Facilitating the evolution of resistance to avian malaria in Hawaiian birds

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A B S T R A C T

Research has shown that avian malaria plays an important role in limiting the distribution and population sizes of many Hawaiian birds, and that projected climate change is likely to eliminate most disease-free habitat in Hawai'i in the next century. I used a modeling approach, parameterized with demographic data from the literature and the field, to examine alternate management scenarios for the conservation of native Hawaiian birds. I examined the feasibility of using management in the form of rodent control to facilitate the evolution of resistance to malaria by increasing the survival and reproduction of native birds. Analysis of demographic data from seven native species, Akepa (*Loxops coccineus*), 'Akohekohe (*Palmeria dolei*), Elepaio (*Chasiempis sandwichensis*), Hawai'i'amakihi (*Hemignathus virens*), Hawai'i creeper (*Oreomystis mana*), Omao (*Myadestes obscurus*), and Palila (*Loxioides bailleui*), suggest that differences in life history cause some species to be more susceptible to local extinctions from the transmission of malaria. Modeling results demonstrated that rodent control at middle, but not high, elevations can facilitate the evolution of resistance to malaria in several species of Hawaiian birds. Advocating a management approach that encourages evolutionary change in endangered species contrasts with the traditional conservation paradigm but it may be the best strategy to reduce the impacts of one of the multiple stressors that have devastated the native bird community of Hawai'i.

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1.   Introduction

Many endangered species are threatened by novel stressors: prairie dogs (*Cynomys* spp.) by plague (*Yersinia pestis*) (Biggins and Kosoy, 2001); Kirtland's warbler (*Dendroica kirtlandii*) by Brown-headed cowbirds (*Molothrus ater*) (Probst, 1986); and plants by unprecedented browsing pressure from white tailed deer (*Odocoileus virginianus*) (Rooney and Gross, 2003; Rooney and Waller, 2003, Knight, T., Holt, R.D., Barfield, M., unpublished data). In some cases it is the combination of multiple

stressors that lead to the decline of species or groups of species; e.g., amphibians by nematodes (Johnson et al., 1999), increased UV radiation and pollutants (Hatch and Blaustein, 2003; Beebee and Griffiths, 2005), and disease (Collins and Storfer, 2003; Daszak et al., 2003). Conserving endangered species and facilitating their recovery is one the main environmental challenges of the 21st century. Unfortunately, history has shown that in most cases permanent removal of stressors has not been possible and species often need to be managed indefinitely to ensure their persistence; of the

1263 species listed under the Endangered Species Act of the United States less than 2% have been de-listed (US Fish and Wildlfe Service, 2004).

One of the most threatened group of animals in the United States is native Hawaiian birds. At present 53 of Hawai'i's 71 endemic avian taxa are either extinct or endangered (Jacobi and Atkinson, 1995). The three major stressors causing the decline of Hawaiian birds are habitat loss, predation by 2introduced mammals, and disease (Atkinson, 1977; Scott et al., 1986, 2002; Pratt, 1994; Jacobi and Atkinson, 1995). Currently disease plays a major role in limiting the distribution and population size of a large number of species (Scott et al., 1986; van Riper et al., 1986; Jenkins et al., 1989). There are large areas of apparently suitable habitat for many endangered species that are unoccupied, presumably due to the presence of mosquitoes and disease (Scott et al., 1986) and disease may be causing the continued decline that has been observed in several species of Hawaiian birds (Scott et al., 2002; Foster et al., 2004). The critical state of several endangered species suggests that urgent management action is required to prevent additional extinctions.

Suggestions for management actions to preserve the remaining species include habitat conservation and restoration (including exotic species removal), predator control, and captive propagation (Scott et al., 2002; Groombridge et al., 2004). Current habitat conservation and restoration focuses on high elevation areas where mosquitoes are absent and where the most dense populations of most native birds are found (US Fish and Wildlife Service, 2003). However, recent work suggests that most of the remaining mosquito and disease-free forested area in Hawai'i will be eliminated by a 2 °C rise in global temperatures (Benning et al., 2002), corresponding to recent climate change projections. Consequently, management strategies that do not lead to the evolution of resistance to malaria may fail to preserve many of Hawai'i's native birds. Previous suggestions to address disease as a limiting factor have included reducing mosquito densities through larval habitat reduction and captive propagation of genetically resistant individuals (Cann and Douglas, 1999; Jarvi et al., 2001; US Fish and Wildlife Service, 2003).

In contrast, some researchers have advocated using rodent control to facilitate the evolution of resistance to malaria in the field (Vanderwerf and Smith, 2002). Decreasing predation through rodent control would increase the probability that resistant individuals would be able to reproduce and survive successfully. Rodents (especially rats, *Rattus* spp.) are a significant source of nest and adult mortality for many Hawaiian birds (Atkinson, 1977; Nelson et al., 2002; Vanderwerf and Smith, 2002). Several Hawaiian birds are known to remain on the nest despite the close presence of predators, and this has led to predation on incubating or brooding females and possibly to the skewed sex ratio seen in several species (Lindsey et al., 1995; Vanderwerf and Smith, 2002). As a result, reducing rodent populations, perhaps through aerial broadcast of rodenticides, should reduce predation pressure on native birds (Nelson et al., 2002). However, it is not clear how successful this approach would be for the diverse group of endangered species present in Hawai'i.

Rodent control will facilitate resistance evolution only if it allows populations of a species to persist in the face of the selective pressure from disease-caused mortality. Assessing the likelihood of success thus requires information on the ecology and demography (survival and reproduction) of each species under consideration. Facilitating resistance evolution on a feasible time scale also requires that genes for resistance currently exist in the population. I define individuals as resistant if they can survive the acute (∼30 days) period of malaria infection, because mortality from acute infection appears to be the most important impact of malaria on Hawaiian birds (Kilpatrick, 2003; Kilpatrick et al., 2006). Data suggest that resistant individuals exist in populations of many species that are not commonly found in areas with abundant mosquitoes and disease (van Riper et al., 1986; Atkinson et al., 2005; Woodworth et al., 2005; Kilpatrick et al., 2006). In addition, laboratory challenge experiments confirm that there is significant variability in the resistance to malaria between species (Warner, 1968; van Riper et al., 1986; Atkinson et al., 1995, 2000, 2001a; Yorinks and Atkinson, 2000; Jarvi et al., 2001). Although the genetic basis underlying resistance in Hawaiian birds is not well understood (Jarvi et al., 2001, 2004), research on mice and humans suggest that there are several individual loci that influence susceptibility to malaria (Swardson et al., 1997; Miller, 1999; Burt et al., 2002; Henri et al., 2002). Consequently, I present analyses based on the assumption that a single loci can confer resistance to avian malaria and allow individuals to survive the acute malaria infection.

I employed a modeling approach to address the efficacy of rodent control in facilitating the evolution of resistance to malaria. I attempted to determine whether management efforts could increase population growth rates above replacement in areas where malaria exists. This will help to determine whether it is better to focus management efforts at high elevations where disease is currently absent, or at middle elevations where selective pressure for malaria resistance exists. The results suggests that management of mid-elevation rodent populations may lead to long term persistence of several species' populations and the evolution of resistance to avian malaria.

2. Methods

2.1. Demographic parameters

To address the efficacy of rodent control in facilitating resistance, I parameterized models with demographic data for all species of native Hawaiian birds where estimates of survival and reproduction were available from the same site. I used a model which combined the length of the nesting cycle (nest building, egg laying, incubation period, nestling period, and inter-nest interval) with the length of the breeding season and daily survival rates to generate an estimate of the number of successful nests per season (Pease and Grzybowski, 1995). For species where Birds of North America accounts (Poole and Gill, 2005) were available (except Hawai'i'amakihi, *Hemignathus virens*), I estimated the length of the breeding season by adding the length of the nestling period to the time period when eggs were observed in nests for that species. For other species I used the time period described as the main or peak breeding season. For Hawai'i'amakihi, I used the length of the breeding season at the site where the data for repro-

duction and adult survival were collected (Hawai'i Volcanoes National Park; Kilpatrick et al., 2006). For all species, I multiplied the number of successful broods for each species by the average number of fledglings per successful nest and used this as an estimate of the number of hatch-year birds produced per season. This estimate may be higher than the actual fecundity of a species if the published length of the breeding season did not represent the average length of the breeding season for individuals in the study population.

I found suitable data for six passerine species in three families, Monarchidae: 'Elepaio (*Chasiempis sandwichensis*) (including data from two sites), Turdidae: 'Oma'o (*Myadestes obscurus*), Fringillidae: Palila (*Loxioides bailleui*), Hawai'i'amakihi, Akepa (*Loxops coccineus*), Hawai'i creeper (*Oreomystis mana*), and nearly complete data for one more species, the 'Akohekohe (*Palmeria dolei*) (Table 1). These data on reproduction and survival represent populations with little or no disease, except for Oahu 'Elepaio which occur in middle and low elevations where disease is prevalent (Vanderwerf et al., 2001, in press). It should be noted that the demographic data in Table 1 are estimates from a single location over a relatively short time span (2–7 years), and do not include estimates of spatial variation. As a result, the population growth rates for the studied populations may not always match the population trajectories for the species as a whole (e.g., Palila; US Fish and Wildlife Service, 2003). In addition, measured survival estimates may be biased low for Palila due to Palila removing color bands (Banko, P., personal communication). Nonetheless, I used these survival and reproduction data to estimate the per capita population growth rate using the following expression:

$$\lambda = S_A + FS_J,$$

where S_A is yearly adult survival, S_J is yearly juvenile survival and F is the per capita fecundity or number of females produced by each female, which is calculated as the product of the number of successful nests per year multiplied by the number of female offspring per successful nest. I estimated the standard error of the per capita population growth rate, λ, of each species using a Taylor-series expansion approximation. The variance in λ can be estimated as

$$\text{var}(\lambda) = \sum_{i=1}^{n} \frac{d\lambda}{dx_i} * \text{var}(x_i),$$

where x_i represents each variable in the expression for λ, and the derivatives are evaluated at the mean value for each x_i. I used published estimates, where available, for var(S_A) and var(S_J). Otherwise, I estimated the variance of the adult and juvenile survival using the expression for the variance of a binomial variable which has a value of 1 with probability p: $p(1 - p)/N$, where N is the number of trials (or birds used to estimate the survival). To estimate the variance of F, I used published estimates of the variance of the daily nest survival rates and drew from a normal distribution with the same variance. I used these draws in the seasonal fecundity model to generate an estimate of the variance in the number of successful nests per year. For species where only the nesting success

Table 1 – Demographic parameters for seven species (and two subspecies) of native Hawaiian birds

Species	Akepa	'Akohekohe	'Elepaio (HFNWR)	'Elepaio (Oahu)	Hawai'i'amakihi	Hawai'i creeper	Oma'o	Palila
Length of breeding season	63	141	66	76	107	140	154	179
DPR (incubation)[a]	0.007	0.011	0.014	0.0303	0.010	0.040	0.023	0.057
DPR (nestlings)[a]	0.007	0.011	0.014	0.0303	0.027	0.040	0.024	0.016
Nest building	7	11	13	13	11	15	11	14
Egg laying	2	2	2	2	2	2	1	2
Incubation period	15	17	18	18	14	13	16	17
Nestling period	18	22	16	16	17	20	19	25
DFSN[b]	30	7	30	30	5	23	36	30
DFUN[c]	2	2	2	2	2	7	2	2
% Nesting success	0.785	0.650	0.611	0.330	0.542	0.240	0.385	0.219
# Successful nests/year[d]	0.889	1.765	0.734	0.535	1.295	0.748	1.203	0.942
# F fledglings/successful nest	0.83	0.70	0.59	0.56	1.21	0.86	0.75	0.70
# F fledglings/year	0.735	1.235	0.433	0.297	1.564	0.641	0.902	0.659
Adult survival/year	0.82	0.95	0.83	0.5	0.75	0.88	0.55	0.63
Juvenile survival/year	0.42[f]	0.25[d]	0.33	0.33[e]	0.35[f]	0.32[f]	0.4	0.36

All parameters are in days except where noted.
Data sources: Akepa- (Lepson and Freed, 1997); Akohekohe – (Berlin and Vangelder, 1999; Simon et al., 2001); Elepaio (HFNWR) – (van Riper, 1995; Vanderwerf, 1998a; Vanderwerf, 2004); Elepaio (Oahu) – (van Riper, 1995; Vanderwerf, 1998a; Vanderwerf and Smith, 2002); Hawai'i'amakihi – (Lindsey et al., 1998; Kilpatrick, 2003; Kilpatrick et al., 2006); Hawai'i creeper – (Vanderwerf, 1998b; Woodworth et al., 2002); Omao – (Ralph and Fancy, 1994b; Wakelee and Fancy, 1999); Palila – (van Riper, 1980; Pletschet and Kelly, 1990; Lindsey et al., 1995).
a DPR – Daily predation rate for the incubation (including egg laying) and nestling stages.
b DFSN – Days following a successful nests before starting a new nest. Most values were taken from descriptions of the length of parental care. I ensured that the number of successful broods modeled using this time period agreed with that seen in the field.
c DFUN – Days following unsuccessful nests before starting a new nest. The only robust data available for this parameter was for Hawai'i'amakihi so I used this value (2 days) for the other species where this period was unknown.
d Juvenile survival for Akohekohe was unknown, so I used a conservative estimate of 0.25.
e Unknown for this subspecies so the estimate from the Hakalau Forest National Wildlife Refuge (HFNWR) population was used.
f Estimated using enumeration, and thus represents a minimum estimate.

was reported, I estimated the variance of this binary variable using the same method I used for the survival probabilities and used this to generate estimates of the variance in the daily nest survival rate. Finally, I used published estimates of the variance in the number of nestlings per successful nest and combined this with the variance in the number of successful nests per year to estimate the variance in the yearly fecundity, F.

2.2. Model description

To investigate the possible benefits of rodent control on resistance evolution, I modeled two populations connected by dispersal. One population existed at middle elevation (~800–1200 m) and was subject to selective mortality due to malaria, while the other population existed at high elevation (>1400 m) where little or no mosquitoes or malaria occur (van Riper et al., 1986; Atkinson et al., 2005). This two population model is a simplification of the true state of most species which occur in one or more populations that are spread across elevational gradients. However, the model described here captures the qualitative dynamics present in the more complex continuous population model (Kilpatrick, A.M., unpublished data).

2.2.1. Genetic model
Populations are composed of resistant individuals (genotype rr), susceptible individuals (ss) and heterozygotes (rs). In the model, resistant (rr) individuals survive the acute phase of malaria infections and become chronically infected, while all susceptible (ss) individuals that are infected with malaria die. Below, I present results for the two extreme cases of heterozygote dominance (heterozygotes are either completely susceptible or fully resistant). I modeled chronically infected individuals as immune to malaria superinfection because they do not appear to suffer additional morbidity or mortality and there was no rise in the concentration of *Plasmodium* in their blood following superinfection (Atkinson et al., 1995, 2001a,b).

2.2.2. Age structure
This model divides each population into juvenile (J) or hatch year birds and adults (A), which enabled parameterization of the model with the demographic data collected from the field. In addition, resistant adults are divided into unexposed or naïve individuals (subscript n) and infected individuals (subscript i) which survived the acute phase of infection. Populations are counted just after the breeding season is complete but before any transmission of malaria or mortality has taken place. Adults and juveniles are subsequently exposed to malaria before being counted the next year. Under these conditions, populations of resistant and susceptible (subscripts r and s, respectively) adults and juveniles (subscripts A and J, respectively) at time $t + 1$ are given by

where α is the transmission rate or fraction of individuals infected with malaria each year, S_m is the probability of resistant birds surviving the acute phase of malaria (=1 for the analyses presented here), S is the survival of individuals from one year to the next, and F is the per capita fecundity or number of offspring produced. I parameterized this simple model with the demographic data from Table 1 with a single estimate of fecundity for birds breeding in their second year (F_J) and older birds (F_A). I assumed that chronic malaria infections had no negative effects on reproduction (Kilpatrick et al., 2006) but decreased survival by 17%, based on a field study that showed that chronic malaria infections decreased the survival of adult Hawaii amakihi from $0.75 \pm SE = 0.029$ to 0.62 ± 0.043 (Kilpatrick, 2003). Although the quantitative effect of chronic malaria infection on other species is likely to differ, these estimates were the only published data available and so I used them for other species as well.

2.2.3. Density dependence
I assumed density dependent reproduction (which is equivalent in this model to density dependent juvenile survival) by calculating yearly reproduction as:

$$F = F_m e^{-N/K},$$

where F_m is the measured reproductive output described above, N is the adult population size at the beginning of reproduction, and K is a constant. To facilitate comparison between species and different sets of parameter value, I set K for all simulations so that the equilibrium population size was one thousand individuals for all species for the high elevation population:

$$K = \frac{1000}{\ln\left(\frac{F_m S_J}{1 - S_A}\right)}.$$

In simulating the effects of rodent control on mid elevation populations (see below), I used the same value for K for the high and mid-elevation populations. This resulted in the high elevation equilibrium population size remaining fixed at 1000 individuals (if $\lambda > 1$ without rodent control), but the middle elevation equilibrium population size was increased by rodent control so that it was greater than 1000 individuals.

2.2.4. Dispersal
I modeled migration in both elevational directions as is commonly done in two-population models of insecticide resistance using treatment and refuge areas (May and Dobson, 1986; Ives and Andow, 2002). I assumed that some fraction f_d of the hatch year individuals born each year were exchanged between the two populations. These individuals

$$
\begin{bmatrix} J_r \\ A_{rn} \\ A_{ri} \end{bmatrix}^{t+1} =
\begin{bmatrix}
[F_{Jrn}(1-\alpha) + F_{Jri}(\alpha \cdot S_{mJ})]S_{Jr} & [F_{Arn}(1-\alpha) + F_{Ari}(\alpha \cdot S_{mA})]S_{Arn} & F_{Ari}S_{Ari} \\
S_{Jr}(1-\alpha) & S_{Arn}(1-\alpha) & 0 \\
S_{Jr}(\alpha \cdot S_{mA}) & S_{Arn}(\alpha \cdot S_{mA}) & S_{Ari}
\end{bmatrix}
\begin{bmatrix} J_r \\ A_{rn} \\ A_{ri} \end{bmatrix}^{t},
$$

$$
\begin{bmatrix} J_s \\ A_s \end{bmatrix}^{t+1} =
\begin{bmatrix}
F_{Js}S_{Js}(1-\alpha) & F_{As}S_{As}(1-\alpha) \\
S_{Js}(1-\alpha) & S_{As}(1-\alpha)
\end{bmatrix}
\begin{bmatrix} J_s \\ A_s \end{bmatrix}^{t},
$$

$$(3a, b)$$

then mated randomly with individuals in each population. The dispersal fraction, f_d, was calculated as a fraction of the difference between juvenile and adult mortality for each species. In doing so, I assumed that dispersal is density dependent (Lin and Batzli, 2001; Gaggiotti et al., 2002; Rhainds et al., 2002) and that dispersing individuals were not part of the local population. As a result, I did not decrease juvenile survival below that measured empirically. I based these assumptions on the fact that survival estimates based on mark–recapture data cannot distinguish between mortality and permanent dispersal (White and Burnham, 1999), and most dispersing birds are young of the year who have yet to establish a territory. It therefore seems likely that some fraction of the difference between adult and juvenile mortality is due to permanent dispersal of juveniles.

2.2.5. Initial conditions

For the demographic traits of each species I modeled the population and genetic dynamics of the two populations connected by post-reproductive dispersal with a yearly time step. I assumed that each species occurred primarily at high elevations and that the frequency of the resistant allele was low (~1%). The high elevation population started with 1000 individuals (the equilibrium high elevation population size), with 990 homozygous susceptible (ss) individuals and 10 heterozygote individuals (rs), approximately corresponding to an allele frequency of $f(r) = 0.01$. The middle elevation population started with 1 homozygous susceptible individual (ss). I present results for a base or reference parameter set (50 year simulation, $\alpha = 0.3$, $f_d = 0.25$), and three variants where a single parameter was varied from the base run: reduced malaria transmission ($\alpha = 0.1$), reduced dispersal ($f_d = 0.1$), and an extended length run (100 years). I do not present results for Oahu Elepaio, which only occurs in an area with mosquitoes and disease. In addition, I omit results where all levels of rodent control I simulated resulted in the decline of both populations below 5 individuals, since these would almost certainly result in eventual extinction.

The output of these simulations was the size of the mid-elevation population and the frequency of the resistant allele in the populations after 50 or 100 years. The latter time span roughly corresponds to the period over which global warming is expected to eliminate most mosquito and malaria free habitat in Hawai'i (Benning et al., 2002). I examined several parameter combinations, including varying levels of inter-population dispersal, the fraction of individuals that became infected with malaria each year, the dominance of the resistant allele (or the susceptibility of the heterozygote) and the magnitude of the effects of rodent control.

2.3. Model assumptions

I made three key assumptions in this modeling approach in order to simplify the analysis. First, I have assumed that the demographic parameters in Table 1 are constant across an elevational gradient, except for mortality due to malaria

transmission. If mid-elevation habitat differs in food resources, nesting sites, or other factors that affect survival and reproduction then using rodent control at middle elevations will differ from the results I present here. Some aspects of habitat that are important to Hawaiian birds (nectar production and insect densities) have been measured (Baldwin, 1953; van Riper, 1984; Ralph and Fancy, 1994a; Fretz, 2002), and could be determined for middle elevation habitat before making a decision where to expend valuable resources to implement rodent control.

Second, in modeling the demographic parameters as constant with density, I have also assumed that mortality due to malaria is additive, rather than compensatory. If some fraction of the individuals that I modeled as being killed by malaria would have died of predation or starvation, then the transmission rates I have used will be overestimates of the selective pressure on the population. However, the same selective pressure as I have modeled would be present at a lower elevation where transmission was higher.

Third, in this model reproduction and survival are assumed to be the same for susceptible and uninfected resistant birds, which assumes that there is no cost of resistance. Consequently, if resistant infected birds have fecundity and survivorship greater than zero, then the fraction of the population that is resistant will increase as long as there is some malaria transmission (if $\alpha > 0$). However, the rate of evolution, and the size of the population are determined by the interplay of demography, migration, genetics, and malaria transmission (Tabashnik et al., 2004; Vacher et al., 2004).

2.4. Rodent control

I used the model to address the feasibility of using rodent control to reduce predation rates and facilitate the evolution of resistance. As I result, I altered the survival and reproduction of the mid-elevation population while keeping the demographic characteristics of the high elevation population at the measured values. In forests on the island of Oahu, Hawai'i, rodent control led to a 67% decrease in adult female mortality and a 37% decrease in nest predation for 'Elepaio (Vanderwerf and Smith, 2002). Here I examine the possible effects of rodent control by modeling the impact of 25%, 50% and 75% reductions in survival paired with 20%, 35%, and 50% reductions in nest predation on middle elevation populations. This acknowledges the fact that rodent control may have smaller or larger effects on some species in other areas compared to 'Elepaio on Oahu (Vanderwerf, 1998a; Vanderwerf and Smith, 2002). In addition, if a species has higher nesting success or adult survival than 'Elepaio, then a 50% reduction in nest predation or survival will be smaller numerically than those for 'Elepaio. Finally, although these may appear to be large effects, it should be noted that prior to the introduction of mammalian predators (primary rats, mice, cats and mongeese), the only predators on nests and adult birds in Hawai'i were avian predators including an eagle, several long legged owls, the Hawai'ian hawk or Io (*Buteo solitarius*), and the Short-eared owl or Pueo (*Asio flammeous sandwichensis*). All of these likely occurred at relatively low densities

Table 2 – Population growth rates, k, (±1SE) estimated from high elevation populations with disease transmission low or absent (except 'Elepaio-Oahu), and with three levels of simulated rodent control (numbers in parentheses refer to percent decreases in nest predation (NP) and adult mortality (AM))

Species	λ (±1SE)	λ (−20%NP, −25%AM)[1]	λ (−35%NP, −50%AM)[1]	λ (−50%NP, −75%AM)[1]
Akepa	1.13 ± 0.065	1.18	1.23	1.28
Akohekohe	1.26 ± 0.136	1.29	1.31	1.33
'Elepaio-HFNWR	0.97 ± 0.037	1.03	1.08	1.13
'Elepaio-Oahu	0.60 ± 0.137	0.75	0.89	1.03
Hawai'i'amakihi	1.30 ± 0.093	1.46	1.56	1.67
Hawai'i creeper	1.09 ± 0.079	1.21	1.29	1.38
'Oma'o	0.91 ± 0.147	1.09	1.25	1.40
Palila	0.87 ± 0.081	1.03	1.18	1.32

compared with mainland avian nest predators (Atkinson, 1977).

3. Results

The estimated population growth rates, λ, in the absence of disease (except Oahu Elepaio) differed substantially between species (Table 2, Column 2). Some species (e.g., Hawai'i'amakihi, Akohekohe) appeared to have a greater chance to survive moderate levels of malaria transmission and still persist. In contrast, the demographic data suggested that study populations of other species appeared to have declining populations, even in the absence of malaria (Table 2). Due to the limitations of the data (see Section 2), these estimates may not match population trajectories for species as a whole.

For species with $\lambda \gg 1$, the model suggested that dispersal and natural selection would lead to the establishment of resistant populations at middle elevations if resistance was a dominant trait. For example, after 100 simulated years, high and mid elevation populations of Hawai'i'amakihi had resistant allele frequencies of 0.36 and 0.67, respectively and over

half of both populations were resistant (heterozygous or homozygous for the resistant allele) (Fig. 1). However, without rodent control, evolution of resistance was possible on a 100 years timescale only for Hawai'i'amakihi and Akohekohe.

Fortunately, the simulated impacts of rodent control substantially increased population growth rates for most species (Table 2, columns 3–5). If the quantitative effects of rodent control on other species were at least half that measured on Elepaio on Oahu (Table 2, column 3; Vanderwerf and Smith, 2002), several species' populations that appeared to be declining would have $\lambda > 1$ and some would be capable of significant population growth (Table 2, columns 4 and 5). For these species, rodent control allowed the establishment of resistant populations at middle elevations within a 50 or 100 years time span (Fig. 2). The evolutionary trajectory of these species was qualitatively similar to Fig. 1, with the rise of the frequency of the resistant allele being proportional to the population growth rate (Fig. 2 and Table 2).

I altered parameters of the model and identified four important results. First, the ending frequency of resistant alleles increased substantially with the effectiveness of rodent control, except for species (Hawai'i'amakihi, Akohekohe) with
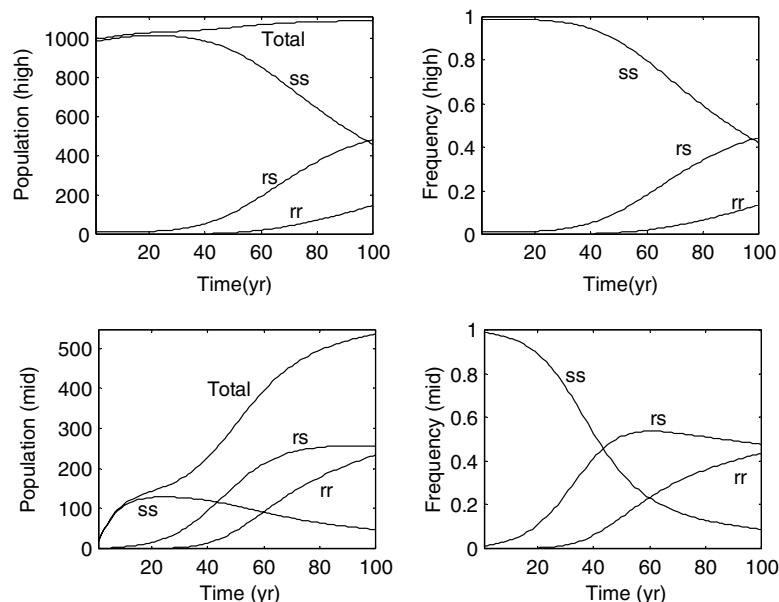

Fig. 1 – Simulated population (left) and genetic (right) trajectories of high (top) and middle (bottom) elevation populations of Hawai'i'amakihi in the absence of rodent control. Parameter values were: $a = 0.3$, $f_d = 0.25$.
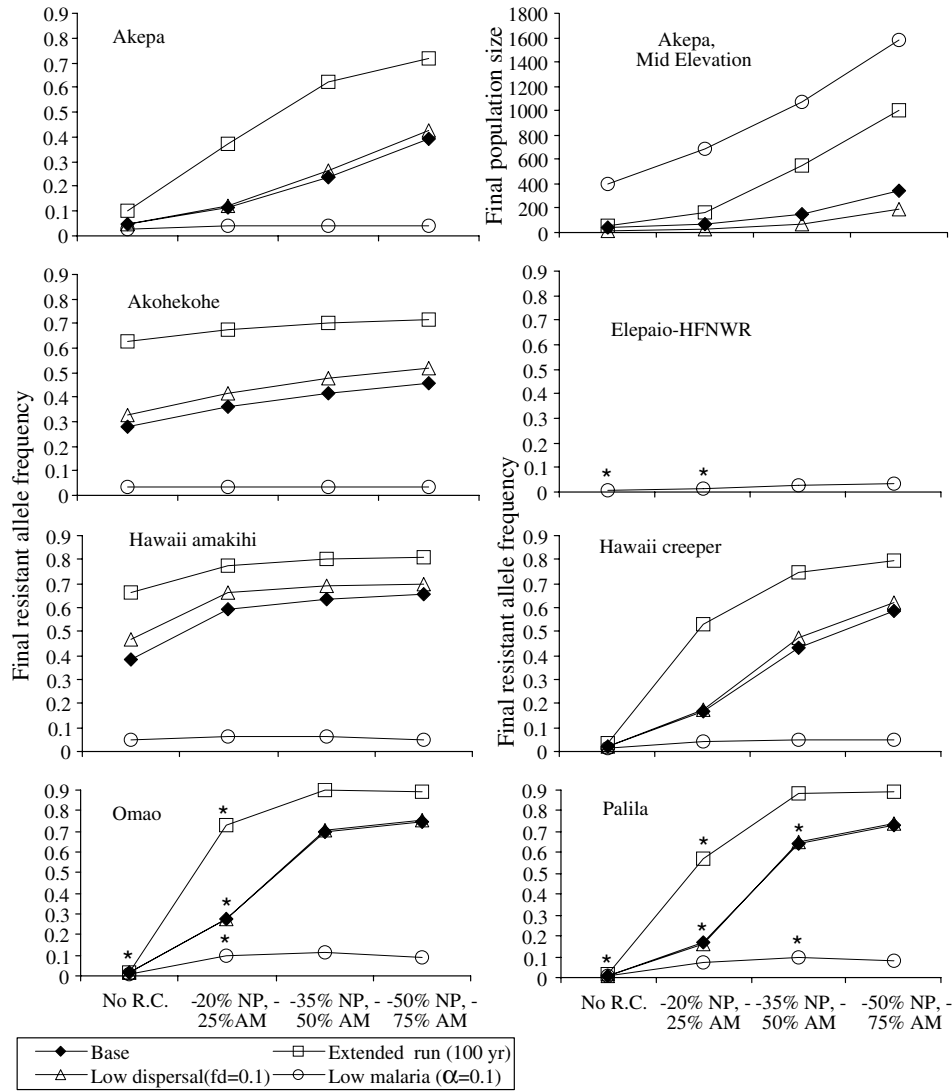
Fig. 2 – Ending mid-elevation population frequencies of the resistant allele, $f(r)$, for seven species for unmanipulated (No R.C.) and three levels of simulated rodent control. Base parameter values were: $a = 0.3$, $f_d = 0.25$ and 50 years simulation. Upper right panel shows ending mid-elevation population sizes for Akepa, corresponding to ending gene frequencies in the upper left panel. Asterisks denote simulations where the combined (high and mid elevation) populations fell below 20 individuals during the simulation.

already large population growth rates (Fig. 2). Second, lower malaria transmission ($\alpha = 0.1$) resulted in the highest ending mid-elevation population sizes (Fig. 2; upper rightmost panel), but the lowest ending resistant allele frequency. This demonstrates that using rodent control in higher elevation areas with little or no malaria would not facilitate the evolution of resistance. Third, decreasing the dispersal fraction slightly increased the ending frequency of the resistant allele (Fig. 2) but substantially lowered the ending population sizes (Fig. 2, upper right panel). Increasing the dispersal fraction ($f_d = 0.5$) had similar converse effects (results not shown). Fourth, extending the length of the simulation to 100 years substantially increased the ending frequency of the resistant allele and the ending mid-elevation population size.

In some simulations (marked by asterisks in Fig. 2) combined high and low elevation populations declined to such low levels (<25 individuals) that extinction would be likely

to occur, even if the mid-elevation population had a long term growth trajectory. In particular, Elepaio on Hawai'i (Elepaio-HFNWR) were only able to persist above 20 total individuals (for both populations) under the low transmission simulation, with moderately effective rodent control ($\geqslant 35\%$ reduction in nest predation (NP) and $\geqslant 50\%$ reduction in adult mortality (AM); Fig. 2). Similarly, populations of Elepaio on Oahu, which already reflect the impacts of disease, were only stabilized ($\lambda \geqslant 1$) by highly effective rodent control (Table 2), in agreement with published results (Vanderwerf and Smith, 2002).

Finally, if malaria resistance is a recessive trait (heterozygote susceptibility = 1), then none of the species evolved substantial levels of resistance (all had ending resistant allele frequencies <2% for all of the simulations and rodent control levels). In short, model results suggested that evolution of malaria resistance on a 100 years timescale required that resistant alleles show at least partial dominance.

4. Discussion

The declines and extinctions of most of the unique avifauna of Hawai'i as a result of introduced predators, habitat degradation and disease makes this group one of the most important focuses for conservation in the United States (Jacobi and Atkinson, 1995; US Fish and Wildlife Service, 2003). The most urgent aspect of this conservation challenge is the recent work that suggests that most mosquito and disease free habitat will be eliminated by a 2 °C rise in global temperatures that is projected to occur in the next century (Benning et al., 2002). Clearly, management approaches that aim to preserve high elevation populations of native Hawaiian birds will likely fail to conserve these species due to the expansion of malaria and its devastating effects.

The demographic characteristics of the species considered here suggest that most species are unable to persist in areas with malaria transmission. However, my results suggest that control of rodent predators, which was extremely effective in increasing reproduction and survival of Elepaio on Oahu (Vanderwerf and Smith, 2002), may offer an opportunity for facilitating the evolution of resistance to malaria in several currently endangered Hawaiian birds. A key requirement of this strategy is that rodent control be used in areas where there is moderate malaria transmission, rather than at high elevations where most conservation effort is currently expended (US Fish and Wildlife Service, 2003).

This assertion is based on several key assumptions. Most importantly, the model demonstrated that the evolution of resistance will not occur on a reasonable timescale for any of the species examined if malaria resistance is governed by a single recessive allele. This is a result of the relatively low reproductive potential of the species considered here, especially compared to insects evolving resistance against pesticides (Ives and Andow, 2002; Tabashnik et al., 2004). However recent research provides evidence that resistance has evolved in Hawaiian birds over the last century. Populations of Hawai'i'amakihi now persist at low elevations and have increased in abundance in the last decade (Woodworth et al., 2005). In addition, the prevalence of malaria antibodies (indicative of exposure and survival or resistance, as I have defined it here) in several populations of Hawaii amakihi, two populations of Elepaio and one populations of Apapane (Himatione sanguinea) was 20–100% (Atkinson et al., 2005; Woodworth et al., 2005; Kilpatrick et al., 2006, Vanderwerf, E.A., Burt, M.D., Rohrer, J.L., Mosher, S.M., unpublished data). The existence of these populations with a significant proportion of resistant individuals combined with model results from a wide range of parameter values suggests that resistance is unlikely to be governed by a single recessive allele.

Nonetheless, if current work on the genetic basis for malaria resistance (Jarvi et al., 2001, 2004) determines that for some species resistance is governed by a single recessive allele then a different strategy would be more effective. This would entail breeding a captive population of resistant individuals and then introducing them into an area of habitat where they would be unlikely to breed with susceptible individuals, since this would lead to the production of susceptible heterozygotes and the rapid decline of populations of most species considered here.

Three other points merit discussion. First, the results presented here show that there is an important tradeoff in the implementation of rodent control. Facilitating population growth through rodent control in areas with low levels of malaria transmission (e.g., 10% yearly transmission rate) leads to larger minimum and final populations, since the malaria-induced mortality is lower. However, the selection pressure from this level of transmission is weak. As a result, this strategy does not lead to evolution or resistance on a reasonable time scale and should only be employed for populations or species whose population growth rates or populations are too low to permit them to survive under higher levels of malaria transmission. For other species, reducing rodent predators in areas where malaria transmission is higher (e.g., 30% yearly transmission rate) are more likely to result in the evolution of resistant populations (Fig. 2).

Second, I have deliberately focused on controlling rodent predators, rather than habitat restoration, because previous data existed with measured and demonstrated effects on demography. However, habitat restoration which also led to increases in survival and reproduction would likely have similar effects to those suggested here for predator control.

Finally, the models presented here ignore evolution on the part of the parasite. It is well known that pathogens can evolve at rates several orders of magnitude faster than their hosts, and consequently, evolution for increased virulence by the parasite could counteract evolution for resistance in the host population (Ewald, 1994; Gog and Grenfell, 2002; Mackinnon and Read, 2004). However, Hawaiian birds which survive acute malaria infections maintain chronic viremias that are still infectious to mosquitoes (Atkinson et al., 2001b; Jarvi et al., 2002). Consequently, the evolution of resistance in hosts should not lead to decreased transmission of the parasite and may in fact increase transmission due to a longer lifespan while remaining infectious. This suggests that facilitating the evolution of resistance in Hawaiian birds should not be hampered by evolution for increased virulence of the pathogen.

Using rodent control to aid populations of native Hawaiian birds is not without difficulties. First, using aerially deployed rodenticides carries a possible risk of exposing non-target species to the poisons used to kill the rodents. In addition, there are several other predators in Hawaiian forests including cats (Felis catus) and mongeese whose populations are partially supported by rats. Reducing or eliminating rats from an area runs the risk of shifting the predation pressure from rats to birds, which might abrogate the benefits of rodent control. In addition, predator birds that eat rats, including Ios (Buteo solitarius) and Pueos (Asio flammeus sandwichensis) could be poisoned through secondary consumption. Successfully controlling rodents to aid bird populations would require addressing these challenges.

Finally, some populations of Hawaiian birds may be limited more by available habitat than by disease. For example, Palila are confined to areas of sub-alpine Mamane (Sophora chrysophylla)-Naio (Myoporum sandwicense) woodlands, and these occur primarily at elevations where mosquitoes are absent (Banko et al., 2002). Consequently, using rodent control to facilitate the evolution of disease resistance would first require habitat restoration at middle elevations. Selecting for

disease resistant Palila would safeguard against the invasion of high elevation habitat by mosquitoes projected under the global warming scenario (Benning et al., 2002). Other species such as the 'Oma'o appear to be relatively resistant to acute malaria infections, at least in the benign laboratory environment (Atkinson et al., 2001a), and so their distributions may also be limited by other factors. If 'Oma'o populations are limited by predators rather than disease, then rodent control aimed at facilitating resistance evolution in other species will still improve their survival and reproduction for the length of the control program. In summary, if the technical and logistical challenges of deploying rodenticides can be overcome, rodent control should have positive benefits for most native species, and may be the best strategy for facilitating the evolution of resistance to malaria for many species that might otherwise be driven extinct by rising global temperatures and the invasion of mosquitoes to high elevation habitat.

In a broader context, advocating a management approach that encourages evolutionary change in endangered species contrasts with the traditional conservation paradigm. However, for species impacted by multiple stressors, reducing the impact of one factor and encouraging evolutionary change that allows a species to deal with other stresses may allow for long-term recovery of a species. A great number of endangered species are threatened either due to a novel stressor that they have not yet been able to adapt to (Gomulkiewicz and Holt, 1995), or by multiple stressors that together lead to their decline. The results presented here suggest that reducing one stressor may allow for these species to recover by evolving behavioral (Short et al., 2002), or physiological adaptations. This strategy requires that species have the evolutionary potential to adapt to factors causing their decline and underscores the value of preserving sub-specific diversity in declining species. However, removing one of the stressors has the advantage that, even in the absence of evolutionary change, it should help to stabilize the focal population. The modeling approach I have presented here offers a framework for choosing which stressor to manage that has the greatest chance to lead to long term recovery of populations.

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