although no tests were conducted specifically for these two chemicals, dyes are required by law to be added when these chemicals are mixed with grains in California. Ingestion of the treated grain by waterfowl normally produces staining of the proventriculus and gizzard: green for strychnine and blue for fumarin. No stain was seen in any of the necropsied swans. Further, with a fast-acting toxin such as strychnine, one would expect to find some of the grain bait still in the birds; no grain was seen in the swans. With a slow-acting toxin such as fumarin, one would have expected a scattering of dead swans, rather than finding all of the carcasses concentrated at one site. Thus, it is unlikely that either of these two toxins was involved.

Gizzard contents consisted largely of sand; 36 (41%) of the 88 gizzards contained *Eleocharis* seeds. A single lead pellet was found in each gizzard of two of the 88 swans. Unidentified nematodes were found under the gizzard lining of three swans. Blackening of the gizzard lining was found in 19 (22%) of the 88 swans. Sloughing of the gizzard lining was observed in 17 (19%) of the 88 swans. Absence of other tissue changes suggests these were postmortem sequelae.

The only consistent lesions seen in the swans were fluid-filled, reddened lungs. Of the 53 lungs evaluated for fluid content, an average of 16.5 g of fluid was drained from each lung (Table 1). The amount of fluid present may have been influenced by postmortem exudation or immersion in salt water. Some fluid may have been pulmonary edema, either independent of, or associated with, water aspiration. Freezing and thawing may also have contributed to the fluids present. The reddened appearance of the lungs may have been influenced by postmortem imbibition of hemoglobin pigment, as well as the freezing and thawing process. However, these changes also would appear compatible with saltwater drowning.

Sand was noted in the intact tracheas of two unscavenged swans. Fluid was observed in the intact trachea of one unscavenged swan.

In addition to the lungs, hemorrhage was observed in other areas in 26 (30%) of the 88 swans. Five swans had hemorrhage on the gizzard; six on the heart; six at the femoral-pelvic joint; six in the chest cavity dorsal to the lungs and 13 on the liver. Four swans had hemorrhages in more than one area. Recently broken ribs were seen in three swans, but no other broken bones were found.

We found evidence of previous shootings in three swans. One #4 lead pellet was found lying outside the gizzard in one swan; a second swan had one pellet embedded in the pectoralis muscle, a third had one pellet embedded in the ventral portion of peritoneum. These all appeared to be old wounds.

Several observations seem pertinent to determining the cause of death of the swans. The good general condition of the swans indicated that death occurred rapidly. No sick birds were seen. The carcasses were all found in one location and all exhibited a similar state of postmortem changes, suggesting the swans died simultaneously. Based on gizzard contents, there

TABLE 1. Fluid loss from 53 individual lungs taken from 27 tundra swans found dead 7 January 1984 at the mouth of the Eel River, Humboldt County, California.

	$\bar{x}$	SD	Range
Initial lung weight (g)	56.8	$\pm 12.9$	36.3–84.5
Fluid loss (g)	16.5	$\pm 9.6$	3.8–46.7
Change due to fluid loss (%)	26.5	$\pm 11.9$	8.8–57.1

was no evidence the swans had ingested toxic substances and toxins and pesticides were not evident in tissues. Taken together, these factors seem to eliminate the likelihood of ingested poisons.

Weather and tide conditions at the time of the swan mortality are also a consideration. During the previous week swans had been observed at dusk, swimming and preening near the mouth of the Eel River, where the swans were later found dead. Some of the live swans were seen drifting beyond the mouth of the river into the surf.

Due to recent storms, the river current was fast during this time period. River flow is monitored at Scotia, approximately 25 km upstream from the mouth of the Eel River. Between 1 and 7 January 1984, recorded river flow ranged from 10,300 to 37,300 cubic feet per second (cfs). In contrast, the year-round mean river flow is 7,370 cfs. There was also a large tidal range of  $-0.2$  m to  $2.3$  m during the first week of January, with the low tides in the evening hours. This combination can create a damming effect on the river at high tide, with fast currents then releasing the water during low tide (Borgeld, pers. comm.). These currents may have carried the swans into rough surf. Wooten (1954, op. cit.) also reported that the heaviest observed losses from drowning among ducks in Humboldt County occurred near the mouth of the Eel River. He further noted that most losses occurred on the beaches, immediately following low tides.

The saturation of plumage and skin with sand, and the presence of hemorrhage on internal organs and around limbs, are conditions similar to those reported by Wooten (1954, op. cit.) for drowned ducks in Humboldt County.

To our knowledge, drowning has been reported only rarely in waterfowl as large as swans. However, considering all of the information, drowning appears to have

been the most likely cause of mortality in the tundra swans.

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### **Idiopathic Enteropathy in the Larval Pacific Herring, *Clupea harengus pallasii* (Valenciennes)**

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The Pacific herring constitutes an important commercial fishery around the North Pacific rim, as well as being an important forage species. Various factors which may affect early life stages and thus recruitment into the adult population have been studied. These include predation on eggs (Taylor, 1964, Fish. Res. Board Can. Bull. 143: 1-81) and larvae (Stevenson, 1962, J. Fish Res. Board Can. 19: 735-810; Fraser, 1969, J. Fish. Res. Board Can. 26: 1743-1762), high density of egg masses (Taylor, 1971, Rapp. Cons. Explor. Mer 160: 34-41), and temperature and salinity (e.g., Alderdice et al., 1971, J. Fish. Res. Board Can. 28: 1545-1562).

The purpose of this paper is to report preliminary observations of a severe necrotizing enteropathy in laboratory-reared Pacific herring hatchlings. Although several diseases have been reported in adult herring, diseases of larval herring, which may be significant determinants of a given

year class success, are apparently unstudied and not reported in the literature. Difficulties in the collection, identification, and preparation of larval fishes from mixed wild populations have precluded the study of these life stages. Thus, this report is the first description of a disease of larval herring and, as well, demonstrates methods for the effective study of diseases of these animals.

Adult herring were obtained for laboratory conditioning and spawning from Puget Sound stocks in both 1982 and 1983. Conditioning, spawning and egg incubation were identical in each year and were effected using methods similar to those described by Alderdice et al. (1971, op. cit.). Animals examined in this study included those whose eggs were incubated under apparently optimal conditions as well as those used to examine the effects of environmental contaminants on egg development. Larval herring were collected for histological and ultrastructural studies of tissues between 18 and 30 hr post-hatching. All animals were fixed whole in

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