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THE EPIZOOTIOLOGY AND ECOLOGICAL SIGNIFICANCE OF MALARIA IN HAWAIIAN LAND BIRDS¹

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Abstract. Laboratory and field experiments were conducted on the island of Hawaii from 1977–1980 in an effort to determine the impact of avian malaria on the forest birds. At 16 study sites from sea level to tree line in mesic and xeric habitat, birds were captured and bled to determine the host and altitudinal distribution of blood parasites. In the laboratory, six bird species were challenged with malarial parasites to measure host susceptibility. Distributions, activity cycles, and transmission potentials of malarial parasite vectors were also analyzed.

One species of *Plasmodium* was present from sea level to tree line, concentrated in the mid-elevational ranges in the ecotonal area where vectors and native birds had the greatest overlap. Native forest birds were: (a) more susceptible to malaria than were introduced species; (b) most likely to have malaria during the nonbreeding, wet season; (c) found ranging lower in xeric than in mesic forests; and (d) found to have a lower prevalence of malaria in xeric forests. Temporal as well as elevational differences in prevalence and parasitemia levels of wild birds were apparent throughout the annual cycle, a result of differing host and parasite responses to biotic and abiotic factors.

Avian malaria probably did not reach epizootic proportions on Hawaii until after ≈ 1920 . However, since that time it has had a negative impact on the population dynamics of the native forest birds and is today a major limiting factor, restricting both abundance and distribution of these species on the island. In response, a number of native bird species have developed immunogenetic and behavioral responses that reduce the impact of the parasite on host populations.

Key words: avian malaria; disease; extinction; Hawaiian Islands; host-parasite relationships; island birds; *Plasmodium*; population regulation.

INTRODUCTION

A larger portion of the endemic bird species of the Hawaiian Islands have become extinct in historical times than in any other comparable region of the world (Greenway 1958). Hypotheses to explain why so many species succumbed in the short time following discovery of the Islands by Captain Cook in 1778 include: habitat destruction by humans and introduced ungulates; indiscriminate killing of birds; competition with introduced birds; introduced predators; and introduced diseases.

Much concern has been expressed over a possible link between introduced diseases and the depletion of the native Hawaiian birds (Scott et al. 1982), but few data are available to prove or disprove the relationship. Warner's (1968) study of the presumed role that introduced malaria and Avian Pox played in the extinc-

tion of the native Hawaiian birds is the only major published work dealing with this subject. He proposed that an "imaginary line" existed at ≈ 600 m, above which there were no mosquitoes and below which native birds were not found, presumably because they had succumbed to introduced diseases. Warner's theory has found wide acceptance as an example of how disease can limit a host population, this despite the fact that some observations appear not to demonstrate the validity of his model (Berger 1975, 1981).

In this paper we attempt to clarify the impact that an introduced malarial parasite has had on the native Hawaiian avifauna. We examine: (1) the susceptibility of extant bird species to the malarial parasite; (2) prevalence and intensity of *Plasmodium* infections in birds; (3) distribution of *Plasmodium*, its vectors, and its hosts; and (4) the epizootiology of avian malaria in the Hawaiian Islands. We also present evidence that the avian disease problem in Hawaii is more complex than originally proposed by Warner (1968), and we will show that malaria probably did not have a major impact upon the numbers of Hawaiian birds in the late 19th

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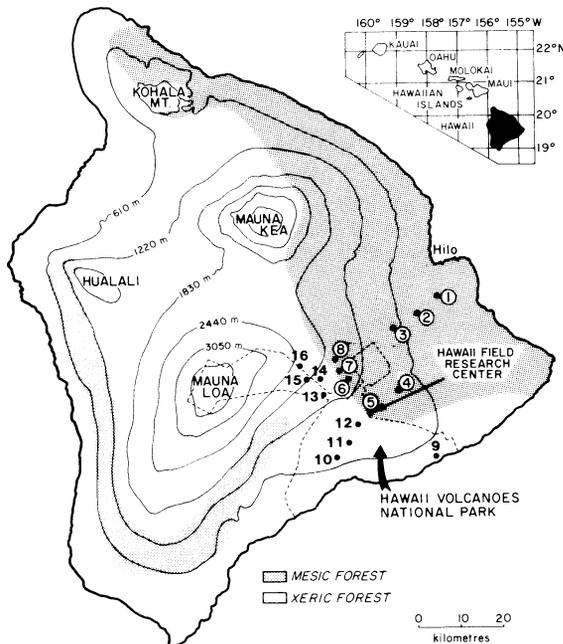


FIG. 1. Location of study area and sampling stations on Mauna Loa Volcano, Hawaii.

century, but is presently limiting some native bird populations as well as affecting their distributional and behavioral patterns.

METHODS

Study areas consisted of 16 sampling stations, established at intervals of 300 m in elevation on the southern and eastern slopes of Mauna Loa Volcano, Hawaii (Fig. 1). The southern stations spanned principally xeric forest habitat, while the eastern stations crossed mesic forest. Detailed descriptions of these vegetation types may be found in Mueller-Dombois and Fosberg (1974).

Field techniques

During 1978 and 1979 wild birds were mist-netted every two months at each station, except station 5 (at 1200 m elevation), where they were netted monthly over a 3-yr period (1978–1980). At each station, 10 mist-nets on long bamboo poles were erected independently, in close proximity to a flowering or fruiting tree. Depending on the size of the clearing in the forest, 6-, 11-, or 15-m nets of 5.6 mg/m (50 denier), 2-ply, 36-mm mesh were used. Each bird was bled by clipping a toenail. A thin blood smear was taken, and the slide fixed for 30 s in absolute methyl alcohol. Birds were measured, then banded with unique combinations of color bands plus a single United States Fish and Wildlife Service metal band. We collected moribund and dead birds at each station throughout the study. Each bird was necropsied following the procedures outlined by van Riper and van Riper (1980) and impression

smears were made from blood-associated internal organs.

Ten each of Apapane (*Himatione sanguinea*), Iiwi (*Vestiaria coccinea*), Japanese White-eye (*Zosterops japonicus*), Red-billed Leiothrix (*Leiothrix lutea*), and Common Amakihi (*Hemignathus virens*) were collected from high-altitude forest habitats on Mauna Loa, where avian malaria is virtually absent, and transported in mosquito-proof cages to the Avian Disease Laboratory at the Hawaii Field Research Center. In addition, 15 Common Amakihi were taken from the xeric forest of Mauna Kea where malaria is absent (van Riper 1975). This Common Amakihi population was sampled to discern if a differential resistance to the malarial parasite has developed in host subpopulations as a result of exposure histories. We hypothesized that, because the Mauna Loa population is continuous from tree line to sea level, while the Mauna Kea population is restricted to a high, dry forest area, the former would have developed immunogenetic responses to the malarial parasite because of gene flow from low elevations. Forty Laysan Finches (*Telespiza cantans*) were captured on mosquito- and malaria-free Laysan Island and transported by boat in mosquito-proof cages to the island of Hawaii.

At each sampling station artificial mosquito oviposition sites were maintained with a constant supply of water. All sites were checked for eggs, larvae, and pupae each month during 1977 through 1979. At each station potential natural oviposition sites were identified and examined regularly for larval activity. Counts of adult mosquitoes from a series of 30 light traps run 24 h/d by the Hawaii Department of Health (see Goff and van Riper 1980 for locations), were supplemented by monthly trapping at each of the 16 study stations using Standard New Jersey Mosquito Light Traps. Biting collections on humans were taken over a 24-h period on a monthly basis for both *Culex quinquefasciatus* and *Aedes albopictus* (Goff and van Riper 1980).

Laboratory techniques

Cultures of malarial organisms were established from wild-caught, moribund, introduced and endemic Hawaiian forest birds. The cultures were maintained through subinoculations of infected blood into canaries, a standard method employed in *Plasmodium* research (Garnham 1966). We observed no differences in morphology or pathogenicity between these cultures.

After capture, all experimental birds were allowed a closely monitored acclimation period of no less than 2 mo, during which time they were fed food ad libitum. During this acclimation period body masses were taken every 2nd d, and blood smears taken on a regular weekly basis and searched for the presence of haematozoan infections. To ensure that no birds had infections that were not detected by microscopy, blood was extracted from select individuals and subinoculated back to canaries, a technique that has been shown to reveal latent

infections (Herman et al. 1966). After stabilization of body mass, all birds were treated with Solutmet and Tramasol to clear them of helminth endoparasites and coccidians, in an effort to minimize the contributions of these maladies.

Following the parasite-clearing process, five captive Apapane, Iiwi, Red-billed Leiothrix, Japanese White-eye, Laysan Finches, and Mauna Loa Common Amakihi, and six Mauna Kea Common Amakihi, were inoculated intramuscularly with 0.1 mL of first-passage infected canary blood. Each species group was challenged within a 2-d period with blood of similar parasitemia levels from the same canary host; ≈ 16 schizonts were subinoculated into each bird. After day 4 each bird was bled daily until the host succumbed or parasitemia levels fell to a chronic state. Throughout the entire challenge period body mass and parasitemia levels were monitored daily. All birds that died during the experiment were necropsied in an effort to determine the cause of death. Blood impression smears were made from the liver, lung, spleen, kidney and brain, all endoparasites noted, and swabs taken for bacterial analysis. Five other individuals of each group constituted controls, which were maintained in mosquito-proof cages and monitored throughout the experiment. In addition, diel periodicity was examined during peak levels of parasitemia in the Canary, Apapane, and Common Amakihi, of which the former two were bled every 4 h over two 24-h periods.

Blood slides were treated for 30 min with Giemsa stain in a solution buffered with Na_2PO_4 and KH_2PO_4 to a pH of 7.17. Each slide was read under oil immersion at $1000\times$ for 10 min, or until a minimum of 25 000 red blood cells (RBC) had been examined. All parasites were classed as either gametocytes, schizonts, or trophozoites. In the case of heavy infections, 100 parasites were counted and categorized. The average number of RBC per field was recorded for each slide, as was the mean number of fields read per minute by the observer. The number of cells examined on each slide (C_e) was determined using the following formula:

$$C_e = (B_f)(F_m)(M_r)$$

where B_f = number of RBC per field; F_m = fields per minute read; and M_r = minutes slide was read. The formula used to determine the number of parasites per 10 000 RBC was:

$$\text{Parasites}/10\,000\text{ RBC} = (P_o) \times \frac{10\,000}{C_e}$$

where P_o = number of parasites observed. To take into account the refractory ability of a bird species against the malarial parasite, and the ability of that host to survive once infected, the following formula was developed for an Index of Adaptation (I_a):

$$I_a = \frac{(\alpha + \beta)}{2n}$$

where α = number of individuals surviving the challenge; β = number of refractory individuals, and n = sample size of the challenge group. In this manner, species lacking any immunogenetic capacity would have an $I_a = 0$. The closer I_a approached 1, the greater the ability of the host group to survive the disease.

Mosquito colonies of *C. quinquefasciatus* and *A. albopictus* were established in the laboratory from larvae collected in the wild. Newly emerged adults were removed to biting cages and maintained on a sugar and water combination. From 4 to 6 d after emergence, females were offered blood meals from infected birds, and following engorgement were removed to separate cages. At 12–24 h after engorgement, diluted smears of mosquito stomach blood were fixed in methyl alcohol, stained with Giemsa and examined for ookinetes. Stomachs were examined for oocysts via wet mounts, and salivary glands were stained with Giemsa to show any sporozoites, following the techniques outlined in Garnham (1966). Females previously engorged on infected birds were placed in biting cages with non-infected birds to complete laboratory transmission.

Daily activity cycles of *C. quinquefasciatus* and *A. albopictus* were determined in the laboratory. Three recently emerged mated females of each species were placed in sealed glass jars containing water plus sugar and allowed to acclimate for 3 d. The jars were maintained at ambient temperature with a humidity of 100%, and sealed to eliminate problems with air movement and CO_2 attraction. During the first 10 min of each hour over a 24 h period, the total number of seconds any one of the three experimental mosquitoes were flying was recorded. Four replications were made on each species.

All statistical analyses were computed on a Burroughs 6700 computer using SPSS programs (Nie et al. 1975). Where data were not normally distributed, logarithm transformations were used before application of statistical tests. The level of statistical significance was accepted when $P \leq .05$.

RESULTS

Field results

During 14 027 net-hours from 1978 to 1979, individuals of 18 wild bird species were captured and bled. In this paper we deal with 2 365 blood samples taken from the 11 most common species on Mauna Loa (the native Apapane, Common Amakihi, Iiwi, Elepaio [*Chasiempis sandwichensis*], Hawaiian Thrush [*Phaethornis obscurus*], and the introduced House Finch [*Carpodacus mexicanus*], House Sparrow [*Passer domesticus*], Nutmeg Mannikin [*Lonchura punctulata*], American Cardinal [*Cardinalis cardinalis*], Japanese White-eye, and Red-billed Leiothrix). None of the remaining seven species (see Table 1) were captured with enough frequency to warrant their inclusion in the blood data analysis.

TABLE 1. Bird capture rate from 1978 to 1979 in mist nets on Mauna Loa, Hawaii.

Bird species	Percent of total	Capture rate (birds/100 net-hours)					
		Jan–Feb	Mar–Apr	May–Jun	Jul–Aug	Sep–Oct	Nov–Dec
Apapane*	11.4	32.6	23.1	60.1	40.5	32.9	14.2
Common Amakihi*	23.3	107.2	65.7	78.5	105.1	107.9	85.8
Japanese White-eye	43.6	85.6	175.3	188.5	70.6	155.9	120.8
Elepaio*	2.6	6.3	5.9	2.9	12.3	16.2	6.7
Omao*	2.0	3.1	13.1	5.2	2.7	3.7	7.7
Iiwi*	3.9	16.9	9.0	10.1	20.7	14.7	6.0
House Finch	2.9	3.2	12.5	17.3	11.5	12.2	2.1
House Sparrow	2.2	5.4	9.5	6.7	0.8	2.6	9.8
American Cardinal	1.7	5.3	7.7	4.9	1.2	3.8	5.7
Red-billed Leiothrix	1.4	0.4	4.3	7.5	5.0	3.5	1.7
Nutmeg Mannikin	4.4	3.8	15.0	11.1	12.0	15.7	18.3
Misc. species†	0.6	1.3	1.1	5.4	1.4	0.3	7.2
Total	100.0	271.1	342.2	398.2	283.8	369.4	286.0

* Denotes native Hawaiian species.

† Zebra Dove (*Geopelia striata*), Spotted Dove (*Streptopelia chinensis*), Blue Pheasant (*Phasianus versicolor*), California Quail (*Lophortyx californicus*), Common Mynah (*Acridotheres tristis*), Hawaiian Creeper (*Oreomystis mana*), and Akiapolaau (*Hemignathus munroi*).

Each of the 16 study locations was sampled with similar effort, yielding a mean of 877 net-hours per station; however, the number of birds and species captured at each location varied. The only malarial parasite found during this study (Laird and van Riper 1981) was *Plasmodium relictum capistranoae*. Microfilaria, babesoids, and trypanosomes were not detected, and appear to be absent in the birds on Hawaii, although it is recognized that the last-mentioned group, which are seldom seen in routine blood films, might well be demonstrated by appropriate flagellate-concentration techniques. However, *Atoxoplasma* sp. was identified in blood smears from the House Sparrow, Nutmeg Mannikin, and House Finch, these being the first records of this parasite in the Hawaiian Islands (van Riper and van Riper 1985).

The total number of birds captured per 100 net-hours showed highs in March through June and September through October, with lows in November through February and July through August (Table 1). The January–February and July–August decreases were attributable to the low numbers of Japanese White-eyes captured during those 2-mo periods. There was a difference between the numbers and distribution patterns of native and introduced birds captured at different elevations, especially in mesic areas (Fig. 2). More introduced birds occurred in the lowland mesic habitat, but their numbers generally decreased with higher elevation. Native species were generally absent in mesic forest below 1000 m, after which their numbers increased with elevation. Introduced birds were more uniformly distributed throughout all elevational ranges of the xeric habitat; native birds ranged lower in the xeric forest (when compared to their distribution in mesic habitat), but reached their highest numbers above 1500 m.

Prevalence of Plasmodium in hosts.—Of the 2365 wild birds analyzed for blood parasites during this study,

7.8% were infected with *Plasmodium*. The native Apapane population had the highest percentage of infected individuals; the introduced House Finch and House Sparrow populations also had high percentages, although they were almost two-thirds lower than that of the Apapane (Table 2). The greatest percentage of individuals was infected with *Plasmodium* during November–December, the lowest percentage was infected in July–August (Table 3), but, there was no significant heterogeneity in prevalence among months ($\chi^2 = 10.98$; $df = 5$; $P \leq .10$). However, most native species had increases in the prevalence of *Plasmodium* in their respective populations sometime during the months of July to December (Fig. 3), indicating that during this time of the year native birds were most likely to be exposed to the malarial parasite. On the other hand, introduced birds tended to have a more uniform prevalence throughout the annual cycle, and in those species that did have peaks, a yearly pattern was not evident.

Avian populations from the mesic and xeric forests of Mauna Loa had significantly different levels of *Plasmodium* infections in the two habitat types (Table 3: $\chi^2 = 11.24$; $df = 1$; $P \leq .001$). The wet forest consistently supported higher prevalence levels, which was probably related to the increased availability of vector breeding sites. Elevation also had an extremely important influence upon population infection rates on Mauna Loa (Fig. 4: $\chi^2 = 149.96$; $df = 15$; $P \leq .001$). This suggests that elevation is also tied closely to *Plasmodium* infection levels, as are the time of year and the forest type.

There was no significant difference between the number of young and adults that were infected with *Plasmodium* in either native ($\chi^2 = 0.0$; $df = 1$; $P = 1.0$) or introduced species ($\chi^2 = 0.49$; $df = 1$; $P = .48$), which indicates that young birds are not being infected preferentially, as has been shown by Blackmore and Dow (1958) in North America. However, *Plasmodium* is

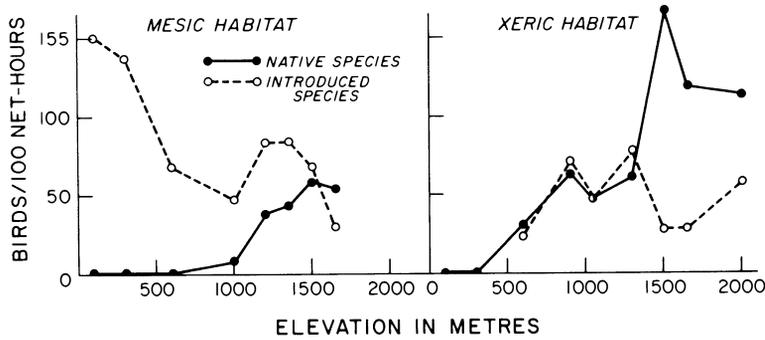


FIG. 2. Total introduced and native birds captured per 100 net-hours from 1978–1979 at 16 sampling stations on Mauna Loa, Hawaii.

more pathogenic for young birds, and the mortality of those dying soon after contracting the disease might have been overlooked in this type of mistnetting study. A recaptured bird was not more likely to have malaria than was a first capture ($\chi^2 = 2.93$; $df = 1$; $P = .09$). Birds ($n = 148$) that had one, two, or more visible lesions either on their feet, legs, or face were, however, more likely to have malaria than were individuals without lesions ($\chi^2 = 152.6$; $df = 1$; $P \leq .001$). If lesions are associated with diseases (such as Avian Pox) carried by airborne vectors, birds with lesions would perhaps have been exposed to mosquitoes and thereby have had a greater chance of exposure to the malarial parasite. Moreover, if a bird has another disease, it may also be more susceptible to developing avian malaria because of a lowered resistance caused by the current disease.

Birds that were collected moribund ($n = 10$) or that were killed by automobiles ($n = 31$) had higher prevalences of malaria than did birds captured in mist-nets ($\chi^2 = 24.2$; $df = 1$; $P \leq .001$). Perhaps the malarial parasite debilitates a bird so that diseased individuals are more likely to be struck by cars. However, birds accidentally killed in mist-nets ($n = 64$) were not more likely to have malaria than were normally processed

birds ($\chi^2 = 0.06$; $df = 1$; $P = .80$). Low, medium, and high fat levels in a bird had no relationship to *Plasmodium* infections ($\chi^2 = 3.81$; $df = 3$; $P = .28$), nor did light, medium, or heavy molts ($\chi^2 = 1.75$; $df = 3$; $P = .63$).

Parasitemia levels.—The overall parasitemia level from the entire avian community on Mauna Loa was 2.3 parasites per 10 000 RBC. The heaviest parasitemia levels occurred in September–October (Table 4), and there was a significant difference in those levels among months (ANOVA; $F = 24.7$; $df = 11$; $P \leq .001$). Elevation also had a marked influence on parasitemia levels in the birds on Mauna Loa (Fig. 4), and there was a significant difference in parasitemia levels among stations (ANOVA; $F = 5.73$; $df = 15$; $P \leq .001$). The highest parasitemia levels occurred between 900 and 1500 m elevation, that area where native bird and breeding mosquito distributions overlapped. Low parasitemia levels at elevations above 1500 m are probably owing to decreased vector abundances (see Goff and van Riper 1980).

Parasitemia levels varied significantly among species on Mauna Loa (ANOVA; $F = 9.2$; $df = 10$; $P \leq .001$). Apapane had the highest overall parasitemia levels throughout the year (Fig. 3), most likely due to the nomadic nature of this species. Introduced species had low numbers of parasites per 10 000 RBC throughout

TABLE 2. Average yearly prevalence of *Plasmodium relictum capistranoae* infections in the 11 most common bird species captured from 1978 to 1979 on Mauna Loa, Hawaii.

Bird species	No. of birds examined	No. of birds infected	Percent of population infected
Apapane*	366	107	29.2
Common Amakihi*	518	38	7.3
Iiwi*	81	5	6.1
Elepaio*	67	4	6.0
Omao*	47	1	2.1
House Finch	69	8	11.6
House Sparrow	70	8	11.4
Nutmeg Mannikin	121	3	2.5
American Cardinal	45	1	2.2
Japanese White-eye	961	9	0.9
Red-billed Leiothrix	20	0	0.0

* Denotes native Hawaiian species.

TABLE 3. Annual pattern of *Plasmodium relictum capistranoae* prevalence in the 11 most common bird species from Mauna Loa, Hawaii ($n =$ number of birds).

Months	Prevalence of <i>Plasmodium</i> infection (% of birds)					
	Mesic forest		Xeric forest		All habitats	
	%	<i>n</i>	%	<i>n</i>	%	<i>n</i>
January–February	8.2	244	6.9	180	7.5	424
March–April	10.2	257	4.0	150	7.9	407
May–June	11.0	96	3.9	79	8.1	175
July–August	6.1	231	4.7	131	6.0	362
September–October	9.3	404	5.0	180	8.0	584
November–December	11.0	289	6.6	124	9.8	413

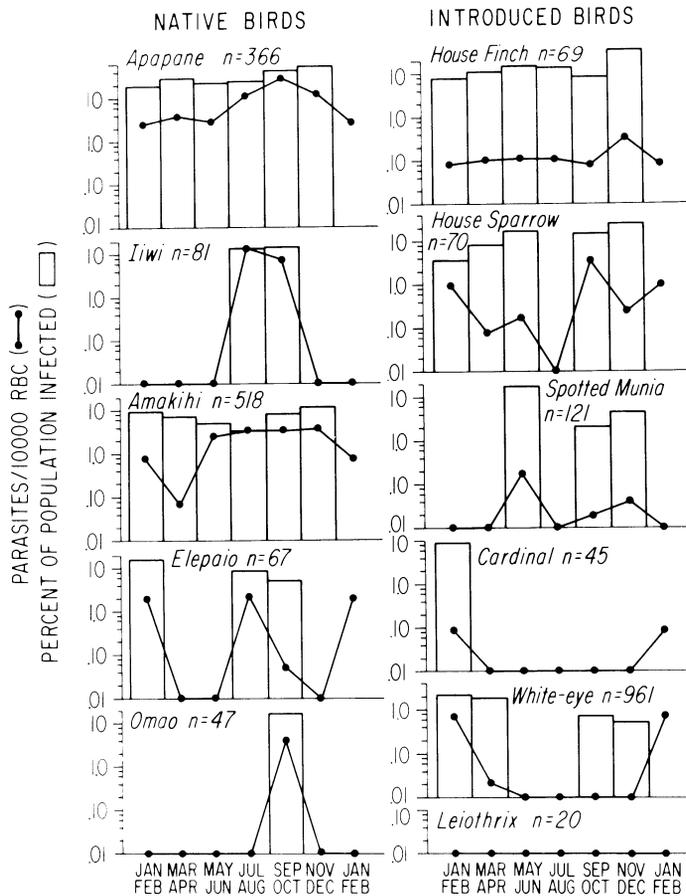


FIG. 3. Bimonthly rates of malaria infection in avian hosts on Mauna Loa, Hawaii, as compared to parasitemia levels (parasites/10000 red blood cells) in those infected individuals.

the year, indicating primarily latent infections. Native species had higher parasitemia levels, indicating the greater susceptibility of the endemic birds. A multiple range (LSD) test showed that only native species formed subgroups, with the Apapane's parasitemia levels significantly different from those of the Red-billed Leiothrix, Nutmeg Mannikin, Omaa, Japanese White-eye, House Sparrow, and Common Amakihi. The Iiwi formed the second subset and differed from the Nutmeg Mannikin, Omaa, and Japanese White-eye. The third subset was formed by the Elepaio, which had significantly different parasitemia levels than the Nutmeg Mannikin and Japanese White-eye. The last subset was formed by the Common Amakihi, which differed significantly from the Nutmeg Mannikin and the Japanese White-eye.

The principal breeding period of native Hawaiian birds is December to May (Baldwin 1953, Eddinger 1970, Conant 1977, van Riper 1978, 1980, van Riper and Scott 1979, Berger 1981), and this was the time of year when we found the lowest parasitemia levels. Patterns of feather molt and the presence of a cloacal protuberance or brood patch were used as an index to measure the effect of breeding on parasitemia levels in

the birds. We found no significant difference in parasitemia levels between breeding and nonbreeding birds using these parameters ($\chi^2 = 1.75$; $df = 3$; $P = .63$). While young were not more likely to have malaria than were older birds, parasitemia levels of 1st-yr birds ($n = 256$) were up to six times greater than those in older birds, particularly the native species. This suggests that younger birds have less resistance to the malarial parasite once contracted.

Vector distributions.—Because of the porosity of the volcanic substrate in Hawaii, which results in a patchy distribution of potential breeding sites, mosquito distribution was not uniform over the altitudinal range of the forests on Mauna Loa (see also discussion by Goff and van Riper 1980). Greatest mosquito densities were found to be associated with kipukas (an island of older vegetation surrounded by a more recent lava flow with younger vegetation) and human habitation (most drinking water in rural Hawaii is in water tanks that store rainfall collected from house roofs).

Two mosquito species were collected regularly throughout this study, *Aedes albopictus* and *Culex quinquefasciatus*. Larvae and pupae of *C. quinquefasciatus* were present from sea level to 1350 m elevation

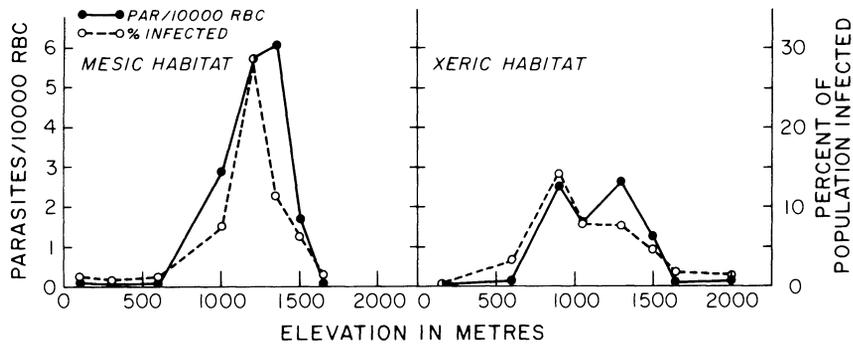


FIG. 4. Percentages of all birds captured on Mauna Loa Volcano, Hawaii, from 1978–1979 that were infected with malarial parasites, and their concomitant parasitemia levels (parasites/10 000 red blood cells), both expressed along an elevational gradient from sea level to tree line.

throughout each month of this study (Table 5). During July–August this mosquito was found breeding up to 1650 m elevation, the highest reaches of extant mesic forest on Mauna Loa. In addition to these data from our artificial oviposition sites, “natural” oviposition sites were found to be 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 1350 m in xeric habitat and 1500 m in mesic habitat on Mauna Loa. Other findings from the island of Hawaii record breeding at even higher elevations. Swezey and Williams (1932) found egg rafts in a rain barrel at 1829 m on Mt. Hualalai and larvae at 1981 m on Mauna Kea, and Komatsu (1966) reported egg rafts from concrete pools at 1981 m on Mauna Kea.

Adult *C. quinquefasciatus* were collected in light traps operated by the Hawaii State Department of Health at elevations below 900 m. This mosquito showed increasing adult populations from January until July–August, after which numbers fell precipitously (Fig. 5). We operated standard New Jersey Mosquito Light Traps at all stations above 600 m for a 6-mo period, but no mosquitoes were collected, even when a CO₂ attractant was employed. These data point to the generally low density of mosquitoes at higher elevations on Hawaii.

During this same period biting collections using humans at all stations above 300 m elevation yielded negative results for *C. quinquefasciatus*. For example, during three 24-h biting collections at 1200 m elevation, only two adult mosquitoes were observed and neither alighted. Even though adult *C. quinquefasciatus* reach high levels at lower elevations during the warmer months of the year, densities at higher elevations are apparently quite low.

Larvae and pupae of *A. albopictus* were recovered from our artificial oviposition sites only up to 300 m during all months of the study, but were found up to 900 m during the warmer months. Other oviposition sites were associated with human activity and consisted primarily of construction equipment, discarded bottles, tires, cans and sundry human refuse. No *A. albopictus* larvae were found above 900 m elevation. *Aedes albopictus* has been reported to be a minor vector of avian malaria (Boyd 1949), and our attempts to transmit *P. r. capistranoae* with this vector were unsuccessful. However, Huff et al. (1950) were able to infect 73% of *A. albopictus* in the laboratory with *Plas-*

TABLE 4. Parasitemia levels of *Plasmodium relictum* in the 11 most common bird species from Mauna Loa, Hawaii. Sample sizes as in Table 3.

Months	Parasites/10 000 red blood cells		
	Mesic forest	Xeric forest	Both forests
January–February	1.21	0.47	0.97
March–April	0.95	0.04	0.60
May–June	0.87	0.81	1.27
July–August	2.48	1.60	3.26
September–October	3.46	0.78	4.15
November–December	1.51	1.82	2.11

TABLE 5. Maximum elevations of mosquito larvae and pupae collected from artificial oviposition sites at stations from sea level to 2000 m elevation on Mauna Loa Volcano, Hawaii, from November 1977 through January 1979.

Months	Species	Max. elev. (m)
January–February	<i>Culex quinquefasciatus</i>	1500
	<i>Aedes albopictus</i>	900
March–April	<i>C. quinquefasciatus</i>	1350
	<i>A. albopictus</i>	300
May–June	<i>C. quinquefasciatus</i>	1500
	<i>A. albopictus</i>	300
July–August	<i>C. quinquefasciatus</i>	1650
	<i>A. albopictus</i>	900
September–October	<i>C. quinquefasciatus</i>	1500
	<i>A. albopictus</i>	600
November–December	<i>C. quinquefasciatus</i>	1500
	<i>A. albopictus</i>	600

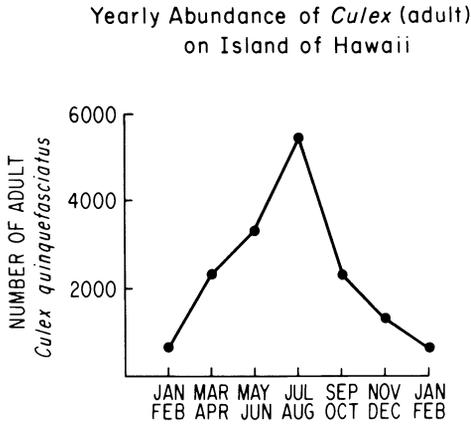


FIG. 5. Numbers of adult *Culex quinquefasciatus* captured in light traps operated by the Vector Control Branch, Hawaii State Department of Health on the island of Hawaii from November 1977 through January 1979.

modium fallax. This vector, therefore, may be responsible for some malaria transmission in wild Hawaiian birds.

Experimental results

Asexual malaria cycle.—Each avian host used in our experiments was put through a rigid acclimatization period after capture from the wild. These periods were quite variable among the seven challenged host species. Stabilization of body mass was achieved in the Canary, Red-billed Leiothrix, Japanese White-eye, Laysan Finch, and Common Amakihi within 1 wk after capture. In fact, the Laysan Finch spent 3 wk in small holding cages on the ocean voyage from Laysan Island, and arrived on Hawaii Island in excellent condition. The Iiwi adjusted more slowly, and mass stabilization

was not achieved until an average of 76.6 d (SE = 5.27 d) following capture. Apapane, the most common hon-eycreeper, also experienced difficulty in adjusting to captivity; an average of 71.4 d (SE = 2.97 d) elapsed before this species was ready for challenge experiments with the malarial parasite.

The five experimental Japanese White-eyes and Red-billed Leiothrix all were refractory to *P. r. capistranoae* and each of these species thus had an Index of Adaptation of 1.0. All other challenged species contracted malaria, although inter- and intraspecific survival rates were variable (Fig. 6). The Canary had an Index of Adaptation of 0.50. Because two of the Mauna Loa Common Amakihi were refractory, their Index of Adaptation ($I_a = 0.60$) was the highest of the native species, followed in decreasing order by the Mauna Kea Common Amakihi ($I_a = 0.33$), Apapane ($I_a = 0.30$), and the Iiwi ($I_a = 0.20$). The Laysan Finch totally lacked any immunogenetic capacity against *P. r. capistranoae* ($I_a = 0$).

Each of the experimental birds that succumbed was found upon necropsy to have an extremely high level of malarial parasites. The liver and spleen were usually swollen and discolored and extensive tissue damage had occurred, especially in the latter organ. All of the birds had packed cell volumes of <20%, but none had any endoparasites, secondary bacterial infections, or symptoms of other debilitating maladies. It was, therefore, assumed that these birds died from the malaria infection.

Although survival differed among the challenged native host species, parasitemia levels throughout the patent period were similar (Fig. 7). Peak parasitemia levels for the Common Amakihi, Apapane and Iiwi all fell within 400 parasites per 10 000 RBC of one another.

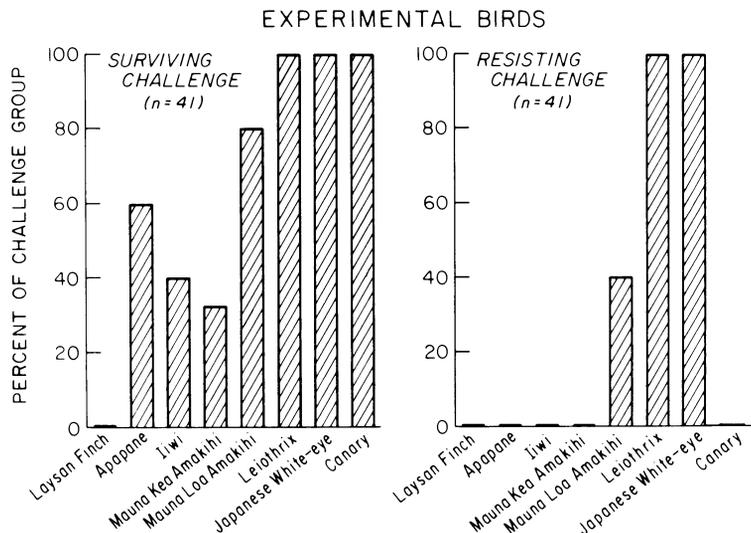


FIG. 6. Percentage of species that were resistant to and percentage that survived a challenge with avian malarial parasites (*Plasmodium relictum capistranoae*). All species had five individuals challenged except for the Mauna Kea Common Amakihi, which had six.

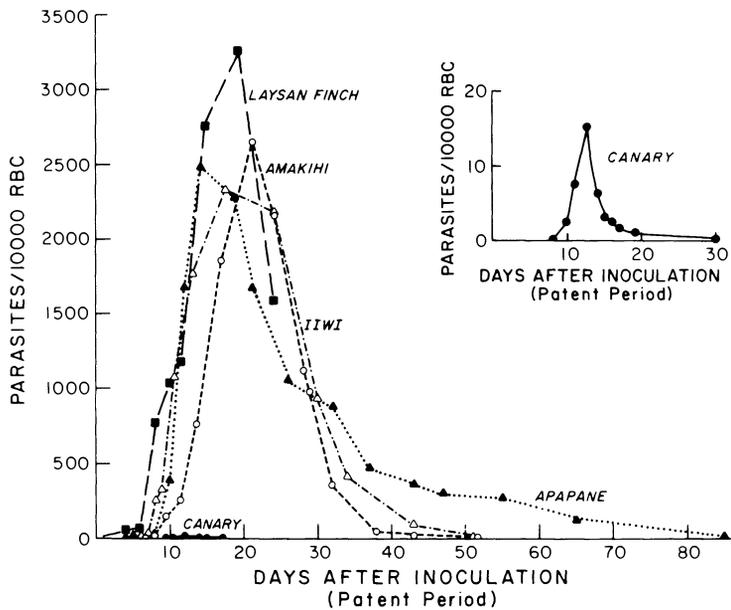


FIG. 7. Parasitemia levels over the patent period (interval during which parasites can be demonstrated in the blood) of four native Hawaiian bird species ($n = 26$) as compared to that of the Canary ($n = 5$), following challenge with the malarial parasite *Plasmodium relictum capistranoae*.

In the one Laysan Finch that survived past day 20 of the patent period, parasitemia levels were dropping, although the bird did not survive. The disparity between parasitemia levels in the native Hawaiian species and the Canary indicates the very high susceptibility of the native birds to this introduced malarial parasite. Moreover, the patent period (the period during which parasites can be demonstrated in the blood) was over three times as long in the Hawaiian species. The duration of the patent period in the main island Hawaiian birds did not correspond to the number of individuals that survived. Fewer Iiwi than Common Amakihi or Apapane survived the infection, but the last species carried much higher parasitemia levels for a longer time period. However, analysis of the overall course of the infection does show that the prepatent period was shorter in the Iiwi, as was the initial period of rise. In addition, the length of the Iiwi crises was almost double that of the other two species.

In an analysis of immature (trophozoite), sexual (gametocyte), and asexual (schizont) forms present throughout the primary attack period in the Hawaiian bird hosts, trophozoites generally outnumbered the other two groups of parasites (Fig. 8). Parasite abundance patterns were similar in the Common Amakihi and Iiwi, except that during the Iiwi crisis period schizonts outnumbered gametocytes. In the Apapane, gametocytes and schizonts comprised a much larger percentage of the total parasites than in any of the other species. The highly susceptible Laysan Finch, in addition to having an earlier expression of parasites in the peripheral blood, had a very different pattern of

parasite abundances, with immature parasites still increasing at the time that the last individual succumbed. Only in the Laysan Finch did we observe a decreased food intake just prior to death, and this was the only species that exhibited an appreciable decline in mass over the patent period.

The daily schizogonic cycle in the blood was asynchronous when the parasites were analyzed as percentages of those counted on a single occasion, as recommended by Garnham (1966). However, when expressed as the number of parasites per 10 000 RBC over a 24-h cycle, periodicity of *P. r. capistranoae* in the Apapane, Common Amakihi, and Canary exhibited a quotidian cycle, with peaks occurring at ≈ 12 -h intervals (Fig. 9). Peak levels of gametocytes in the peripheral blood occurred from the late morning to the early afternoon hours.

Sexual malaria cycle.—The daily activity patterns of *C. quinquefasciatus* and *A. albopictus* were antipodal (Fig. 10). *Culex quinquefasciatus* were more active during the cooler night (i.e., 1800–0600), whereas *A. albopictus* exhibited the greatest activity during the warmer daylight hours. The only period when the activity of the two species overlapped was from 1800 to 2200. *Culex quinquefasciatus* would not feed during the day ($n = 12$ replications), and it was necessary to leave infected birds in the biting cages overnight. Thirty-nine separate feedings (of which 25 were successful) employing 57 mosquitoes were conducted during the course of this study.

The sexual stage of the malaria cycle was quite difficult to complete, probably because of the low ambient

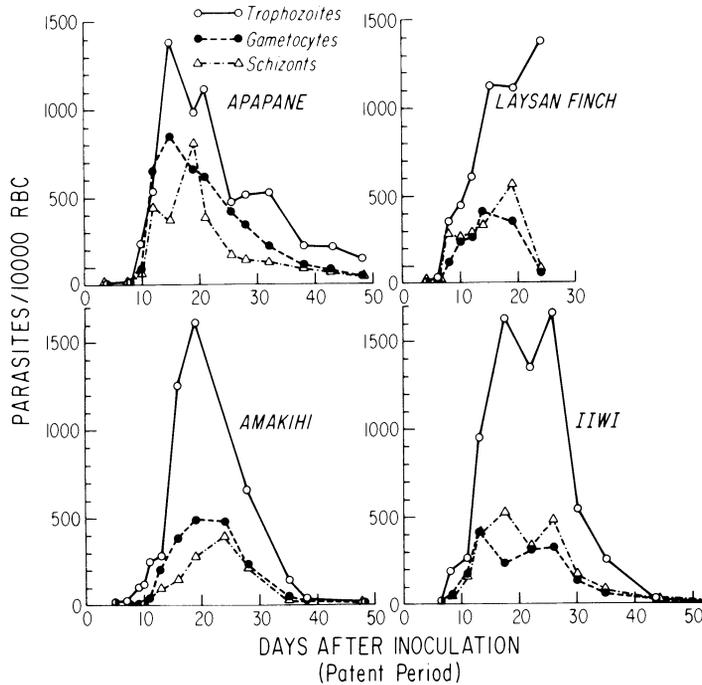


FIG. 8. Trophozoite, gametocyte, and schizont levels in four species of native birds ($n = 26$) from Hawaii challenged with the malarial parasite *Plasmodium relictum capistranae*. The patent period is the interval during which parasites can be demonstrated in the blood.

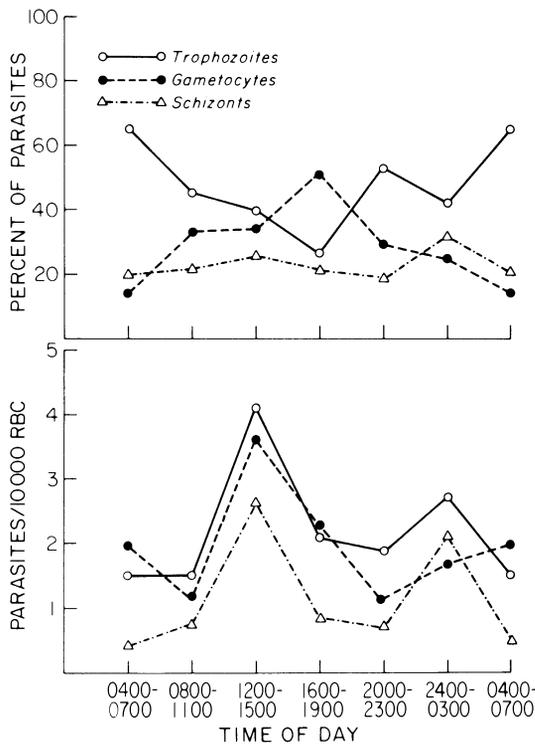


FIG. 9. The daily schizogonic cycle of *Plasmodium relictum capistranae* parasite in the peripheral blood of the Apapane, Common Amakihi, and Canary (RBC = red blood cells).

temperatures at the laboratory ($\approx 15^{\circ}\text{C}$). Colder temperatures are well known to inhibit parasite development in the mosquito (Hewitt 1940, Ball and Chao 1964, Huff 1968). The earliest ookinete development that we observed was 16 h after engorgement, and the parasite reached this state of development in numerous *C. quinquefasciatus*. Early ookinete measurements (\bar{X} length = $6.5 \mu\text{m}$, $\text{SE} = 0.61 \mu\text{m}$; \bar{X} width = $2.2 \mu\text{m}$, $\text{SE} = 0.22 \mu\text{m}$) were similar to those given for other strains of *P. relictum* (Corradetti et al. 1970). However, we found a very low prevalence of oocyst development in the gut wall. Sporozoite development was quite slow, and it was not until day 16 following the infected blood meal that completion of the sexual cycle in the mosquito was achieved (Table 6).

Successful transmission of *P. r. capistranae* by *C. quinquefasciatus* was first demonstrated in this study on 28 September 1979. The mosquito took its initial blood meal on 12 September from a Common Amakihi with a parasitemia level of 1500 parasites per 10 000 RBC. Sixteen days later the mosquito engorged a second time on a noninfected Laysan Finch. Through day 4 the Laysan Finch exhibited no signs of parasitemia in the peripheral blood. The first parasites were observed on day 5, the infection climax was reached on day 7, but the bird did not die until day 21. When compared to the cycle of subinoculated Laysan Finches (see Fig. 7), the malaria cycle in a sporozoite infected individual took longer to develop.

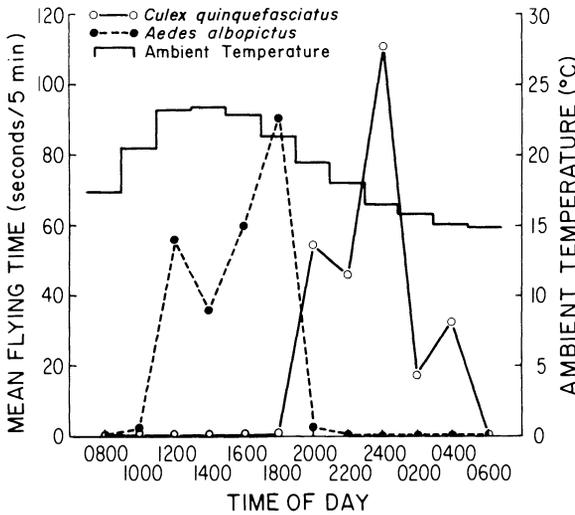


FIG. 10. Daily activity cycles of 12 female *Culex quinquefasciatus* and 12 *Aedes albopictus* over four 24-h cycles, at 1200 m elevation on Mauna Loa, Hawaii.

DISCUSSION

Four questions will now be answered: (1) What is the present-day distribution of the malarial parasite on the island of Hawaii? (2) How susceptible to malaria are the native land birds, when compared to their introduced counterparts? (3) What selective forces resulting from malaria are currently operative on the bird populations? (4) What role has malaria played in the decline of the endemic Hawaiian avifauna?

Distribution of malaria

Although Warner (1968) failed to find malarial parasites in wild native birds, and presented few data on the altitudinal distribution of mosquitoes in Hawaii, he suggested that the vector, *C. quinquefasciatus*, was "functionally" absent from the forests above 600 m elevation; therefore, this habitat was a "safe" or malaria-free zone. Our data indicate a quite different picture. Breeding populations of the vector were present throughout much of the extant native bird habitat on Mauna Loa, and infected birds were found at all elevations on the mountain. During the warmer months of the year *C. quinquefasciatus* was found breeding even in the uppermost reaches of the mesic forest. However, we rarely observed free-flying adult *C. quinquefasciatus*, and the negative results of our biting experiments show how difficult it is to detect the presence of this vector, the daily activity cycle of which is asynchronous with humans'. Furthermore, Tempelis et al. (1970) showed that this mosquito prefers to bite birds, thus further reducing the chance of human detection. Bennett and Coombs (1975) found a similar situation in Newfoundland, where very low densities of ornithophilic vectors still maintained a high prevalence of blood parasites in bird populations.

Possibly *C. quinquefasciatus*, in the absence of nat-

ural selective forces that are present in its native North American habitat, has recently undergone an altitudinal expansion in Hawaii. Mosquitoes could have been restricted to elevations below 600 m from 1826 until ≈1960, after which they suddenly tripled their altitudinal range during the following 15 yr period. But available data do not support this hypothesis. The likelihood of this rapid expansion is further diminished by the fact that extensive mosquito control programs were initiated in Hawaii during the early 1960's. We feel that mosquitoes were present above 600 m when Warner (1968) conducted his initial malarial survey, but, as is the case today, in such low densities that they were not detected on a regular basis. Furthermore, as noted by Goff and van Riper (1980), the distribution of mosquitoes is not uniform at higher elevations, but coincides with the distribution of kipukas. This patchy distributional pattern, combined with primary breeding sites other than ground pools, would make the detection of mosquitoes at higher elevations difficult. This is supported by the pattern we see today, with casual observations of mosquitoes at higher elevations (Komatsumi 1966, Banko in Berger 1981) which are similar to the earlier observations of Swezey and Williams (1932).

TABLE 6. Success rate of sexual cycle transmission with *Plasmodium relictum capistranoae* in 57 *Culex quinquefasciatus* and *Aedes albopictus* at 1200 m elevation on Mauna Loa, Hawaii.

Days between infective blood meal and analysis	Vector species	No. successful feeding trials*	Success rate	
			No. engorged	No. positive†
4	<i>C. quinquefasciatus</i>	4	17	0
	<i>A. albopictus</i>
6	<i>C. quinquefasciatus</i>	1	1	0
	<i>A. albopictus</i>	1	1	0
8	<i>C. quinquefasciatus</i>	4	10	0
	<i>A. albopictus</i>
9	<i>C. quinquefasciatus</i>	2	5	0
	<i>A. albopictus</i>
10	<i>C. quinquefasciatus</i>	3	8	0
	<i>A. albopictus</i>	1	1	0
11	<i>C. quinquefasciatus</i>	1	1	0
	<i>A. albopictus</i>
12	<i>C. quinquefasciatus</i>	3	3	0
	<i>A. albopictus</i>
14	<i>C. quinquefasciatus</i>	2	6	0
	<i>A. albopictus</i>
16	<i>C. quinquefasciatus</i>	1	1	1
	<i>A. albopictus</i>
17	<i>C. quinquefasciatus</i>	2	3	2
	<i>A. albopictus</i>	1	1	0

* A successful trial is one in which > 1 mosquito engorged.
 † A "positive" is a mosquito that had sporozoites in its salivary glands and/or that successfully transmitted malaria to a noninfected avian host.

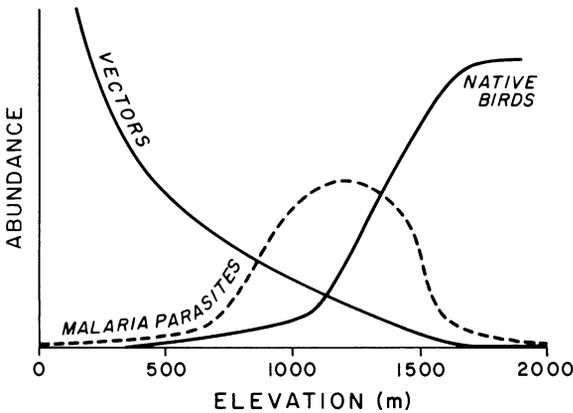


FIG. 11. A generalized model of native bird abundances, malarial parasite incidence, and mosquito vector levels along an elevation gradient on Mauna Loa, Hawaii.

The presence of a vector does not necessarily imply that parasites can be transmitted at that location. However, we found *Plasmodium* present in birds at all locations sampled from sea level to forests at high elevations. Some birds at higher elevations undoubtedly contracted malaria during their visits to lower forests, particularly those infected individuals captured at upper xeric stations. The extremely high prevalence in the Apapane population is probably related to the nomadic behavior of this species, which frequently brings the birds to lower elevations. But the high prevalence in all native bird species at 1350 m in the xeric and 1500 m elevation in the mesic forest can be attributed in large part to the fact that *C. quinquefasciatus* breeds at those locations throughout the year. Furthermore, species such as the Elepaio and Omao are not considered migratory, yet we found infected individuals at upper elevations.

The present-day altitudinal distribution of the avian malarial parasite on Mauna Loa is not a direct reflection of vector densities (Fig. 11). *Culex quinquefasciatus* is numerous at lower elevations, yet the prevalence of *Plasmodium* in avian populations from those localities is quite low. It is not until the mid-elevational ranges are reached that prevalence appreciably changes. These are also the lowest elevations at which native birds are presently found. In this region of overlap, prevalence increases disproportionately to the number of available vectors. As noted by Goff and van Riper (1980), it is at these elevations that kipukas become sites of increased contact between vector and host, thus increasing the potential for small vector populations to transmit the malarial parasite to a large number of hosts, especially with this vector being ornithophilic. Apparently, pressure exerted by the pathogenicity of *Plasmodium* is presently restricting the native avifauna to high-elevation forests. The distributional pattern of malaria in Hawaii today is by no means a static situation. During the warmer fall months *C. quinquefasciatus* breeds at higher densities in upper forests (Goff and van Riper 1980), and survival and completion of the parasite's sexual cycle in this insect host are probably enhanced. Concomitantly, the highest monthly prevalence of malaria (Table 3) in avian populations occurred subsequent to the upper altitudinal movement of the vector.

Susceptibility of Hawaiian birds to malaria

Our field data and supplementary laboratory experiments have shown that in all cases, once infected, native Hawaiian birds carry higher parasitemia levels than do most introduced species (Fig. 12). The total lack of resistance to malaria in the Laysan Finch, whose population has never been exposed to this parasite,

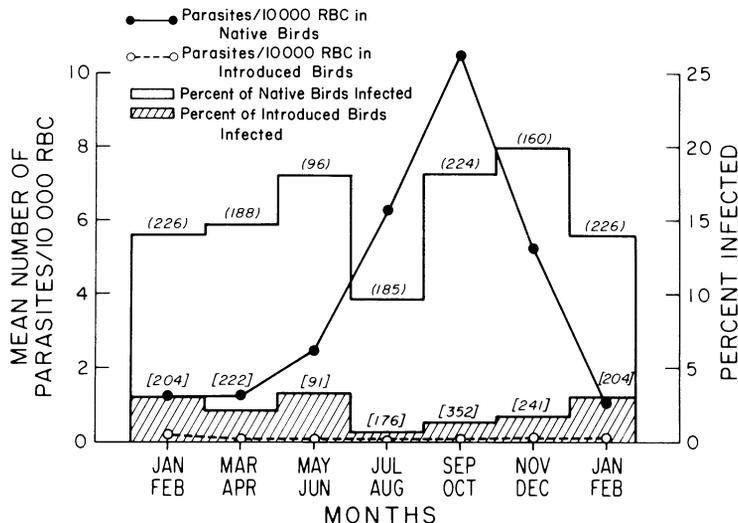


FIG. 12. A comparison of native and introduced birds' parasitemia levels and infection rates over the 1978-1979 annual cycle on Mauna Loa, Hawaii. Numbers in brackets or parentheses are sample sizes.

provides an insight into the probable resistance of the pre-malaria Hawaiian avifauna; it must have been extremely low. The explosive increase of parasites following the prepatent period (see Fig. 7) predetermined that birds in the pre-malaria avifauna either survived or succumbed; a gradual adaptation was rarely possible. Absence of loss of mass and continued high levels of fat in our challenged birds shows that infections are still acute in nature. From those few early individuals that did survive, the immunogenetic capability to cope with the malarial parasite has now spread throughout portions of the extant populations.

We found that each challenged species exhibited resistance to the malarial parasite in direct proportion to the present-day abundance of that species. For example, the Iiwi, the most susceptible of the challenged birds from the main islands, has undergone the greatest population declines. In 1903 Perkins wrote: "The 'Iiwi' is one of the most abundant and generally distributed of all of the Drepanid birds, being found throughout the woods of all the forest-clad islands." Iiwi are now rare on Oahu (Shallenberger 1978) and Molokai (Scott et al. 1977), and apparently extinct on Lanai (Hirai 1978). Since Baldwin's 1953 work on the island of Hawaii, the species has also undergone drastic population reductions and range contractions (Conant 1981, Scott et al., *in press*). On the other hand, the Common Amakihi, which we found to be the most resistant native bird, is still found in fairly high numbers and has reinvaded areas from which it was earlier extirpated (Pedley 1961, van Riper 1973, Berger 1981).

Each bird species coped with high parasitemia levels in a different fashion. The Common Amakihi on Mauna Loa was the only native bird that was refractory to *Plasmodium*, and all but one challenged individual survived. The relatively high degree of resistance developed by this species can be linked directly to its natural history. Unlike the Apapane and Iiwi, Common Amakihi populations are fairly sedentary and do not undergo regular massive altitudinal movements (Baldwin 1953). This would mean that any population found within the "malaria zone" would be continually exposed throughout the year. Thus, selection for resistance would be greater than for species that migrate out of the zone. Moreover, this would also decrease the chance of genetic swamping.

The differences that have developed in regard to the immunogenetic capabilities of a species are exemplified by the comparison of the wet forest Mauna Loa and dry forest Mauna Kea Common Amakihi populations. Malaria is absent in the dry forest of Mauna Kea (van Riper 1975), and the Common Amakihi from this region, unlike the Mauna Loa Common Amakihi, were more susceptible to malaria (Fig. 6). This suggests that intraisland gene flow is quite slow in Hawaii, as recently suggested by Pratt (1980). However, some immunogenetic resistance to *P. relictum* is now present in a number of Hawaiian birds, and should stand them

well barring the future introduction of another malaria species or a temperate zone vector.

Responses of native birds to malaria

The results of this study clearly demonstrate that malaria is present in native bird populations and can readily kill these birds. In addition to causing mortality (thus lowering population numbers), the malarial parasite is not uniformly distributed at all elevations, and thus may be restricting native species to the higher elevations and drier areas. Also, host behavioral patterns have been modified so that birds now minimize their temporal contact with malarial vectors.

Immunogenetic responses.—The mesic forests of Mauna Loa occupy a much larger area, have a greater foliage height diversity, and a higher insect biomass and standing crop of nectar-producing flora than does the xeric forest (Mueller-Dombois et al. 1981). One would, therefore, expect a greater density of birds in mesic habitats. A comparison of capture rates of birds during the same time periods at mesic and xeric stations of similar elevations indicates that this was not the case. For example, the Apapane, which is principally a mesic forest bird (Berger 1981), was captured more often at xeric sites than at mesic sites of similar elevations (5:4 ratio). The Iiwi, another mesic forest bird, also had higher capture rates at xeric sites. Higher capture rates in xeric forest may be a result of the catchability of the birds in that habitat, but nevertheless does suggest that mesic forest birds are presently numerous throughout the year in drier areas. A recent census of birds on Mauna Loa, Hawaii, also found that the highest densities of native species occurred in the upper elevation xeric forests of Puna (Scott et al., *in press*). Because of the availability of vector breeding sites, both prevalence and parasitemia levels were uniformly higher in the mesic areas. It therefore appears that as well as restricting the birds altitudinally, pressure from malarial parasites favors native birds that can live in drier areas.

Introduced birds, except for the House Finch, which is primarily a dry-forest species, were consistently found in higher numbers in mesic forests. For example, the Japanese White-eye, which is ubiquitous throughout the islands (Guest 1973), was captured more often in mesic sites (9:1 ratio). It therefore appears that introduced bird species are occupying those areas where native species occur in lower numbers, and the refractory ability of many introduced species to the malarial parasite certainly benefits them in these areas.

Behavioral responses.—Warner (1968) noted in his experimental Hawaiian birds that none slept with their bills and faces tucked into the fluffed back feathers and that their legs were continually exposed, whereas the introduced species all slept with their heads and feet buried. We have made extensive observations on sleeping postures of captive Hawaiian birds from Kauai and Hawaii, and our results are quite different. Kauai Com-

mon Amakihi and Apapane frequently maintained a sleeping posture where the bill and face were tucked into the back feathers, and often had one leg raised into the abdominal feathers. In the birds maintained on Hawaii Island during this study, the Common Amakihi, Apapane, and Iiwi, all slept in this latter fashion. On a few occasions, individuals were found clinging to the sides of the cages while asleep. But for the most part, all Drepanidinae that we have observed slept with their heads tucked into ruffled back feathers and one leg raised into their abdominal feathers.

Warner's (1968) observations on the sleeping postures of his captive birds led him to speculate that native birds were more likely to be exposed to malarial vectors. Assuming that Warner's observations were correct, this would mean that those native birds that slept with their soft integumentary structures exposed were more likely to be infected with malaria, and were subsequently eliminated. Selection should favor a modification of the native birds' sleeping behavior.

A number of native birds rely upon nectar as a food source and follow the altitudinal flowering sequence of nectar-bearing trees (Perkins 1903, Munro 1944, Baldwin 1953). Lamoureux et al. (1981) showed 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 highest altitude trees flowering during the winter months. This means that to obtain maximum quantities of nectar, birds must move to lower elevations during the fall period. For example, it is during this time that the greatest number of Iiwi are found with malaria (Table 3). The birds are, therefore, forced into the lower malaria belts during their fall sojourns in search of nectar, and are met at this time by an expanding vector population. As a result, the zone of mosquito-bird overlap is greatly increased and the potential for malaria transmission is much higher than it otherwise would be. It can be seen that interacting environmental factors and behavioral activities maximize the spread of avian malaria.

Some native Hawaiian birds move daily to and from the malaria zone. MacMillen and Carpenter (1980) have shown that the Apapane and Iiwi presently undergo daily altitudinal migrations. Individuals gradually move downslope during the day, then just prior to dusk gather from lower elevations (≈ 1200 m) and migrate upslope. Although daily flights are energetically costly, these authors felt that overnight energy savings, as a result of the thermal protection afforded by the mature forest, compensated for the movements. Apparently these flights occur only in the warmer and usually drier months of the year. Were the selective force for this movement energetically based, it could be predicted that such flights would occur during the colder and wetter winter months. We found that the Apapane and Iiwi regularly breed during the cold, wet winter months and our mist-net recapture data and color-band ob-

servations show that most birds are also sedentary during this period.

The daily evening movement of birds to higher elevations seems to have been influenced by the selection pressure exerted by malarial parasites at lower elevations. The birds leave their overnight area early in the morning, when *C. quinquefasciatus* activity levels are decreasing (see Fig. 10), gradually work downslope, and reach lower elevations when *C. quinquefasciatus* is at its lowest activity levels. MacMillen and Carpenter (1980) showed that evening flights began at 1600, and that most birds reached upper elevations by 1830. Our data show that *C. quinquefasciatus* activity does not begin until 2000, by which time birds have left the principal malaria zone.

What was once probably a gradual movement of the birds downslope following the flowering of nectar-producing trees, has now evolved into a daily circular pattern. In the past, those birds that either remained in the upper elevation breeding areas or that moved laterally across the mountain (Baldwin 1953, van Riper 1978) would not have been appreciably affected, nor would have the small segment of the early Apapane and Iiwi populations that returned upslope each evening. However, those individuals that gradually moved downslope would eventually enter malaria zones. Owing to the presumed extreme susceptibility of early drepanids, the great majority of those individuals most likely succumbed to malaria. At present, the portion of the population that still undergoes a gradual downslope movement is being further reduced in numbers. Because of the selective pressure exerted by the malarial parasite at lower elevations, we are, therefore, left today with a pattern in which a large majority of the Apapane and Iiwi undergo daily altitudinal migrations.

Malaria arrival date

After the introduction of *C. quinquefasciatus* in 1826, this mosquito rapidly spread over the main Hawaiian islands (Hardy 1960). Workers have, therefore, deduced that avian malaria closely followed the spread of this mosquito throughout the archipelago (Warner 1968). Avian malaria was undoubtedly present in Hawaii during the 1800's because of the arrival from time to time of parasitized migrants. However, we doubt that a large enough reservoir was present at this early date for malaria to have spread to the native birds and result in the mass extinctions of the late 1800's.

If in fact malaria was a causative factor in the demise of the native birds at the turn of the century, one surely would have expected it to be common on Hawaii island during the late 1930's. This was not the case. P. H. Baldwin (*personal communication*) did a survey of blood parasites throughout Hawaii Volcanoes National Park in 1938 and 1939, collecting samples from 88 introduced and native birds from 70 to 2000 m elevation. The only species in which he detected blood

parasites were the introduced Red-billed Leiothrix (collected at 1350 m elevation) and the California Quail (collected at 2000 m elevation). Baldwin concluded his study by saying: "This is at least some indication that there was no high incidence of bird malaria in the native birds and possibly none at all." In the same areas where we found an extremely high prevalence of *Plasmodium* in native birds, he failed to find any. It is unlikely that he could have overlooked this parasite if it had been present.

The largest number of migrants that regularly travel to Hawaii from North America and Asia are sea- and shorebirds. Recent exhaustive summaries of hematozoa distribution in bird families across North America (Greiner et al. 1975), Asia (McClure et al. 1978), and Great Britain (Peirce 1980) have shown that both groups of birds are nearly hematozoan-free. Moreover, not one of the individuals examined by these authors was found to harbor *P. relictum*, the only malarial parasite presently known to occur in Hawaii (Laird and van Riper 1981). Ducks reach Hawaii each year on a regular basis, and are the only migrant host group known to carry *P. relictum*, although *P. r. capistranae* has never been reported from them. The strenuous (≈ 4500 km) trip from the mainland would certainly select against the arrival of any individual with a high malarial parasitemia. Moreover, there is relatively little habitat suitable for migrating waterfowl in Hawaii, and these areas are restricted to select locations along the shorelines. Even during the late 1800's the native passerines rarely frequented these shoreline areas unless blown from the higher forests by storms (Henshaw 1902). It therefore seems unlikely that migratory birds acted as a substantial reservoir for avian malaria in Hawaii.

The complete asynchrony of *P. r. capistranae* gametocyte periodicity and *C. quinquefasciatus* activity levels also suggests that malaria has been a recent arrival to the Hawaiian Islands. Gametocyte production peaks during 1200–1500 (see Fig. 9), whereas vector activity levels peak during 2400–0300 (see Fig. 10). This means that when *C. quinquefasciatus* is actively seeking blood meals, sexual malarial parasites are at their lowest levels in avian hosts, thus decreasing the possibility of successful completion of *Plasmodium*'s sexual cycle. There apparently has been insufficient time for the parasite and its Hawaiian vector to synchronize.

We propose that the vector, *C. quinquefasciatus*, arrived well before an adequate reservoir of malarial parasites was present. It seems likely that avian malaria did not reach epizootic proportions in Hawaii until after the 1900's, following the numerous releases of introduced birds, particularly those from Asia. Although early release records are extremely fragmentary, Caum (1933) listed 93 exotic birds that had been released. Bryan (1958) included 95 species of introduced or escaped cage birds in his checklist, and Walker (1967) noted 78 kinds of potential game birds released in Ha-

waii. Most introduced bird species of Asian origin that successfully established populations in the native forests after release were liberated during the early 1900's (Berger 1981). It is also interesting to note that the type host of *P. r. capistranae*, the Painted Quail (*Coturnix chinensis*), was first introduced into Hawaii from the Orient in 1910 (Laird and van Riper 1981), although this bird never became established in the islands. Certainly by 1920 a large enough pool of infected introduced avian hosts was present in Hawaii to begin the spread of malaria to native bird species.

Role of malaria in native bird declines

The pattern of historical native Hawaiian bird decline is bimodal. This supports our hypothesis of a late malaria introduction to the archipelago (Table 7). The initial reduction of native birds occurred in the mid- and late 1800's. It was unlikely to have been the result of malaria, because many of the species that were dying off during this period (e.g., the finch-billed drepanids from Kona) were historically confined to elevations well above 600 m. The second extinction period started in the early 1900's, and was most likely the result of the newly introduced *P. r. capistranae* parasite. Birds that succumbed during this period were principally species that were found in the mid-elevational forests, where we found the highest prevalences of avian malaria.

In concert with the second phase of extinction was a continued range reduction in many extant bird populations. This too correlates with the later introduction of malaria. Munro (1944) spent many years on Lanai and documented the status of native birds on that island. In 1923 he wrote that, if anything, he saw the native birds increasing, but by 1932, they were again declining in numbers. The decline of the Ou (*Psittirostra psittacea*) throughout the islands is characteristic of species that were greatly impacted by avian malaria. For example, Bryan (1908) found the bird quite abundant on Molokai. However, Richardson (1949) failed to find the Ou in 1948. It has not been observed on the island since (Pratt 1973, Scott et al. 1977). Donn Carlsmith (*personal communication*) observed the Ou in the Puna district of Hawaii island (elevation 900 m) with regularity in the 1930's. By 1940 the species was no longer present in those forests. Baldwin found Ou commonly in Hawaii Volcano National Park in 1936 and 1938–1940 (Richards and Baldwin 1953), but it became much less common thereafter (van Riper 1978, Conant 1981).

Baldwin (1941, 1953), the first ornithologist to conduct systematic censuses of the Hawaiian avifauna, found Creeper, Akepa (*Loxops coccineus*), Iiwi, Apapane, Common Amakihi, and Elepaio from 600–900 m elevation along the Hilina Pali Road in Hawaii Volcanoes National Park. Conant (1975) recently censused Baldwin's original study plots and found that the

TABLE 7. Presumed extinction dates of native land birds from the main Hawaiian Islands (from Bryan 1908, Munro 1944, Bryan 1958, Greenway 1958, and Berger 1981).

Species	Pre-malaria extinction period	Post-malaria extinction period
Rallinae		
Hawaiian Rail (<i>Porzana sandwicensis</i>)	c. 1885	
Turdinae		
Oahu Thrush (<i>Phaeornis obscurus oahensis</i>)	c. 1825	
Lanai Thrush (<i>P. o. lanaiensis</i>)		c. 1931
Meliphagidae		
Oahu Oo (<i>Moho apicalis</i>)	c. 1840	
Bishop's (Molokai) Oo (<i>M. bishopi</i>)	c. 1905	
Hawaii Oo (<i>M. nobilis</i>)		c. 1935
Kioea (<i>Chaetoptila angustipluma</i>)	c. 1860	
Drepanidinae		
Greater Amakihi (<i>Hemignathus sagittirostris</i>)	c. 1900	
Lanai Creeper (<i>Paroreomyza montana newtoni</i>)		c. 1935
Oahu Akepa (<i>Loxops coccineus rufa</i>)	c. 1900	
Oahu Akiāloa (<i>Hemignathus obscurus ellisianus</i>)		c. 1935
Lanai Akiāloa (<i>H. o. lanaiensis</i>)	c. 1900	
Hawaii Akiāloa (<i>H. o. obscurus</i>)		c. 1935
Oahu Nukupuu (<i>H. lucidus affinis</i>)	c. 1875	
Oahu Ou (<i>Psittirostra psittacea</i>)	c. 1900	
Lanai Ou		c. 1930
Molokai Ou		c. 1920
Maui Ou	c. 1900	
Greater Koa Finch (<i>Rhodacanthis palmeri</i>)	c. 1895	
Lesser Koa Finch (<i>R. flaviceps</i>)	c. 1895	
Grosbeak Finch (<i>Chloridops kona</i>)	c. 1895	
Molokai Crested Honeycreeper (<i>Palmeria dolei</i>)		c. 1915
Ula-ai-hawane (<i>Ciridops anna</i>)	c. 1895	
Hawaii Mamo (<i>Drepanis pacifica</i>)	c. 1895	
Black Mamo (<i>D. funerea</i>)		c. 1915

Creeper, Akepa, and Iiwi had disappeared. Our mist-net data support these findings. Furthermore, the native bird species that presently remain between 600 and 900 m elevation have undergone sizeable reductions in population numbers. This pattern of bimodal native bird extinction and decline appears to be consistent with the hypothesis of a later malaria arrival date.

Malaria has had and is presently having a significant negative impact upon the native Hawaiian avifauna. The extinction of many species during the second and third decades of the 1900's, and subsequent range reductions of other species, is correlated temporally with the presumed introduction date of *P. r. capistranae*. However, we must look elsewhere to explain the reduction of the Hawaiian avifauna prior to 1910. We

are only now appreciating the impact that Polynesians had upon the native birds prior to European settlement of the Hawaiian Islands (Olson and James 1982a, b). The introduction of *Rattus rattus* and *R. norvegicus* following the arrival of Europeans no doubt was also a major factor that contributed to this early decline 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, mongoose, and feral cat (Tomich 1969, van Riper and van Riper 1982). If disease did play a role in the initial decline of birds, a logical explanation would be a virus, such as Avian Pox. Early collectors noted lesions similar to those caused by Avian Pox virus on the native birds (Wilson and Evans 1890–1899, Rothschild 1893–1900, Perkins 1903, Munro 1944). With the annual altitudinal migrations and flocking behavior of many birds, this particular pathogen could have spread rapidly throughout the forests. We are, therefore, left with a complex picture for the demise of so many native Hawaiian birds. Avian malaria, although probably introduced during recent times, has been only one of many reasons for the decline of this unique avifauna. However, malaria is currently one of the major factors affecting avian population patterns in the Hawaiian Islands.

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