



The role of native and introduced birds in transmission of avian malaria in Hawaii

KATHERINE M. MCCLURE ^{1,2,3,4} ROBERT C. FLEISCHER ² AND A. MARM KILPATRICK ¹

¹Department Ecology and Evolutionary Biology, University of California, 130 McAllister Way, Santa Cruz, California 95060 USA

²Center for Conservation Genomics, Smithsonian Conservation Biology Institute, Washington, USA

Citation: McClure, K. M., R. C. Fleischer, and A. M. Kilpatrick. 2020. The role of native and introduced birds in transmission of avian malaria in Hawaii. *Ecology* 101(7):e03038. 10.1002/ecy.3038

Abstract. The introduction of nonnative species and reductions in native biodiversity have resulted in substantial changes in vector and host communities globally, but the consequences for pathogen transmission are poorly understood. In lowland Hawaii, bird communities are composed of primarily introduced species, with scattered populations of abundant native species. We examined the influence of avian host community composition, specifically the role of native and introduced species, as well as host diversity, on the prevalence of avian malaria (*Plasmodium relictum*) in the southern house mosquito (*Culex quinquefasciatus*). We also explored the reciprocal effect of malaria transmission on native host populations and demography. Avian malaria infection prevalence in mosquitoes increased with the density and relative abundance of native birds, as well as host community competence, but was uncorrelated with host diversity. Avian malaria transmission was estimated to reduce population growth rates of Hawai'i 'amakihi (*Chlorodrepanis virens*) by 7–14%, but mortality from malaria could not explain gaps in this species' distribution at our sites. Our results suggest that, in Hawaii, native host species increase pathogen transmission to mosquitoes, but introduced species can also support malaria transmission alone. The increase in pathogen transmission with native bird abundance leads to additional disease mortality in native birds, further increasing disease impacts in an ecological feedback cycle. In addition, vector abundance was higher at sites without native birds and this overwhelmed the effects of host community composition on transmission such that infected mosquito abundance was highest at sites without native birds. Higher disease risk at these sites due to higher vector abundance could inhibit recolonization and recovery of native species to these areas. More broadly, this work shows how differences in host competence for a pathogen among native and introduced taxa can influence transmission and highlights the need to examine this question in other systems to determine the generality of this result.

Key words: community composition; demography; dilution effect; distribution; Hawaiian honeycreeper; introduced species; reservoir.

INTRODUCTION

Changes in the abundance and distribution of native biodiversity and the widespread introduction of nonnative species have resulted in substantial changes in community composition throughout most habitats on earth (Dornelas et al. 2014, Newbold et al. 2015). How these widespread changes in communities will affect disease is a key unanswered question for the 21st century (Young et al. 2016). Host community composition plays a fundamental role in regulating pathogen transmission in multi-host disease systems because species vary in

contact rates, competence, and abundance (LoGiudice et al. 2003, Kilpatrick et al. 2006a, Hoyt et al. 2018). However, our understanding of the role of introduced and native species in pathogen transmission is still surprisingly limited (Young et al. 2016). Detailed studies of transmission in host communities that examine the roles of native and introduced species are needed to increase our understanding of how species turnover will impact disease ecology.

Species introductions can influence multi-host vector-borne pathogen transmission by several mechanisms that depend on how introductions change host and vector communities in abundance, competence, contact rates, and other traits (Keesing et al. 2006, Young et al. 2016, Kilpatrick et al. 2017). One hypothesis suggests that life-history traits that favor rapid growth and reproduction might reduce allocations to immune function and result in species that are both more likely to become invasive and have higher host competence, or capacity to

Manuscript received 15 February 2019; revised 9 January 2020; accepted 30 January 2020. Corresponding Editor: Viviana Ruiz-Gutierrez.

³Present address: Cornell Wildlife Health Center and Cornell Atkinson Center for Sustainability, Cornell University, Ithaca, New York, 14850USA

⁴E-mail: kat.m.mcclure@gmail.com

maintain and transmit pathogens (van Kleunen et al. 2010, Young et al. 2016). Empirical evidence for this hypothesis is limited and includes mixed results (Johnson et al. 2009, Ostfeld et al. 2014). A second hypothesis suggests that native species that have been isolated on islands with depauperate parasite communities will evolve lower immune function (Wikelski et al. 2004). These species are hypothesized to have higher infection intensity resulting in higher host competence for introduced pathogens compared to introduced species that have coevolved with the introduced pathogen (van Riper et al. 1986, Wikelski et al. 2004, Matson 2006). Empirical evidence for this hypothesis has also been mixed (Matson 2006, Beadell et al. 2007). Finally, the dilution effect hypothesis, which suggests that disease risk is reduced by increases in species richness, predicts either a decrease or increase in transmission depending on the net gain or loss of species following species introductions (Sax et al. 2002, Keesing et al. 2006, Vilà et al. 2011). Few studies have been carried out that measure pathogen transmission intensity and host community competence in communities with native and introduced host communities (Young et al. 2016). Even fewer account for factors that co-vary with changes in host community composition that is often associated with anthropogenic habitat alteration such as increased vector abundance (Andreadis et al. 2004, Kilpatrick et al. 2017, Kovach and Kilpatrick 2018, McClure et al. 2018). Here we examine the role of native and introduced birds in the transmission of avian malaria in Hawaii.

Avian malaria is caused by multi-host mosquito-borne protozoan parasites in the genera *Plasmodium* (Bennett et al. 1993, Valkiunas 2005). Avian malaria parasites are present on most continents and many islands, including the main Hawaiian Islands, and commonly infect both introduced and native bird species (Bennett et al. 1993, Valkiunas 2005, Howe et al. 2012, Ventim et al. 2012, Clark et al. 2014). *Plasmodium relictum*, the causative agent of avian malaria in Hawaii, was likely introduced in the 20th century and is transmitted by the introduced southern house mosquito, *Culex quinquefasciatus* (van Riper et al. 1986, LaPointe et al. 2005). Transmission of avian malaria and avian pox (*Avipoxvirus* spp.; Van Riper et al. 2002) caused the extirpation of most native bird populations from areas of Hawaii below 1,200 m, in conjunction with habitat loss and introduced predators (Warner 1968, Atkinson 1977, Olson and James 1982, Scott et al. 1986, van Riper et al. 1986, Atkinson et al. 1995). Avian malaria transmission continues to contribute to rapid declines of multiple bird species as climates warm (Atkinson et al. 2014, Paxton et al. 2016). As native bird species disappeared from lowland and mid-elevation forests, more than 150 exotic bird species were deliberately introduced to Hawaii, resulting in at least 54 nonnative bird species becoming established on one or more of the main Hawaiian islands (Long 1981, Moulton and Pimm 1983, Pyle 2002). Most bird communities in low elevation forests in Hawaii are now

composed of entirely introduced species. However, there are two species of endemic Hawaiian honeycreeper (Fringillidae; subfamily Drepanidinae), the Hawai'i 'amakihi, *Chlorodrepanis virens*, and 'apapane, *Himatione sanguinea*, that persist at moderate abundance in lowland forest fragments (Scott et al. 1986, Woodworth et al. 2005, Spiegel et al. 2006). This landscape of forested areas with avian communities composed of either entirely introduced bird species or mixed native and introduced bird species provides an opportunity to examine the role of native and introduced species in the transmission of an important vector-borne pathogen, avian malaria.

In lowland Hawaii, variation in host community composition, particularly native and exotic bird species abundance, likely influences the transmission of *P. relictum*. Native Hawaiian birds appear to be more competent hosts for avian malaria than introduced species. Parasitemia, the concentration of the parasite in blood, increases avian malaria host infectivity to mosquitoes in *P. relictum*, and contributes to host competence (Pigeault et al. 2015). Parasitemia is very high in Hawaiian honeycreepers during the acute phase of infection (i.e., 3–38% of red blood cells are infected in the 30 d post-infection in 'amakihi and 'apapane; Atkinson et al. 2000, Yorinks and Atkinson 2000), whereas parasitemias in introduced bird species are usually much lower and are thought to be transient in the introduced species that have been studied (van Riper et al. 1986, Atkinson et al. 1995, Samuel et al. 2011). Native birds that survive the acute phase are chronically infected for life and likely continue to infect a fraction of biting mosquitoes (Atkinson et al. 2001). This suggests that malaria transmission from hosts to mosquitoes should increase with higher native bird density, whereas in communities composed solely of introduced birds host competence might be too low to sustain transmission (van Riper et al. 1986). However, anthropogenic land use that facilitates invasive species also increases the abundance of some mosquito species (Andreadis et al. 2004, Gottdenker et al. 2014), including *Cx. quinquefasciatus* in Hawaii (McClure et al. 2018), and with increased mosquito abundance, transmission may be sustained or even higher in fully exotic avian communities.

We examined drivers of malaria infection in mosquitoes and birds across a gradient of native and introduced host species composition in lowland Hawaii. We tested the hypothesis that avian malaria prevalence in mosquitoes increases with host community competence, which increases with the density of native birds. We also examined the effect of spatial variation in malaria transmission on the population growth rate of the most abundant native bird in lowland Hawaii, the Hawai'i 'amakihi, using a demographic model. We hypothesized that avian malaria in lowland forests limits the distribution of Hawai'i 'amakihi (which suffers mortality from acute and chronic infections; Kilpatrick 2006, Atkinson et al. 2013), such that 'amakihi density would decrease

with the abundance of malaria-infected mosquitoes, and 'amakihī would be absent in areas with the highest malaria transmission.

MATERIAL AND METHODS

Study sites

We sampled birds and mosquitoes at eight forested sites ranging from 18 to 349 m in elevation on the Island of Hawai'i between May and August in each of three years, 2011–2013 (Appendix S1: Fig. S1). We selected sites so that four had appreciable numbers of native birds and four did not. Sites had similar vegetative communities that were dominated by native 'ōhi'a (*Metrosideros polymorpha*) trees that provide food and nesting sites for many native and nonnative birds (Baldwin 1953, Kern and van Riper 1984). The understory was composed of shrubs, small trees, and ferns, including the native fern, uluhe (*Dicranopteris linearis*), and invasive shrubs such as strawberry guava (*Psidium cattleianum*), melastoma (*Melastoma septemnervium*), and Coster's curse (*Clidemia hirta*) (Zimmerman et al. 2008). Average monthly temperatures ranged from 21.9°C to 24.6°C, and annual cumulative rainfall ranged from 2,363 to 3,876 mm across the study area (Giambelluca et al. 2013). *Cx. quinquefasciatus* are present and active year-round, and avian malaria transmission is thought to exhibit somewhat limited seasonality in low elevation forests where it has been previously studied (Ahumada et al. 2004, Woodworth et al. 2005, Samuel et al. 2011).

Mosquito capture

To estimate relative mosquito abundance and avian malaria infection prevalence in mosquitoes, we collected mosquitoes using modified CDC Miniature Light Traps and CDC gravid traps (John W. Hock, Gainesville, Florida, USA), which target adult female host-seeking and egg-laying mosquitoes, respectively. Light traps were baited with ~ 1 kg of dry ice (CO₂) and gravid traps were baited with hay-infused water. Gravid traps attract species such as *Cx. quinquefasciatus* that utilize organic-rich water for oviposition (egg-laying) sites (Reiter 1983, Bentley and Day 1989). One CO₂ and one gravid trap were set at sunset at each of four to six sampling stations at each site every three to four weeks. Trapped mosquitoes were killed by placing them in a –80°C freezer and then sorted by species before being stored in a –80°C freezer for later analysis. We quantified the abundance of host-seeking *Cx. quinquefasciatus* using the number of mosquitoes collected per CO₂-baited trap-night. We estimated malaria infection prevalence in mosquitoes by testing individual whole-body mosquitoes or pools of two to five mosquitoes caught in either gravid and CO₂ traps (see below for details). We tested a subset of mosquitoes from each site (10–20% of trapped mosquitoes)

until we obtained an estimate of prevalence with a coefficient of variation of approximately 0.19–0.36.

Bird censuses and bird capture

We conducted unlimited-distance point counts and used distance sampling to estimate bird abundance (Thomas et al. 2010). Surveys were conducted at five or six fixed sampling points per site two or three times between May and August in 2012 and 2013, between the hours of 05:00 am and 11:00 am. Points were >150m from one another and were established randomly within each site. We estimated densities of each bird species for which there were >50 detections using the generalized hierarchical distance-sampling model fitting functions in package unmarked (Fiske and Chandler 2011) in program R (version 3.31). These models incorporate abundance and detection covariates into distance sampling models and accommodate replicated distance sampling observations (e.g., multiple visits per season; Fiske and Chandler 2011). We included forest canopy structure (open or closed) and site as a covariate to the detection and abundance parameters, respectively. We fit half-normal, hazard, and exponential detection functions for each species separately, added covariates to the model with the best-supported detection function, and selected the best-fitting model using Akaike's information criterion (AIC; Akaike 1973). We estimated site-level host diversity using the Shannon diversity index and Chao's richness estimator, a non-parametric method for estimating species richness that accounts for imperfect detection by using information on rare species to adjust for undetected species (Chao 1984, Oksanen 2013).

To examine malaria infection prevalence in birds, we captured birds at six of the eight sites using 12 m long 38-mm mesh mist nets from May to August in 2012 and 2013, and at a seventh site in February 2014. We aged and sexed birds using morphometric and plumage characteristics (Pyle 1997, Woodworth et al. 2005) and banded them with an aluminum U.S. Fish and Wildlife leg band for individual identification. We drew approximately 0.1ml of blood by brachial venipuncture. Whole blood was spun in the field in a centrifuge to separate red blood cells from plasma. Red blood cells were placed in lysis buffer (0.1 mol/L Tris, 0.1 mol/L EDTA, 0.01 mol/L NaCl, and 0.5% SDS, pH 8) and stored in a –80°C freezer. Work was performed under BBL banding permit 23600 and the University of California Santa Cruz IACUC protocol kilpm112.

Laboratory methods

We extracted DNA from bird blood samples and mosquito pools using Qiagen DNeasy Blood and Tissue Kits (Qiagen, Hilden, Germany) and the Qiagen tissue extraction protocol with an overnight incubation period. We ground one to five whole-bodied *Cx. quinquefasciatus* prior to incubation using a sterilized 7-cm

polypropylene pestle or by homogenizing the mosquitoes using steel balls and a Bullet Blender Homogenizer bead mill (Next Advance Lab Instruments). We screened samples for *P. relictum* using primers that target a 160-bp non-coding region of mitochondrial ribosomal RNA of avian haemosporidians (213F/372R; Beadell and Fleischer 2005). PCR bands were visualized using gel electrophoresis of ethidium bromide- or GelRed- stained agarose gels, and we included positive and negative controls for the extraction and PCR reactions. We estimated prevalence of avian malaria in groups of pooled samples of one to five mosquitoes by maximum likelihood (Walter et al. 1980).

Statistical analysis

To examine the influence of avian host community composition on avian malaria transmission, we analyzed infection prevalence in pooled samples of *Cx. quinquefasciatus* using generalized linear mixed models (package lme4 in R v3.31; Bates et al. 2015, R Core Team 2017) with a binomial distribution, a logit link, and used log (pool size) as an offset. We examined nine predictors by fitting mixed effects models (with site as a random effect to account for repeated sampling over time) with a single fixed effect in each, to avoid overfitting. Predictors included the density or relative abundance of native birds, estimated bird species richness or Shannon diversity, community competence, *Cx. quinquefasciatus* abundance, Hawai'i 'amakihi density or relative abundance, and the density and relative abundance of the most common introduced species, the Japanese White-eye (*Zosterops japonicus*). We examined both bird density and relative abundance as predictors of mosquito infection prevalence because density is more relevant if mosquitoes search for hosts by randomly moving through space and choosing to bite (or not bite) hosts as they encounter them, and relative abundance is more relevant if mosquitoes are able to detect multiple hosts from further away and actively seek out species they prefer (Takken and Verhulst 2013). There was little evidence that infection prevalence in mosquitoes varied significantly among sampling years or with date within a year (all $P > 0.08$ for year, date and date² terms, in additive and interactive models with other fixed effects, Appendix S1: Fig. S2), so we did not include fixed effects for these predictors, but results including these temporal predictors were qualitatively identical. We combined mosquito infection prevalence data from gravid and CO₂ traps because trap type was not statistically significant in any models (all $P > 0.06$) and results were qualitatively similar when including trap type. We evaluated the relative support of infection prevalence models using AIC (Akaike, 1973).

We estimated avian host community competence using published parasitemia data from >2,000 individuals of five native and five introduced bird species (van Riper et al. 1986). Species-specific host competence was

estimated as the average monthly parasitemias for each reported species found at our sites (van Riper et al. 1986), for all months where parasitemias were non-zero. Ideally, we would have estimated host competence for each species as the integral of the parasitemia over time from experimental infection studies, translated into infectiousness to biting mosquitoes (Kilpatrick et al. 2007). However, avian malaria experimental infection studies are lacking for most introduced species and no study has quantified vector competence across a range of host parasitemia. Nonetheless, the large sample sizes obtained by van Riper et al. provide a relatively robust estimate of the parasitemias of species captured in the field during that study, and qualitatively match the contrast in parasitemia between native and introduced birds observed in the species that have been experimentally infected (van Riper et al. 1986, Atkinson et al. 1995). We calculated site-level host community competence by summing the product of site- and species-specific relative abundance estimates and species-specific competence estimates, divided by the total site-level bird abundance. We took the mean parasitemia of the five introduced bird species present at our study sites as the estimated competence of the introduced bird species that were not sampled in van Riper et al. (van Riper et al. 1986). The elevated uncertainty in estimates of host competence should reduce the power to detect a correlation with mosquito infection prevalence, rather than increase it. To explore differences in parasitemias in native and introduced bird species, we analyzed untransformed and log-transformed parasitemia data from van Riper et al. (1986), separately, using general linear mixed models, with native or introduced origin as a fixed effect, and month of sampling and species as random effects.

To estimate disease risk to the avian community, we calculated the abundance of infected mosquitoes by multiplying mosquito infection prevalence by host-seeking mosquito abundance from CO₂ traps. Abundance of infected mosquitoes, sometimes referred to as entomological risk or vector index, is often strongly correlated with host incidence over space (Kilpatrick et al. 2006b, Kilpatrick and Pape 2013). We calculated a single site-level abundance of infected mosquitoes (across all years) as the average of annual estimates of abundance of infected mosquitoes, weighted by the inverse of the squared standard error of the estimate. We used simple linear correlations to examine relationships between mosquito abundance, the abundance of infected mosquitoes, and Hawai'i 'amakihi density. We analyzed malaria infection prevalence in Hawai'i 'amakihi and Japanese White-eye separately using generalized linear mixed models with a binomial distribution and a logit link, with abundance of infected mosquitoes as a fixed effect and site as a random effect, to examine the relationship between the abundance of infected mosquitoes and malaria infection prevalence in these species. We analyzed avian malaria infection prevalence in Hawai'i 'amakihi and Japanese White-eye using generalized

linear mixed models with a binomial distribution and a logit link, with species as a fixed effect and site as a random effect, to explore differences in malaria infection prevalence between these two species.

To examine if spatial variation in malaria transmission impacts the distribution of native birds in lowland Hawaii, we estimated site-specific per capita population growth rates for ‘amakihi that incorporated malaria-induced mortality and site-specific ‘amakihi infection prevalence rates. We calculated per capita population growth rate (Caswell 2001, Kilpatrick 2006) at each site using

$$\lambda = S_A(1 - I\mu) + FS_J(1 - I\mu) \quad (1)$$

here S_A and S_J are adult and juvenile disease-independent annual survival rates, respectively (0.75 ± 0.29 and 0.35 ± 0.46 [mean \pm SE], estimated for a site ~36 km away in Hawaii Volcanoes National Park; Kilpatrick 2006). This adult survival rate is similar to an estimate of annual population survival of lowland ‘amakihi, in the absence of malaria mortality, using data collected at three of our four sites where ‘amakihi were abundant (0.73 ; Samuel et al. 2015). I is the annual force of infection, or per capita probability of becoming infected (see equation 2), μ is the disease infection fatality ratio for ‘amakihi from low elevation populations (Atkinson et al. 2013), and F is the annual per capita fecundity (number of females produced by each female, 1.56 ± 0.11 , which was unaffected by chronic malaria infections; Kilpatrick 2006). We estimated the force of infection, I , from measured malaria prevalence, p , after correcting for acute mortality due to malaria, μ (Komar et al. 2005)

$$I = p / (1 - \mu + (p\mu)) \quad (2)$$

This expression is accurate as long as acute malaria mortality is <100%; laboratory infection studies suggest that μ for lowland ‘amakihi is 0.17 (Atkinson et al. 2013). We estimated uncertainty in λ using a Taylor series expansion approximation (Kilpatrick 2006).

RESULTS

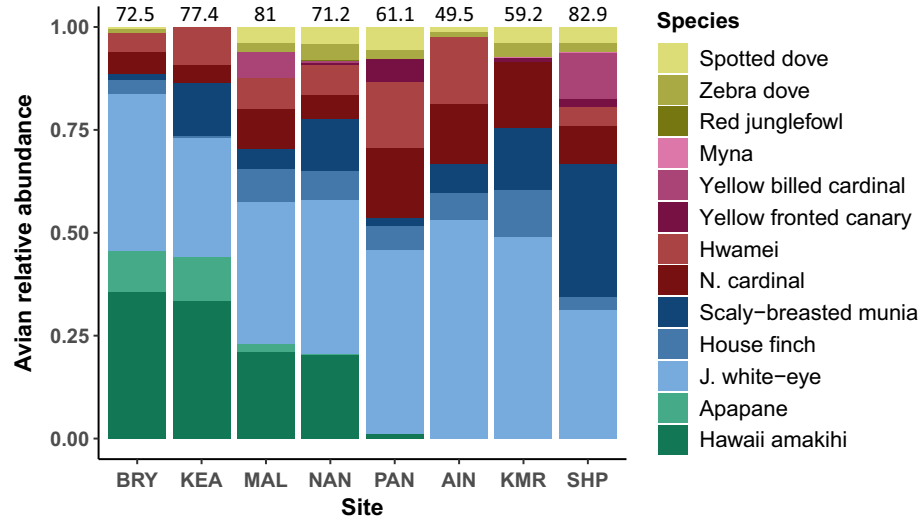
The bird censuses indicated that at four of the sites, native birds, primarily Hawai‘i ‘amakihi and ‘apapane, made up 21–46% of the avian community, whereas the other four sites were composed of almost entirely introduced species (Fig. 1). Japanese White-eye was the dominant introduced species, making up 29–53% of all birds across all eight study sites (Fig. 1). Other species included introduced birds from Asia (e.g., Hwamei or Melodious Laughing Thrush, *Garrulax canorus* and Scaly-breasted Munia or Nutmeg Mannikin, *Lonchura punctulata*), North America (e.g., Northern Cardinal, *Cardinalis cardinalis* and House Finch, *Haemorhous mexicanus*), South America (e.g., Yellow-billed Cardinal,

Paroaria capitata), and Africa (e.g., Yellow-fronted Canary, *Serinus mozambicus*; see Appendix S1: Table S1 for all bird species observed during point counts).

We caught 16,664 *Cx. quinquefasciatus* mosquitoes in CO₂ and gravid traps during 1,585 trap nights from 2011–2013, and screened 1,275 individual mosquitoes (mean = 156.9 mosquitoes screened per site, SD = 61.9, range = 92–288). Infected *Cx. quinquefasciatus* were captured at all sites and mosquito infection prevalence ranged eight-fold across sites (Fig. 2a, b).

Avian malaria infection prevalence in mosquitoes was significantly correlated with four variables in univariate analyses that were themselves correlated (Figs. 2a, b,3, and Appendix S1: Fig. S3). Mosquito infection prevalence increased with the density (and relative abundance) of native birds (Fig. 2a), increased with host community competence (Fig. 3), decreased with mosquito abundance (Fig. 2b), and decreased with the relative abundance of Japanese White-eye (logit (prevalence) = $0.85 - 7.5x$ white-eye relative abundance slope SE = 2.62; $P = 0.004$), but not with white-eye density ($P = 0.57$). The positive correlation with native birds suggested that prevalence in *Cx. quinquefasciatus* would be, on average, 69% lower if native birds were not present at the four sites where they currently exist in appreciable numbers (Fig. 2a). Host species diversity was not significantly correlated with avian malaria infection prevalence in mosquitoes or the abundance of infected mosquitoes, regardless of whether diversity was measured as estimated species richness or Shannon diversity (Fig. 4). See Appendix S1: Table S2 for AIC model selection results.

Hawai‘i ‘amakihi density was negatively correlated with both mosquito abundance (Fig. 2c) and the abundance of infected mosquitoes (Fig. 2d). Infection prevalence in Hawai‘i ‘amakihi was significantly higher than in Japanese White-eyes (‘amakihi average across sites \pm SE = 0.75 ± 0.05 , $N = 73$; Japanese White-eyes = 0.20 ± 0.02 , $N = 345$; species coefficient: ‘amakihi compared to White-eye = 2.48 ± 0.32 , $N = 418$, $P < 0.001$). Infection prevalence in Hawai‘i ‘amakihi and Japanese White-eyes increased with the abundance of infected mosquitoes, but the relationship was not significant for either species, possibly due to lower power from the small sample size of sites with Hawai‘i ‘amakihi ($N = 4$) or the small sample size of each species sampled (Appendix S1: Fig. S4). The variation we observed in malaria transmission was projected to reduce ‘amakihi per capita population growth rate from 1.3 (± 0.42) with no malaria transmission to 1.19 (± 0.79) – 1.1 (± 0.94) as malaria prevalence in ‘amakihi increased from 0.46 (± 0.02 , $N = 13$) to 0.85 (± 0.005 , $N = 26$) with estimated yearly probability of infection of 0.4 (± 0.05 , $N = 5$) and 0.77 (± 0.01 , $N = 13$), respectively, based on prevalence in young of the year birds). If the probability of infection was 100% (i.e., all birds were infected with malaria in their first year), ‘amakihi per capita population growth rate would be 1.07.



(FIG. 1). Relative abundance of bird species at eight sites in Hawaii based on point counts and distance sampling. Native Hawaiian birds are shown in green, and all other colors represent introduced species. Numbers at the top of bars indicate the total bird density at that site, calculated as the sum of all species' density estimates at each site, in individuals/ha. Rare species for which density estimates could not be accurately estimated are not included. AIN, Ainako UH-Hilo; BRY, Bryson's Cinder Cone (Pu'u Kali'u); KEA, Keau'ohana Forest Reserve; KMR, Keaukaha Military Reservation; MAL, Malama Kī Forest Reserve; NAN, Nānāwale Forest Reserve; PAN, Pana'ewa Waiakea Forest Reserve; SHP, W.H. Shipman property

DISCUSSION

We found that mosquito infection prevalence increased significantly with the density and relative abundance of native birds, despite mosquito abundance being two orders of magnitude lower at sites with native birds (Appendix S1: Fig. S5). The positive correlation with native bird abundance suggests that native Hawaiian birds increase pathogen transmission to mosquitoes in lowland areas where they occur (Atkinson et al. 2005, Atkinson and Samuel 2010, Samuel et al. 2011, Hobbs et al. 2012), and that the abundance of nonnative bird species overall, including individual species such as Japanese White-eyes, decrease transmission in this system (Dobson 2004, Samuel et al. 2011; Appendix S1: Fig. S5). Three lines of evidence support this idea. First, Hawai'i 'amakihi had high infection prevalence at our sites (0.75 ± 0.003 [mean \pm SE]) and in an earlier study at some of the same sites (0.85; Woodworth et al. 2005, Samuel et al. 2015), suggesting they are frequently fed on by infected mosquitoes. Second, although published experimental infection studies for many abundant exotic birds in lowland Hawaii are lacking, native species appear to be more competent hosts for avian malaria than most introduced species (Appendix S1: Fig. S6). These data do not support the hypothesis that invasive species, which often have high reproductive capacity (van Kleunen et al. 2010), also have higher host competence. Third, infection prevalence in mosquitoes was negatively correlated with the relative abundance of Japanese White-eyes. We found no support for the dilution effect hypothesis that suggests that disease risk decreases with host diversity or species richness. Instead,

in our work, details of host community composition (i.e., avian host identity, abundance, and competence) and data on vector abundance were needed to understand variation in malaria transmission to mosquitoes and disease risk (the abundance of infected vectors), respectively (Appendix S1: Fig. S5).

Counterintuitively, we found that mosquito infection prevalence was negatively correlated with mosquito abundance. This suggests that other factors that were themselves negatively correlated with mosquito abundance, including native host abundance and host competence, outweighed the positive effect of increasing mosquito abundance on avian malaria transmission expected from theory (MacDonald 1952). Other factors likely also influence pathogen transmission at our sites, such as mosquito survival and vector competence. In addition, variation in the abundance of different introduced bird species also likely influence transmission because some introduced species appear to be more infectious than others (Appendix S1: Fig. S6). Determining the precise role of each species in transmission will require three types of studies: (1) experimental infections of unstudied introduced species; (2) quantifying the relationship between host parasitemia and vector competence to translate parasitemia data into infectiousness; and (3) documenting mosquito-feeding patterns in relation to host abundance.

We found that the abundance of infected mosquitoes was negatively associated with Hawai'i 'amakihi density, but in contrast to our hypothesis, mortality from malaria transmission alone could not explain the absence of 'amakihi from sites where they were not present. Our calculations suggest that although increased

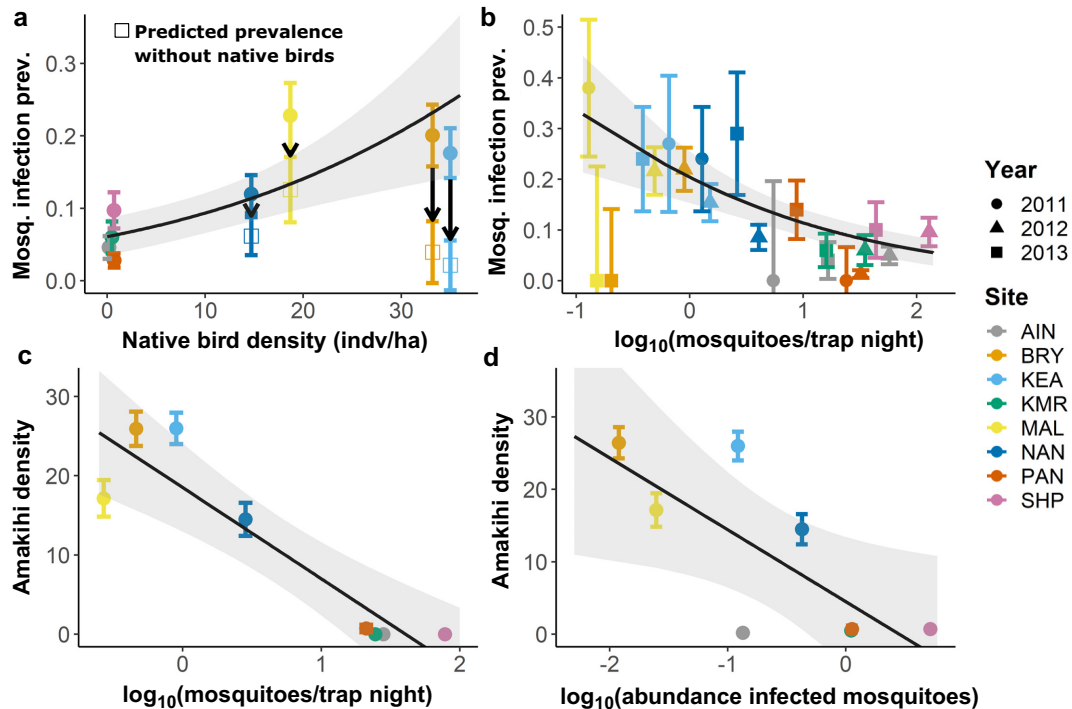


FIG. 2. Avian malaria infection prevalence in mosquitoes, mosquito abundance, and native bird density Slope SE = 0.011. (a) Prevalence of avian malaria in mosquitoes plotted against the density of the native birds. Solid circles are maximum likelihood estimates of prevalence in mosquito pools from individuals captured in gravid and CO₂ traps from 2011–2013 (± 1 binomial SE). The fitted line ($\text{logit}(\text{prevalence}) = -2.7 + 0.05 \times \text{native bird density}$, slope SE = 0.011; $P = 4.62 \times 10^{-4}$; $N_{\text{pools}} = 812$ at eight sites) includes site as a random effect, and the shaded area shows the 95% CI of the predicted line. Points are slightly jittered along the x-axis to improve visualization. Open squares show predicted prevalence estimates using the fitted regression model if native bird density was zero. Relationships with mosquito infection prevalence were similar with relative abundance (instead of density) of native birds ($\text{logit}(\text{prevalence}) = -2.73 + 0.23 \times \text{native relative abundance}$, slope SE = 0.88; $P = 8.86 \times 10^{-5}$), ‘amakihi density ($\text{logit}(\text{prevalence}) = -2.8 + 0.06 \times \text{‘amakihi density}$, slope SE = 0.0013; $P = 3.42 \times 10^{-6}$), or ‘amakihi relative abundance ($\text{logit}(\text{prevalence}) = -2.78 + 4.5 \times \text{‘amakihi relative abundance}$, slope SE = 1.03; $P = 1.13 \times 10^{-5}$). (b) Prevalence estimates of avian malaria in mosquitoes (± 1 binomial SE) plotted against host-seeking mosquito abundance for each of three years (2011–2013) at eight sites. The fitted line ($\text{logit}(\text{prevalence}) = -1.24 - 0.79 \times \text{log}_{10}(\text{Cx. quinquefasciatus abundance})$, slope SE = 0.07; $P = 1.26 \times 10^{-7}$; $N_{\text{pools}} = 812$ at eight sites, includes site as a random effect). (c) Hawai‘i ‘amakihi density (individuals/ha) plotted against host-seeking mosquito abundance ($r = -0.92$, $t = -5.65$, $df = 6$, $P = 0.0013$). Points are Hawai‘i ‘amakihi density (individuals/ha, ± 1 SE) estimated by fitting distance sampling models to point count data. The fitted line ($\text{‘amakihi density} = 18.51 - 11.5 \times \text{log}_{10}(\text{Cx. quinquefasciatus})$, slope SE = 2.037) and 95% CI of the line are shown. Results were qualitatively similar in a separate analysis of ‘amakihi density where $\text{log}_{10}(\text{Cx. quinquefasciatus})$ was included as an abundance covariate in distance sampling models ($\text{log}(\text{‘amakihi density}) = 2.79 - 1.12 \times \text{log}_{10}(\text{Cx. quinquefasciatus})$, slope SE = 0.08; $P < 0.0001$). (d) Hawai‘i ‘amakihi density (individuals/ha, ± 1 SE) plotted against the abundance of infected mosquitoes ($r = -0.75$, $t = -2.8$, $df = 6$, $P = 0.03$). The fitted line ($\text{‘amakihi density} = 4.5 - 9.91 \times \text{log}_{10}(\text{abundance of infected mosquitoes})$, slope SE = 3.5) and the 95% CI of the line are shown. Results were qualitatively similar in a separate analysis where $\text{log}_{10}(\text{abundance of infected mosquitoes})$ was an abundance covariate in distance sampling models ($\text{log}(\text{‘amakihi density}) = 1.74 - 1.13 \times \text{log}_{10}(\text{abundance of infected mosquitoes})$, slope SE = 0.09; $P < 0.0001$).

malaria transmission reduces the per capita growth rate of ‘amakihi, even if 100% of the population were infected each year, the population growth rate would still be above one. This suggests that additional factors are necessary to explain the absence of ‘amakihi from our sites where they were not present, including variation in other factors that affect the local geographic distribution of native Hawaiian birds, such as predation, interspecific competition, or food resource availability (Mountainspring and Scott 1985, Vanderwerf and Smith 2002, Wolfe et al. 2017). Mosquito abundance at our sites increased with developed land (McClure et al. 2018), and anthropogenic land use may have also

increased predators or degraded habitats for ‘amakihi. Low elevation ‘amakihi populations on the Island of Hawai‘i have increased in abundance and distribution in recent decades despite high malaria infection prevalence (Woodworth et al. 2005, Spiegel et al. 2006), but broader-scale recovery of ‘amakihi throughout lowland forests remains uncertain (and Kaua‘i ‘amakihi are declining quickly; Paxton et al. 2016). Facilitating the recovery of lowland ‘amakihi into these habitats may require actions that reduce these other stressors, but management actions that reduce mosquito abundance and malaria transmission would also aid conservation efforts.

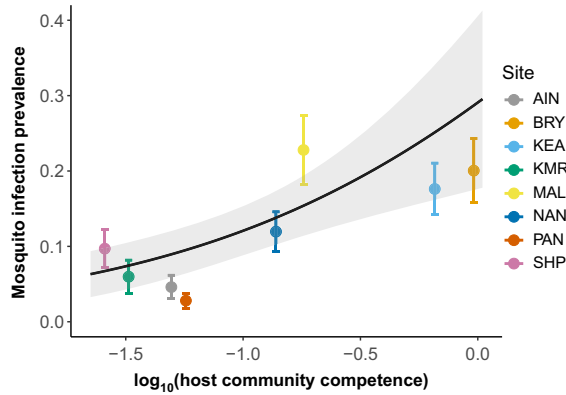


FIG. 3. Avian malaria infection prevalence in mosquitoes and host community competence. The prevalence of avian malaria in mosquitoes plotted against the \log_{10} (host community competence). Solid circles are maximum likelihood estimates of prevalence in mosquito pools from individuals captured in gravid and CO_2 traps from 2011–2013 (± 1 binomial SE). The fitted line ($\text{logit}(\text{prevalence}) = -0.89 + 1.09 \times \log_{10}(\text{host community competence})$, slope SE = 0.275; $P = 6.76 \times 10^{-5}$; $N_{\text{pools}} = 812$ at eight sites) includes site as a random effect, and the shaded area shows the 95% CI of the line. Mosquito infection prevalence was also significantly correlated with untransformed host community competence estimates, where the fitted line includes site as a random effect ($\text{logit}(\text{prevalence}) = -2.55 + 0.58 \times \text{host community competence}$, slope SE = 0.16; $P = 2.89 \times 10^{-4}$; $N_{\text{pools}} = 812$ at eight sites).

The increase in malaria transmission with native bird abundance, combined with variable mortality from infection in native species, suggests the potential for ecological feedback on the host community, as others have suggested in other systems (Dobson 2004). Low elevation ‘amakihi suffer 16.7% mortality rates from acute malaria infections (Atkinson et al. 2013), yet both survivors and those that succumb to infection mount parasitemias during the acute phase of infection that are an order of magnitude higher than that of introduced species that have been studied (Appendix S1: Fig. S6; van Riper et al. 1986, Atkinson et al. 1995). By increasing pathogen transmission within these communities, our results suggest that lowland ‘amakihi act as the main amplification hosts in areas where they occur (Atkinson et al. 2005, Atkinson and Samuel 2010, Samuel et al. 2011, Hobbelen et al. 2012), which could ultimately depress their own populations. Introduced species could play a key role in this ecological feedback, with incompetent introduced hosts reducing malaria transmission, allowing for the initial re-invasion of natives and potential evolution of more resistant or tolerant native birds (Woodworth et al. 2005, Kilpatrick 2006).

Intense year-round transmission of avian malaria in lowland Hawaii likely exerts a strong selective force on ‘amakihi for the evolution of malaria tolerance or resistance in lowland ‘amakihi populations (Atkinson et al.

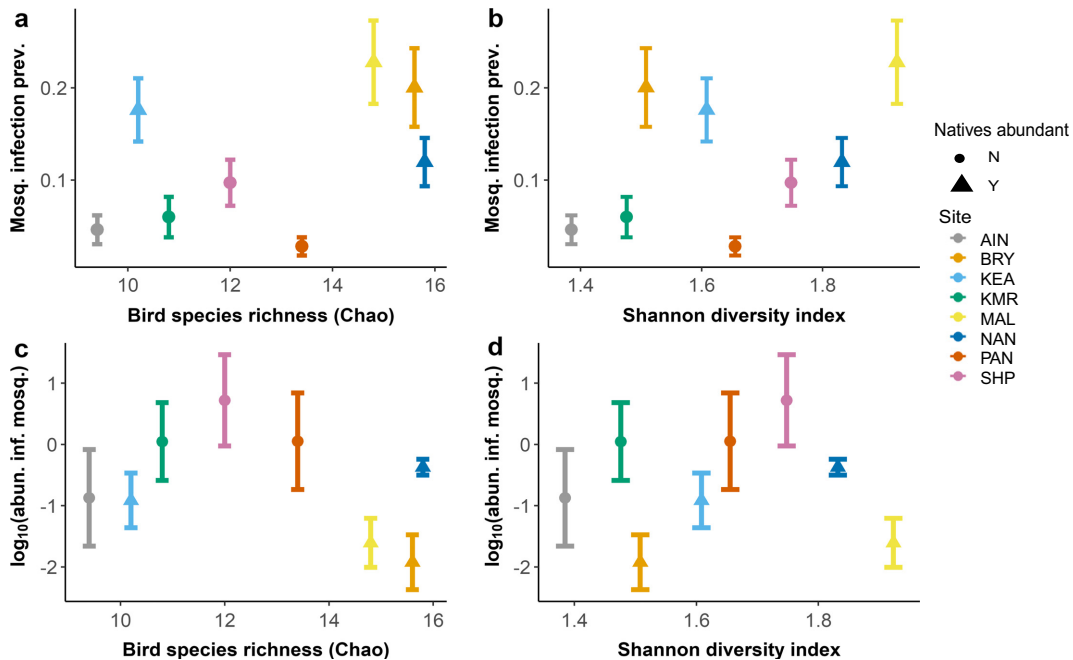


FIG. 4. Host diversity, avian malaria infection prevalence, and disease risk. Avian malaria infection prevalence in mosquitoes (± 1 binomial SE) plotted against host diversity at sites with (Y = yes) and without (N = no) $>15\%$ relative abundance of native birds within the avian community measured using (a) estimated species richness (Chao’s; Pearson’s correlation: $r = 0.48$, $t = 1.33$ $df = 6$, $P = 0.23$) or (b) Shannon diversity ($r = 0.442$, $t = 1.21$, $df = 6$, $P = 0.27$). Disease risk measured as \log_{10} (abundance of infected mosquitoes) (± 1 SE) plotted against host diversity measured using (c) estimated species richness (Chao’s, $r = -0.335$, $t = -0.87$, $df = 6$, $P = 0.42$) or (d) Shannon diversity ($r = 0.052$, $t = 0.13$, $df = 6$, $P = 0.9$). Estimated and raw species richness were significantly correlated ($r = 0.89$, $t = 4.85$, $df = 6$, $P = 0.9$).

1995, Woodworth et al. 2005, Foster et al. 2007, Cassin-Sackett et al. 2019). An experimental infection study showed that a smaller fraction of low elevation birds, 16.7% compared to 50% for high elevation birds, died from acute malaria infection, and birds dying from malaria had much higher parasitemias (Atkinson et al. 2013). This work suggests that selection has increased the fraction of lowland amakihi that are able to limit parasitemias from reaching high levels, increasing their probability of surviving malaria. Other work has demonstrated that lowland 'amakihi populations are genetically distinct and have genomic differences in a suite of immune-related genes relative to high elevation populations with low malaria exposure (Foster et al. 2007, Eggert et al. 2008, Cassin-Sackett et al. 2019). While the evolution of malaria host defenses offers promise for the persistence of some honeycreepers, the presence of native birds with still high parasitemias and chronic infections could also impede the dispersal and recovery of more susceptible native species to the lowlands (Hobbelen et al. 2012) such as the 'iwi, *Drepanis coccinea* (which suffers >90% mortality from acute infection; Atkinson et al. 1995).

CONCLUSION

Introduced species have well-documented impacts on ecosystems and native species through predation and competition (Salo et al. 2007, Kenis et al. 2009, Vilà et al. 2011, Croxall et al. 2012), but the role of introduced and native species in pathogen transmission and resulting reciprocal impacts on native species are poorly understood (Young et al. 2016). We found avian malaria infection prevalence in mosquitoes increased with native bird density due to native birds having higher host competence than introduced birds, as has been suggested by others (van Riper et al. 1986, Atkinson et al. 2005, Atkinson and Samuel 2010, Samuel et al. 2011, Hobbelen et al. 2012). However, we also found that covarying patterns of mosquito abundance led to higher infected mosquito abundance in fully introduced bird communities, which reduced the population growth rate of native birds. More broadly, the role of introduced species on pathogen transmission depends critically on their host competence relative to native hosts, as we demonstrate, as well as their contact rates with native hosts and vectors. Additional studies from other systems that combine data on host competence of native and introduced species and their impact on transmission (Kilpatrick et al. 2006a, 2007, Hamer et al. 2011) are needed to determine whether introduced species are more likely to increase or decrease transmission in general.

ACKNOWLEDGMENTS

We thank Keaukaha Military Reservation, State of Hawaii Division of Forestry and Wildlife, University of Hawai'i at Hilo, and Kamehameha Schools for providing access to lands and permission to sample. Many thanks to the hardworking interns

and volunteers, without whom this work would not have been possible, especially Marie Russell, Mike McFarlin, Katy Ward, Kacie Jonasen, Charlotte Lawrence, and Chris Davis. Mahalo nui loa to Part Hart at the University of Hawai'i at Hilo for logistic and field support. Funding was provided by a Smithsonian Institution pre-doctoral fellowship, University of California Cota-Robles fellowship, an American Ornithologists' Union Research Award, NSF GAANN fellowship grants A16-0061-002 and P200A030188, Smithsonian Conservation Biology Institution's Center for Conservation Genomics, NSF grants EF-0914866 and DEB 1717498, and NIH grant 1R01AI090159.

LITERATURE CITED

- Ahumada, J. A., D. LaPointe, and M. D. Samuel. 2004. Modeling the population dynamics of *Culex quinquefasciatus* (Diptera: Culicidae), along an elevational gradient in Hawaii. *Journal of Medical Entomology* 41:1157–1170.
- Akaike, H. 1973. Information theory and an extension of the maximum likelihood principle. Pages 267–281 in E. Parzen, K. Tanabe, and G. Kitagawa, editors. *Selected papers of Hirotugu Akaike*. Springer, New York, New York, USA.
- Andreadis, T. G., J. F. Anderson, C. R. Vossbrinck, and A. J. Main. 2004. Epidemiology of West Nile virus in Connecticut: A five-year analysis of mosquito data 1999–2003. *Vector-Borne and Zoonotic Diseases* 4:360–378.
- Atkinson, C. T., R. J. Dusek, and J. K. Lease. 2001. Serological responses and immunity to superinfection with avian malaria in experimentally-infected Hawaii Amakihi. *Journal of Wildlife Diseases* 37:20–27.
- Atkinson, C. T., R. J. Dusek, K. L. Woods, and W. M. Iko. 2000. Pathogenicity of avian malaria in experimentally-infected Hawaii Amakihi. *Journal of Wildlife Diseases* 36:197–201.
- Atkinson, C. T., J. K. Lease, R. J. Dusek, and M. D. Samuel. 2005. Prevalence of pox-like lesions and malaria in forest bird communities on leeward Mauna Loa Volcano, Hawaii. *Condor* 107:537–546.
- Atkinson, C. T., K. S. Saili, R. B. Utzurrum, and S. I. Jarvi. 2013. Experimental evidence for evolved tolerance to avian malaria in a wild population of low elevation Hawai'i 'Amakihi (*Hemignathus virens*). *EcoHealth* 10:366–375.
- Atkinson, C. T., and M. D. Samuel. 2010. Avian malaria *Plasmodium relictum* in native Hawaiian forest birds: epizootiology and demographic impacts on apapane *Himatione sanguinea*. *Journal of Avian Biology* 41:357–366.
- Atkinson, C. T., R. B. Utzurrum, D. A. Lapointe, R. J. Camp, L. H. Crampton, J. T. Foster, and T. W. Giambelluca. 2014. Changing climate and the altitudinal range of avian malaria in the Hawaiian Islands- an ongoing conservation crisis on the island of Kaua'i. *Global Change Biology* 20:2426–2436.
- Atkinson, C., K. Woods, R. Dusek, L. Sileo, and W. Iko. 1995. Wildlife disease and conservation in Hawaii: Pathogenicity of avian malaria (*Plasmodium relictum*) in experimentally infected Iwi (*Vestiaria coccinea*). *Parasitology* 111:S59–S69.
- Atkinson, I. 1977. A reassessment of factors, particularly *Rattus rattus* L., that influenced the decline of endemic forest birds in the Hawaiian Islands. *Pacific Science* 31:109–133.
- Baldwin, P. H. 1953. Annual cycle, environment and evolution in the Hawaiian honeycreepers (Aves: Drepaniidae). *University of California Publications in Zoology* 52:286–398.
- Bates, D., M. Maechler, B. Bolker, and S. Walker. 2015. Fitting linear mixed-effects models using lme4. *Journal of Statistical Software* 67:1–48.
- Beadell, J. S., C. Atkins, E. Cashion, M. Jonker, and R. C. Fleischer. 2007. Immunological change in a parasite-impooverished

- environment: Divergent signals from four island taxa. *PLoS ONE* 2:e896.
- Beadell, J. S., and R. C. Fleischer. 2005. A restriction enzyme-based assay to distinguish between avian hemospodians. *Journal of Parasitology* 91:683–685.
- Bennett, G. F., M. A. Bishop, and M. A. Peirce. 1993. Checklist of the avian species of *Plasmodium Marchiafava & Celli*, 1885 (Apicomplexa) and their distribution by avian family and Wallacean life zones. *Systematic Parasitology* 26:171–179.
- Bentley, M. D., and J. F. Day. 1989. Chemical ecology and behavioral aspects of mosquito oviposition. *Annual Review of Entomology* 34:401–421.
- Cassin-Sackett, L., T. E. Callicrate, and R. C. Fleischer. 2019. Parallel evolution of gene classes, but not genes: Evidence from Hawaiian honeycreeper populations exposed to avian malaria. *Molecular Ecology* 28:568–583.
- Caswell, H.. 2001. Matrix population models. Sinauer Associates, Sunderland, Massachusetts, USA.
- Chao, A.. 1984. Nonparametric estimation the number of classes in a population. *Annals of Mathematical Statistics* 20:265–270.
- Clark, N. J., S. M. Clegg, and M. R. Lima. 2014. A review of global diversity in avian haemosporidians (*Plasmodium* and *Haemoproteus*: Haemosporida): New insights from molecular data. *International Journal for Parasitology* 44:329–338.
- Croxall, J., S. Butchart, B. Lascelles, A. Stattersfield, B. Sullivan, A. Symes, and P. Taylor. 2012. Seabird conservation status, threats and priority actions: a global assessment. *Bird Conservation International* 22:1–34.
- Dobson, A. 2004. Population dynamics of pathogens with multiple host species. *American Naturalist* 164:64–78.
- Dornelas, M., N. J. Gotelli, B. McGill, H. Shimadzu, F. Moyes, C. Sievers, and A. E. Magurran. 2014. Assemblage time series reveal biodiversity change but not systematic loss. *Science* 344:296–299.
- Eggert, L. S., L. A. Terwilliger, B. L. Woodworth, P. J. Hart, D. Palmer, and R. C. Fleischer. 2008. Genetic structure along an elevational gradient in Hawaiian honeycreepers reveals contrasting evolutionary responses to avian malaria. *BMC Evolutionary Biology* 8:315.
- Fiske, I. J., and R. B. Chandler. 2011. unmarked: An R package for fitting hierarchical models of wildlife occurrence and abundance. *Journal of Statistical Software* 43:1–23.
- Foster, J. T., B. L. Woodworth, L. E. Eggert, P. J. Hart, D. Palmer, D. C. Duffy, and R. C. Fleischer. 2007. Genetic structure and evolved malaria resistance in Hawaiian honeycreepers. *Molecular Ecology* 16:4738–4746.
- Giambelluca, T., Q. Chen, A. Frazier, J. Price, Y.-L. Chen, P.-S. Chu, J. Eischeid, and D. Delparte. 2013. Online rainfall atlas of Hawai'i. *Bulletin American Meteorological Society* 94:313–316.
- Gottdenker, N. L., D. G. Streicker, C. L. Faust, and C. R. Carroll. 2014. Anthropogenic land use change and infectious diseases: a review of the evidence. *EcoHealth* 11:619–632.
- Hamer, G. L., L. F. Chaves, T. K. Anderson, U. D. Kitron, J. D. Brawn, M. O. Ruiz, S. R. Loss, E. D. Walker, and T. L. Goldberg. 2011. Fine-scale variation in vector host use and force of infection drive localized patterns of West Nile virus transmission. *PLoS ONE* 6:e23767.
- Hobbelen, P. H. F., M. D. Samuel, D. A. LaPointe, and C. T. Atkinson. 2012. Modeling future conservation of Hawaiian honeycreepers by mosquito management and translocation of disease-tolerant Amakihi. *PLoS ONE* 7:1–9.
- Howe, L., I. C. Castro, E. R. Schoener, S. Hunter, R. K. Barraclough, and M. R. Alley. 2012. Malaria parasites (*Plasmodium spp.*) infecting introduced, native and endemic New Zealand birds. *Parasitology Research* 110:913–923.
- Hoyt, J., et al. 2018. Cryptic connections illuminate pathogen transmission within community networks. *Nature* 563:710–713.
- Johnson, P. T. J., et al. 2009. Living fast and dying of infection: Host life history drives interspecific variation in infection and disease risk. *Ecology Letters* 106:20853–20858.
- Keesing, F., R. D. Holt, and R. S. Ostfeld. 2006. Effects of species diversity on disease risk. *Ecology Letters* 9:485–498.
- Kenis, M., M.-A. Auger-Rozenberg, A. Roques, L. Timms, C. Péré, M. J. W. Cock, J. Settele, S. Augustin, and C. Lopez-Vaamonde. 2009. Ecological effects of invasive alien insects. *Biological Invasions* 11:21–45.
- Kern, M., and C. van Riper. 1984. Altitudinal variations in nests of the Hawaiian honeycreeper *Hemignathus virens virens*. *Condor* 86:443–453.
- Kilpatrick, A. M. 2006. Facilitating the evolution of resistance to avian malaria in Hawaiian birds. *Biological Conservation* 128:475–485.
- Kilpatrick, A. M., P. Daszak, M. J. Jones, P. P. Marra, and L. D. Kramer. 2006a. Host heterogeneity dominates West Nile virus transmission. *Proceedings of the Royal Society B* 273:2327–2333.
- Kilpatrick, A. M., L. D. Kramer, M. J. Jones, P. P. Marra, and P. Daszak. 2006b. West Nile virus epidemics in North America are driven by shifts in mosquito feeding behavior. *PLoS Biology* 4:606–610.
- Kilpatrick, A. M., S. L. LaDeau, and P. P. Marra. 2007. Ecology of West Nile virus transmission and its impact on birds in the western hemisphere. *Auk* 124:1121–1136.
- Kilpatrick, A. M., and W. J. Pape. 2013. Predicting human West Nile virus infections with mosquito surveillance data. *American Journal of Epidemiology* 178:829–835.
- Kilpatrick, A. M., D. J. Salkeld, G. Titcomb, and M. B. Hahn. 2017. Conservation of biodiversity as a strategy for improving human health and well-being. *Philosophical Transactions of the Royal Society B* 372:20160131.
- Komar, N., N. A. Panella, S. A. Langevin, A. C. Brault, M. Amador, E. Edwards, and J. C. Owen. 2005. Avian hosts for West Nile virus in St. Tammany Parish, Louisiana 2002. *American Journal of Tropical Medicine and Hygiene* 73:1031–1037.
- Kovach, T., and A. Kilpatrick. 2018. Increased human incidence of West Nile Virus disease near rice fields in California but not in southern United States. *American Journal of Tropical Medicine and Hygiene* 99:222–228.
- LaPointe, D. A., M. L. Goff, and C. T. Atkinson. 2005. Comparative susceptibility of introduced forest-dwelling mosquitoes in Hawai'i to avian malaria, *Plasmodium relictum*. *Journal Parasitology* 91:843–849.
- LoGiudice, K., R. S. Ostfeld, K. A. Schmidt, and F. Keesing. 2003. The ecology of infectious disease: effects of host diversity and community composition on Lyme disease risk. *Proceedings of the National Academy of Sciences USA* 100:567–571.
- Long, J. 1981. *Introduced birds of the world: the worldwide history, distribution, and influence of birds introduced to new environments*. Universe Books, New York, New York, USA.
- MacDonald, G. 1952. The analysis of equilibrium in malaria. *Tropical Diseases Bulletin* 49:813–829.
- Matson, K. D. 2006. Are there differences in immune function between continental and insular birds? *Proceedings of the Royal Society B* 273:2267–2274.
- McClure, K. M., C. Lawrence, and A. M. Kilpatrick. 2018. Land use and larval habitat increase *Aedes albopictus* (Diptera: Culicidae) and *Culex quinquefasciatus* (Diptera: Culicidae) abundance in lowland Hawaii. *Journal of Medical Entomology* 55:1509–1516.
- Moulton, M. P., and S. L. Pimm. 1983. The introduced Hawaiian avifauna: biogeographic evidence for competition. *American Naturalist* 121:669–690.

- Mountainspring, S., and J. M. Scott. 1985. Interspecific competition among Hawaiian forest birds. *Ecological Monographs* 55:219–239.
- Newbold, T., et al. 2015. Global effects of land use on local terrestrial biodiversity. *Nature* 520:45–50.
- Oksanen, J. 2013. R package Vegan: ecological diversity:1–12. <https://cran.r-project.org>
- Olson, S. L., and H. F. James. 1982. Fossil birds from the Hawaiian Islands: Evidence for wholesale extinction by man before Western contact. *Science* 217:633–635.
- Ostfeld, R. S., T. Levi, A. E. Jolles, L. B. Martin, P. R. Hosseini, and F. Keesing. 2014. Life history and demographic drivers of reservoir competence for three tick-borne zoonotic pathogens. *PLoS ONE* 9:e107387.
- Paxton, E. H., R. J. Camp, P. M. Gorresen, L. H. Crampton, D. L. Leonard, and E. A. VanderWerf. 2016. Collapsing avian community on a Hawaiian island. *Science Advances* 2:E1600029.
- Pigeault, R., J. Vézilier, S. Cornet, F. Zélé, A. Nicot, P. Perret, S. Gandon, and A. Rivero. 2015. Avian malaria: A new lease of life for an old experimental model to study the evolutionary ecology of *Plasmodium*. *Philosophical Transactions of the Royal Society B* 370:201440300.
- Pyle, P. 1997. Identification guide to North American birds. Slate Creek Press, Bolinas, California, USA.
- Pyle, R. L. 2002. Checklist of the birds of Hawaii. *Elepaio* 62:137–148.
- R Core Team. 2017. R: A language and environment for statistical computing. R Foundation for Statistical Computing, Vienna, Austria. www.R-project.org
- Reiter, P. 1983. A portable, battery-operated trap for collecting gravid *Culex* mosquitoes. *Mosquito News* 43:496–498.
- Salo, P., E. Korpimäki, P. B. Banks, M. Nordstrom, and C. R. Dickman. 2007. Alien predators are more dangerous than native predators to prey populations. *Proceedings of the Royal Society B* 274:1237–1243.
- Samuel, M. D., P. H. F. Hobbelen, F. DeCastro, J. A. Ahumada, D. A. LaPointe, C. T. Atkinson, B. L. Woodworth, P. J. Hart, and D. C. Duffy. 2011. The dynamics, transmission, and population impacts of avian malaria in native Hawaiian birds: a modeling approach. *Ecological Applications* 21:2960–2973.
- Samuel, M. D., B. L. Woodworth, C. T. Atkinson, P. J. Hart, and D. A. LaPointe. 2015. Avian malaria in Hawaiian forest birds: infection and population impacts across species and elevations. *Ecosphere* 6:104.
- Sax, D. F., S. D. Gaines, and J. H. Brown. 2002. Species invasions exceed extinctions on islands worldwide: a comparative study of plants and birds. *American Naturalist* 160:766–783.
- Scott, J. M., S. Mountainspring, F. L. Ramsey, and C. B. Kepler. 1986. Forest bird communities of the Hawaiian Islands: their dynamics, ecology, and conservation. *Studies in Avian Biology* 9:1–431.
- Spiegel, C. S., P. J. Hart, B. L. Woodworth, E. J. Tweed, and J. J. LeBrun. 2006. Distribution and abundance of forest birds in low-altitude habitat on Hawai'i Island: evidence for range expansion of native species. *Bird Conservation International* 16:175–185.
- Takken, W., and N. O. Verhulst. 2013. Host preferences of blood-feeding mosquitoes. *Annual Review of Entomology* 58:433–453.
- Thomas, L., S. T. Buckland, E. A. Rexstad, J. L. Laake, S. Strindberg, S. L. Hedley, J. R. B. Bishop, T. A. Marques, and K. P. Burnham. 2010. Distance software: Design and analysis of distance sampling surveys for estimating population size. *Journal of Applied Ecology* 47:5–14.
- Valkiunas, G. 2005. Avian malaria parasites and other Haemosporidia. CRC Press, Boca Raton, Florida, USA.
- van Kleunen, M., E. Weber, and M. Fischer. 2010. A meta-analysis of trait differences between invasive and non-invasive plant species. *Ecology Letters* 13:235–245.
- van Riper, C., S. G. van Riper, M. L. Goff, M. Laird, L. M. Goff, M. Laird, M. L. Goff, and M. Laird. 1986. The epizootiology and ecological significance of malaria in Hawaiian land birds. *Ecological Monographs* 56:327–344.
- van Riper, C., S. van Riper, and W. Hansen. 2002. Epizootiology and effect of avian pox on Hawaiian forest birds. *Auk* 119:929–942.
- Vanderwerf, E. A., and D. G. Smith. 2002. Effects of alien rodent control on demography of the O'ahu 'Elepaio, an endangered Hawaiian forest bird. *Pacific Conservation Biology* 8:73–81.
- Ventim, R., L. Mendes, J. A. Ramos, H. Cardoso, and J. Pérez-Tris. 2012. Local haemoparasites in introduced wetland passerines. *Journal of Ornithology* 153:1253–1259.
- Vilà, M., J. L. Espinar, M. Hejda, P. E. Hulme, V. Jarošík, J. L. Maron, J. Pergl, U. Schaffner, Y. Sun, and P. Pyšek. 2011. Ecological impacts of invasive alien plants: A meta-analysis of their effects on species, communities and ecosystems. *Ecology Letters* 14:702–708.
- Walter, S. D., S. W. Hildreth, and B. J. Beaty. 1980. Estimation of infection rates in populations of organisms using pools of variable size. *American Journal of Epidemiology* 112:124–128.
- Warner, R. E. 1968. The role of introduced diseases in the extinction of the endemic Hawaiian avifauna. *Condor* 70:101–120.
- Wikelski, M., J. Foufopoulos, H. Vargas, and H. Snell. 2004. Galapagos birds and diseases: Invasive pathogens as threats for island species. *Ecology and Society* 9:5.
- Wolfe, J. D., C. J. Ralph, and A. Wiegardt. 2017. Bottom-up processes influence the demography and life-cycle phenology of Hawaiian bird communities. *Ecology* 98:2885–2894.
- Woodworth, B. L., et al. 2005. Host population persistence in the face of introduced vector-borne diseases: Hawaii amakihi and avian malaria. *Proceedings of the National Academy of Sciences USA* 102:1531–1536.
- Yorinks, N., and C. T. Atkinson. 2000. Effects of malaria on activity budgets of experimentally infected juvenile Apapane (*Himatione Sanguinea*). *Auk* 117:731–738.
- Young, H. S., I. M. Parker, G. S. Gilbert, A. Sofia Guerra, and C. L. Nunn. 2016. Introduced species, disease ecology, and biodiversity–disease relationships. *Trends in Ecology & Evolution* 32:1–14.
- Zimmerman, N., R. F. Hughes, S. Cordell, P. Hart, H. K. Chang, and D. Perez. 2008. Patterns of primary succession of native and introduced plants in lowland wet forests in eastern Hawai'i. *Biotropica* 4:277–284.

SUPPORTING INFORMATION

Additional supporting information may be found in the online version of this article at <http://onlinelibrary.wiley.com/doi/10.1002/ecy.3038/supinfo>