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Modelling the Future of the Hawaiian Honeycreeper: An Ecological and Epidemiological Problem

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Abstract

The Hawaiian honeycreeper (*Drepanididae*) faces the threat of extinction; this is believed to be due primarily to predation from alien animals, endemic avian malaria (*Plasmodium relictum*) and climate change. A deterministic SI modelling approach is developed, incorporating these three factors and a metapopulation approach in conjunction with a quasi-equilibrium assumption to simplify the vector populations. This enables the qualitative study of the behaviour of the system. Numerical results suggest that although (partial) resistance to avian malaria may be advantageous for individual birds, allowing them to survive infection, this allows them to become carriers of infection and hence greatly increases the spread of this disease. Predation obviously reduces the life-expectancy of honeycreepers, but in turn this reduces the spread of infection from resistant carriers; therefore the population-level impact of predation is reduced. Various control strategies proposed in the literature are also considered and it is shown that predation control could either help or hinter, depending upon resistance of the honeycreeper species. Captive propagation or habitat restoration may be the best feasible solution to the loss of both heterogeneity within the population and the loss of the species as a whole.

Keywords: Avian malaria, Hawaii, quasi-equilibrium assumption, climate change

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1. Introduction

The IUCN Red List of Threatened Species suggests that 41% of amphibians, 25% of mammals and 13% of birds are threatened. This is due to a mixture of reasons, from hunting to habitat loss, from invasion of exotic species to climate change; with human activity at the heart of most. The native and largely endemic fauna of Hawaii typifies the range of threats faces; the limited area, the introduction of predators and disease, have all contributed to multiple extinctions. It is now estimated that over half of all Hawaii's 142 native bird species which were extant pre-human colonisation, are now extinct (Banko et al., 2001). The Hawaiian honeycreeper is a typical example; of the over 35 original species (all of which were endemic to the area), currently just 22 are listed as extant; only 2 are of least concern, 15 are threatened and 5 are thought to be recently extinct (see Appendix A, Table 1).

There are several reasons for the decline in the numbers of endemic avifauna in Hawaii, however there are three main aspects to consider; predation (particularly from rats (*Rattus*) (Kilpatrick, 2006), cats (*Felis catus*) and mongooses (*Herpestes aureopunctatus*) (Scott et al., 2001), the introduction of avian malaria to the Hawaiian islands (Atkinson et al., 2000) and loss of habitat (Benning et al., 2002). Each of these three factors is considered.

The native birds' major predators (rats, cats and mongooses) are all non-native species introduced to the islands after Europeans made contact in 1778 (Scott et al., 2001). Rats prey mainly on eggs and nestlings but will also kill adult birds. As is the case worldwide, one of the cats' main prey is birds and this is no different in Hawaii, where this alien species is now found on all of the eight main islands. The mongoose preys mainly on ground nesting birds and so has had a great effect on species such as Hawaiian goose (*Branta sandvicensis*), although probably a lesser effect on Hawaiian honeycreepers. These three predators are part of a complex trophic web in which all three share common prey species. Out of all extinctions of native avifauna in Hawaii, 54% are attributed to the rat and 26% to cats (van Riper and Scott, 2001), showing the potential harm these species can cause.

Infectious diseases are considered one of the dominating factors in the vulnerability of Hawaii's population of endemic avifauna (Scott et al., 2001). Although the honeycreeper population experiences the effects of multiple diseases such as avian pox, by far the most important is avian malaria (Atkinson et al., 1995). The malaria carrying mosquito (*Culex quinquefasciatus*) was introduced to Hawaii in the 1920s and currently only Laysan Island (one of the Southwestern smaller islands) remains mosquito-free (Scott et al., 2001). As with all malaria, transmission only occurs when female mosquitoes take a blood meal before laying eggs; this biting acts to both transfer the protozoan malaria parasites from an infected host to the mosquito and from an infected mosquito to the bitten host (Lovette, 2005). Only a single strain of malaria (*Plasmodium relictum*) currently exists in Hawaii (?), and this strain is specific to birds, hence zoonotic transmission to humans does not occur. Mosquitoes thrive in humid conditions and so there are large populations living at lower and middle elevations (below 1500 metres) on many islands due to the tropical climate (Kilpatrick, 2006). Currently there are relatively few mosquitoes able to survive at the lower temperatures of the higher elevation regions and, even if these vectors survive, there is strong evidence that protozoan parasites cannot develop (and hence there is no spread of malaria) at these cooler temperatures (Benning et al., 2002).

Many non-native birds have some resistance to malaria (Benning et al., 2002), however, once infected, the probability of mortality for Hawaiian honeycreepers in general is high,with experimental estimates ranging from 63-90% and varying between species (Atkinson et al., 2000). In particular, species of honeycreeper found at high elevations have little protection against the disease (?) due to the absence of both the disease and therefore its selective effects at these altitudes (Lovette, 2005). Acute malaria infection lasts around 18-24 days (Atkinson et al., 2000); the symptoms start with decreased food consumption and activity, and infected birds develop a prominent sternum. Subsequently death occurs, usually due to anaemia caused directly from the destruction of red blood cells by the malaria parasite (?). Although many birds die with acute infection, others (generally termed resistant birds) survive but remain infected and are able to transmit malaria to any biting mosquitoes for the rest of their lives. It is believed that in some species this resistance is a genetic trait (Kilpatrick, 2006; Westerdahl et al., 2005), which suggests that the prevalence of resistance

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should increase over time due to the greater mortality suffered by those birds not carrying the resistance gene or genes. The interaction between malaria and the honeycreeper in Hawaii, is therefore very different to avian malaria in mainland USA where the disease is very common but levels of mortality are far lower (Kilpatrick, 2006).

Hawaii has a highly heterogeneous landscape which has led to an array of unique fauna and flora. The islands rise to 4,205 metres above sea level, which gives rise to a range of habitats with pronounced differences in temperature. At present the average temperature ranges from $0°C$ to $29°C$; however these temperatures are predicted to increase by approximately $2°C$ over the next hundred years due to climate change (Benning et al., 2002), with an associated change in habitat. Such changes are not new to Hawaii; since the arrival of the Polynesians in approximately 400 AD the landscape has altered dramatically (Banko et al., 2001) and more recent changes in farming methods have devastated much of Hawaii's natural forest, especially in low to mid elevations (van Riper and Scott, 2001). In addition, alien plants and animals have taken over much of the lower elevations driving native species to higher areas or to extinction through competition (**?**). In turn these processes have limited the honeycreepers' habitat and food resources, reducing its population carrying capacity.

One of the most important uses of mathematical models is to test a range of scenarios or strategies that would be too costly or too time consuming to test in reality. Currently many suggestions exist to preserve the honeycreeper populations and these are briefly outlined.

Captive propagation programmes in which eggs produced either in the wild or captivity are incubated and chicks are hand-reared, already exist on a small scale on both Hawaii's main island and Maui (Kuehler et al., 2001) for various birds including some species of honeycreeper. Such programmes effectively increase the birth rate by insuring the safety of both the vulnerable eggs and chicks which have low survivorship in the wild. Comparable breeding programs of Nene (Hawaiian Goose) have already been highly successful (Banko et al., 2001).

Traditionally, to control the spread of malaria, the number of mosquitoes is sought to be reduced and, given the lack of control that exists over the other parameters (such as bite rate or trans-

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mission probabilities), this may still prove to be the most sensible choice. While insecticides may cause irreversible damage to much of Hawaii's unique plant and animal life, other methods exist. Genetically modified sterile-male mosquitoes could be developed and introduced (Scott et al., 2001), although complications include time to engineer a suitable mosquito and potential accompanying controversy. A reduction in mosquito habitat, such as removal of feral pigs (VanderWerf, 2001), is potentially a way forward without disturbing the delicate balance of this ecosystem (feral pigs cause habitat degradation via destruction of vegetation which in turn leads to muddy areas of suitable mosquito larval habitat (Scott et al., 2001)).

Small scale predation control via trapping has shown success for other native Hawaiian bird species (Hodges and Nagata, 2001). Other suggestions to prevent predation include rodenticides (Kilpatrick, 2006) and mongoose poisons (Banko et al., 2001). Any of these methods can be implemented either island-wide (across all elevations), or be targeted at specific areas, and given that they target non-native species are unlikely to meet opposition.

Due to the action of climate change it is inevitable that habitat previously above the temperature threshold for transmission will experience some transmission in the future, effectively reducing the amount of 'disease-free' habitat. Habitat conservation schemes to sustain areas of high elevation habitat for the honeycreeper such as replanting trees (Scott et al., 2001) are suggested approaches to take to preserve malaria-free habitat.

2. Model Formulation

Mathematical models have two main uses: to develop a more intuitive understanding of the mechanistic processes governing the behaviour of a system; or to utilised these processes to extrapolate the behaviour to a new scenario or situation. Here both of these approaches are adopted by developing a continuous-time deterministic model for the interaction between a host (the Hawaiian honeycreeper), predators and disease (malaria), which is parameterised using available data. This model provides a robust framework to explore the potential interactions between these three elements, and to assess the likely impacts of climate change in the near future.

The fundamental building-block of the model is the long-term population dynamics of the honey-creeper; in the absence of infection or predation this is modelled as a simple density-dependent logistic model:

$$\frac{\mathrm{d}N}{\mathrm{d}t} = rN(1 - \frac{N}{k}) = bN(1 - \frac{N}{K}) - dN \tag{2.1}$$

where N is the population size, r is the growth rate, and k is the carrying capacity. To assist with later developments, we partition the logistic model into a density-dependent birth rate and a density-independent death rate governed by the parameters b, K and d. Although this model ignores much biological realism, such as age-structure, gender or stochastic/environmental fluctuations, it provides a suitable basis onto which the impacts of predation and infection can be grafted.

Predation is believed to significantly reduce the numbers of native birds; it is therefore important to correctly assess how predation pressure is affected by prey abundance (in this case the honeycreeper). Here we use a Type III predator functional response (Murray, 2002; Britton, 2003), which assumes that the predator may change to an alternative food source when the prey is at lower densities, but also that predation saturates for large enough prey densities. The rate at which honeycreepers are consumed is then given by $NP(N)$ where:

$$P(N) = \frac{AN}{B^2 + N^2} \tag{2.2}$$

Here, A is related to the abundance of predator – which is assumed constant, independent of honeycreeper numbers, due to the availability of other food sources. The parameter B is a measure of the abundance of these other food sources, and determines when the predator is likely to switch its preference between preying upon honeycreepers to other species. It is noted that the rate of predation is not assumed to be affected by the factors such as age or whether an individual is suffering from the adverse effects of disease.

To capture the dynamics of malaria, the population is initially partitioned, according to the classic SI model into two classes: those susceptible to malaria (S) and those infected with malaria (I). Where $S(t)$ and $I(t)$ are taken to represent numbers of individuals rather than proportions or

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densities. Honeycreepers are assumed to be born susceptible as there is no evidence for vertical transmission. An exposed (or latent) class has not been included, as this period is negligible compared to the other epidemiological time scales. In the case of malaria, an exposed individual is one that has been bitten and has the protozoan developing with its organs but is not yet in its blood stream (it is infected but not infectious). This latency period is short, about 2 days, whereas the entire infectious period lasts approximately 22 days for acute infection and is lifelong for chronic infections (**?**).

The population of birds can be further subdivided into two classes N and R depending on whether they are resistant or not. Resistant birds can still become infected but suffer negligible disease and mortality, instead they become chronically infected with malaria for life. Such chronically infected birds retain excellent body condition (**?**) and have a similar reproductive success to uninfected birds (Kilpatrick et al., 2006). In contrast, non-resistant birds rapidly succumb to disease-induced mortality (at a rate D). It is believed that despite chronically infected (resistant) birds displaying lower levels of parasitaemia than their acutely infected (non-resistant) counterparts (Atkinson et al., 2000), that they act as reservoirs of malarial infection and are still infectious to mosquitoes (**?**). Within the model we assume that both have the same probability of transmitting infection to a biting mosquito.

Resistance to malaria appears to be both an inherited genetic trait potentially governed by the number of major histocompatibility complex (MHC) alleles (Westerdahl et al., 2005; Atkinson et al., 2000), but also a property that can spontaneously arise, dependant on general fitness and factors such as age, weight and sex (Atkinson et al., 1995). In the model resistance is therefore governed by two parameters: the probability η that resistance is inherited from the parent (if the parent is resistant); and θ the probability that if resistance is not inherited, that is it occurs randomly.

In the absence of predation, the equations for the numbers of infected, susceptible, resistance and

non-resistant birds becomes:

$$
\begin{aligned}
\frac{dS_N}{dt} &= b(1 - \frac{N_H}{K})((1-\theta)S_N + (1-\eta)(1-\theta)(S_R + I_R)) - dS_N - \lambda_H S_N \\
\frac{dI_N}{dt} &= \lambda_H S_N - (D+d)I_N \\
\frac{dS_R}{dt} &= b(1 - \frac{N_H}{K})(\theta S_N + [\eta + \theta(1-\eta)](S_R + I_R)) - dS_R - \lambda_H S_R \\
\frac{dI_R}{dt} &= \lambda_H S_R - dI_R
\end{aligned}
\tag{2.3}
$$

where birth and "natural" death rates are given (as in equation 2.1) by b and d respectively and N_H is the total host population size. λ_H is the force of infection on the host (honeycreeper) population (Anderson and May, 1992); this is the rate at which a susceptible host becomes infected, the derivation of which follows. It is assumed that infected non-resistant birds fail to breed or successfully raise offspring.

For malaria, the force of infection (λ_H) is due to the biting rate of infected mosquitoes, while the rate that mosquitoes get infected is governed by their biting rate and the proportion of infected honeycreepers in the population. Hence we observe the standard *criss-cross* transmission matrix associated with many vector born infections (MacDonald, 1957). The transmission dynamics are determined by the mosquito biting rate (a), the probability of a bite leading to infection for the host or vector (p_H and p_V respectively) and the numbers of susceptible and infectious hosts (S_H and I_H) and vectors (S_V and I_V). We also allow for mosquitoes feeding on other host species (such as mammals) to obtain their blood meal; this relieves some of the biting pressure on the bird population, but does not contribution to onwards transmission as the strain of malaria being considered is avian specific. Here the number of other hosts will be denoted by O and assumed constant. This gives a force of infection on the each avian hosts as:

$$
\lambda_H = a p_H \frac{I_V}{N_H + O}
\tag{2.4}
$$

It is proportional to the number of infected vectors I_V, and assumes that vectors bite at a constant

rate a irrespective of host density. Similarly the force of infection on each susceptible mosquito vector is:

$$\lambda_V = ap_V \frac{I_H}{N_H + O} \tag{2.5}$$

where $I_H = I_N + I_R$ is the total number of infected hosts.

To close the dynamics it is necessary to additionally include the mosquito population dynamics, again assuming SI epidemiological behaviour. It will be initially assumed that mosquitoes have a constant carrying capacity K_V, and that infection does not affect fecundity or survival (although see Ferguson and Read (2002)). The governing equations for the mosquito population numbers are:

$$\begin{aligned}
\frac{\mathrm{d}S_V}{\mathrm{d}t} &= b_V K_V - d_V S_V - \lambda_V S_V \\
\frac{\mathrm{d}I_V}{\mathrm{d}t} &= \lambda_V S_V - d_V I_V
\end{aligned} \tag{2.6}$$

where the birth and death rates b_V and d_V are assumed equal.

The six-dimensional system (equations (2.3) and (2.6)) can be reduced by means of a quasi-equilibrium assumption (Keeling and Rohani, 2008). This enables the elimination of the two mosquito equations, at the expense of more complex transmission functions, by assuming that the mosquito dynamics are sufficiently fast that they rapidly reach equilibrium. This assumption is reasonable given that the life expectancy of a mosquito is approximately 1-4 weeks (Styer et al., 2007), such that during its short lifetime a mosquito sees a sustained level of infection in the bird population. Setting the two rates of change in equation (2.6) equal to zero we obtain the quasi-equilibrium solutions which are functions of the current honeycreeper population:

$$\begin{aligned}
S_V^*(I_H, N_H) &\approx \frac{b_V K_V}{d_V + ap_V \dfrac{I_H}{N_H + O}} \\
I_V^*(I_H, N_H) &\approx \frac{ap_V K_V I_H}{(N_H + O)(d_V + ap_V \dfrac{I_H}{N_H + O})}
\end{aligned} \tag{2.7}$$

hence the force of infection acting on a susceptible honeycreeper (independent of resistance) is given by the non-linear function:

$$\lambda_H = a^2 p_H p_V K_V \frac{I_H}{(N_H + O)(a p_V I_H + d_V (N_H + O))} \tag{2.8}$$

where $I_H = I_N + I_R$ is the total number of infected honeycreepers.

Two remaining factors need to be included to more accurately capture the specific behaviour of honeycreepers and avian malaria in Hawaii: the impact of seasonality on the transmission of infection, and the spatial partitioning of the population into regions based on temperature ranges.

The impact of seasonality is important in many vector-borne infectious diseases (Keeling and Rohani, 2008), particularly so for avian malaria in Hawaii as there is a critical temperature ($13°C$) which determines whether or not the *Plasmodium* can develop (Benning et al., 2002). In mid elevation areas where the temperature fluctuates near this critical level the spread of malaria is very much dependent on seasonal changes, with peak transmission occurring during the warmer summer months (**?**).

To incorporate this biological feature into the model, the transmission rate (or, in this case, the bite rate a) which was previously taken to be a constant, can be considered to be a function of time, and will be taken as sinusoidal :

$$a(t) = \alpha_0(1 + \alpha_1 cos(\omega t)) \tag{2.9}$$

where α_0 is the half the maximum bite rate, $\omega(= 2\pi$ per year) is the period of forcing and α_1 is the amplitude of seasonality. Although there are many elaborate functions that could be taken to describe the temporal forcing, this simple sinusoidal wave should be sufficient in the case of malaria as it is driven by annual temperature cycles.

As the temperatures for the Hawaiian islands vary little over the course of the year (the North American National Weather service estimates around $4°C$ difference between summer and winter),

only mid elevations experience temperatures that fluctuation across the critical 13°C boundary. Therefore, when adding seasonality it is also important to spatially partition the population into low elevation regions that are permanently above 13°C, high elevations that are permanently below 13°C, and mid elevations that annually cross this threshold.

We utilise a simple metapopulation framework (Grenfell and Harwood, 1997) to capture these distinct spatial regions; with high, mid and low elevations corresponding to habitat above 1500 metres, between 900 and 1500 metres and lower than 900 metres respectively. The associated population at each elevation is denoted by 1, 2 or 3 as a further subscript; and the values of the biting rate, $a(t)$, are set according ($a(t) = 0$ at high elevation, $a(t)$ is sinusoidally forced at mid elevation, and $a(t)$ is constant at low elevation). Since there are movements of birds between elevations, these different subpopulations do not behave independently of each other, instead there is a low-level of population exchange between neighbouring altitudes controlled by the parameter ε.

Finally, to include the impact of climate change, it is considered how the areas permanently above, permanently below and around the critical 13°C will change. In particular we define L_i to be the area of land corresponding to each of the three elevations. Climate change acts by moving the temperature-based boundaries between the three regions and hence changing the respective areas. In particular, once climate change starts we assume that:

$$L_i = \hat{L}_i \left[1 + (c_{i-1} - c_i)t\right] \tag{2.10}$$

which captures the gain and loss of land in one temperature band to land directly above and below. Associated with these changes are modifications to the associated carrying capacities and population levels in each region; in particular we will assume that these scale linearly with the area such that:

$$K_i = \hat{K}_i L_i \qquad O_i = \hat{O}_i L_i \qquad B_i = \hat{B}_i L_i \qquad K_{V,i} = \hat{K_{V,i}} L_i \qquad A_i = \hat{A}_i L_i \tag{2.11}$$

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Incorporating all discussed features gives a twelve dimensional set of ODEs. Parameters (see Table 2) are taken to be representative of a "typical" honeycreeper however estimates vary across sub-species.

There are three elevations $i = 1, 2, 3$ (high, mid, low) and at each elevation the system is given by:

$$
\begin{aligned}
\frac{\mathrm{d}S_{iN}}{\mathrm{d}t} &= b_H(1 - \frac{N_i}{K_i})((1-\theta)S_{iN} + (1-\eta)(1-\theta)(S_{iR} + I_{iR})) - dS_{iN} - \lambda_i S_{iN} \\
&\quad - P_i S_{iN} + \sum_{j \neq i} (\varepsilon_{jj} S_{jN} - \varepsilon_{ij} S_{iN}) + c_{i-1} S_{(i-1)N} - c_i S_{iN} \\
\frac{\mathrm{d}I_{iN}}{\mathrm{d}t} &= \lambda_i S_{iN} - (d+D) I_{iN} - P_i I_{iN} + \sum_{j \neq i} (\varepsilon_{ji} I_{jN} - \varepsilon_{ij} I_{iN}) + c_{i-1} I_{(i-1)N} - c_i I_{iN} \\
\frac{\mathrm{d}S_{iR}}{\mathrm{d}t} &= b_H(1 - \frac{N_i}{K_i})(\theta S_{iN} + [\eta + \theta(1-\eta)](S_{iR} + I_{iR})) - dS_{iR} - \lambda_i S_{iR} \\
&\quad - P_i S_{iR} + \sum_{j \neq i} (\varepsilon_{ji} S_{jR} - \varepsilon_{ij} S_{iR}) + c_{i-1} S_{(i-1)R} - c_i S_{iR} \\
\frac{\mathrm{d}I_{iR}}{\mathrm{d}t} &= \lambda_i S_{iR} - dI_{iR} - P_i I_{iR} + \sum_{j \neq i} (\varepsilon_{ji} I_{jR} - \varepsilon_{ij} I_{iR}) + c_{i-1} I_{(i-1)R} - c_i I_{iR}
\end{aligned}
\tag{2.12}
$$

where the subpopulation specific terms for the force of infection (λ_i), predation rate (P_i) and bite rate $(a_i(t))$ are given by:

$$
\lambda_i = a_i(t)^2 p_H p_V K_{V,i} \frac{I_{iR} + I_{iN}}{(N_i + O_i)(a_i(t) p_V I_i + d_V(N_i + O_i))}
\tag{2.13}
$$

$$
P_i = \frac{A_i N_i}{B_i^2 K_i^2 + N_i^2}
\tag{2.14}
$$

and

$$
a_i(t) = \begin{cases} 0 & i = 1 \\ \alpha_0(1 + \alpha_1 \cos(\omega t)) & i = 2 \\ \alpha_0(1 + \alpha_1) & i = 3 \end{cases}
\tag{2.15}
$$

while parameters K_i, $K_{V,i}$, A_i, B_i, and O_i are related to the areas of the three regions as specified

in equation (2.11).

3. Results

3.1. Calculating R_0

The *basic reproductive ratio*, R_0, is defined as the expected number of infected hosts that are produced from a single infected host in a completely susceptible population of both hosts and vectors (Keeling and Rohani, 2008). This useful quantity can determine whether or not an infection can invade and persist in a population, but it can also be used to compare spread of disease from different populations. The R_0 values for this model can be calculated from first principles by examining the chain of transmission from mosquitoes to birds and back to mosquitoes.

Calculating the number of infected birds from a single infected mosquito using its life expectancy (LE):

$$
\begin{aligned}
\text{infected birds} \quad &= \quad \text{LE of a mosquito} \times \text{bite rate} \times \text{prob of transmission} \times \frac{\text{susceptible birds}}{\text{total birds}} \\
&= \quad \frac{1}{b_V} a_0 p_H \frac{N_H}{N_H + O} \\
&= \quad \frac{a_0 p_H N_H}{b_V (N_H + O)} \quad\quad\quad\quad\quad\quad\quad\quad\quad\quad\quad (3.1)
\end{aligned}
$$

Calculating the number of infected mosquitoes from a single infected resistant bird:

$$
\begin{aligned}
\text{infected mosquitos} \quad &= \quad \text{LE of bird} \times \text{bite rate} \times \text{prob of transmission} \times \frac{\text{susceptible mosquitoes}}{\text{total birds}} \\
&= \quad \frac{1}{d} a_0 p_V \frac{N_V}{N_H + O} \quad\quad\quad\quad\quad\quad\quad\quad\quad\quad (3.2)
\end{aligned}
$$

Calculating the number of infected mosquitoes from a single infected non-resistant bird:

$$\text{infected mosquitos} \quad = \quad \text{LE of bird} \times \text{bite rate} \times \text{prob of transmission} \times \frac{\text{susceptible mosquitoes}}{\text{total birds}}$$

$$= \quad \frac{1}{d+D} a_0 p_V \frac{N_V}{N_H + O} \tag{3.3}$$

And so R_0 is given by:

$$R_{0R} = \frac{a_0^2 p_H p_V N_V N_H}{d b_V (N_H + O)^2} \quad \text{and} \quad R_{0N} = \frac{a_0^2 p_H p_V N_V N_H}{(d+D) b_V (N_H + O)^2} \tag{3.4}$$

for resistant and non-resistant populations respectively.

Hence

$$R_{0R} = (1 + \frac{D}{d}) R_{0N} \tag{3.5}$$

and so using values for the natural and disease induced mortality, d and D, given in Table 2, R_{0R} is approximately 70 times larger than R_{0N}. This indicates that the resistant honeycreepers are acting as carriers for malaria which results in much greater spread of disease within the total population when they are present.

It can be seen that whilst R_0 increases with the number of mosquitoes (N_V), the larger the comparative size of the other animal populations (O) compared to the honeycreeper population (N_H), the lower it will be. Assuming the honeycreeper population sizes are always smaller than numbers of other animals (as would be expected), the smaller the honeycreeper population (N_H) the smaller R_0 and so the disease alone is unable to push honeycreepers to extinction, but the population sizes can be pushed to very low levels.

3.2. Numerical Simulation

Numerical analysis of the model (equation 2.12) was performed using Matlab software with parameters chosen such that they match data where available, reflect plausible values and show typical

behaviour of this system (see Table 2). It is predicted that there are only a limited number of infected non-resistant honeycreepers, due to the high rate of disease induced mortality, moreover such infected birds are extremely rare at high elevations where transmission is impossible. Annual oscillations occur as a result of the temporal malarial forcing at mid elevation; however despite the small amount of mixing (5%), there is minimal transfer of these fluctuations to either of the other regions. At low and mid elevations the majority of the resistant population consists of infected birds and even at high elevations there is a substantial persistent infected population (due to mixing) despite the absence of transmission (see Figure 1) .

To determine the impact of various parameters for which data was limited, simulations were run to examine the change in honeycreeper population sizes across a range of plausible parameter values.

3.2.1. Inheritance of Genetic Resistance

The three most abundant species of honeycreeper today are Apapane, Amahiki and Iiwi (see Table 1). The first two have both shown experimentally high resistances to malaria relative to other Hawaiian avifauna (approximately 35% chance of developing chronic malaria rather than acute disease), whereas the latter has little resistance (10% chance of chronic infection) (Atkinson et al., 2000). While such estimates give a population-level quantification of resistance, the causative mechanism (or the precise mix of chance and genetic resistance – governed by θ and η) is unclear. A contour plot of total population-level resistance in the (θ, η) plane enables sets of (θ, η) parameter pairs to be determined that correspond to both these high and low total resistances (Figure 2); this is performed for the populations once at equilibrium (simulations were ran for 100 years to ensure this steady state was achieved..

3.2.2. Introductions

Using equations (2.12) the effects of introducing sequentially predation, malaria and climate change into a population at the relevant times can be simulated. Populations with 35% and 10% resistance which are equivalent to Apapane/Amahiki and Iiwi resistances;are chosen to be $(\theta, \eta) = (0.03, 0.21)$

and $(\theta, \eta) = (0.03, 0.06)$ for high and low respectively. The only variable altered between these two simulations was the genetic inheritance of resistance to malaria (η), enabling the effects of this particular trait to be elucidated.

For both populations the introduction of alien predators in 1778 reduces the equilibrium value for the population size. Up until the introduction of the mosquito, the populations are identical as fitnesses of resistant and non-resistant birds are assumed to be equal in the absence of disease. However, after this point, a difference in the sizes of these two populations is seen; with the addition of malaria, a considerable impact is felt by the low and mid elevation populations. Even before the effects of climate change, the total population size for lower-resistance birds is less than for that of the high-resistance birds. After 1950 the loss of the high elevation region due to climate change impacts greatly on both populations primarily due to the substantial loss of land (show by a decrease in high elevation population for both resistances); at this height both resistances display similar characteristics. At mid elevation there are minor declines for lower-resistant birds however numbers of honeycreepers remain relatively consistent for the more resistant species. At low elevations there is growth in the population sizes, again due to temperature isocline changes, however this does not necessarily indicate a net growth for the total population. By observing the total population size (black line), it is seen that for higher-resistance birds climate change leads to a slight net increase over time whereas there is a net decline for low-resistance species (Figure 3).

3.2.3. Varying Predation

Predation is known to have had a significant impact on the native fauna of Hawaii (van Riper and Scott, 2001), here the interaction of predation with infection is considered in detail; to elucidate the underlying mechanisms the impact of climate change is excluded. The expected trend of a negative correlation between total population sizes and predation is observed for the most part, although the impact of predation is somewhat weaker than expected and there are even regions of plausible parameter space for which increasing predation yields an increase in population size (see Figure 4). These results, which initially appear paradoxical, can be explained; it should be noted that for

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very high levels of predation (such as values of A above 1.5×10^6), whilst this is deleterious for honeycreeper population sizes, it does lead to extinction of malaria infection. This trade-off with high-levels of predation depressing the population size, but in turn reducing the force of infection and hence disease-induced mortality, is evident throughout parameter space.

There are distinct differences between the three elevations; the greatest impact of increasing predation is for high elevation populations due to the absence of malaria transmission at these altitudes. Low elevation populations respond differently to predation depending on whether high or low resistance honeycreeper species are considered. For low-resistance species increasing predation may even lead to an increased population size at low elevations by depressing the impact of infection, in contrast for high-resistance species (where infection has a more limited impact), lower predation results in a drop in population size however as predation becomes large the same beneficial effects are seen (due to essentially culling a substantial portion of the host reservoir of infection). Similar, but less marked effects can be observed at mid-elevation .

3.3. Conservation Strategies

Here the mathematical model is used to quantify the impact of several conservation strategies that have been suggested in the literature and outlined in the introduction. The relative honeycreeper population size at equilibrium with controls in place compared to the default model is examined; this is done for two strengths of control (reflecting a 5% and 50% change in parameters) as well as for low and high resistance populations and includes the effect of climate change.

Results of increasing b_H (corresponding to an increase in the birth rate via captive propagation programmes) show enormous benefit for high resistant species (Figure 5). However, the effect is far weaker for lower-resistance species as any increase in birth-rate leads to an associated rise in infection and therefore mortality.

Reducing the carrying capacity of mosquitoes (K_V) by 50% leads to vastly increased honeycreeper population sizes for both high and low resistance species. It should be noted that reducing the bite

rate (a) or the transmission probabilities (p_H or p_V) all achieve a reduced rate of transmission and hence very similar effects.

Wide-spread predation control corresponds to the reduction in parameter A_i (for all i) and so the previously seen behaviour (as in Figure 4) would be excepted; high predation rates can control levels of malarial infection within the population and therefore even substantial levels of prediction control have limited impact. With climate change incorporated over 100 years, loss of high elevation area to mid and mid to low results in predation control having a negative effect on population size for low-resistant populations, but still a positive one for high-resistant ones (Figure 5).

Alternatively predation control can be targeted solely at one elevation (changing A_i for just one i value); primarily it can be seen that while reducing the predation A_2 (just mid elevation) is predicted to leave total population sizes largely unchanged for high resistant birds, for less resistant species loss of predation is again detrimental to population sizes due to the interplay between infection and predation. Controlling predation at only high elevations (changing A_1) yields the outcome that would naturally be expected (due to the absence of transmission), with an increase in the total population of honeycreepers, albeit a very minor one.

Finally, increasing the carrying capacity at high elevation, K_1, shows potential over many of the other strategies, leading to increases in both low and high-resistant populations.

4. Conclusions

It has been proposed by Kilpatrick (2006) that predation control at mid elevations may allow the evolution of resistance to malaria when resistance is always genetically inherited. In contrast, the results here suggest that minimising predation at high elevation is more beneficial as predation has the greatest impact on population size in this region as predators are not experiencing interference from infection.

Facilitating predation control at high elevations is the only method that results in a positive change for both 10% and 35% resistant species and however this change is only marginally better than no

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control at all. These results suggest that whilst predation reduction may save some native species from extinction, it may not benefit those species which cannot readily confer genetic resistance to malaria. It also highlights that predation control may not be the most resource-efficient method to use; either blanket control or elevation specific. This model indicates that reducing the predation solely at mid elevation is not effective and may be of significance when planning control methods.

Despite only a small improvement to population sizes of low-resistant populations, captive propagation is still likely to be beneficial, especially as it is comparatively easy to implement. In contrast vector control has very good effects upon honeycreeper population sizes for both high a low resistant species but is much harder to apply in practise; it would be highly beneficial to find a suitable method to gain such control over the factors of mosquito carrying capacity, bite rate of transmission probabilities.

Finally the results indicate that habitat conservation and restoration might enable the protection of the non-resistant honeycreepers, thus preserving the heterogeneity within the population. For low-resistant populations this can be seen as an improvement over strategies such as predation which may even hinder growth or captive propagation which may be only mildly helpful.

5. Discussion

At the moment, a lack of field data for the honeycreeper population sizes prevents rigourous statistical fitting of the model, however the results (see Figure 3) do match currently accepted trends; according to a technical report by Camp et al. (2011) between the years of 1976-2006, both Apapane and Amakihi (higher resistant species) have shown stable or growing populations throughout their ranges on the island of Hawaii and are often detected at the high malaria-prevalent, low elevation, whereas the less resistant species, Iiwi, has declining numbers across the island and has a contracting area in which it is detected. Such changes match the basic qualitative predictions of the model derived from using two varying parameter pairs (θ, η) for high and low resistances and also indicates that resistance enables low elevation population survival congruent with much of the literature.

Mosquito control has commonly been used as a means of limiting human disease in many areas; reducing the mosquito population in the model shows similar benefits. However, implementing such a control strategy is an almost insurmountable task, given the ability of mosquitoes to breed in any small pools of standing water. The removal of the pigs (as suggested by VanderWerf (2001)) has potential benefits for both mosquito control and habitat conservation. It is noted, however, that such techniques would need be applied to a widespread area to facilitate general reduction in the mosquito population (Banko et al., 2001).

Both biologically and mathematically, this complex system is a challenging one to model. Throughout, relatively simple assumptions have been made to keep the model transparent and tractable. There are many other modelling concepts that could be included and these are now discussed.

Predation models of this type are additive in nature and do not take into account that some birds which are consumed by predators may have succumbed to disease-induced mortality or "natural" death anyway and may overestimate the deleterious effects on the population. Conversely, the assumption that Honeycreepers are preyed upon equally regardless of infection status my underestimate predation of birds, potentially more vulnerable due to the adverse effects of avian malaria. The predator population will likely fluctuate over time, however in this scenario it is assumed that these fluctuations are not driven by the Honeycreeper population. A full model including both predators and honeycreepers and a measure of vulnerability dependent on infection status would be of interest for future research.

The use of full stochastic equations (see Bailey (1982)) may lead to quantitatively better results for this relativity small population, potentially below the ambiguous threshold at which deterministic modelling becomes a good approximation of the stochastic dynamics. Even when modelling a larger population size, stochastic dynamics can arise from many sources, such as external climatic effects. Such noise term may be fairly easily be incorporated within the equations, but lead to a loss in transparency of the results and are difficult to parameterise.

Age structure of the honeycreeper population is also omitted from this model. Demographic aspects such as low juvenile survivorship for honeycreepers during their first year (Kilpatrick, 2006), varia-

tion in fecundity and age-dependant mortality for both honeycreepers and mosquitoes (Styer et al., 2007) have been identified in the field and could be dealt with either by higher-dimensional compartmental models (such as Kilpatrick (2006)) or a PDE type model with age-dependant parameters. Similarly, modifications could be made to births rates to include temporal forcing, leading to a more natural pulsed birth rate (Kilpatrick, 2006)); although the precise shape of such a function may need some consideration as would sensitivity to the functional form. However, given the general uncertainty surrounding many of the fundamental parameters it is questionable if such additional realism is justified.

The findings indicate a need for further data to be able to infer more accurately the value of parameters; moreover a need for better parameter estimation is necessary to gain accurate predictions. Parameters used are indicative of "typical" low and high resistant sub-species, which allows broad qualitative analysis across the honeycreeper family. As such the results show general behaviour of populations but unfortunately lack of precision leads to low confidence numerically. However, other parameters such at predation levels or the propensity of mosquitoes to feed on other species is less well determined and would require more field experiments to parameterise.

The use of microscopy to confirm malarial infection, where visual conformation of the parasites can confirm diagnosis, is effective for acute infections, but 70% or more of chronic infections may be missed due to low numbers of parasites (**?**). Other more costly and time-consuming procedures are more accurate at detecting chronic infections, but these may fail to spot the early stages of the disease and cannot distinguish between acute and chronic infections.

Due to the restricted range a mosquito can travel, transmission of disease between islands is assumed to be negligible. Since malaria is vector-borne even the migration of infectious birds will not cause spread of disease to areas with no mosquitoes (as shown with mosquito-free Laysan Island). Mixing between islands may be possible for the honeycreeper population but, as the distance between neighbouring islands is up to 80 miles (between Kaua'i and O'ahu), this is again assumed to be insignificant compared to within island mixing. This model focuses on individual islands as independent systems, in particular the dynamics on the island of Hawaii.

The modelling of the complex relationships between the Hawaiian honeycreepers, their predators, malaria and climate change has multiple benefits despite the inherent complexity. In particular, it enables an investigation of how conservation measures might affect the delicate balance between honeycreeper populations and their environment without endangering an already diminishing population; in this case indicating the potential advantages of habitat control and captive propagation over the other posed methods and highlighting the problems that could arise by facilitating predation control. Moreover, the act of formulating a mathematical model forces a crystalisation of the assumptions regarding the basic biological processes, while repeated simulation helps to highlight parameter sensitivity and the impact of parameter uncertainty, suggesting new directions for future field or laboratory studies. Finally, although Hawaiian honeycreepers have faced (and continue to face) numerous challenges, the models developed here suggest that there is the potential for the species to be saved if conservation measures are carefully targeted.

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Table 1: Wild pop honeycreepers still extant (or recently extinct), CE - critically endangered, E - endangered, V - vulnerable, LC - Least concern, Ex - Thought to be extinct

Subfamily	Genus	Scientific Name	Common Name	Approx. No. (Camp et al., 2011)	Status (?)
Drepanidini	Himatione	Himatione sanguinea	Apapane	1300000	LC
	Melamprosops	Melamprosops phaeosoma	Po'ouli	0	Ex
	Palmeria	Palmeria dolei	Crested honeycreeper	3800	CE
	Vestiaria	Vestiaria coccinea	Iiwi	360000	V
Hemignathini	Hermignathus	Hermignathus flavus	O'ahu 'Amakihi	50000	V
		Hermignathus kauaiensis	Kaua'i 'Amakihi	51000	V
		Hemignathus lucidus	Nukupu'u	0	Ex
		Hermignathus virens	Hawai'i 'Amakihi	800000	LC
		Hemignathus parvus	Anianiau	37500	V
		Hemignathus munroi	Akiapola'au	1900	E
	Loxops	Loxops caeruleirostris	'Akeke'e	7900	CE
		Loxops coccineus coccineus	Hawai'i 'Akepa	12000	E
		Loxops coccineus ochraceus	Maui 'Akepa	0	Ex(1988)
		Loxops coccineus wolstenholmei	O'ahu 'Akepa	0	Ex
	Oreomystis	Oreomystis bairdi	'Akikiki	3600	CE
		Oreomystis mana	Hawai'i Creeper	14000	E
	Paroreomyza	Paroreomyza montana	Maui Nui 'Alauahio	35000	E
Psittirostrini	Loxioides	Loxioides bailleui	Palila	3900	CE
	Pseudonestor	Pseudonestor xanthophrys	Maui parrotbill	500	CE
	Psittirostra	Psittirostra psittacea	'O'u	0	Ex(1987)
	Telespyza	Telespyza cantans	Laysan finch	5000-20000(?)	V
		Telespyza ultima	Nihoa finch	2800(?)	CE

Table 2: Parameters and their estimates used in the numerical analysis of the model (unless otherwise specified)

Parameters	Description	Estimate
b_H	birth rate of honeycreepers	$1.5/year$ [a]
d	"natural" death rate	$0.25/year$ [a]
$\hat{L}_i$	initial area of the three temperate regimes	$\begin{cases} 4 \times 10^5 & i = 1 \\ 10 \times 10^5 & i = 2 \\ 14 \times 10^5 & i = 3 \end{cases}$
$\hat{K}_i$	natural carrying capacity per area	1 for all i
$\hat{A}_i$	predation rate per area	0.1 for all i
$\hat{B}_i$	relative predation switching point	0.548 for all i
D	malaria mortality rate	$17.4/year$ [b]
ω	period of forcing (disease)	2π
η	probability of inherited resistance	-
θ	probability of chance resistance	-
$\hat{K}_{V,i}$	carrying capacity of mosquitoes per area	500 for all i
d_V	mosquito birth/death rate	$11.8/year$ [c]
α_0	half maximum bite rate	$45.5/year$ [d]
α_1	relative amplitude of seasonality	1
p_H, p_V	probability of infection for hosts/vectors	0.8 for H and V
$\hat{O}_i$	relative abundance of "other" hosts	60
ε_{ij}	rate at which honeycreeper move from region i to region j	$\begin{cases} 0.05 & |i - j| = 1 \\ -0.1 & i = j \\ 0 & \text{otherwise} \end{cases}$
c_i	rate of loss of area i	$\begin{cases} 0.006 & i = 1, 2 \\ 0 & i = 0, 3 \end{cases}$ [e]

[a] Uses data for Amakihi (Kilpatrick, 2006)
[b] (?)
[c] Estimates taken from life expectancy of *Aedes aegypti* (Styer et al., 2007)
[d] Estimates taken from bite rate of *Aedes aegypti* (McClelland and Conway, 1971)
[e] This approximate rate is based upon predictions for loss of forested habitat over the next 100 years (Benning et al., 2002)
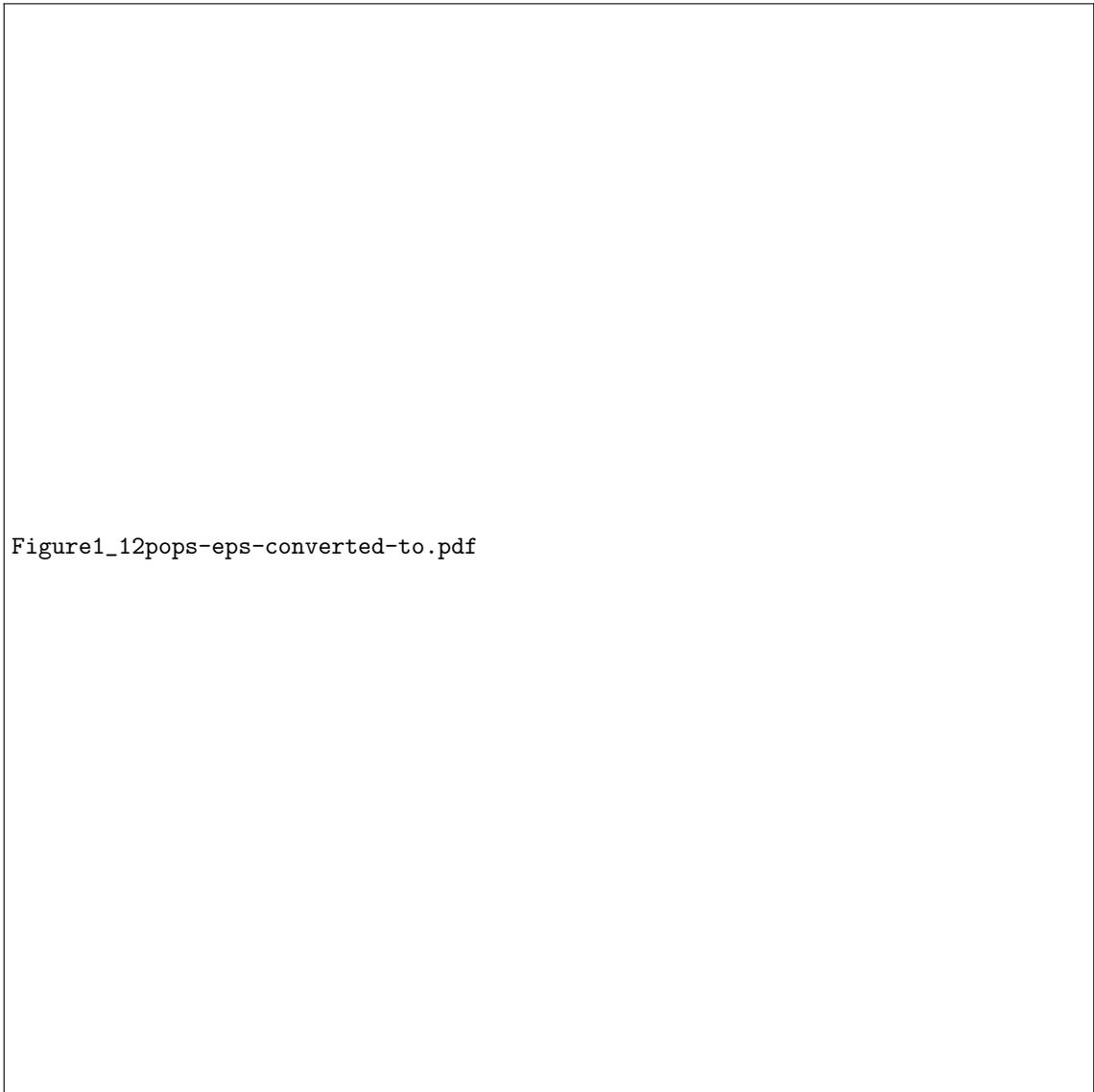
Figure 1: Results of simulation demonstrating the variations at each elevation of numbers of resistant/non-resistant and susceptible/infected honeycreepers. Top, middle and bottom rows correspond to high, mid and low elevations; left and right columns correspond to non-resistant and resistant birds respectively. $((\theta, \eta) = (0.15, 0.03)$; $c_i = 0$ so as to observe effects of malaria on the population in the absence of climate change).
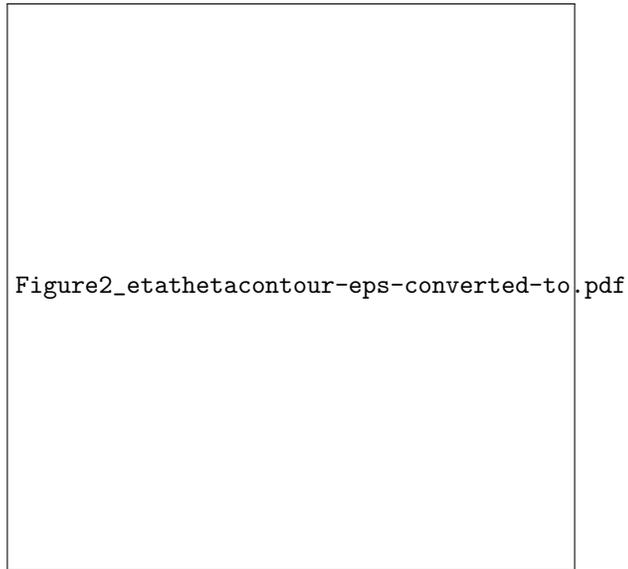
Figure 2: Region of (θ, η) parameter space that yields overall resistances (as a percentage) which are assumed to be at equilibrium by the end of a 100 year simulation. Documented resistance values of 10% and 35% resistant birds in the population are highlighted. Again the effects of climate change are not included here ($c_i = 0$).

Figure3a_Intro35res-eps-converted-to.pdf

Figure3b_Intro10res-eps-converted-to.pdf

(a) High resistance $(\theta, \eta) = (0.03, 0.25)$

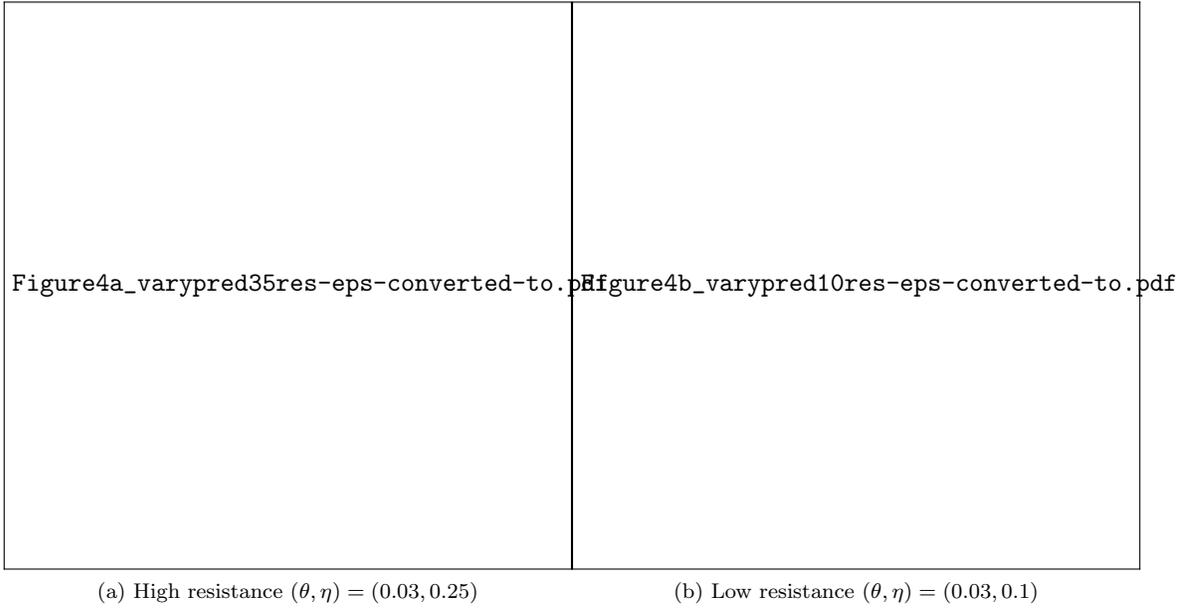(b) Low resistance $(\theta, \eta) = (0.03, 0.1)$

Figure 4: Changes to the equilibrium population size as predation is varied for both high (left column) and low (right column) resistant species. Vertical dashed lines indicate where the chosen value of predation, A, used in the other simulations lies.

(a) High resistance $(\theta, \eta) = (0.03, 0.25)$ (b) Low resistance $(\theta, \eta) = (0.03, 0.1)$

Figure 5: Results of varying controllable parameters to promote population levels of the honey-creeper. The parameters varied are shown on the x-axis, while the bars represent the relative increase in the total population size at equilibrium at the new parameter values compared to the default. For the honeycreeper birth rate, b_H, and the carrying capacity at high elevations, K_1, the conservation strategy involves increasing these parameters by 5 and 50%, while all other conservation parameters K_V and A are decreased by similar percentages.