The Auk 117(3):731–738, 2000

EFFECTS OF MALARIA ON ACTIVITY BUDGETS OF EXPERIMENTALLY INFECTED JUVENILE APAPANE (*HIMATIONE SANGUINEA*)

NANCY YORINKS[1,3] AND CARTER T. ATKINSON[2,4]

[1]*Department of Zoology, University of Wisconsin, Madison, Wisconsin 53706, USA; and*
[2]*United States Geological Survey, Pacific Island Ecosystems Research Center, P.O. Box 218, Hawaii National Park, Hawaii 96718, USA*

ABSTRACT.—We used behavioral, physiological, and parasitological measures to document effects of acute malarial infections on activity budgets of experimentally infected juvenile Apapane (*Himatione sanguinea*). Five of eight birds died within 20 to 32 days after exposure to a single infective mosquito bite. Infected Apapane devoted less time to locomotory activities involving flight, walking or hopping, and stationary activities such as singing, preening, feeding, and probing. The amount of time spent sitting was positively correlated with parasitemia and increased dramatically after infection and between treatment and control groups. Birds that succumbed to infection experienced a significant loss of body mass and subcutaneous fat, whereas surviving Apapane were better able to maintain body condition and fat levels. When rechallenged with the parasite five months after initial infection, surviving birds experienced no increase in parasitemia, indicating that they had become immune to reinfection. Regardless of the outcome, infected birds experienced acute illness that would have left them unable to forage or to escape from predators in the wild. *Received 8 March 1999, accepted 11 January 2000.*

NATIVE HAWAIIAN forest birds were first exposed to vector-borne parasitic diseases after the accidental introduction of mosquitoes in 1827 and the subsequent importation of avian pox virus and malaria (*Plasmodium relictum*) in cage birds and domestic fowl (Zimmerman 1948, Warner 1968). Warner (1968) provided the first detailed evidence that diseases from vector-borne pathogens were influencing native forest birds by exposing Laysan Finches (*Telespiza cantans*) from a disease-free population on Laysan Island to mosquitoes at low elevations on the island of Kauai. All caged individuals died from fulminating pox and malarial infections within 21 days of initial exposure, suggesting that high vector populations and a reservoir of disease in non-native birds maintained active cycles of disease transmission at these elevations.

Studies conducted since then have shown that pox and malaria, in conjunction with habitat destruction and the introduction of mammalian predators and avian competitors, have caused dramatic changes in the distribution and abundance of highly susceptible native forest birds (Scott et al. 1986, van Riper et al. 1986, Atkinson et al. 1995). Most extant populations of Hawaiian honeycreepers are confined to shrinking tracts of native forest at elevations above 1,200 m, where cooler temperatures begin to limit populations of the primary vector of pox and malaria, *Culex quinquefasciatus* (Goff and van Riper 1980, van Riper et al. 1986).

Recent surveys of native Hawaiian birds have shown that populations of a few species are able to survive in mid- and low-elevation habitats below 1,200 m, where mosquitoes are seasonally common and malaria and pox transmission have become endemic, suggesting that the birds have developed resistance to lethal effects of the diseases (Scott et al. 1986, van Riper et al. 1986, Jarvi et al. 2000). On the island of Hawaii, Apapane (*Himatione sanguinea*) and Hawaii Amakihi (*Hemignathus virens*) are the only honeycreepers still commonly found in mid- and low-elevation habitats. These species, as well as Omao (*Myadestes obscurus*) and Elepaio (*Chasiempis sandwichensis*), have become primary reservoirs for malaria because of their high susceptibility to infection (van Riper et al. 1986, Atkinson et al. 2000). Based on blood smears, the prevalence of infection in these na-

[3] Present address: 1208 Tamarack Drive, Moscow, Idaho 83843, USA.

[4] Address correspondence to this author. E-mail: carter_atkinson@usgs.gov

tive species ranges as high as 40% at elevations between 900 and 1,500 m, with most infected individuals carrying chronic, low-intensity infections characteristic of birds that have recovered from acute malaria and have some degree of resistance to reinfection.

The effect of disease on juvenile birds is of particular interest because a change in their behavior potentially could translate into decreased fitness as adults. Parasitemias in hatching-year Hawaiian honeycreepers are up to six times higher than those in adults, suggesting the younger birds have less resistance to infection and are the age class that is hardest hit during malarial epidemics (van Riper et al. 1986). Findings from other studies have been similar. For example, infections of *Haemoproteus prognei* in Purple Martins (*Progne subis*) are believed to be more virulent in young birds (Davidar and Morton 1993). Sublethal infections with *Trichinella pseudospiralis* in American Kestrels (*Falco sparverius*) influenced juvenile fitness through altered incubation behavior and parental care by infected adults, and through altered care of infected nestlings by uninfected parents (Saumier et al. 1991). In this study, we used behavioral, physiological, and parasitological measures to quantify the effects of acute malarial infections on morbidity and mortality in juvenile Apapane under controlled experimental conditions.

METHODS

We caught 16 hatching-year Apapane in mist nets in ohia (*Metrosideros polymorpha*)/koa (*Acacia koa*) forests at 1,800 m in the Upper Waiakea Forest Reserve on the eastern slopes of Mauna Loa in March and April 1993. Birds were captured at three sites that were approximately 3 km apart. This area is relatively free of *Culex* mosquitoes, and the prevalence of malarial infection in forest birds is less than 5% (C. Atkinson unpubl. data). All 16 birds were in juvenal plumage and had not started their first prebasic molt (Fancy et al. 1993).

Birds were transferred to a mosquito-proof aviary at Hawaii Volcanoes National Park (elevation 1,200 m) and allowed to acclimate under conditions of ambient temperature and light for one to four weeks before use in experiments. This was sufficient time for mass and food consumption to stabilize. During the acclimatization period, birds were housed individually in cages measuring 1 × 0.5 × 0.5 m and fed a diet of artificial nectar (Nector Plus, Necton USA, Tarpon Springs, Florida) and fresh oranges and papaya. Birds were color banded and bled from the jugular vein with a 27.5-gauge syringe within two to three days of being moved to captivity. A blood smear was prepared, fixed in absolute methanol, stained with 2% buffered Giemsa (pH 7.0), for 1 h, and examined for 10 min with a 400× objective to diagnose patent malarial infections. Heparinized whole blood was centrifuged in microhematocrit tubes and plasma was collected and screened for antibodies to *P. relictum* with an immunoblot technique (Atkinson et al. 1995) to confirm that the birds had no prior history of exposure to malaria.

The behavioral experiment was conducted in a mosquito-proof flight cage (5.5 × 2.4 × 3.6 m) that contained various types of local vegetation. Birds were fed Nector Plus in eight 40-mL feeders, and eight other feeders containing water were placed in the room. Locations of the 16 feeders were changed each morning to mimic the transitory conditions of natural food sources. A fine mist of water vapor was spayed into the flight cage for 15 to 30 min each morning to simulate rain forest conditions. Feeding and misting ended at least 2 h before behavioral observations.

Eight Apapane were assigned randomly to the experimental group and eight to the control group. Experimental birds were infected by the bite from a single *Plasmodium relictum*-infected mosquito using colonized *Culex quinquefasciatus* (see Atkinson et al. 1995). Control birds were bitten by a single uninfected mosquito. Morphological measurements and behavioral observations were taken before and after infection.

No more than eight birds were present in the flight cage at one time, and equal numbers of infected and control birds were present at the beginning of each of two successive trials. Because densities of Apapane in the wild exceed 3,000 birds/km^2 (Ralph and Fancy 1995), density in the flight cage was comparable to natural conditions. Behavioral observations were not taken for the first three to four days after birds were moved to the flight cage to allow them to habituate to their surroundings. This was sufficient time for the birds to locate food sources in the aviary and exhibit behaviors comparable to what we have observed in the wild. After this acclimation period, observations were taken for four to eight days (preinfection period). Birds were then infected and subsequently observed for the next 24 days. The first four days were the "pre-patent" period of infection where the parasite was undergoing development in fixed tissues in the host and had not reached detectable concentrations in blood. The next 20 days were taken during the "patent" period when intraerythrocytic parasites could be found on blood films of peripheral blood.

Behavior was quantified with a laptop computer through a program that assigned different keys on the keyboard to specific behaviors. The program kept track of the time between keystrokes so that the total

time spent on each behavior could be determined at the end of an observation period. Observations took place through a one-way mirror at one end of the flight cage. Observed behaviors were defined as either locomotory (flying, flapping, hopping, walking) or stationary (preening, probing, singing, feeding, scratching, sitting, miscellaneous). Each bird was observed for 10 to 20 min per day for an average 7.7 h per bird. All birds were observed for approximately the same amount of time per day, and the order of observation was determined randomly. Observations were conducted between 0900 and 1700 Hawaii Standard Time.

Every four days, Apapane were captured in the flight cage with a small mist net. We weighed captured birds to the nearest 0.1 g and estimated the amount of furcular fat on a four-point scale (1 = no visible fat, 4 = fat bulging beyond the plane of the chest). In addition, smears were made from a drop of blood collected from the brachial vein to follow the progress of malarial infection in infected birds and to confirm absence of infection in control birds. We also measured tarsus, wing, and culmen length every eight days. Morphological measurements and blood samples were taken either after observations were finished, or at least 2 h prior to the beginning of observations in the morning. Parasitemia was quantified as the number of infected erythrocytes per 1,000 erythrocytes on Giemsa-stained blood smears (see Godfrey et al. 1987).

We averaged the proportion of time spent on each behavior over all observation periods for each bird within four-day blocks. Results from the two trials were similar, so data from all 16 birds were combined for analysis. Each block had corresponding measurements of growth and parasitemia because these parameters were obtained every four days. The postinfection period consisted of five time blocks in the 20 days after the pre-patent period. Because pathogenic effects of *P. relictum* are associated with erythrocytic stages of infection (Atkinson et al. 1995), the four days before infection and the pre-patent block were combined as the preinfection period.

Because we were concerned primarily with effects of malarial infection on behavior of these birds, the eight infected birds were considered the infected group, regardless of their later survival status, and were compared with the eight uninfected control birds. We also divided the infected group into two subgroups: (1) birds that survived to be experimentally reinfected five months later, and (2) birds that died toward the end of the experiment.

Behavioral and morphological measurements were analyzed for the effects of time and infection group. Because time of day and weather were distributed similarly among observation periods, we assumed that any effects they may have had on behavior also were uniform across experimental treatments. Thus, we did not include time and weather as independent variables in tests of treatment effects.

Differences in mortality between infection groups were examined with a Fisher's exact test. We analyzed behavioral and morphological data by repeated-measures ANOVA (PROC GLM; SAS 1985) using time block as the repeated unit and individual birds as the experimental units. The repeated-measures design allows detection of a difference in behavior between infection groups over time. We then used a contrast analysis to examine effects within groups more carefully. Characteristics before infection were compared with those after infection within and between infection groups. Pre- and postinfection characteristics also were compared between infection groups.

All birds that died during the experiment were necropsied to determine cause of death. Representative pieces of major organs were fixed in 10% buffered formalin, embedded in paraffin, sectioned, and stained with hematoxylin and eosin. Birds that recovered from acute malarial infection were rechallenged with the same isolate of *P. relictum* five months postinfection to assess immunity to reinfection. Three uninfected Apapane also were exposed to single infective mosquito bites at the same time to act as positive controls.

Results

All eight infected Apapane developed patent erythrocytic infections within eight days postinfection, whereas all control birds remained uninfected throughout the duration of the experiment. Mean parasitemia in three of the infected individuals peaked at 10% 12 days postinfection and subsequently declined (Fig. 1). The remaining five infected birds succumbed to acute anemia during the course of the experiment, with mean parasitemias as high as 28% before death (Fig. 1). Three of these birds survived the duration of the five time blocks of postinfection behavioral observations, whereas two (25%) died during the fourth postinfection block (Fig. 1). None of the uninfected control birds died during the experiment (Fisher's exact test, $P = 0.026$).

Gross and microscopic lesions in the five fatalities were characteristic of severe malaria. All five individuals were emaciated at necropsy and had enlarged and blackened livers, spleens, and kidneys from extensive deposition of malarial pigment. Microscopic changes were characteristic of severe acute anemia described in other species of honeycreepers with malarial infections, including diffuse extramedullary

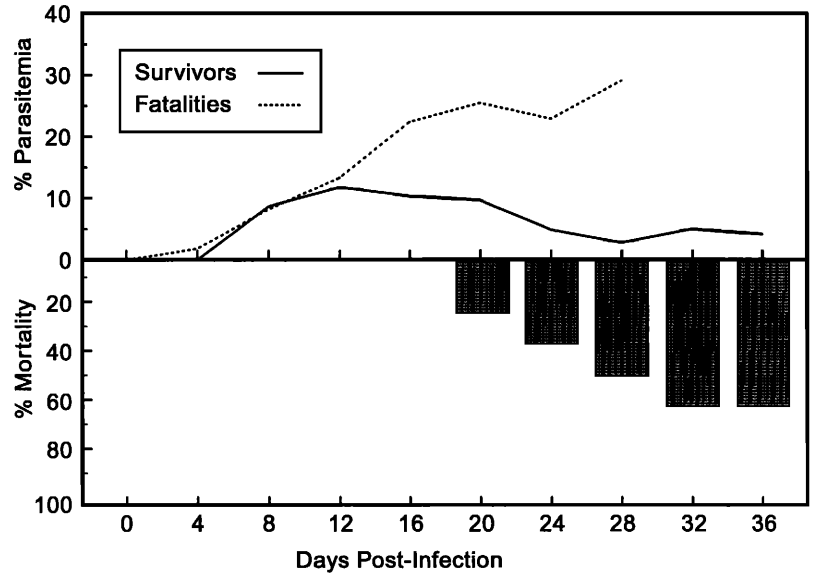

FIG. 1. Mortality and mean parasitemia for surviving (n = 3) and dying (n = 5) Apapane after exposure to a single infective mosquito bite.

erythropoiesis in the hepatic sinusoids and occasional areas of extramedullary erythropoiesis in the hepatic parenchyma (Atkinson et al. 1995).

Before exposure to mosquitoes, probing, sitting, and preening were the predominant behaviors among birds in the control and infection groups. Locomotory behaviors were infrequent, so they were analyzed together as the category "locomotion." The behaviors scratching, stretching, and "miscellaneous" also were infrequent and were dropped from the analysis.

Because two birds died before the fifth time block, we used type III sums of squares so that these birds could be included in the repeated-measures ANOVA despite the missing cells. A significant interaction ($P < 0.05$) occurred between time and experimental group for sitting, preening, probing, and singing (Table 1). Preening activity increased for control birds and decreased for infected birds. Probing and singing activity decreased for infected birds, and the amount of time spent sitting increased (Fig. 2A). None of these three behaviors changed significantly for control birds. Although neither locomotion nor feeding showed significant treatment × time interactions, they were significantly different between treatment groups, and locomotion also was significantly different over time (Table 1, Figs. 2B,C).

We then analyzed behavioral data by contrasts. We found no differences in behavior between infected and control groups before infection, but after infection all behaviors differed significantly between treatment groups ($P < 0.05$). Among infected birds, all behaviors changed significantly after infection ($P < 0.05$). Among control birds, probing and preening declined after exposure to uninfected mosquitoes and were the only behaviors to change significantly ($P < 0.05$).

When infected birds were subdivided into survivors and mortalities, a significant treatment × time interaction occurred for mass ($P < 0.01$), sitting ($P < 0.01$), and preening ($P < 0.05$). When the three surviving birds were compared with the eight control birds by contrast analysis, no characteristics were significantly different between groups before infection, whereas all variables except preening and mass differed after infection ($P < 0.05$). Although the size of the surviving group was small, the results concur with those obtained when all eight infected birds were considered together (Table 1). A positive relationship occurred between parasitemia and sitting, and negative relationships occurred between para-

TABLE 1. Results of repeated-measures ANOVA for differences in behavior and mass of Apapane by time and experimental group.

Characteristic	Treatment[a]		Time[b]		Treatment × time[c]	
	F	*P*	*F*	*P*	*F*	*P*
Sitting	49.03	0.0001	16.04	0.0001	11.03	0.0001
Locomotion	21.52	0.0004	6.42	0.0001	1.24	0.294
Preening	0.36	0.558	0.67	0.672	4.28	0.001
Probing	14.25	0.002	9.35	0.0001	2.65	0.021
Singing	2.71	0.122	2.91	0.013	2.79	0.016
Feeding	4.94	0.043	1.91	0.088	1.40	0.224
Mass	1.39	0.258	8.92	0.0001	13.27	0.0001

[a] df = 1 and 14.
[b] df = 6 and 84.
[c] df = 6 and 84.

sitemia and all other behaviors (Table 2). All of the behavioral correlations with parasitemia (except feeding) were significant ($P < 0.05$; Table 2).

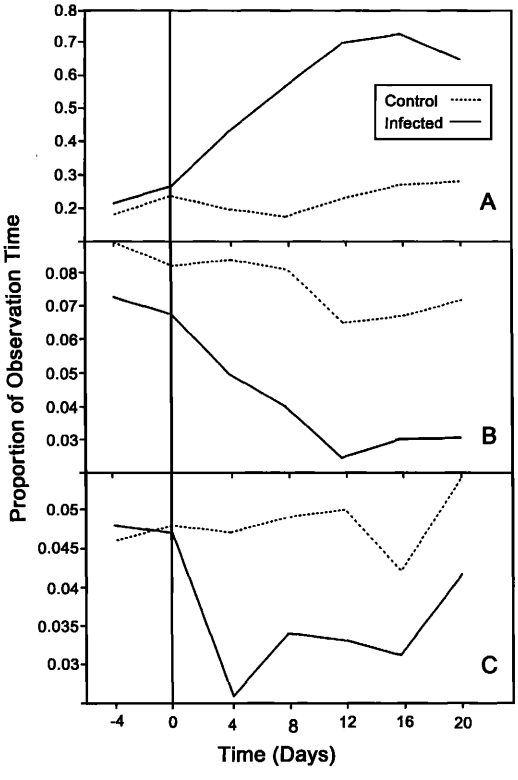

FIG. 2. Behavioral effects of malarial infection on eight control and eight experimentally infected Apapane during five four-day time blocks between exposure to mosquitoes at day 0 and termination of experiment at day 20. (A) Proportion of time sitting still; (B) proportion of time engaged in locomotory activities; and (C) proportion of time spent feeding.

Each four-day time block had corresponding measurements of body mass, tarsus, wing, culmen, and fat. None of the birds grew during the experiment, so tarsus, wing, and culmen lengths were not included in the analysis. Mass was analyzed by the same repeated-measures method used for the behavioral data and showed a significant interaction between time and infection group (Table 1, Fig. 3A). Following contrast analysis, mass differed significantly between treatment groups after infection and within the infected group before and after infection ($P < 0.05$). Fat scores declined significantly in infected birds relative to controls (Mann-Whitney $U = 57.5$, $n_1 = 8$, $n_2 = 8$, $P < 0.05$; Fig. 3B).

Of the three birds that recovered from acute malarial infection, one died at day 155 postinfection from an impacted gizzard that was not related to malarial infection. Parasitemias in the two survivors remained below 2% when they were rechallenged with the same isolate of *P. relictum* on days 142 and 171 postinfection. One of the two rechallenged birds experienced a relapse at day 200 postinfection and died from acute anemia complicated by a partially

TABLE 2. Pearson correlation coefficients between parasitemia and proportion of time devoted to each behavior by infected Apapane.[a]

Characteristic	Correlation	Bonferroni *P*
Sitting	0.720	<0.001
Locomotion	−0.531	0.001
Preening	−0.449	0.016
Probing	−0.514	0.002
Singing	−0.422	0.034
Feeding	−0.139	1.000

[a] Overall test of significance, Bartlett $\chi^2 = 495.5$, df = 21, $P < 0.0001$.

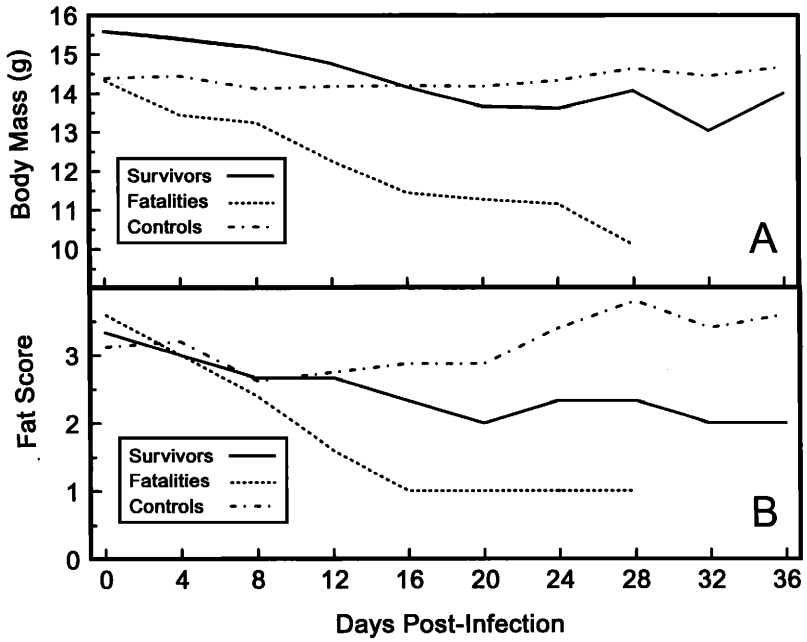

FIG. 3. Effect of malarial infection on (A) mean body mass and (B) mean fat score for eight uninfected control Apapane, three infected survivors, and five mortalities. Measurements were taken at four-day intervals beginning at day 0.

impacted gizzard and poor body condition. Parasitemia at death was 18%. The second rechallenged bird survived until day 605 postinfection, when it died from accidental causes during blood sampling. All three positive control birds developed acute infections and succumbed within 14 days postinfection with parasitemias as high as 18% at death. Mean body mass of these three birds at death was 8.55 ± SD of 0.49 g, which was slightly lower than the mean mass of the five original fatalities (Fig. 3A).

DISCUSSION

We assessed the effects of acute malarial infections on juvenile Apapane under controlled experimental conditions with a combination of behavioral, physiological and parasitological measures. We observed significant declines in activity levels of infected birds, with all birds becoming moribund during the period when parasitemia reached a peak. Infected birds underwent an acute illness with significant loss of body mass and fat over its duration. Loss of body mass and declines in activity were correlated with parasitemia, indicating that sublethal effects of the disease are closely linked to level of resistance in a particular individual.

In general, the behavioral changes we detected suggest an overall decrease in activity among infected birds. Particularly significant was the negative influence of acute infections on surviving birds. All individuals passed through a period of severe physiological stress where they were almost totally inactive. In the wild, they would have been extremely vulnerable to predation.

Apapane feed primarily on nectar of ohia flowers and may conduct elevational migrations in search of ohia blooms (MacMillen and Carpenter 1980, Ralph and Fancy 1995). Uninfected birds may follow periodic blooms of ohia trees to lower elevations, become exposed to malaria, and then migrate upslope during the pre-patent period of the disease before behavioral effects of the infection have been manifested. Once the infection becomes patent, decreased mobility and feeding effort (Figs. 2A–C) would place acutely ill individuals at a competitive disadvantage with Iiwi (*Vestiaria coccinea*) and other native nectarivores that still maintain dense populations in high-elevation montane forests (Carpenter and MacMillan

1976). Acute illness also would leave infected Apapane more susceptible to thermal stresses (Hayworth et al. 1987).

Factors that determine whether a bird will survive infection are poorly understood. Such factors may be related to genetics of the host and parasite and to overall physiological condition of the host (Gustafsson et al. 1994, Sorci et al. 1997). It is interesting that body mass of surviving Apapane did not differ significantly from that of control birds, suggesting that stored fat reserves and overall body condition are critical for surviving the acute phases of the infection when foraging behavior declines sharply. Atkinson et al. (1995) found a similar relationship in experimentally infected Iiwi, where preinfection body mass was a good predictor of survival time.

Recovery from acute malarial infection may benefit an individual by conferring acquired resistance to reinfection with homologous isolates of the parasite. This phenomenon, termed concomitant immunity or premunition, was first described in avian host-parasite models more than 70 years ago (Taliaferro and Taliaferro 1929) and is based on persistence of a subclinical infection that stimulates strain-specific humoral and cell-mediated immunity. It is likely that these chronically infected individuals form the core breeding population in low- and mid-elevation habitats where malaria transmission has become endemic. It is not clear, however, whether chronic infections are sufficiently virulent to influence host fitness, although recent studies suggest that a clear physiological tradeoff exists between host defense against parasites and the stresses associated with reproduction (Norris et al. 1994, Oppliger et al. 1996, Allander 1997).

We provided additional experimental evidence documenting the pathogenicity of avian malaria in Hawaiian honeycreepers. These findings support the idea that this introduced disease continues to have profound effects on native forest birds at lower elevations (Warner 1968, van Riper et al. 1986, Atkinson et al. 1995). Efforts to restore populations of native forest birds in mid- and low-elevation habitats will continue to be hampered unless effective techniques for managing vector populations and interrupting transmission of this introduced disease can be developed.

ACKNOWLEDGMENTS

We thank Michael Samuel and Dong Xiang for assistance with the statistical analysis and Louis Sileo, Robert Dusek, William Iko, Julie Lease, Christine Gaasch, and Bonnie Lewis for help in capturing and caring for Apapane used in the study. John Coleman, Department of Wildlife Ecology, University of Wisconsin-Madison, kindly provided the computer program to quantify behavioral observations. Bethany Woodworth and Donald Forrester provided helpful comments on earlier drafts of the manuscript.

LITERATURE CITED

ALLANDER, K. 1997. Reproductive investment and parasite susceptibility in the Great Tit. Functional Ecology 11:358–364.

ATKINSON, C. T., R. J. DUSEK, K. L. WOODS, AND W. M. IKO. 2000. Pathogenicity of avian malaria in experimentally infected Hawaii Amakihi. Journal of Wildlife Diseases 36:197–204.

ATKINSON, C. T., K. L. WOODS, R. J. DUSEK, L. S. SILEO, AND W. M. IKO. 1995. Wildlife disease and conservation in Hawaii: Pathogenicity of avian malaria (*Plasmodium relictum*) in experimentally infected Iiwi (*Vestiaria coccinea*). Parasitology 111:S59–S69.

CARPENTER, F. L., AND R. E. MACMILLEN. 1976. Threshold model of feeding territoriality and test with a Hawaiian honeycreeper. Science 194: 639–642.

DAVIDAR, P., AND E. S. MORTON. 1993. Living with parasites: Prevalence of a blood parasite and its effect on survivorship in the Purple Martin. Auk 110:109–116.

FANCY, S. G., T. K. PRATT, G. D. LINDSEY, C. K. HARADA, A. H. PARENT, Jr., AND J. D. JACOBI. 1993. Identifying sex and age of Apapane and Iiwi on Hawaii. Journal of Field Ornithology 64:262–269.

GODFREY, R. D., Jr., A. M. FEDYNICH, AND D. B. PENCE. 1987. Quantification of hematozoa in blood smears. Journal of Wildlife Diseases 23: 558–565.

GOFF, M., AND C. VAN RIPER III. 1980. Distribution of mosquitoes (Diptera: Culicidae) on the east flank of Mauna Loa Volcano, Hawaii. Pacific Insects 22:178–188.

GUSTAFSSON, L., D. NORDLING, M. S. ANDERSSON, B. C. SHELDON, AND A. QVARNSTRÖM. 1994. Infectious diseases, reproductive effort and the cost of reproduction in birds. Philosophical Transactions of the Royal Society of London Series B 346:323–331.

HAYWORTH, A. M., C. VAN RIPER III, AND W. W. WEATHERS. 1987. Effects of *Plasmodium relictum* on the metabolic rate and body temperature in

Canaries (*Serinus canarius*). Journal of Parasitology 73:850–853.

JARVI, S. I., C. T. ATKINSON, AND R. C. FLEISCHER. 2000. Immunogenetics and resistance to avian malaria (*Plasmodium relictum*) in Hawaiian honeycreepers (Drepanidinae). In press *in* Conservation and ecology of Hawaiian birds (J. M. Scott, S. Conant, and C. van Riper III, Eds.). Studies in Avian Biology.

MACMILLEN, R. E., AND F. L. CARPENTER. 1980. Evening roosting flights of the honeycreepers *Himatione sanguinea* and *Vestiaria coccinea* on Hawaii. Auk 97:28–37.

NORRIS, K., M. ANWAR, AND A. F. READ. 1994. Reproductive effort influences the prevalence of haematozoan parasites in Great Tits. Journal of Animal Ecology 63:601–610.

OPPLIGER, A., P. CHRISTE, AND H. RICHNER. 1996. Clutch size and malaria resistance. Nature 381: 565.

RALPH, C. J., AND S. G. FANCY. 1995. Demography and movements of Apapane and Iiwi in Hawaii. Condor 97:729–742.

SAS INSTITUTE. 1985. SAS user's guide: Statistics, version 5. SAS Institute, Inc., Cary, North Carolina.

SAUMIER, M. D., M. E. RAU, AND D. M. BIRD. 1991. Behavioral changes in breeding American Kestrels infected with *Trichinella pseudospiralis*. Pages 290–313 *in* Bird-parasite interactions: Ecology, evolution, and behaviour (J. E. Loye and M. Zuk, Eds.). Oxford University Press, Oxford.

SCOTT, J. M., S. MOUNTAINSPRING, F. L. RAMSY, AND C. B. KEPLER. 1986. Forest bird communities of the Hawaiian Islands: Their dynamics, ecology and conservation. Studies in Avian Biology No. 9.

SORCI, G., A. P. MØLLER, AND T. BOULINIER. 1997. Genetics of host-parasite interactions. Trends in Ecology and Evolution 12:196–200.

TALIAFERRO, W. H., AND L. G. TALIAFERRO. 1929. Acquired immunity in avian malaria I. Immunity to superinfection. Journal of Preventive Medicine 3:198–208.

VAN RIPER III, C., S. G. VAN RIPER, M. L. GOFF, AND M. LAIRD. 1986. The epizootiology and ecological significance of malaria in Hawaiian land birds. Ecological Monographs 56:327–344.

WARNER, R. E. 1968. The role of introduced diseases in the extinction of the endemic Hawaiian avifauna. Condor 70:101–120.

ZIMMERMAN, E. C. 1948. Insects of Hawaii, vol. 1. University of Hawaii Press, Honolulu.

Associate Editor: E. Danchin