

EPIZOOTIOLOGY AND EFFECT OF AVIAN POX ON HAWAIIAN FOREST BIRDS

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ABSTRACT.—We determined prevalence and altitudinal distribution of forest birds infected with avian pox at 16 locations on Hawaii, from sea level to tree line in mesic and xeric habitats, during 1977–1980. Isolates from lesions were cultured in the laboratory for positive identification of *Poxvirus avium*. Infected birds from the wild were brought into the laboratory to assess differences in the course of infection in native versus introduced species. We also documented distributions and activity cycles of potential avian pox vectors.

Native forest birds were (1) more susceptible to avian pox infection than were introduced species, (2) most likely to be infected during the wet season, and (3) found to have a higher prevalence in mesic when compared to xeric forests. Avian pox occurred in forest birds at all elevations, but highest levels were in the mid-elevational ranges (~1,200 m) where vectors and native birds had the greatest overlap. Temporal and elevational differences in prevalence were apparent throughout the annual cycle. Avian pox probably did not reach epizootic proportions on Hawaii until after introduction of the mosquito and domestic birds in the early 1800s, and since then has had a negative effect on the population dynamics of native forest birds. Today, this introduced disease is an important factor that should be considered in future conservation efforts that are directed at the recovery of native forest birds in Hawaii. Received 8 June 2001, accepted 26 March 2002.

RESUMEN.—Determinamos la prevalencia y distribución altitudinal de aves de bosque infectadas con viruela aviaria (*Poxvirus avium*) en 16 localidades de Hawaii, desde el nivel del mar hasta la línea de bosque en hábitats húmedos y secos, entre 1977 y 1980. Muestras de las lesiones fueron cultivadas en el laboratorio para confirmar la identificación de *Poxvirus avium*. Las aves silvestres infectadas fueron llevadas al laboratorio para evaluar diferencias en el curso de la infección entre especies nativas e introducidas. También documentamos las distribuciones y los ciclos de actividad de los vectores potenciales de *Poxvirus avium*.

Las aves nativas del bosque (1) fueron más susceptibles a infecciones con viruela aviaria que las aves introducidas, (2) presentaron mayor probabilidad de ser infectadas durante la estación húmeda, y (3) tuvieron mayor prevalencia en bosques húmedos que en bosques secos. La viruela aviaria fue registrada en aves de bosque a todas las elevaciones, pero los niveles más altos se registraron a elevaciones medias (~1,200 m), donde los vectores y las aves nativas presentaron una mayor superposición. Las diferencias temporales y de elevación en prevalencia fueron evidentes a lo largo del ciclo anual. La viruela aviaria probablemente no alcanzó proporciones epizooticas en Hawaii hasta después de la introducción del mosquito y de aves domésticas a principios de 1800, y desde entonces ha tenido un efecto negativo en la dinámica poblacional de las aves nativas de bosque. Hoy en día, esta enfermedad introducida es un factor importante que debería ser considerado en futuros esfuerzos de conservación dirigidos a recuperar las aves nativas de bosque en Hawaii.

INTRODUCED AVIAN DISEASES have been suggested as one factor responsible for depletion of native Hawaiian birds (e.g. Warner 1968, Scott et al. 2001a, Ralph and van Riper 1985, Pratt 1994). There has been considerable research conducted on the effect that malarial

parasites have on the native avifauna (van Riper et al. 1986; Atkinson et al. 1995, 2000; Yorinks and Atkinson 2000; Jarvi et al. 2001; Shehata et al. 2001), but less attention has been given to the study of avian pox (*Poxvirus avium*).

Avian pox is a viral infection of birds caused by one of the larger viruses of the poxvirus

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group (Wilner 1969). The virus particle is large, oval, or brick-shaped, about 150 to 250 μm by 265 to 350 μm in size, covered with irregularly spaced surface knobs. Coupar et al. (1990) identify the genome of the avian pox virus as composed of a single double-stranded, 300 Kb DNA molecule. This DNA-containing, enveloped virus develops in the cytoplasm of infected avian epithelial cells (Tripathy and Reed 1997). Infected cells characteristically contain large acidophilic intracytoplasmic inclusions called "Bollinger bodies."

The geographic distribution of the avian poxes is worldwide (van Riper and Forrester 2003). This widespread avian disease appears able to infect all bird families, with some (e.g. Phasianidae and Fringillidae) seeming more susceptible. Avian pox occurs in two forms: (1) the more common skin form, in which discrete, warty, proliferative lesions develop on the skin; and, (2) the more advanced diphtheritic form in which moist, necrotic lesions develop on the mucous membranes of the mouth and upper respiratory tract. Lesions are most common on the unfeathered parts of the body—the legs, feet, eyelids, base of the beak, and the comb and wattles of gallinaceous birds. The disease in most birds is mild and rarely results in death. However, when infections are on the eyelids or mucous membranes of the oral or respiratory cavities, mortality rates are higher. Avian pox lesions heal following degeneration and sloughing of the abnormally proliferated epithelium.

The observation of visual lesions on wild birds does not represent positive diagnosis of an avian pox infection. At a minimum, histopathologic examination with demonstration of typical intracytoplasmic inclusions is sufficient for a preliminary diagnosis of avian pox. Whenever possible, isolation via the propagation of virus on chorioallantoic membranes of chicken embryos should be used as the definitive diagnosis of choice (Hansen 1987).

Like many other diseases that are density dependent, avian pox transmission is enhanced with increasing vector and host densities. Probably the most common method of transmission is by means of biting insects such as mosquitoes, midges, and flies (Forrester 1991). All biting insects have been shown to be mechanical vectors only, transferring virus from infected to susceptible birds by contamination of their

skin-piercing mouthparts. Transmission can also occur directly by contact between infected and susceptible birds or by contact with contaminated objects, such as bird-feeder perches (e.g. Bleitz 1958). Aerosol transmission, although rare, can occur from viruses being carried along with dust, particularly in confined situations (i.e. aviaries). Avian pox virus is unable to penetrate unbroken skin, but small abrasions are sufficient to permit infection.

In Hawaii, numerous authors have mentioned pox-like lesions on native birds (e.g. Wilson and Evans 1890–1899, Rothschild 1893–1900, Perkins 1903, Munro 1944, Warner 1968, VanderWerf 2001), but few examples exist of positive identification of *P. avium* in Hawaii. The earliest identification of avian pox in native Hawaiian birds is that of Henshaw (1902), who submitted an Akepa (*Loxops coccineus*) to the Bureau of Animal Industry in Washington D.C. The diagnosis was that of "bird pox," an introduced disease that the Bureau had demonstrated during the previous summer in domestic chickens from Honolulu. Locke et al. (1965) identified avian pox from a Red-tailed Tropicbird (*Phaethon rubricauda*) on Midway Island. It was not until the 1980s that the next positive published identification of *P. avium* was made, from a Hawaiian Crow (*Corvus tropicus*) on the island of Hawaii (Jenkins et al. 1989). Tripathy et al. (2000) have recently characterized poxviruses from forest birds in Hawaii. To our knowledge, there have been no other published accounts of positively identified *P. avium* from wild birds on the Hawaiian Islands.

In this article, we document the temporal distributional patterns of avian pox in xeric and mesic forests, along an elevation gradient from sea level to tree line on the island of Hawaii. Specifically, we examine the (1) prevalence of *P. avium* infections in free-ranging avian species, (2) susceptibility of select native and introduced bird species, (3) temporal relationships of avian pox infections and potential vectors, and (4) epizootiology of avian pox on Mauna Loa, Hawaii. We will also present evidence that avian pox is presently a factor that negatively affects distributional and behavioral patterns of native forest bird populations.

STUDY AREAS AND METHODS

To fully document the epizootiology of avian pox in Hawaii, we employed a combination of field and

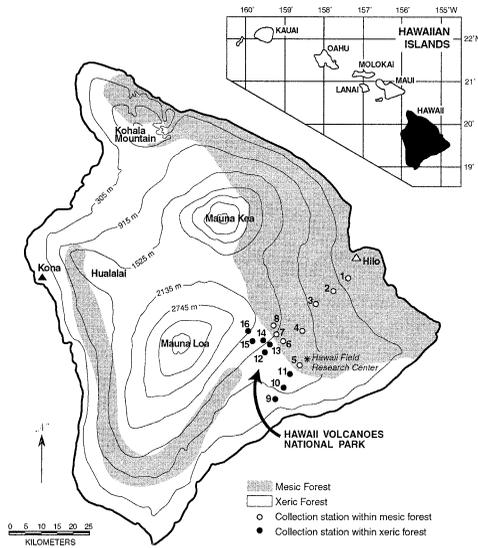


FIG. 1. Location of study areas on Mauna Loa Volcano, Hawaii. Open circles denote sampling sites along an elevational gradient in mesic forest, closed circles are sampling sites over a xeric elevational gradient.

laboratory methodologies. Birds and potential vectors were sampled at 300 m intervals along an elevational gradient from sea level to tree line to document vector abundances and prevalence of avian pox lesions. A subsample of lesions was collected and cultured in the laboratory to prove conclusively that we were documenting infections of *P. avium*. Birds in early stages of avian pox infection were brought into a controlled laboratory situation to document disease progression in native and introduced species. We then used data from each of those methodologies to develop models, using logistic regression, to examine disease potentials in habitats where native birds no longer occur.

Study areas.—We collected birds and vectors from 16 sampling sites, established at 300 m elevation intervals on the southern and eastern slopes of Mauna Loa Volcano, Hawaii (Fig. 1). The southern transect spanned principally xeric habitats, whereas the eastern crossed mesic forest. Detailed descriptions of vegetation communities at our sampling sites on Mauna Loa may be found in Mueller-Dombois et al. (1981).

Field techniques.—Wild birds were mist-netted monthly at 1,200 m elevation over four years (1977–1980) and every other month during 1978 and 1979 at 15 other sampling sites. At each sampling site, 10 mist nets of 6, 11, or 15 m long and of 5.6 mg m⁻¹ (50 denier), two-ply, 36 mm mesh, were erected independently at ground level, but in close proximity to flowering or fruiting trees. We report capture rates

as number of birds caught per 100 net-hours (a 12 × 2.5 m net open for 1 h equals 1 net-hour; see Ralph et al. 1993). Each captured bird was examined for active and inactive avian pox lesions, measured, banded with unique combinations of color bands plus a single metal federal band, and released. Active lesions were defined as areas on the feet, legs, or head that had crusty scabs and were often swollen, whereas areas where digits were missing or that had recently healed marks were classified as inactive sites. One avian pox lesion was classed as a “light” infection, two lesions as “moderate,” and three or more lesions or one lesion on the head as a “heavy” infection. We collected moribund and dead birds at each sampling site throughout the study, with all necropsied and body-washed for ectoparasites following the procedures outlined by van Riper and van Riper (1980). From necropsied birds, 20 samples of what appeared to be avian pox lesions were excised, immediately frozen, and sent to the National Wildlife Health Laboratory in Madison, Wisconsin, for analyses.

Because mosquitoes are known avian-pox vectors, we maintained artificial mosquito oviposition containers at each of our sampling sites. Containers were maintained with a constant supply of water and checked monthly for eggs, larvae, and pupae (see also Goff and van Riper 1980). Counts of adult mosquitoes were taken by monthly trapping at each of the 16 sampling sites using standard New Jersey mosquito light traps, supplemented with data taken from a series of 30 light traps run 24 h day⁻¹ by the Hawaii Department of Health. All hippoboscids and levels of louse and feather mite infestations were recorded from each captured bird. Other potential vectors (e.g. blow flies and house flies) were noted when present at study sites. Nests that had recently fledged young were placed in Berlaise funnels to collect nest ectoparasites.

Laboratory techniques.—Frozen lesions that had been excised from wild-caught birds were used to make avian pox isolates. Larger cutaneous lesions were divided in half for direct histological examination and virus isolation. The portion of tissue used for virus isolation was ground in a tissue grinder with virus transport medium, and centrifuged at 1,500 revolutions per minute (650 g) for 10 min at 4°C to produce supernatant used for egg inoculation. Four, 10 to 15 day old embryonated chicken eggs were each inoculated via dropped chorioallantoic membranes (CAM) with 0.1 mL of sample and incubated at 37°C as described by Senne (1989). Embryos dying after two days were chilled at 4°C and CAMs examined for focal white opaque pock-like lesions or general thickening, when compared to normal chorioallantoic membranes. The CAMs with characteristic avian-pox-like lesions were pooled for each sample and ground for additional egg passages. At the end of the incubation period, inoculated eggs

TABLE 1. Avian Pox prevalences from 3, 122 individuals of the 11 most common bird species captured from 1977–1980 on Mauna Loa Volcano, Hawaii. Numbers in parentheses are comparisons of that number to the total of the respective row. Native species are denoted by an asterisk.

Species	Total number captured	Without avian pox <i>n</i> (% of species)	With avian pox	
			Active lesions <i>n</i> (% of species)	Inactive lesions <i>n</i> (% of species)
Elepaio* (<i>Chasiempis sandwichensis</i>)	77	62 (80.5%)	7 (9.1%)	8 (10.4%)
Omao* (<i>Myadestes obscurus</i>)	74	56 (75.7%)	15 (20.3%)	3 (4.0%)
Iiwi* (<i>Vestiaria coccinea</i>)	107	89 (83.2%)	11 (10.3%)	7 (6.5%)
Hawaii Amakihi* (<i>Hemignathus virens</i>)	626	516 (82.4%)	66 (10.6%)	44 (7.0%)
Apapane* (<i>Himatione sanguinea</i>)	601	391 (65.1%)	85 (14.1%)	125 (20.8%)
Spotted Munia (<i>Lonchura punctulata</i>)	137	137 (100.0%)	0 –	0 –
Red-billed Leiothrix (<i>Leiothrix lutea</i>)	46	46 (100.0%)	0 –	0 –
Northern Cardinal (<i>Cardinalis cardinalis</i>)	51	50 (98.0%)	0 –	1 (2.0%)
House Sparrow (<i>Passer domesticus</i>)	81	75 (92.6%)	2 (2.5%)	4 (4.9%)
House Finch (<i>Carpodacus mexicanus</i>)	79	62 (78.5%)	5 (6.3%)	12 (15.2%)
Japanese White-eye (<i>Zosterops japonicus</i>)	1,243	1,216 (97.8%)	17 (1.4%)	10 (0.8%)
Total	3,122	2,700 (86.5%)	208 (6.7%)	214 (6.8%)

that did not show signs of infection were chilled at 4°C and the CAMs harvested, ground, and blind passaged in additional eggs.

All samples, embryo mortality, and virus passage material were monitored for bacterial contamination by inoculation of a standard nutrient broth and thioglycollate medium that was incubated at 37°C. Any inoculum or harvested egg material showing bacterial growth at 48 h of incubation were filtered through a 0.45 µm filter and reinoculated into eggs.

Suspect avian pox lesions and pieces of egg passage from two infected CAMs were fixed in 10% buffered formalin, paraffin imbedded, sectioned for slide mounting, and stained with hematoxylin-eosin for histological examination. Tissues were considered positive for *P. avium* if intracytoplasmic type-A (Bollinger) inclusion bodies were found in the tissue sections.

Captive bird experiments.—Three Hawaii Amakihi, three Apapane, and four House Finches, each captured at 1,220 m elevation with one small active lesion (<1 mm diameter), were brought into the laboratory to document the course of avian pox infection in native and introduced host species. Equal numbers of control birds with no visible lesions were captured at the same locations and transported in separate cages. All birds were isolated in sterilized individual 12 × 6 × 10" bamboo cages that were covered in mosquito netting, and fed *ad libitum* (sugar water with vitamin supplements, fruit, Gerber's high protein baby cereal with deca-visol vitamins, finch seed, and fruit fly larvae). Body mass of all birds was measured every second day. Following stabilization, here defined as maintenance of captured body mass, all birds were treated with Solutet and Tramasol (Schering-Plough Corp., Kenilworth, New Jersey) to reduce helminth endoparasites and coccidians, respectively.

Statistical analyses.—We examined several models using logistic regression to identify factors associated with the distributional pattern of avian pox. Because we recorded many parameters in this study (e.g. age, gender, weight, multiple bird species, time of year, elevation, forest type, breeding condition, physical measurements, breeding status, etc.) necessitating multiple comparisons, we thus chose to lump active and inactive avian pox infections in order to provide more power in predicting large-scale patterns. We used Akaike's information criterion (AIC) for model selection and likelihood-ratio tests and Wald statistics for hypothesis testing (see Burnham and Anderson 1992). Our use of ecological terms, in a parasitological framework, follow the suggestions of Bush et al. (1997). Statistical analyses were computed with SPSS software (SPSS 1995). Where data were not normally distributed, transformations were used prior to application of statistical tests. Level of statistical significance was accepted when $P \leq 0.05$.

RESULTS

Field results.—From 1977–1980 during 15,000 net-hours, 3,122 birds of the 11 most common species on Mauna Loa were captured and analyzed for avian pox lesions (Table 1). There was a difference between numbers and distribution patterns of native and introduced birds captured at different elevations (Fig. 2). In mesic habitat, more introduced birds occurred in the lowlands, with their numbers generally decreasing with higher elevation (Fig. 2A), whereas native species were generally absent below 1,000 m, after which their numbers in-

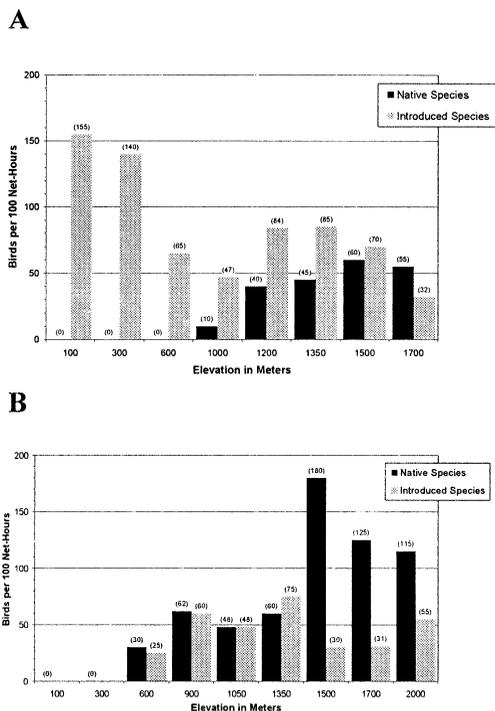


FIG. 2. Total birds captured per 100 net-hours, from 1977–1980 at 16 study locations on Mauna Loa Volcano, Hawaii. (A) Native and introduced birds captured from sea level to tree line in mesic forest on Mauna Loa; (B) depicts native and introduced birds captured along an elevational gradient in xeric forest.

creased with elevation. Within xeric habitats (Fig. 2B), introduced birds were more uniformly distributed throughout all elevational ranges whereas native birds ranged lower, when compared to their distribution in mesic habitat.

Prevalence and intensity of avian pox infections.—Among the 3,122 wild birds captured during this study, 13.5% were found to have avian pox lesions (Table 1). Native birds had a significantly higher prevalence of avian pox than did introduced species ($\chi^2 = 319$, $df = 1$, $P \leq 0.001$). Within native bird populations, Apapane (see Table 1 for scientific names) had the highest prevalence (34.9%), followed by Omao (24.4%), Elepaio (19.5%), Hawaii Amakihi (17.5%), and then Iiwi (16.8%). Within the introduced species, we failed to find lesions on the Red-billed Leiothrix and Spotted Munia. The Northern Cardinal and Japanese White-eye had prevalences of 2%, the House Sparrow 7.4%, and the House Finch 21.5% (Table 1).

TABLE 2. Bimonthly prevalences of avian pox from 3,122 birds of 11 species captured between 1977 and 1980 on Mauna Loa Volcano, Hawaii.

Month	Total number examined (% of total captured)	Without avian pox	With avian pox (% with lesions)
January–February	488 (15.6%)	405	83 (19.6%)
March–April	460 (14.7%)	395	65 (14.2%)
May–June	548 (17.5%)	497	51 (12.1%)
July–August	460 (14.7%)	399	61 (14.4%)
September–October	675 (21.6%)	593	82 (19.4%)
November–December	491 (15.7%)	411	80 (18.9%)
Total	3,122 (100%)	2,700	208 (100%)

Prevalence of avian pox varied significantly over the annual cycle ($\chi^2 = 77.3$ $df = 5$, $P \leq 0.001$; Table 2). The greatest percentage of infected individuals occurred September–February, the lowest in May–June (Table 2). That pattern resulted almost entirely from native bird infections, because there was no annual pattern for introduced birds.

Avian populations from mesic and xeric forests of Mauna Loa had significantly different levels of avian pox infections ($\chi^2 = 11.80$, $df = 1$, $P \leq 0.001$; Fig. 3). Mesic forest consistently supported higher prevalence levels, which may have been related to an increased transmission potential within that habitat. Elevation also had an extremely important influence upon population infection rates on Mauna Loa ($\chi^2 =$

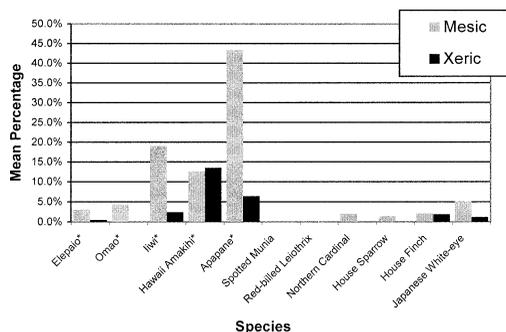


FIG. 3. Prevalence of avian pox infections from 3,122 individuals of 11 bird species collected from 1977–1980 on Mauna Loa Volcano, Hawaii. Avian pox prevalences of each species from mesic forests are lighter shaded bars, whereas prevalences from xeric forests are darker shaded bars. Native species are denoted with an asterisk.

TABLE 3. Avian pox prevalences of 3,122 native and introduced birds captured along an elevational gradient from 1977–1980 on Mauna Loa Volcano, Hawaii. Numbers in parentheses are prevalences of introduced and native birds at that elevation.

Elevation	Introduced birds		Native birds	
	With- out le- sions	With lesions (% with lesions)	With- out le- sions	With lesions (% with lesions)
0–500 m*	530	18 (3.4%)	17	4 (23.5%)
500–1,000 m*	182	2 (1.0%)	92	30 (32.6%)
1,000–1,200 m*	376	11 (2.9%)	284	159 (56.0%)
1,200–1,350 m*	229	3 (1.3%)	90	37 (41.1%)
1,350–1,500 m*	147	12 (8.2%)	277	88 (31.8%)
1,500–1,650 m*	85	2 (2.4%)	214	44 (20.6%)
1,650–2,000 m	38	3 (7.9%)	139	9 (6.5%)

*P ≤ 0.01.

106.12, df = 6, P ≤ 0.001; Table 3), with greatest prevalence at 1,000–1,200 m elevation.

We found no significant difference in infection rates between breeding and nonbreeding birds ($\chi^2 = 0.137$, df = 1, P = 0.711). However, active infections of first-year birds (n = 31) were up to 3× greater in older birds, particularly native species. A recaptured bird was not more likely to have avian pox than was a first capture ($\chi^2 = 2.56$, df = 1, P = 0.11). Birds accidentally killed in mist nets ($\chi^2 = 1.55$, df = 1, P = 0.21, n = 94) or killed by automobiles (n = 31) were not more likely to have lesions than were normally processed birds. Low, medium, and high fat levels in a bird had no relationship to *P. avium* infections ($\chi^2 = 4.34$, df = 3, P = 0.23), nor did light, medium, or heavy molts (χ^2

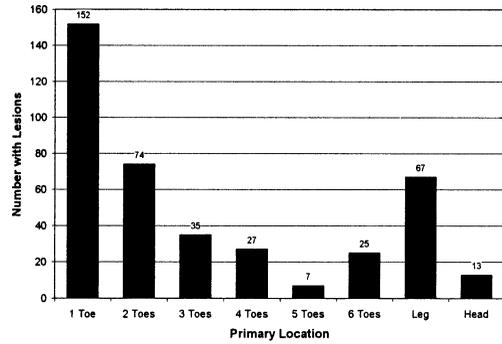


FIG. 4. Location of avian pox infections from 3,122 birds of 11 species captured from 1977–1980 on Mauna Loa Volcano, Hawaii. Numbers of avian pox lesions on infected birds were recorded as being on from 1–6 toes, on the leg, or on the head (particularly around the eyes and mouth area). Numbers in parentheses at the top of each bar are total birds recorded with lesions in that location.

= 1.75, df = 3, P = 0.63). However, birds that were found moribund (n = 10) had a significantly higher prevalence of avian pox than did birds captured in mist-nets ($\chi^2 = 15.58$, df = 1, P ≤ 0.001).

When classified as heavy, moderate, or light, intensities of avian pox infections were significantly different among species ($\chi^2 = 464.7$, df = 30, P ≤ 0.001). Most species had a preponderance of light infections (n = 214), whereas 112 birds had moderate and 96 had heavy infections (Table 4). As was the case with prevalence, native birds were more greatly affected, having 94 of the 96 heavy infections. The majority of lesions occurred on toes (Fig. 4). We rarely encountered avian pox lesions on the

TABLE 4. Avian pox incidence from 3,122 birds, of the 11 most common species, from Mauna Loa Volcano, Hawaii. Information was taken between 1977 and 1980, with infections categorized as 1 lesion being "Light", 2 lesions being "Moderate", and 3+ lesions or 1 on the head as "Heavy". Native species are denoted by an asterisk.

Species	None	Light	Moderate	Heavy
Elepaio*	62	5	6	4
Omao*	56	5	6	7
Iiwi*	89	10	2	6
Hawaii Amakihi*	516	62	24	24
Apapane*	391	95	62	53
Spotted Munia	137	0	0	0
Red-billed Leiothrix	46	0	0	0
Northern Cardinal	50	1	0	0
House Sparrow	75	2	3	1
House Finch	62	12	4	1
Japanese White-eye	1,216	22	5	0

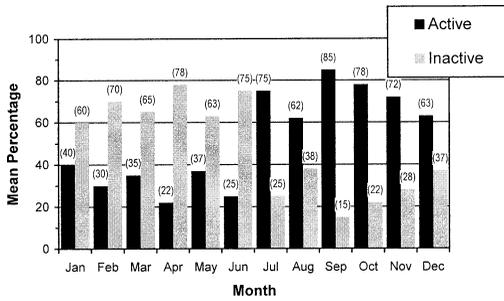


FIG. 5. A monthly comparison of active versus inactive (healed) avian pox lesions encountered on 422 birds captured from 1977–1980 on Mauna Loa Volcano, Hawaii. Each month totals 100%, with dark shaded histogram bars representing active and light shaded bars inactive avian pox lesions. Sample sizes of total captured birds for that month are given in parentheses at the top of each bar.

head ($n = 13$), and those were principally on native birds (Hawaii Amakihi = 7; Apapane = 4). We never encountered the diphtheritic form of avian pox (where moist, necrotic lesions develop on the mucous membranes of the mouth and upper respiratory tract) in the field, but that form did develop in our experimental birds.

Of the 422 individuals recorded with lesions, 208 (49.3%) were classified as having active lesions. The Omao, Hawaii Amakihi, and Iiwi had higher prevalences of active lesions, whereas the House Finch, Elepaio, and Apapane had higher prevalences of inactive (i.e. healed) lesions (Table 1). When comparing between prevalence of active versus inactive lesions, there was no significant difference between gender, wet versus dry habitat, or among elevations. Although a greater percentage of native than introduced birds were infected, there was no significant difference between active versus inactive lesions within each group. There was a difference in active versus inactive lesions among months ($\chi^2 = 77.33$, $df = 5$, $P \leq 0.001$), with more birds having active lesions from July to December (Fig. 5).

Potential vector distributions.—Two mosquito species (*Aedes albopictus* and *Culex quinquefasciatus*) that could potentially act as mechanical transmission vectors for avian pox, were collected regularly in our artificial oviposition sites throughout this study (Fig. 6). Larvae and pupae of *C. quinquefasciatus* were present from sea level to 1,350 m elevation on Mauna Loa

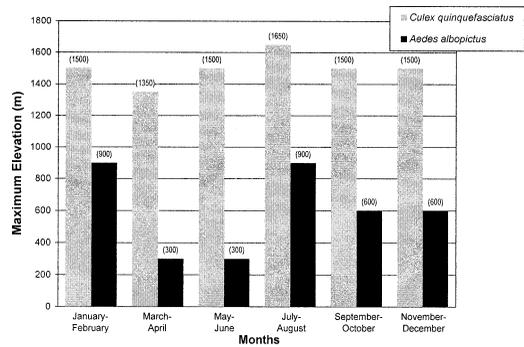


FIG. 6. Maximum elevation of mosquito larvae collected at 16 study sites from 1977–1979 on Mauna Loa Volcano, Hawaii. Numbers in parentheses represent maximum elevation for each type of larvae.

throughout each month of the year, and during July–August mosquitoes were found breeding up to 1,650 m elevation, the highest reaches of extant mesic forest. *Aedes albopictus* was recorded only up to 900 m elevation. In addition to data from artificial oviposition sites, we documented “natural” oviposition sites as pools of water on nonporous lava and felled trees, tree holes, ground pools, tree fern stumps, pig wallows, rain barrels, and cattle watering troughs. Goff and van Riper (1980) documented breeding of *C. quinquefasciatus* throughout the entire year at elevations of 1,350 m in xeric habitat and 1,500 m in mesic habitat on Mauna Loa, whereas adult mosquitoes showed increasing adult populations from January until July–August, after which numbers fell precipitously.

We found feather mites (Acaridia) and feather lice (Mallophaga) on all species of birds and at every site, but hippoboscids were rarely encountered on native birds (van Riper and van Riper 1985). Gamasida mites were collected in very low numbers from the nests of the Hawaii Amakihi and Apapane at 1,200 m elevation. Actiniedida mites were found in body washes of Iiwi and Hawaii Amakihi, from 1,650 to 1,200 m elevation, respectively (Goff 1980).

Laboratory P. avium isolations.—Twenty of 22 wart-like lesions from the legs of Hawaiian forest birds were confirmed as *P. avium* infected by histological examination of the original bird tissue, infected CAMs, or both. Two samples were considered negative after three blind passages in eggs that did not produce characteristic avian pox lesions on the CAM. Therefore, >90% of the submitted samples

from the field, that were initially thought to be caused by *P. avium*, were positively identified as that pathogen.

Course of avian pox infections in the laboratory.—All Apapane, House Finch, and Hawaii Amakihi brought into the laboratory with early stages of avian pox developed advanced symptoms of infection. None of the control birds became infected with *P. avium*. There was a significant decrease between capture and end weights in Hawaii Amakihi (t -test = 9.7, $P \leq 0.01$), whereas House Finches did not lose weight over the experimental period (t -test = 0.76, $P = 0.53$). All House Finches survived. Two of the three Apapane died of complications not associated with avian pox, 10 and 13 days after being brought into captivity, so weight changes could not be tested. All three of the experimental Hawaii Amakihi succumbed to *P. avium* infections.

Modeling results.—In modeling avian pox over an elevational gradient using likelihood-ratio tests and Wald statistics, we found that the addition of a species \times elevation interaction term was significant, when elevation was considered a continuous variable (likelihood-ratio test, $\chi^2 = 9.86$, $df = 4$, $P = 0.04$) and when considered a categorical variable (likelihood-ratio test, $\chi^2 = 22.8$, $df = 12$, $P = 0.03$). Akaike's information criterion (AIC) indicated that the model of species and continuous elevation main effects and their interaction was the most parsimonious (AIC = 1175.4).

The pattern of avian pox prevalence over elevation differs substantially between the Apapane and Hawaii Amakihi. Because of variable capture rates and sites (particularly lower elevations) where species were absent, we were interested to know if this difference below 1,200 m elevation was significant. Therefore, we examined an indicator variable "highlow" (denoting above and below 1,200 m) within the model, along with elevation as a continuous variable. A full model up to the three-way interaction of species, elevation, and highlow was a better model (AIC = 1121.2; and analysis of deviance) than the full interaction model of just species and categorical elevation. Consequently, there appears to be evidence that the different observed pattern between the two species is not an artifact of absences and uneven sample sizes. The Apapane had a much higher prevalence of lesions below 1,200 m than did

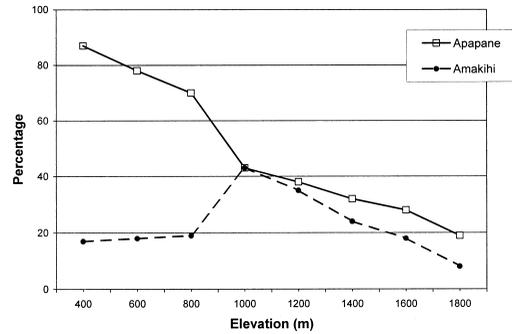


FIG. 7. A model of predicted avian pox prevalences in native Apapane and Hawaii Amakihi. Data are extrapolated from prevalences of 1,227 birds captured from 1977–1980 on Mauna Loa Volcano, Hawaii. Present-day populations of these birds are, in the most part, presently confined above 1,000 m elevation.

the Hawaii Amakihi, only slightly higher after 1,200 m, and decreased monotonically whereas Hawaii Amakihi lesions peaked at 1,200 m elevation (Fig. 7). That could mean that either Apapane from higher elevations contribute to greater prevalence below 1,200 m elevation, or that Hawaii Amakihi populations at lower elevations are more resistant to *P. avium*.

Because so many introduced species did not exhibit avian pox infections, only the Japanese White-eye and House Finch could be contrasted for differences in prevalence along the forest elevational gradient. Although the percentage of individuals infected with avian pox differed between the species, for three models with low AIC statistics—species only (AIC = 322.3), species and elevation (AIC = 322.4), and the species by elevation interaction (AIC = 323.1)—the likelihood-ratio test and analysis of deviance between the models were not significant ($P > 0.1$). Consequently, there was no evidence that the prevalence of *P. avium* differed by elevation in the introduced, as it did with native Hawaiian bird species.

DISCUSSION

HOW SUSCEPTIBLE TO AVIAN POX ARE NATIVE LAND BIRDS COMPARED TO THEIR INTRODUCED COUNTERPARTS?

Native Hawaiian birds are more likely to be infected with avian pox than are their introduced counterparts. For example, 34.9% of all

native Apapane; 24.3% Omao; and almost 20% of the Hawaii Amakihi, Elepaio, and Iiwi that we captured had *P. avium* infections. In comparison, within the introduced birds we failed to find any lesions on Red-billed Leiothrix and Spotted Munia, and only 2% of the Japanese White-eye and Northern Cardinal were infected. House Finches were the only introduced species with a high prevalence (21.5%), and that bird appears to also be highly susceptible to avian pox on the North American continent (Power and Human 1976). In continental regions, the most commonly reported (modal) avian pox prevalence of lesions on wild birds varies between 0.5 and 1.3%. However, in some populations from 25% (e.g. House Finch in California; McClure 1989) to 50% (Davidson et al. 1980) of the birds can support active lesions.

The ability to survive an avian pox infection should be reflected in the number of birds throughout the population with healed lesions. Almost 60% of native Apapane had healed lesions, suggesting a somewhat enhanced immunogenetic capability to cope with this virus strain. A number of our Apapane recaptures that formerly were recorded active had healed lesions at subsequent recaptures. One Apapane also survived our laboratory experiment. This pattern of resistance was also present in the introduced House Finch: 70.6% of infected birds had healed lesions and all four test-individuals in our laboratory experiment survived.

On the other hand, we argue that a higher level of active compared to healed lesions suggests less resistance to avian pox. Even though the Iiwi is presently restricted only to higher elevations (Scott et al. 1986), we still found 61.1% of infected birds with active lesions versus 38.9% with healed lesions. A second native species that exhibited high susceptibility to avian pox was the Omao, with 83.3% of infected individuals with active versus only 16.7% with healed lesions. Thus, our data suggest less resistance to *P. avium* in these two native species.

WHAT ARE PRESENT-DAY DISTRIBUTIONAL AND TEMPORAL DIFFERENCES IN PREVALENCE OF AVIAN POX ALONG MOISTURE AND ELEVATION GRADIENTS?

Temporal differences.—Native birds had increases of active avian pox lesions in their populations during July–September (see Fig. 5), in-

dicating during that time of the year birds were most likely to be exposed to new infections. Ectoparasites, present on birds at all elevations and at similar levels throughout the annual cycle, are probably not a major cause of differences of avian pox prevalence over elevation and time. In that the breeding season of native birds on Mauna Loa is October–March (Baldwin 1953, Berger 1981), recently fledged birds are not temporally associated with the July–September increase in avian pox. It seems rather that the abundance and activities of *C. quinquefasciatus* are most closely correlated with the prevalence of avian pox during different periods of the annual cycle.

One reason for differences in temporal patterns of avian pox on Mauna Loa is undoubtedly related to transmission potentials. During warmer months of the year, Goff and van Riper (1980) found that *C. quinquefasciatus* expanded its breeding range to the uppermost reaches of mesic forest (1,650 m). If in fact *C. quinquefasciatus* is the principal vector of avian pox, transmissions are undoubtedly enhanced by this mosquito being ornithophilic (Bohart and Washino 1978) and having a daily activity cycle that is asynchronous with that of native bird activity (van Riper et al. 1986). Thus, mosquito feeding and avian pox transmission is enhanced when birds are sedentary on night roosts. There is also the possibility that the strong trade winds in Hawaii may be responsible for increasing the spread of mosquitoes up elevational gradients. Swezey and Williams (1932) and Komatsu (1966) reported egg rafts above 1,800 m elevation on Hawaii. It thus appears that mosquitoes can be aerially transported, from higher density cells at lower elevations to upper forest areas on Hawaii, and that certainly accentuates temporal differences in avian pox prevalence.

Moisture gradients.—During the moister winter months, *C. quinquefasciatus* breeds at higher densities in forests (Goff and van Riper 1980), and potential transmission by this mosquito is undoubtedly enhanced, particularly in the continuous forest areas (e.g. Stainbeck highway and Kulani forest). Concomitantly, the highest monthly prevalences of avian pox that we found in avian populations (see Table 2) occurred from September to February, the wettest months of the year in Hawaii.

The mesic forests of Mauna Loa occupy a much larger area, have a greater foliage-height diversity, a higher insect biomass, and standing crop of nectar-producing flora than does the xeric forest (Mueller-Dombois et al. 1981). One would expect a greater density of native birds in mesic habitats. However, a comparison of our capture rates during the same time periods at mesic and xeric sampling sites of similar elevations resulted in a greater number of native birds captured per unit effort in xeric habitat (see Fig. 2). Scott et al. (1986) also documented higher densities of native birds in xeric habitat on Mauna Loa. The higher prevalence of avian pox in mesic areas appears to be a factor contributing to lower native bird numbers, when compared to high-elevation xeric habitats on Hawaii.

Introduced birds, except for the House Finch (primarily a dry-forest species), were consistently found in higher numbers in mesic forests (see Fig. 2). For example, the Japanese White-eye, which is ubiquitous throughout the islands (van Riper 2000), was captured more often in mesic sites (9:1 ratio). It appears that introduced bird species are occupying those localities where native species occur in lower numbers, and the refractory ability of many introduced species to the avian pox certainly benefits them in those areas.

Elevational gradients.—We found greatest prevalences of avian pox in the mid-elevation reaches, that zone of maximum native bird and vector overlap (see Table 3, Fig. 8). However, the present-day elevational distribution of this disease on Mauna Loa is not static nor a direct reflection of vector densities. Mosquitoes are numerous at lower elevations, yet the prevalence of avian pox in bird populations from lowland forests is quite low. It is not until the mid-elevation ranges are reached that prevalence appreciably changes. These are also the lowest elevations at which we captured native birds in any numbers (see also data from Scott et al. 1986). In that region of overlap, where mosquitoes and susceptible native birds occur, avian pox prevalence increases disproportionately (Fig. 8).

A number of native birds rely upon nectar as a food source and follow the elevational flowering sequence of nectar-bearing trees (Perkins 1903, Munro 1944, Baldwin 1953). Lamoureaux et al. (1981) and van Riper (1980) demonstrated

that Hawaiian nectar-producing trees bloom along an elevational gradient, with greatest flowering at lower elevations during the summer and fall, gradually progressing upslope, with trees at highest elevation flowering later. This means that to obtain maximum quantities of nectar, native birds must move to lower elevations during the fall. It was during fall that we found the greatest number of Iiwi with avian pox (see Table 2), and that undoubtedly contributed to the high prevalence in the latter part of the September–December peak. The birds are, therefore, drawn into the lower disease zones during their fall journeys in search of nectar, and are met at that time by an expanding vector population. As a result, the altitudinal band of mosquito–bird overlap is greatly increased and the potential for avian pox transmission is much higher during that time-period, than it otherwise would be. Thus, interacting environmental factors and behavioral activities presently influence temporal differences and maximize spread of avian pox to native birds in Hawaii.

WHAT ROLE DID SELECTIVE FORCES FROM AVIAN POX PLAY IN THE OBSERVED DECLINE AND PRESENT-DAY DISTRIBUTION OF ENDEMIC HAWAIIAN AVIFAUNA?

Avian pox was undoubtedly introduced into Hawaii during the 1800s with the importation of domestic avian stock (Alicata 1964). If *C. quinquefasciatus* acts as a vector of avian pox in Hawaii, as it does in other locations, the potential for spread of this virus would have been greatly enhanced with its introduction in 1827 to the islands (Hardy 1960). We argue that a large enough reservoir in domestic avian stock was present at the time of *C. quinquefasciatus* introduction for avian pox to have spread to native birds and contribute to the numerous extinctions of the late 1800s. Birds that succumbed during that period (e.g. *Moho* spp., *Chaetoptila angustipluma*) were principally species found in the mid- to low elevational forests (Scott et al. 2001b, Berger 1981). With the annual altitudinal migrations and flocking behavior of many native species, coupled with the presence of a suitable vector, avian pox could have spread rapidly throughout the forests. It was during that period that early collectors noted pox-like lesions on many native

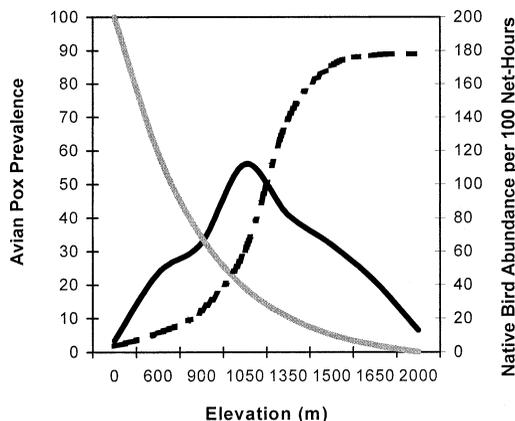


FIG. 8. A comparison of avian pox prevalence with relative mosquito and native bird abundances, from sea level to tree line on Mauna Loa Volcano, Hawaii. Values for the solid black line were derived from avian pox prevalences of 3,122 wild forest birds captured from 1977–1980, along an elevation gradient on Mauna Loa Volcano, Hawaii. Gray line indicates relative mosquito abundance, and broken line denotes native bird abundance.

birds (Wilson and Evans 1890–1899, Rothschild 1893–1900, Perkins 1903, Munro 1944).

The pattern of historical native Hawaiian bird decline is bimodal (Ralph and van Riper 1985, van Riper and Scott 2001). The initial historical reduction of native birds occurred in the mid- and late 1800s and introduced avian pox probably played a significant role. That supports the hypothesis of an earlier avian pox and later malarial parasite (see van Riper et al. 1986) introduction to the archipelago.

It appears that the native Hawaiian avifauna is still in a declining mode because of the effect of introduced diseases. Baldwin (1941, 1953), the first ornithologist to conduct systematic censuses of Hawaiian avifauna, found Hawaii Creeper (*Oreomystis mana*), Akepa, Iiwi, Apapane, Hawaii Amakihi, and Elepaio from 600 to 900 m elevation in Hawaii Volcanoes National Park. Conant (1981) revisited Baldwin's study areas and found that the Creeper, Akepa, and Iiwi had disappeared. Furthermore, the native bird species that presently remain between 600 and 900 m elevation have undergone sizeable reductions in population numbers (Scott et al. 1986). That pattern of declining native bird populations and local extinctions, focused at middle elevation forests, appears to be consistent with the hypothesis that avian diseases are

presently limiting native bird populations in Hawaii.

Avian pox has had and continues to have a negative effect upon the native Hawaiian avifauna. The extinction of many native bird species during the second and third decades of the 1800s, and subsequent range reductions of other species, is correlated temporally with the introduction of domestic poultry and arrival of avian pox. However, we should not ignore other factors that probably negatively influenced the Hawaiian avifauna. We are only now appreciating the effect that Polynesians had upon the native birds prior to European settlement of the Hawaiian Islands (Olson and James 1982a, b). The introduction of *Rattus exelans* following the Polynesian arrival no doubt was also a factor that contributed to a reduction of the native avifauna (Atkinson 1977). Major habitat modifications by humans and by exotic ungulates certainly played a part in reducing bird population levels, as have the introduced pig (*Sus scrofa*), mongoose (*Herpestes auropunctatus*), and feral cat (*Felis catus*) (Tomich 1969, van Riper and van Riper 1982). However, we argue that disease was a major culprit in the initial decline of native birds, with avian pox playing a pivotal role in early historical declines. We are, therefore, left with a complex picture for the demise of so many native Hawaiian birds. Avian pox appears to be only one factor that has contributed to native avian population declines, but because this introduced disease continues to influence present-day numbers and distributional patterns, it should be recognized as a factor in all future conservation planning efforts in Hawaii.

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