

RESPONSE OF A REMNANT POPULATION OF ENDANGERED WATERBIRDS TO AVIAN BOTULISM

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ABSTRACT: The endangered Hawaiian stilt (*Himantopus mexicanus knudseni*) and endangered Hawaiian coot (*Fulica alai*) are uncommon statewide in the Hawaiian Archipelago. On Hawaii Island, these waterbirds are restricted to a few wetlands, including 2 ancient Hawaiian fishponds at Kaloko-Honokohau National Historical Park. An avian botulism outbreak was documented there in 1994, and population estimates before and after the outbreak were made. The outbreak appeared to interrupt the coot's, but not the stilt's, breeding season. The coot population was decimated, but the stilt population appeared to have been less impacted. Both populations have recovered substantially, although the amount of immigration into these populations is unknown. Continuous rigorous removal of fish and bird carcasses appears to have controlled botulism outbreaks at the site. The potential for catastrophic loss of endangered waterbirds at major wetlands on other Hawaiian Islands should be reevaluated for site management and recovery plans.

Key words: avian botulism, endangered species, fishpond, Hawaiian coot, Hawaiian Islands, Hawaiian stilt, waterbirds, wetlands.

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Random events like disease epidemics can have serious effects on small populations of endangered species, such as loss of genetic diversity and even extinction. The objective of my paper is to document the serendipitous decline and recovery of endangered waterbirds caused by avian botulism that occurred during a waterbird breeding study at Kaloko-Honokohau National Historical Park (KAHO), north of Kailua-Kona, on Hawaii Island (Fig. 1), from 1992 through 1995. I thank Chuck and Danny Stone, Cliff Smith, Francis Kuailani, Laura Schuster, Linda Elliott, Thierry Work, Betsy Webb, David Foote, the staff at the Natural Energy Lab of Hawaii, and many others for helping with day-to-day dealings and documentation of this disease disaster. Also thanks to NOAA and Neil Fujii of DLNR for providing weather data, Lyman Abbott for his map-making skills, Joel Simasko for his computer advice, and special thanks to Shannon Hao and Walter Watson for their field support during difficult times. This work was supported by the Research Corporation of the University of Hawaii at Manoa with a grant from the National Park Service.

BACKGROUND

Resident endangered Hawaiian coot (*Fulica alai*) and Hawaiian stilt (*Himantopus mexicanus knudseni*) populations on the island of Hawaii (hereafter Hawaii I.) represent <5% of total statewide populations (Shallenberger 1977, Paton and Scott 1985, Paton et al. 1985, Engilis and Pratt 1993). Little is known about Hawaiian stilt movement throughout the state of Hawaii (Engilis and Pratt 1993, Reed et al. 1994) and nothing is known about their movements between Hawaii I. and the rest of the main islands (Paton et al. 1985), although the relatively

stable population counts on Hawaii I. strongly suggest that such movements are uncommon. Some inter-island movement has been documented for Hawaiian coots (Banko 1987, Engilis and Pratt 1993), but none specifically between Hawaii I. and the other main islands.

Populations of the species are small in KAHO; however, KAHO has the best remaining waterbird habitat on Hawaii I. and maintains roughly 70-80% of the coots and 50% of the stilts for the island. Most of the waterbird

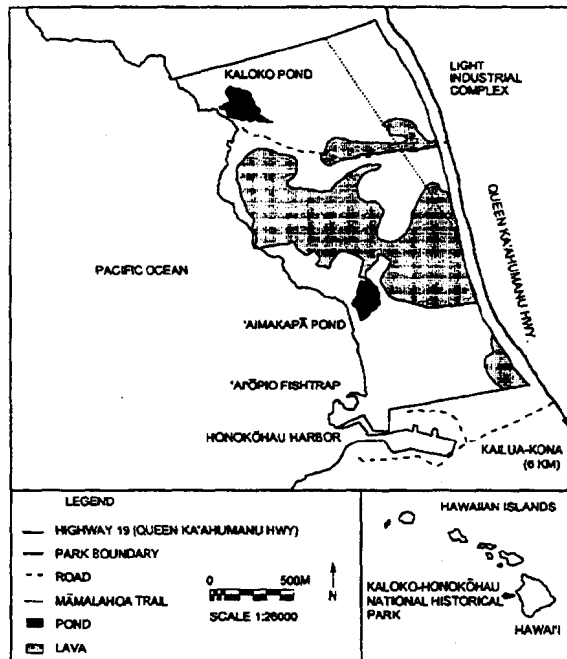


Fig. 1 Map of fishponds at Kaloko-Honokohau National Historical Park on Hawaii Island.

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habitat is associated with KAHO's 2 excellent examples of old Hawaiian fishponds: Aimakapa and Kaloko. Stilt are believed to make regular coastal movements between Aimakapa Fishpond and Opaepala (Makalawena) Fishpond to the north, as well as other fishponds and smaller sites such as anchialine pools along the Kona coast (Shallenberger 1977, Paton et al. 1985). It appears that other waterbirds, including coots and migratory ducks, also move along the coast between Aimakapa and Opaepala (Shallenberger 1977), and is the reason that these two fishponds should be considered as an ecological unit.

Past records indicate that prior to 1977, Hawaiian stilt counts at Aimakapa usually ranged from 4 to 8 birds (with a high count of 11 birds), which was approximately 30-50% of the entire island's population (Shallenberger 1977). Shallenberger (1977) also reported that migratory waterfowl counts rarely exceeded 100 birds, which was similar to my 1992 and 1993 pre-botulism counts and coincides with records on file at Bernice P. Bishop Museum (Robert Pyle, pers. comm.).

Apparently waterbird use relative to Opaepala and Aimakapa fishponds has changed somewhat since the 1960s and early 1970s; it appears that stilt use at Aimakapa has increased and use at Opaepala declined. Stilt counts at Opaepala were reportedly "almost invariably" higher than numbers for Aimakapa (Shallenberger

1977), but in 1993 Aimakapa had a stilt population as large or larger than Opaepala (Morin, pers. obs.).

In late January 1994, dead waterbirds began to be found in 1 of 2 fishponds (Aimakapa) at KAHO; several sick and freshly dead birds were sent to Honolulu for analysis. In early March 1994 avian botulism (*Clostridium botulinum* type C) was confirmed for the first time on Hawaii Island as the cause of death for wild birds (Thierry Work, National Biological Service D.V.M, pers. comm.). Both Hawaiian coots and Hawaiian stilts were affected, as well as common migratory waterfowl, primarily northern shovelers (*Anas clypeata*) and a few northern pintails (*Anas acuta*) (Fig. 2). Dead waterbirds were also recovered or seen in at other wetlands outside KAHO, including an anchialine pool, Opaepala Fishpond, and a nearby sewage treatment plant (Morin, pers. obs.).

Avian botulism has rarely been documented in Hawaii State: before 1987 only 2 outbreaks were reported. The earliest was at Kaelepulu Fishpond (now also called Enchanted Lake) on the island of Oahu from December 1952 to January 1953, but botulism toxin was not verified (Brock and Breese 1953). The second outbreak was in a Lihue settling basin on the island of Kauai that began in 1966 (Swedburg 1967). During the Lihue outbreak, botulism toxin was verified in some carcasses, but the presence of pesticides in those carcasses made it unclear whether the toxin caused the deaths or if the

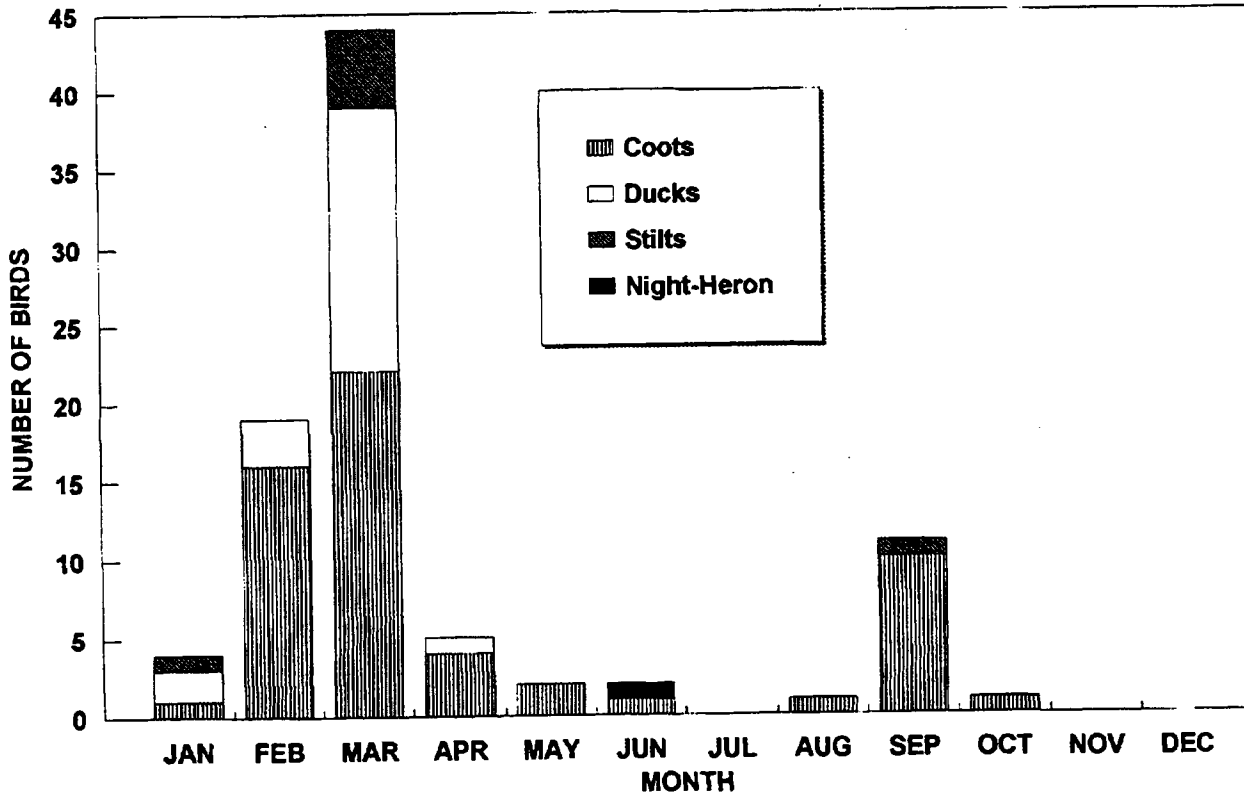


Fig. 2 Dead waterbirds found in west Hawaii Island during 1994, primarily from Aimakapa Fishpond.

toxin was the result of bacterial activity within the carcass after death from other causes. Beginning in 1990, avian botulism has recurred approximately annually in endangered waterbirds at Hanalei National Wildlife Refuge on the island of Kauai (Kathleen Fruth, USFWS, pers. comm.).

In retrospect, other probable outbreaks on Hawaii I. were identified that occurred prior to the 1994 botulism outbreak at Aimakapa Fishpond. The oldest probable (and recurring) outbreak began in some more northerly fishponds at Kona Village Resort in July 1987. This initial outbreak killed 8-12 domestic mallards (*Anas platyrhynchos*) and an introduced black swan (*Cygnus atratus*) (Betsy Webb, D.V.M., pers. comm.). September 1995 was the most recent probable outbreak at these same fishponds, which were heavily stocked with fish and introduced waterbirds. Another probable outbreak killed some Hawaiian stilts at Opaepala Fishpond, also on the western coast of Hawaii I., in the late summer of 1993 (Ron Bachman pers. comm.). In these die-offs, botulism toxin was never clinically verified in fresh carcasses, but symptoms and field signs were consistent with botulism-caused mortality. It is possible that other outbreaks have occurred but have gone unreported. In 1994 the last dead bird was recovered from Aimakapa Fishpond in October (Fig. 2). The outbreak was apparently quiescent during the rest of 1994 and 1995.

SITE DESCRIPTION

KAHO is a newly acquired national park located on the Kona coast immediately north of Honokohau Harbor on Hawaii Island. KAHO lies at the base of the volcano Hualalai, which is still considered to be active. Along the dry Kona coast many old lava flows are evident, often extending down to the sea. One of these flows is immediately adjacent to both Aimakapa and Kaloko fishponds.

KAHO has many sites with outstanding Hawaiian cultural values (Kelly 1971) but also contains some of the most productive endangered waterbird wetlands on the island (Shallenberger 1977; Engilis and Reid in press). Kaloko Fishpond, Aimakapa Fishpond, and Aiopio Fishtrap (Fig. 1), rocky tidal areas, and numerous anchialine pools (Maciolek and Brock 1974, Chai 1991) are situated within the park.

Kaloko Fishpond is approximately 4.5 ha, Aimakapa Fishpond approximately 6 ha of open water and another 6 ha of marshland that was once fishpond, and Aiopio Fishtrap is about 0.7 ha (Kikuchi and Belshe 1971). Although the fishponds contain many rock walls and other constructions made by ancient Hawaiians, the sites were undoubtedly natural wetlands prior to their modification. Aimakapa is considered to have originally been an inland pond behind a barrier sand beach and Kaloko

a natural bay separated from the sea by a man-made rock wall (Wyban 1992). Both fishponds are surrounded by natural anchialine pools.

Aimakapa and Kaloko fishponds experience tidal fluctuations. Both of the fishponds receive substantial subsurface fresh water from upslope and are brackish, although there are considerable variations in salinity due to tidal stage, location, and sample depth (Kikuchi and Belshe 1971). Substantial subsurface freshwater outflows along the Kona coast are a well known local phenomenon (Maciolek and Brock 1974, Kay et al. 1977, Parrish et al. 1990). The brackish water is produced where coastal springs mix with ocean water (Parrish et al. 1990). Brackish fishponds and anchialine pools are scattered all along the Kona coast, even occurring in what appears to be barren lava flows.

Since the 1960s, the seawall at Kaloko Fishpond has been breached and damaged by storms; salt water freely flows into the fishpond, especially during high tides. Aimakapa's subsurface connections to the ocean are not visible and there is a lag in the tidal effects, indicating some resistance.

Water samples taken in 1994 at Aimakapa Fishpond by USGS Water Resources Division (William Meyer, USGS, pers. comm.) indicated that the fishpond had a salinity of about 13 ppt, only slightly saltier than the groundwater (10 ppt) and considerably less salty than seawater (35 ppt). Kaloko Fishpond is currently much more saline than Aimakapa, with salinities ranging from 18-34 ppt. (William Meyer, pers. comm.). In 1971, Kikuchi and Belshe described the central waters of Aimakapa Fishpond as having a temperature of 27.8 °C and a salinity of 7.9 ppt. They described Kaloko Fishpond as a layered body of water, with surface temperatures of 20-24 °C and salinities that ranged from 18-24 ppt in the deeper areas and temperatures of 18-20 °C and salinities of 4-8 ppt near the springs in the shallow back areas. Maciolek and Brock (1974) reported the salinity of Aimakapa as 7-8 ppt. Chai (1991) stated that it appeared that Kaloko Fishpond had more freshwater influx in 1971 than what he found during his 1988 field work.

KAHO is bordered on the south by Honokohau Harbor, by a highway on the east, by a proposed resort development on the north, and by the Pacific Ocean on the west. An industrial park lies immediately east of KAHO above the highway, and on the slopes of Hualalai volcano are numerous housing subdivisions. All of these are currently using cesspools for wastewater. A new wastewater treatment plant just south of KAHO, treating wastewater from the coastal business district in Kona, has been dumping treated sewage water into a pit across the highway immediately south of KAHO since at least March 1994. A golf course which was to be watered

with the treated sewage water has yet to be built. The potential for nutrient loads and chemicals to enter the surface groundwater appears therefore to be very high.

METHODS

Bird censuses were conducted at least weekly at Aimakapa and Kaloko fishponds from February 1992 through July 1993, and from January 1994 through July 1994, and at least twice a month from August 1994 through February 1995, and sporadically thereafter. Counts of all waterbirds and shorebirds were made with binoculars from the western shore (Aimakapa) or the southern shore (Kaloko). Surveys for coot, stilt, and pied-billed grebe (*Podilymbus podiceps*) nests were made at both fishponds by boat (Aimakapa) and from shore (both fishponds) approximately weekly during the stilt breeding season of February through July, and less often otherwise. Nest site, clutch size, chicks, and fledglings were recorded for all nests. I conducted all censuses and nest surveys. Censuses at both fishponds were usually made within an hour of each other. Most dead birds were retrieved from the shoreline, or by boat by technicians checking a predator trap line several times a week; some carcasses were retrieved during the censuses and nest surveys. Most fresh carcasses were packed on ice and flown to Honolulu for verification of botulism toxin.

RESULTS

Although Hawaiian coots were reported to have used Kaloko Fishpond in the past (Shallenberger 1977, Banko 1987), coots and migratory ducks were never seen at Kaloko Fishpond during my 139 censuses. A few Hawaiian stilts periodically used Kaloko Fishpond (average of 1.1 ± 2.64 SD stilt per census, $n = 139$, range 1 to 18), especially the exposed mudflats and shoreline that have been hand-cleared of the alien plant *Batis maritima*. No waterbirds were ever found dead from avian botulism at Kaloko Fishpond.

For all censuses made at Aimakapa Fishpond from 1990 to December 1995, the mean Hawaiian coot count was 45.0 ± 23.94 SD birds ($n = 168$ censuses, range = 0 to 103). However, during the 1980s as many as 188 coots were sighted there (Jaan Lepson, unpubl. data). For the botulism year of 1994, the mean number of coots per count was 20.9 ± 23.22 SD ($n = 48$ censuses), whereas during the baseline censuses from February 1992 through July 1993, the coots averaged 56.5 ± 13.31 SD birds per census ($n = 109$). On 7 January 1994, just prior to the botulism die-off, the Hawaiian coot count was the highest for all my Aimakapa counts (103 birds). On that date there were also 20 Hawaiian stilts, 145 ducks, 1 pied-billed grebe, 4 cattle egrets (*Bubulcus ibis*), and 3 shorebirds. The coot population reached its lowest count

(0) in early November 1994 (Fig. 3), but had recovered to pre-botulism levels by December 1995.

For all censuses made at Aimakapa Fishpond from 1990 to December 1995, the mean Hawaiian stilt count was 12.9 ± 5.01 SD birds ($n = 179$ censuses, range = 0 to 29). Although the stilts are known to have suffered some mortality (Figs. 2 and 4), the mean number of stilts per census during the 1994 botulism year (12.9 ± 5.65 SD birds, $n = 52$ censuses) was virtually identical to the mean number of stilts per pre-botulism census (12.9 ± 4.69 SD birds, $n = 117$ censuses) from February 1992 through July 1993. Use of Kaloko Fishpond by stilts increased to 2.1 ± 3.78 SD stilts per census ($n = 49$) during the botulism year of 1994, in contrast to a mean of 0.3 ± 0.77 SD stilts per census ($n = 82$) from January 1992 through July 1993.

From 1992 through 1995, no coot or stilt nests were found in Kaloko Fishpond. Although Hawaiian coots normally breed year-round (Shallenberger 1977; Morin pers. obs.), coot nests and eggs were not found in Aimakapa from January to April 1994 during the botulism outbreak. Hawaiian stilts continued to nest and breed normally in Aimakapa from March through July during 1994, although there were fewer nests. The number of fledgling stilts per nest was not significantly different from a pre-botulism year (Morin, in prep.)

Carcasses of 56 Hawaiian coot, 6 Hawaiian stilts, 1 Black-crowned Night-Heron (*Nycticorax nycticorax hoactli*), 21 shovelers, and 1 pintail were recovered (Fig. 2). Botulism toxin was verified in most of the fresh carcasses (T. Work pers. comm.). Based on pre-botulism population censuses, I estimate that about only one-half of the carcasses were retrieved, in spite of intensive searching.

DISCUSSION

Almost all of the research and knowledge concerning avian botulism has been gathered in mainland freshwater ecosystems without tidal influence (T. Rocke, NBS, pers. comm.). Aimakapa, Kaloko, and Opaepa fishponds, as well as the numerous anchialine pools that dot the Kona coastline, have connections to underground freshwater sources as well as to the ocean. The uniqueness of this situation, as well as uncertainty as to the origin of the outbreak and the presence of protected archaeological sites, make it less clear what should be done for effective preventative management.

Recent reports do not confirm that the botulism toxin is present in the water or mud except in the immediate vicinity of toxic carcasses or dead animal tissue (Haagsma 1987). Some birds can apparently recover from botulism, if the toxin dose is not too great. However, it is unclear how many wild birds would survive

NUMBER OF HAWAIIAN COOTS

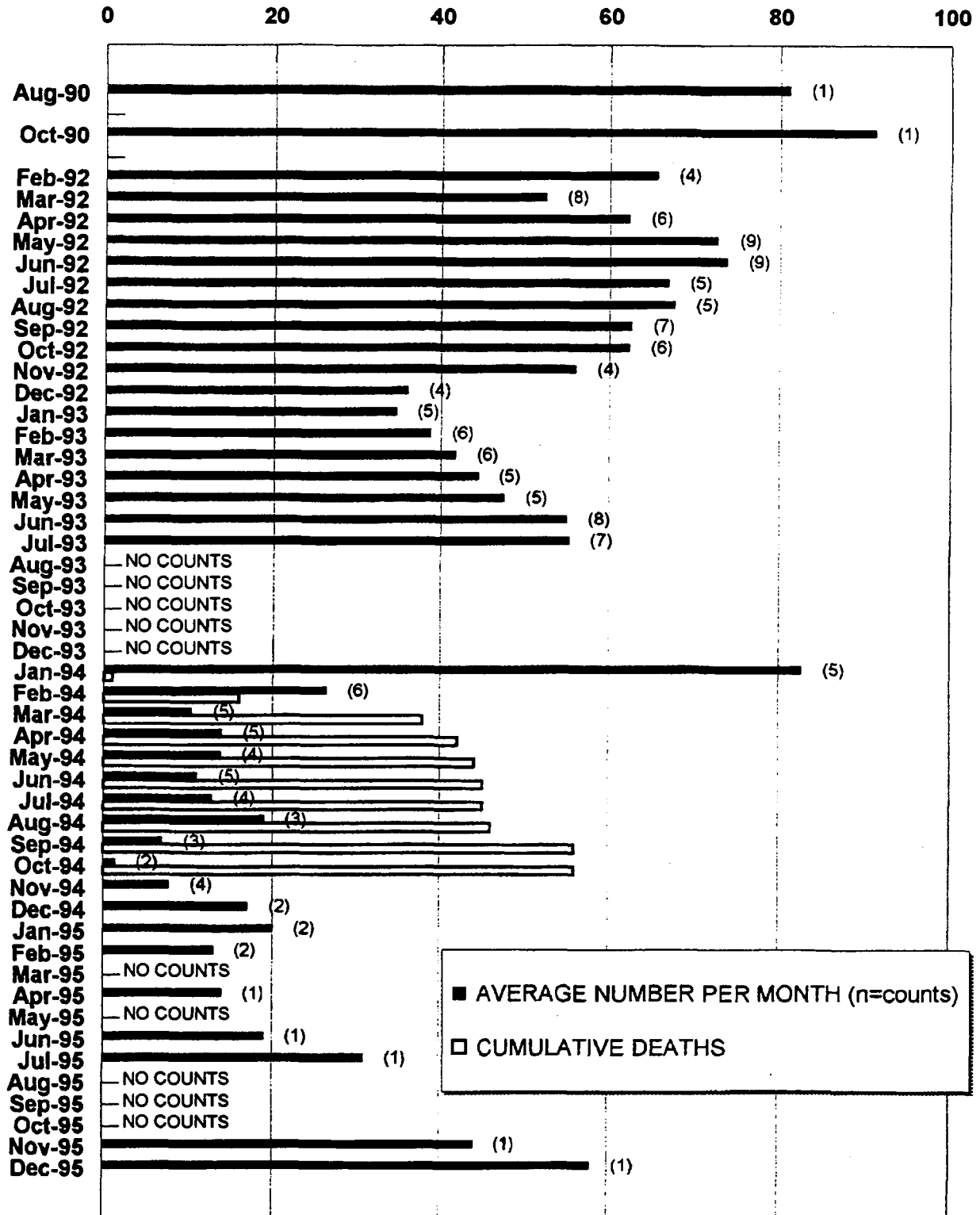


Fig. 3 Hawaiian coot population at Aimakapa Fishpond and cumulative botulism-caused coot mortality, 1990-1995.

without human intervention. After recovering from botulism, mallards were found to have no effective immunity (Haagsma 1987).

Clostridium botulinum type C can be found worldwide in soil, freshwater areas, some marine environments, and even in guts of clinically healthy birds (Haagsma 1987). Although humans are believed to be relatively resistant to type C toxin (Clark 1987), it can be toxic to birds as well as mammals, fish, and amphibians (Asuma and Itoh 1987, Haagsma 1987, Jensen and Price 1987, Mitchell and Rosendal 1987). There are many different situations that can produce an avian botulism die-off and no single set of environmental conditions is associated with every botulism outbreak (Wobeser 1987).

Both macro- and micro-environmental conditions can apparently trigger and/or perpetuate botulism outbreaks. These scenarios include: (1) recently flooded land, possibly where invertebrate carcasses increase the protein available for bacterial growth and toxin production (Clark 1987, Jensen and Price 1987). (2) Infection of the *C. botulinum* bacteria with specific bacteriophages (bacteria viruses) that increase the toxicity of the toxin (Jensen and Price 1987; Wobeser 1987; Chris Brand, pers. comm.). *C. botulinum* bacteria can be found in the environment both with and without these phage infections, and free phage can be found as well (T. Rocke, pers. comm.). However, only botulism bacteria infected by the specific phages produce the deadly forms of toxin (T. Rocke, pers. comm.). These phages may play a primary role. Different strains of botulism type C are infected with 1 or more of these viruses and when they lose these viruses experimentally, the strains stop producing the dominant C1 toxin (Eklund et al. 1987). These authors assert that the bacteria-virus relationships play an important role in the toxic characteristics of the botulism bacteria and botulism outbreaks in natural settings. Different botulism bacteria and phages are moved between distant areas by migratory birds, wind, and floods, which can then infect isolated botulinum bacteria elsewhere (Eklund et al. 1987). The role of phages in producing the C1 toxin may help to explain why botulism outbreaks are difficult to predict by examination of environmental conditions alone and why management actions result in varying amounts of apparent success or failure.

(3) Low pH (e.g., 6.2 to 10.5; Clark 1987, Mitchell and Rosendal 1987). The pH of the environment may not be important if the botulism bacterium is contained within a carcass with the appropriate pH. (4) Optimal environmental temperatures, thought to be about 30-37°C, although toxin production can occur between 10°C and 47°C (Mitchell and Rosendal 1987, Smith 1987). (5) Anaerobic conditions such as those caused by decay-

ing organic matter, especially from sources such as animal plankton (Clark 1987, Mitchell and Rosendal 1987). Oxygen depletion is not necessary for the growth of the botulism bacteria, because both invertebrate and vertebrate carcasses provide a microenvironment that is appropriate for bacterial growth. However, anoxic water may cause animal die-offs that can lead to botulism outbreaks (Wobeser 1987). (6) Die-offs of fish, birds, or mammals from any cause, if left to rot in a wetland, can apparently start an avian botulism outbreak (Clark 1987, Wobeser 1987).

Once there has been an initial botulism outbreak, a secondary microcycle can begin. The toxin within the carcasses is consumed by fly maggots, which apparently concentrate the toxin. A duck that has eaten as few as ten intoxicated maggots can develop avian botulism within a few hours (Clark 1987). This secondary cycle is the reason why the immediate and complete removal of all animal carcasses is so important.

It is still unclear which factors precipitated the KAHO botulism outbreak. Probable causes include:

(1) The combination of low tides during the hottest times of the day when the ambient temperature was also high, fostering the growth of the botulism bacteria. However, before 1994 there were days as hot and tides as low. The average mean daily temperature at nearby Keahole Pt. weather station was actually lower each month in 1994 than the average mean daily temperature each month there for the past 14 years (Neil Fujii, Hawaii State Dept. of Land and Natural Resources).

(2) Very low rainfall which led to drought conditions. West Hawaii was officially declared as having a drought during 1994. In both 1993 and 1994, total yearly precipitation for Honokohau Harbor, Keahole Pt., and Lanihau stations in the KAHO watershed was the lowest it had been during the past 11 years (NOAA; N. Fujii, DLNR). This probably reduced freshwater inflow into the pond, and more importantly, indirectly reduced the subsurface fresh water flows from upslope Hualalai. Low rainfall also assured there would be nonexistent or small amounts of cloud cover. The subsurface fresh water flows keep Aimakapa, Kalokoa, and other KAHO sites brackish. Significant development has occurred on the slopes of Hualalai over the last two decades, increasing the deforestation and altering the hydrology. In addition, water wells are a possible source or future source of subsurface water depletion.

(3) Increased nutrient, pesticide, or contaminant load brought into Aimakapa Fishpond from drainage and cesspools in the upslope development that includes agriculture, housing, and an industrial area. Although open pit dumping of wastewater from the new sewage plant reportedly began after the botulism outbreak, it is possible that increased nutrients in the subsurface water

NUMBER OF HAWAIIAN STILT

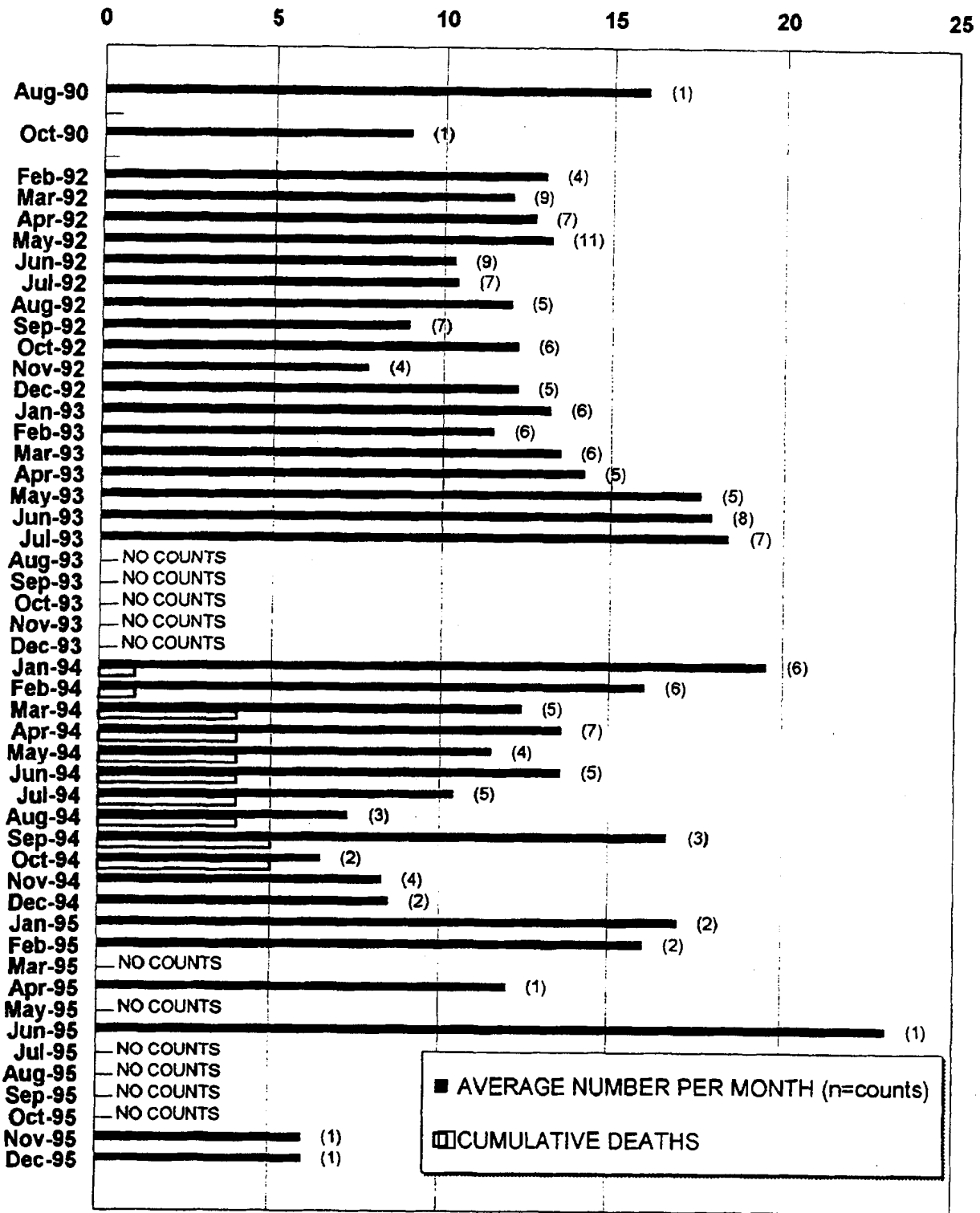


Fig. 4 Hawaiian stilt population at Aimakapa Fishpond and cumulative botulism-caused stilt mortality, 1990-1995.

helped to promote or prolong the die-off. Changes in nutrient and pesticides levels in the water can cause the invertebrate population explosions and die-offs that can lead to botulism outbreaks.

(4) Too much siltation in the pond, allowing the water to warm more easily and anoxic conditions to develop, creating fish or invertebrate die-offs. Of course, these conditions have existed for quite a few years, apparently without previous botulism outbreaks. Dead fish, especially 'ama'ama (mullet: *Mugil cephalus*), were often found along the shoreline of Aimakapa during this outbreak. Dead awa (milkfish: *Chanos chanos*), palani (eye-stripe surgeonfish: *Acanthurus dussumieri*), and dead balloonfish were also found in the fishpond during 1994 and 1995. However, I also saw dead fish at Aimakapa Fishpond during 1992 and 1993 when there was no botulism present. Unfortunately, during the botulism die-off, fish carcasses were not consistently removed until the fall of 1994. In October 1994, 2 freshly dead mullets from Aimakapa were confirmed to have botulism type C toxin in their hearts and guts (Thierry Work, D.V.M., pers. comm.).

(5) Recent management activities within the Park, such as predator trapping, removal of some shoreline canopy-forming trees, and other limited vegetation removal, may have contributed to siltation, water heating, and carcass availability, and may have increased the potential for an outbreak.

(6) Infection by a new bacteriophage into the botulism bacteria in West Hawaii, via migratory waterbirds or introduced waterfowl that were maintained outdoors where contact with wild waterbirds was possible.

Hawaiian coots were never seen, and migratory ducks were very rarely seen in Kaloko Fishpond during my censuses, presumably due to its current high salinity, water depth, and inhospitable non-native shoreline vegetation (pers. obs.), and dead waterbirds were not found in that fishpond during the die-off. Hawaiian stilts forage over a much larger area and variety of habitats than do Hawaiian coots (Morin, pers. obs.) and this generality in foraging behavior may have protected them from getting lethal doses of botulism toxin. The Hawaiian stilt population on Hawaii I. was impacted by the botulism, but it appeared that the population had expanded just prior to the die-off so that the mortality was not obvious. Conversely, the Hawaiian coot population was basically eradicated. On 12 October 1994, I counted only 1 adult Hawaiian coot (plus a coot nest with 6 hatching eggs) at Aimakapa. Eleven Hawaiian stilts were counted during the same census. In November 1994 after an Aimakapa census where no coots were found, a colleague (R. David) and I estimated that fewer than 10 Hawaiian coots remained in West Hawaii and that 20 or fewer remained on the entire island. Because there is

no evidence that regular movements of coots occur between west Hawaii I. and other islands in the state, and because the pre-botulism population size on the Kona coast was so consistent between the two major fishponds, we were very pessimistic about the future of this population. The genetic diversity lost in this subpopulation during the die-off will never be known; the situation was a unique opportunity to document the effects of a genetic bottleneck. The coot population appears to have recovered at Aimakapa (Fig. 3), probably due to increased breeding on or near the site and to immigration from other wetlands on Hawaii I.

MANAGEMENT IMPLICATIONS

It appears that the most consistently effective management practice for controlling botulism outbreaks is to: (1) remove and destroy all carcasses, since each decomposing carcass is a "toxin factory" (Clark 1987). Inspection and removal of vertebrate carcasses appears to have controlled the botulism outbreak at Aimakapa and allowed population recovery. On the mainland, a few waterfowl deaths caused by electrical transmission lines in wetlands have been sufficient to initiate botulism outbreaks (Wobeser 1987). Surveillance must be routine and removal must be immediate and complete, which poses a great logistical problem when resources such as personnel are limited and/or carcasses are spread over a wide area. Carcasses of all vertebrate species must be removed, and it is recommended that the carcasses be buried with quick lime or burned. This has been the management tool used at KAHO, where Aimakapa Fishpond is routinely searched for carcasses and carcasses removed. Inspection must be done carefully so that the waterbird harassment inherent in such activity does not unduly stress the birds, which can weaken sick birds, or cause nest destruction and abandonment. Some successful nesting did occur during the 1994 outbreak, especially by Hawaiian stilts (Morin, in prep.).

(2) Some management practices developed for inland freshwater botulism outbreaks, such as draining a wetland (to prevent or halt an outbreak), or maintaining a single water level (to prevent an outbreak), do not transfer well to intertidal, brackish water habitats where maintaining an intact native ecosystem is a priority.

(3) Construct or modify ponds to have smooth bottoms and steeply sloped sides (Clark 1987, Wobeser 1987). Altering natural wetlands may reduce botulism but may be counterproductive if the primary goal is to maintain a variety of waterbirds in a predominantly native (e.g., plants, invertebrates) habitat. Hawaiian coot and stilt foraging, nesting, and behavioral requirements are not adequately met by one simplistic pond design.

(4) Chase birds away from the area, although habituation by the birds to hazing tactics eventually makes

the tactics ineffective (Clark 1987). Hazing was never considered a viable option for KAHO because Aimakapa is a primary waterbird habitat and there are few other places for waterbirds to go. Hazing should be considered a short-term response technique, although it does create a dilemma as to whether dispersed birds could further disseminate bacteriophages.

(5) Pump water into the wetland so that the source of the toxin is dispersed or placed out of reach of feeding birds (Clark 1987). Pumping sufficient amounts of water into a wetland also potentially could cool elevated temperatures. Large quantities of unchlorinated freshwater were not available at KAHO. In addition there were serious concerns that drastic alteration of salinity in these brackish ponds could actually worsen the botulism outbreak by causing plant, fish, or invertebrate die-offs. Research should be directed at discovering acceptable fishpond modifications that would allow water and sediment to remain cooler (as much as possible) than the temperatures known to be optimal for botulism bacteria growth. Water should be controlled so that excessive nutrients, pesticides, salinities, or abrupt water quality or quantity changes do not allow animal die-offs to occur, which are known to foster botulism outbreaks.

(6) Keeping water fresh by preventing aquatic invertebrate or fish die-offs due to hypoxia, water stagnation, or contaminants such as pesticides (Wobeser 1987). A great deal of discussion at KAHO centered around the idea of reopening the historic fishpond sluice gate, which has been nonfunctional and silted-in for years. The idea was meant to flush silt from the pond and freshen the water by direct tidal action. However, this action was never implemented for a variety of reasons, including questions about wetland habitat changes that would result from altering the fishpond's salinity by opening the sluice gate. Wetland vegetation in Aimakapa is salt tolerant but not salt loving, and vegetation and insect faunal changes will occur (including possibly an invertebrate die-off) if the salinity of the wetland is permanently altered. Salinity changes would also affect waterbird use of the area, since different bird species have different salt tolerances and vegetation preferences.

Future useful management at Aimakapa might include increasing water flows by removal of vegetation mats that currently buffer the freshwater inflows from upslope, or the reconstruction of a sturdy operational sluice gate to allow periodic (but not continuous) flushing of silt during high outgoing tides without allowing saltwater inflow. At Aimakapa Fishpond, restoration of the historic sluice gate site would require a considerable amount of digging in order to allow water flow through the gate, because several hundred feet of silted-in pond are covered with vegetation and would have to be removed or channelized. A water gauge and monitoring

devices should be installed so that water depth, salinity, pH, water and sediment temperatures, and a few other basic parameters can be monitored continuously over long periods of time. Perhaps more importantly, increased nutrients and contaminants from outside (e.g., wastewater treatment plant effluent) need to be monitored and possibly reduced.

(7) Waterbirds, and especially endangered waterbirds, should be rehabilitated if at all possible during die-offs. If waterbirds are being handled in captivity, the possibility of immunizing them with toxoid (Clark 1987) should be further studied, even though this is impractical for birds in the wild. Many sick birds can be rehabilitated using antitoxin and simple good animal husbandry. However, depending on the numbers and types of species involved, this can be very labor intensive and expensive. Rehabilitation (including antitoxin administration) was done for a few KAHO birds during a few months of the die-off (Elliott, unpubl. rep., Kaloko-Honokohau National Historical Report, avian botulism rehabilitation program. Kaloko-Honokohau Natl. Hist. Park, HI, 1994), especially because endangered species were involved.

(8) Immunization with botulinum toxoid (Clark 1987, Shimizu and Kondo 1987). This has not been considered an effective approach for wild birds, since it provides only temporary protection and is logistically a nightmare to administer because the birds must be captured twice during a specified time interval. However, this should be seriously considered in the future for endangered birds brought in for rehabilitation.

Management for botulism requires that an ecosystem approach be taken; the entire watershed of Hualalai volcano is of critical importance to the coastal wetlands' health and maintenance. New water wells and upslope development and deforestation are prime examples of activities which directly affect the wetlands and require proactive input from agencies representatives in order to educate the public.

Numerous experts have emphasized the lack of understanding that still surrounds the role of bacteriophages in initiating and maintaining botulism outbreaks. Avian botulism has been identified as a critical problem facing Hawaiian waterbirds and it has been recommended that this should be recognized in the Waterbird Recovery Plan (Morin, unpubl. rep., Avian botulism report, Kaloko-Honokohau Natl. Hist. Park, HI, 1994). It was agreed during a 1994 interagency meeting that a response plan(s) are needed for other epidemics as well, and that USFWS Ecological Services should take the lead. A response plan for avian botulism was drafted, including these points: (1) Frequent censuses and monitoring of waterbirds should be conducted statewide. (2) Other agencies and private landowners with waterbirds

or wetlands should be alerted and educated about the botulism problem. (3) Sick and dead waterbirds must be removed immediately and the appropriate agencies contacted in order to see if live or dead specimens should be sent. (4) Sick waterbirds, especially endangered species, should be isolated and rehabilitated if at all possible until the area is again "safe" for re-release. (5) Vertebrate carcass and sick waterbird pick-ups can be done most effectively if personnel from many agencies and groups work together.

Whatever management actions are done, there clearly needs to be a coordinated effort among the agencies mandated to govern these wetlands and migratory and endangered birds, most notably U.S. Fish and Wildlife Service, National Park Service, National Biological Service, and the Hawaii State Division of Forestry and Wildlife. However, the private sector must also be actively educated and engaged in helping with management problems such as botulism, as evidenced by the probable botulism outbreaks on private property which preceded the Aimakapa events. The endangered waterbirds and migratory waterfowl in West Hawaii need to be managed as a single population due to the habitat distribution and waterbird movements; effective management must be statewide rather than piecemeal.

LITERATURE CITED

- Azuma, R., and T. Itoh. 1987. Botulism in waterfowl and distribution of *C. botulinum* Type C in Japan. Pages 167-187 in M. W. Eklund and V. R. Dowell, Jr., eds. Avian botulism: an international perspective. Charles C. Thomas, Springfield, Illinois.
- Banko, W. E. 1987. Population histories—species accounts freshwater birds: Hawaiian Coot 'Alae-ke'oke'o. Part I. Cooperative Park Studies Unit, University of Hawaii. Avian History Report 12C. History of endemic Hawaiian birds. Cooperative Park Studies Unit, University of Hawaii Manoa, Department of Botany, Honolulu, Hawaii. 206pp.
- Brock, V. E. and P. L. Breese. 1953. Duck botulism at Kaelepulu Pond, Kailua, Oahu. 'Elepaio 13:80-81.
- Chai, D. K. 1991. An inventory and assessment of Kaloko Pond, marsh, and anchialine pools at Kaloko-Honokohau National Historical Park, North Kona, Hawaii. Tech. Rep. 76, Cooperative Park Studies Unit, Department of Botany, Univ. of Hawaii Manoa, Honolulu, Hawaii. 16pp.
- Clark, W. E. 1987. Avian botulism. Pages 89-105 in M. W. Eklund and V. R. Dowell, Jr., eds. Avian botulism: an international perspective. Charles C. Thomas, Springfield, Illinois.
- Eklund, M., F. Poysky, K. Oguma, H. Iida, and K. Inoue. 1987. Relationship of bacteriophages to toxin and hemagglutinin production by *Clostridium botulinum* types C and D and its significance in avian botulism outbreaks. in M. W. Eklund and V. R. Dowell, Jr., eds. Avian botulism: an international perspective. Charles C. Thomas, Springfield, Illinois.
- Engilis, A., Jr., and F. A. Reid. In press. Hawaiian waterbirds recovery plan. 3rd revision. U.S. Fish and Wildlife Service, Portland, Oregon.
- _____, and T. K. Pratt. 1993. Status and population trends of Hawaii's native waterbirds, 1977-1987. Wilson Bulletin 105:142-158.
- Haagsma, J. 1987. Avian botulism in the Netherlands. Pages 153-165 in M. W. Eklund and V. R. Dowell, Jr., eds. Avian botulism: an international perspective. Charles C. Thomas, Springfield, Illinois.
- Jensen, W. I. and J. I. Price. 1987. The global importance of Type C botulism in wild birds. Pages 33-54 in M. W. Eklund and V. R. Dowell, Jr., eds. Avian botulism: an international perspective. Charles C. Thomas, Springfield, Illinois.
- Kay, E. A., L. S. Lau, E. D. Stroup, S. J. Dollar, D. P. Fellows, and R. H. F. Young. 1977. Hydrologic and ecologic inventories of the coastal waters of West Hawaii. University of Hawaii Water Resources Research Center Technical Report no. 105. 94pp.
- Kelly, M. 1971. Kekaha: 'Aina malo'o. A survey of the background and history of Kaloko and Kukio, North Kona, Hawaii. Report 71-72. Department of Anthropology, Bernice P. Bishop Museum, Honolulu, Hawaii. 61pp.
- Kikuchi, W. K., and J. Belshe. 1971. Examination and evaluation of fishponds on the leeward coast of the island of Hawaii. Hawaii County Planning Commission, Hilo, Hawaii.
- Maciolek, John A. and Richard E. Brock. 1974. Aquatic survey of the Kona Coast ponds, Hawaii Island. Sea Grant Adv. Rep. UNIH-SEAGRANT-AR-74-04. NOAA Grant # 04-3-158-29. Hawaii County Department of Planning, Hilo, Hawaii. 73pp.
- Mitchell, W. R. and S. Rosendal. 1987. Type C botulism: the agent, host susceptibility, and predisposing factors. Pages 55-71 in M. W. Eklund and V. R. Dowell, Jr., eds. Avian botulism: an international perspective. Charles C. Thomas, Springfield, Illinois.
- Morin, M. P. In prep. Endangered Hawaiian Coot and Hawaiian Stilt reproduction in an ancient Hawaiian fishpond. Journal of Field Ornithology.
- Parrish, J. D., G. C. Smith, and J. E. Norris. 1990. Resources of the marine waters of Kaloko-Honokohau National Historical Park. Technical Report 74. Cooperative Park Studies Unit, Depart-

- ment of Botany, University of Hawaii at Manoa, Honolulu, Hawaii. 114pp.
- Paton, P. W. C. and J. M. Scott. 1985. Water birds of Hawaii Island. 'Elepaio 45:69-76.
- _____, J. M. Scott, and T. A. Burr. 1985. American Coot and Black-necked Stilt on the island of Hawaii. Western Birds 16:175-181.
- Reed, J. M., L. W. Oring, and M. Silbernagle. 1994. Metapopulation dynamics and conservation of the endangered Hawaiian Stilt (*Himantopus mexicanus knudseni*). Transactions of The Western Section of the Wildlife Society 30:7-14.
- Shallenberger, R. J. 1977. An ornithological survey of Hawaiian wetlands. Ahuimanu Productions for U. S. Army, Engineer District, Honolulu. Contract DACW 84-77-C-0036. 406pp.
- Shimizu, T., and H. Kondo. 1987. Preparation and evaluation of botulinal type C toxoid for immunization of pheasants. Pages 357-362 in M. W. Eklund and V. R. Dowell, Jr., eds. Avian botulism: an international perspective. Charles C. Thomas, Springfield, Illinois.
- Smith, G. R. 1987. Botulism in waterbirds and its relation to comparative medicine. Pages 73-86 in M. W. Eklund and V. R. Dowell, Jr., eds. Avian botulism: an international perspective. Charles C. Thomas, Springfield, Illinois.
- Swedburg, G. E. 1967. The Koloa, a preliminary report on the life history and status of the Hawaiian Duck (*Anas wyvilliana*). Hawaii Division of Fish and Game, Department of Land and Natural Resources, Honolulu, Hawaii. 56pp.
- Wobeser, G. A. 1987. Control of botulism in wild birds. Pages 339-348 in M. W. Eklund and V. R. Dowell, Jr., eds. Avian botulism: an international perspective. Charles C. Thomas, Springfield, Illinois.
- Wyban, C. A. 1992. Tide and current: fishponds of Hawaii. University of Hawaii Press, Honolulu, Hawaii. 192pp.