

RESEARCH ARTICLE

Interacting Effects of Newcastle Disease Transmission and Illegal Trade on a Wild Population of White-Winged Parakeets in Peru: A Modeling Approach

Elizabeth F. Daut^{1#a*}, Glenn Lahodny, Jr.^{2,4}, Markus J. Peterson³, Renata Ivanek^{4#b}

1 Schubot Exotic Bird Health Center, Department of Veterinary Pathobiology, College of Veterinary Medicine and Biomedical Sciences, Texas A&M University, College Station, Texas, United States of America, **2** Department of Mathematics, Texas A&M University, College Station, Texas, United States of America, **3** Department of Biological Sciences, University of Texas at El Paso, El Paso, Texas, United States of America, **4** Department of Veterinary Integrative Biosciences, College of Veterinary Medicine and Biomedical Sciences, Texas A&M University, College Station, Texas, United States of America

^{#a} Current address: National Socio-Environmental Synthesis Center, Annapolis, Maryland, United States of America,

^{#b} Current address: Department of Population Medicine and Diagnostic Sciences, College of Veterinary Medicine, Cornell University, Ithaca, New York, United States of America

* edaut@sesync.org



OPEN ACCESS

Citation: Daut EF, Lahodny G, Jr., Peterson MJ, Ivanek R (2016) Interacting Effects of Newcastle Disease Transmission and Illegal Trade on a Wild Population of White-Winged Parakeets in Peru: A Modeling Approach. PLoS ONE 11(1): e0147517. doi:10.1371/journal.pone.0147517

Editor: Michelle L. Baker, CSIRO, AUSTRALIA

Received: July 11, 2015

Accepted: January 5, 2016

Published: January 27, 2016

Copyright: © 2016 Daut et al. This is an open access article distributed under the terms of the [Creative Commons Attribution License](https://creativecommons.org/licenses/by/4.0/), which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

Data Availability Statement: All Matlab codes are available as a Supporting Information File ([S1 File](#)) and at the GitHub repository (https://github.com/NDCcodes/Daut_ND-matlab-codes/tree/master).

Funding: E. Daut is grateful 726 for the financial and academic support provided by Applied Biodiversity Science NSF-IGERT 727 program, the Department of Veterinary Pathobiology, and the Schubot Exotic Bird Health Center 728 at Texas A&M University. The funders had no role in study design, data collection and analysis, decision to publish, or preparation of the manuscript.

Abstract

Illegal wildlife-pet trade can threaten wildlife populations directly from overharvest, but also indirectly as a pathway for introduction of infectious diseases. This study evaluated consequences of a hypothetical introduction of Newcastle disease (ND) into a wild population of Peru's most trafficked psittacine, the white-winged parakeet (*Brotogeris versicolurus*), through release of infected confiscated individuals. We developed two mathematical models that describe ND transmission and the influence of illegal harvest in a homogeneous (model 1) and age-structured population of parakeets (model 2). Infection transmission dynamics and harvest were consistent for all individuals in model 1, which rendered it mathematically more tractable compared to the more complex, age-structured model 2 that separated the host population into juveniles and adults. We evaluated the interaction of ND transmission and harvest through changes in the basic reproduction number (R_0) and short-term host population dynamics. Our findings demonstrated that ND introduction would likely provoke considerable disease-related mortality, up to 24% population decline in two years, but high harvest rates would dampen the magnitude of the outbreak. Model 2 produced moderate differences in disease dynamics compared to model 1 ($R_0 = 3.63$ and 2.66 , respectively), but highlighted the importance of adult disease dynamics in diminishing the epidemic potential. Therefore, we suggest that future studies should use a more realistic, age-structured model. Finally, for the presumptive risk that illegal trade of white-winged parakeets could introduce ND into wild populations, our results suggest that while high harvest rates may have a protective effect on the population by reducing virus transmission, the combined effects of high harvest and disease-induced mortality may threaten population survival. These results

Competing Interests: The authors have declared that no competing interests exist.

capture the complexity and consequences of the interaction between ND transmission and harvest in a wild parrot population and highlight the importance of preventing illegal trade.

Introduction

Illegal wildlife trade and infectious diseases are recognized conservation threats affecting wild-life populations [1–3]. Illegal and poorly regulated wildlife trade can result in overharvest and threaten population and species survival [4–7]. Introduced infectious diseases have been linked to major declines of wildlife populations [8–10], and even species extinctions [11,12]. The influence of *legal* harvest or culling on disease dynamics in wildlife populations has been examined [13–15], but the influence of *illegal* wildlife trade on the introduction and spread of infectious diseases has rarely been investigated (but for related topics see: [16–18]). The risks of introducing infectious diseases are particularly high in developing nations where illegal wildlife trade for domestic consumers flourishes and law enforcement and disease surveillance are often lacking [19–22].

Illegal trade in wild-caught birds as pets is common in many regions of the world including southeast Asia [23,24], Africa [25–28], and Latin America [29–35]. Over-exploitation for the pet trade and hunting threatens almost 40% of at-risk birds (i.e., avian species with a conservation status other than least-concern on International Union for Conservation of Nature and Natural Resources (IUCN) Red List) [36]. While songbirds are often illegally harvested in large numbers, particularly for singing competitions [37–39], parrots have historically been and continue to be the most exploited avian family for pets [36,40,41]. As a result of overharvest and other conservation threats such as destruction of suitable habitat [36,42], psittacidae is one of most the threatened bird families in the world, with 152 of the 350 extant species (43%) listed in a threatened category on the IUCN Red List [43,44]. In the Neotropics, Clarke & de By (2013), suggested that poaching is the greatest threat contributing to parrot decline [45].

Peru has among the highest parrot diversities in the world ($n = 53$ species) [46] and a long history of parrot harvest for the pet trade [47–49]. In a recent market study, 65% of the country's parrot species were observed illegally for sale for domestic consumers [29]. As a case study, we used simulation modeling to evaluate a hypothetical introduction and outbreak of Newcastle disease (ND) in a population of Peru's most trafficked psittacine, the white-winged parakeet (*Brotogeris versicolurus*) [48,50]. Threat of infectious disease and illegal harvest of wildlife should be of concern for Peru because it is a megadiverse country and a high priority area for biodiversity conservation [51].

The white-winged parakeet is a small, non-threatened, highly gregarious species that is common throughout most of its Amazonian range [52,53]. Peruvian authorities consider domestic trade of the white-winged parakeet and other native birds illegal because harvest and commercialization are conducted without proper licenses and authorizations [29,54]. During a five-year market survey in Peru, supply of white-winged parakeets was surprisingly constant throughout the year, which may stem from dual harvest techniques—taking nestlings from easily accessible nests located in arboreal termite mounds during the breeding season, and capturing adult parakeets throughout the year using mist nets at roost and feeding sites [49]. It is difficult to determine whether harvest is sustainable [55], but current harvest rates do not appear to negatively influence abundance [53].

Newcastle disease is a highly infectious and fatal viral disease caused by avian paramyxovirus serotype-1 that affects many avian species including parrots and poultry [56]. Large epidemics

have occurred in poultry operations [57,58], racing pigeons [59], and free-ranging double-crested cormorants [60]. Even though vaccination programs have largely prevented recent outbreaks in commercial flocks, ND is still a serious problem in backyard poultry in rural areas throughout the developing world and in the pet trade [61–63]. In 2004, ND virus was isolated in a shipment of imported parrots and other avian species from Pakistan to Italy [64].

The ND virus spreads horizontally between healthy and infected birds through direct contact with bodily secretions from infected birds [65]. Crowded confinement typical of poultry houses or large breeding rookeries provides ideal conditions for virus transmission [65]. Disease in parrots is suspected to result from contact with infected poultry, particularly at animal markets [66,67]. Wild-caught parrots smuggled into the United States in the 1970s were suspected to have acquired ND while at animal markets in South America [58,67,68]. Subsequent outbreaks of ND in chickens cost the U.S. poultry industry millions of dollars [68]. The most common clinical signs in captive psittacine species were respiratory, but ranged from lethargy to limb paralysis [69–72]. Mortality can reach as high as 100% [68], but typically ranged from 20 to 80% [69,70,73].

White-winged parakeets are susceptible to ND [67,74]. During an outbreak in Austria, 53% ($n = 32$) of parakeets died from ND following importation [73]. In the early 1980s, ND was diagnosed in importation lots of white-winged parakeets from Argentina and Bolivia four times according to United States Department of Agriculture (USDA) quarantine records [75]. Newcastle disease is considered endemic in Peru [76,77]. Almost 100 outbreaks have been reported to the World Organization for Animal Health (OIE) during 2008–2014 typically in unvaccinated backyard (*criollo*) chickens and fighting cocks (e.g., [78]), with 34 outbreaks in 2014 alone [76,79]. In animal markets throughout Peru it is common to observe wild-caught parrots alongside *criollo* chickens (E. Daut, personal observation; see S3 for photographs of animal markets), thus providing the opportunity for cross-species ND transmission. Authorities frequently confiscate white-winged parakeets and immediately release individuals into the wild [29,80], typically without health evaluation because they do not have financial or diagnostic means to conduct medical screening [20,81–85]. Although ND has not been identified in the limited studies of wild psittacines to date [86–90], we expect that illegal trade provides a mechanism for ND to reach susceptible populations of white-winged parakeets due to the release of confiscated individuals infected at animal markets.

Infectious disease mathematical modeling is a useful tool for conservationists and epidemiologists to evaluate potential synergistic effects of illegal trade and disease on wildlife populations and to compare mitigating strategies [91,92]. The influence of illegal trade—specifically harvest—on pathogen transmission can be evaluated by comparing the pathogen's basic reproduction number (R_0) at different harvest rates [93]. R_0 is the average expected number of secondary infections produced by one typical infectious individual introduced into a fully susceptible host population [93], and is often used as a threshold value to determine whether or not a pathogen can invade and persist in the population ($R_0 \geq 1$) or fades out ($R_0 < 1$).

Under certain density-dependent pathogen transmission conditions, harvest can decrease transmission and is the reason culling can be effective to prevent disease spread [94,95]. In other cases, where birth rate in a host population is under strong density-dependence so that harvest would stimulate natality, harvest can increase the number of susceptible individuals in the population and subsequent disease prevalence and disease-induced mortality [13]. Because both harvest and diseases can be age selective, age structure can be an important demographic component in mathematical models. Recent studies have demonstrated that incorporating age structure into disease modeling can have strong, yet often unpredictable, influences on wildlife disease dynamics [96,97].

We hypothesized that (1) introducing ND into a susceptible population of white-winged parakeets would result in an outbreak with considerable mortality and (2) increasing harvest

would lower disease transmission and the magnitude of the outbreak. To evaluate these hypotheses, we developed two continuous-time, mathematical models to assess whether predictions about ND dynamics would differ between a simplified model with a homogeneous bird population and a presumably more realistic, but less tractable, model with an age-structured host population. Both models were considered under different harvest scenarios and R_0 and disease-related mortality were assessed using a combination of analytical and numerical approaches. We conducted sensitivity and scenario analyses to evaluate the robustness of the models' results in the presence of uncertainty to individual parameters. Lastly, we discussed the limitations and implications of our results, including the conservation relevance of illegal trade and ND emergence in native populations of white-winged parakeets.

Materials and Methods

Model formulation

Because ND typically results in severe but short-lived epidemics in avian species, we focused our attention on short-term infection dynamics without including density-dependent responses from the host population to the disease-induced mortality (e.g., increased fecundity). However, given the possibility of long-term chronic infections in parrots [69], we did consider an endemic state where ND persists in the population.

In model 1 (Fig 1), we described the parakeet population as homogeneous where the $SEI_A I_C R$ model assumed no differences in the infection—transmission dynamics and harvest

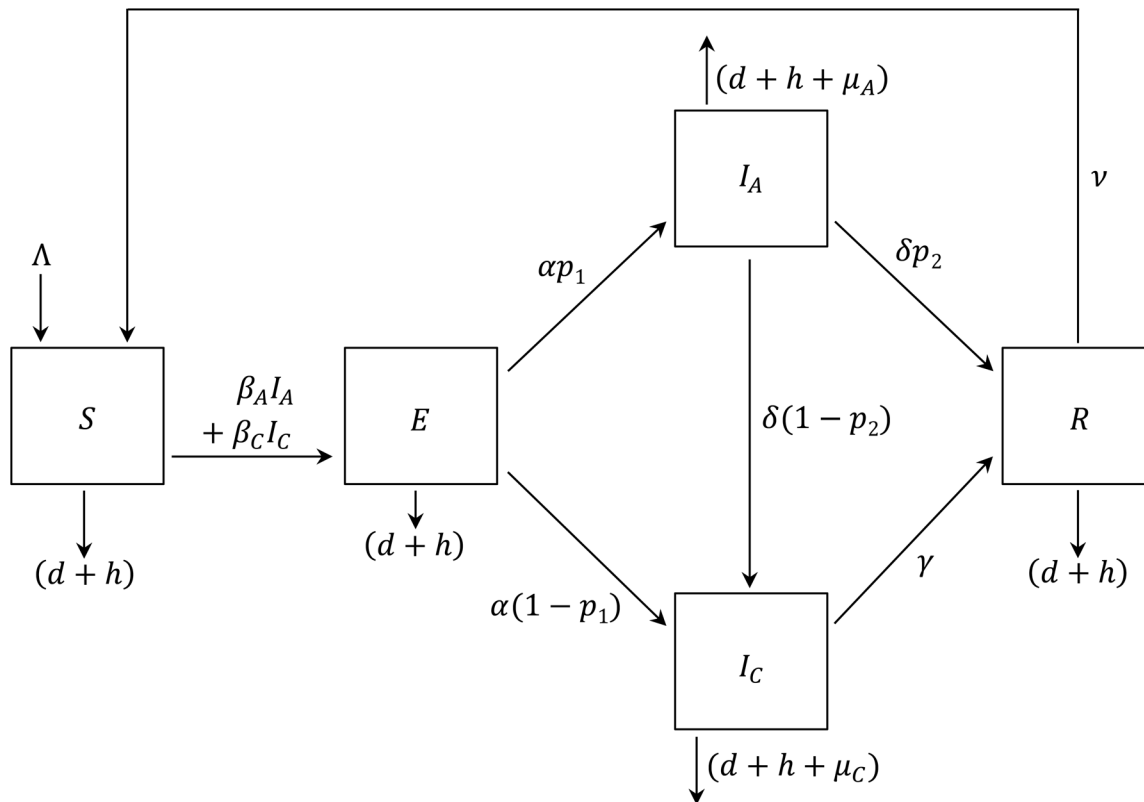


Fig 1. Compartmental diagram of the dynamics of Newcastle disease in a homogeneous population of white-winged parakeets (model 1). Five transition states include: susceptible (S), exposed (E), acutely-infected (I_A), chronically-infected (I_C) and recovered (R). See Table 1 for parameter descriptions.

doi:10.1371/journal.pone.0147517.g001

among age groups. The host population was divided into susceptible (S), exposed (E), acutely-infected (I_A), chronically-infected (I_C), and recovered (R) states [98]. We included two infectious stages (I_A and I_C) because experimental evidence suggested two levels of severity of clinical signs, which we used as a proxy for viral shedding [69,99]. In model 2, we divided the host population into juvenile and adult stages to account for age-related differences in harvest and disease transmission and severity (Fig 2). While model 2 was demographically more realistic than model 1, it was also far more complex and less tractable, which makes its application by non-mathematicians more difficult and brings up a question of whether model 1 could be an acceptable alternative for studying interaction of infection and harvest in a wild bird population. To assure a fair comparison of results between both models, model 2 was structured to collapse into model 1 when the two age stages had the same parameter values. Both models included a baseline harvest rate ($h_b = 1\%$), which we assumed was the current harvest rate of

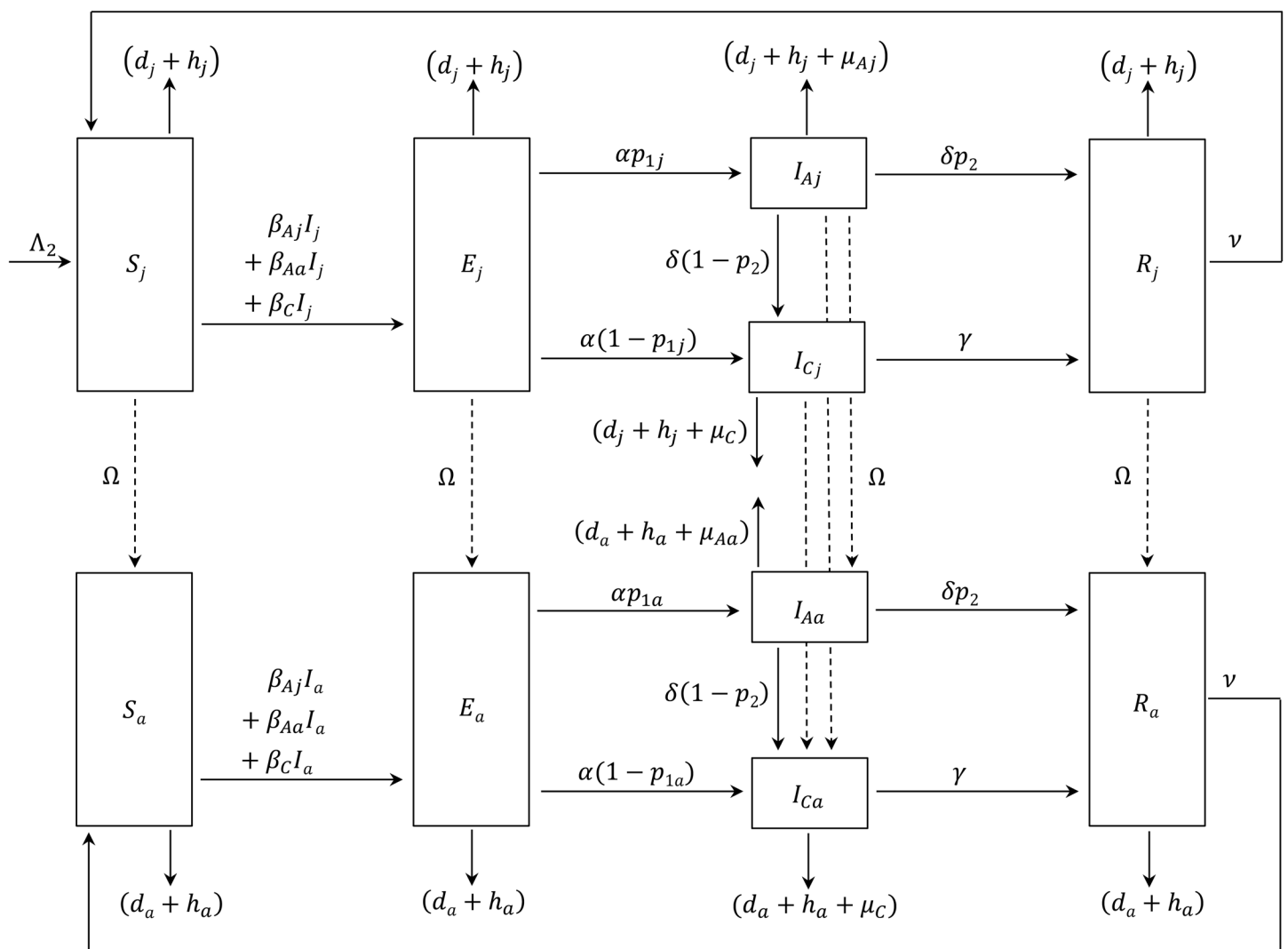


Fig 2. Compartmental diagram of the dynamics of the dynamics of Newcastle disease in an age-structured population of white-winged parakeets (model 2). Transition states for juvenile parakeets are: susceptible (S_j), exposed (E_j), acutely-infected (I_{Aj}), chronically-infected (I_{Cj}) and recovered (R_j) and for adult parakeets the states are: susceptible (S_a), exposed (E_a), acutely-infected (I_{Aa}), chronically-infected (I_{Ca}) and recovered (R_a). See Table 1 for parameter descriptions.

doi:10.1371/journal.pone.0147517.g002

white-winged parakeets in Peru, based on the fact that this species is highly harvested but the population size remains stable [53]. Furthermore, we conducted scenario analyses to evaluate the interaction of ND dynamics and additional plausible harvest (hI) on R_0 and host population size.

Our mathematical framework started with the following assumptions:

1. The host population was a single, free-mixing population and individual parakeets were equally likely to encounter an infected individual. This was a realistic assumption given the gregarious nature of white-winged parakeets and the large number of individuals at communal roost sites, up to 700 to 1,000 individuals [100,101].
2. The host population was stable under initial conditions, closed to immigration and emigration, and parakeet natality and mortality were not under the influence of density dependence. While these conditions may not always hold true, they were useful simplifying assumptions that helped isolate the evaluation of harvest and ND dynamics.
3. Both sexes were equally affected by ND virus [62].
4. At hatching, chicks were susceptible to ND virus. There is limited evidence that psittacine chicks can receive maternal antibodies through the egg [102]; however, no studies have evaluated whether protective maternal ND-antibodies are transferred to psittacine chicks.
5. Transmission of ND was density-dependent where the number of contacts per unit time was proportional to the number of individuals in the population [103]. Density-dependent transmission was a reasonable assumption and has been suggested for systems where the pathogen is transmitted through random contact among individuals and/or by aerial transmission [104,105]. Airborne transmission was considered to contribute to spread of the ND virus near poultry facilities [106,107] and in captive Neotropical psittacines [69].

We used a system of ordinary differential equations (ODEs) to describe transmission of ND in white-winged parakeets for each model (Eqs 1–5 for model 1 and Eqs 6–15 for model 2). For both models, the initial (time $t = 0$) susceptible population, $S(0)$, was set equal to the total population, $N(0)$, minus one individual, which represented an acutely-infectious parakeet introduced into the population, $I_A(0) = 1$ and $I_{Ad}(0) = 1$ for models 1 and 2, respectively. All remaining stages were set to zero. Model simulations and analyses were conducted in Matlab R2015a (MathWorks, USA); see https://github.com/NDCcodes/Daut_ND-matlab-codes/tree/master or [S1 File](#) for Matlab modeling codes.

Model 1

$$\frac{dS}{dt} = \Lambda - (d + h)S - \beta_A SI_A - \beta_C SI_C + \nu R \tag{1}$$

Where $\Lambda = N(d+h)$ and N changes over time as $N(t) = S(t)+E(t)+I_A(t)+I_C(t)+R(t)$

$$\frac{dE}{dt} = \beta_A SI_A + \beta_C SI_C - (d + h + \alpha)E \tag{2}$$

$$\frac{dI_A}{dt} = \alpha p_1 E - (d + h + \mu_A + \delta)I_A \tag{3}$$

$$\frac{dI_C}{dt} = \alpha(1 - p_1)E + \delta(1 - p_2)I_A - (d + h + \mu_C + \gamma)I_C \tag{4}$$

$$\frac{dR}{dt} = \delta p_2 I_A + \gamma I_C - (d + h + v)R \tag{5}$$

Model 2

Juvenile:

$$\frac{dS_j}{dt} = \Lambda_2 - (d_j + h_j + \Omega)S_j - \beta_{Aj}S_jI_{Aj} - \beta_C S_jI_{Cj} - \beta_{Aa}S_jI_{Aa} - \beta_C S_jI_{Ca} + vR_j \tag{6}$$

Where $\Lambda_2 = \frac{N(d_j+h_j+\Omega)(d_a+h_a)}{d_a+h_a+\Omega}$ and N changes over time as $N(t) = N_j(t)+N_a(t)$ where $N_j(t) = S_j(t) + E_j(t) + I_{Aj}(t) + I_{Cj}(t) + R_j(t)$ and $N_a(t) = S_a(t) + E_a(t) + I_{Aa}(t) + I_{Ca}(t) + R_a(t)$

$$\frac{dE_j}{dt} = \beta_{Aj}S_jI_{Aj} + \beta_C S_jI_{Cj} + \beta_{Aa}S_jI_{Aa} + \beta_C S_jI_{Ca} - (d_j + h_j + \Omega + \alpha)E_j \tag{7}$$

$$\frac{dI_{Aj}}{dt} = \alpha p_{1j}E_j - (d_j + h_j + \Omega + \mu_{Aj} + \delta)I_{Aj} \tag{8}$$

$$\frac{dI_{Cj}}{dt} = \alpha(1 - p_{1j})E_j + \delta(1 - p_2)I_{Aj} - (d_j + h_j + \Omega + \mu_C + \gamma)I_{Cj} \tag{9}$$

$$\frac{dR_j}{dt} = \delta p_2 I_{Aj} + \gamma I_{Cj} - (d_j + h_j + \Omega + v)R_j \tag{10}$$

Adult:

$$\frac{dS_a}{dt} = \Omega S_j - (d_a + h_a)S_a - \beta_{Aa}S_aI_{Aa} - \beta_C S_aI_{Ca} - \beta_{Aj}S_aI_{Aj} - \beta_C S_aI_{Cj} + vR_a \tag{11}$$

$$\frac{dE_a}{dt} = \Omega E_j + \beta_{Aa}S_aI_{Aa} + \beta_C S_aI_{Ca} + \beta_{Aj}S_aI_{Aj} + \beta_C S_aI_{Cj} - (d_a + h_a + \alpha)E_a \tag{12}$$

$$\frac{dI_{Aa}}{dt} = \Omega I_{Aj} + \alpha p_{1a}E_a - (d_a + h_a + \mu_{Aa} + \delta)I_{Aa} \tag{13}$$

$$\frac{dI_{Ca}}{dt} = \Omega I_{Cj} + \alpha(1 - p_{1a})E_a + \delta(1 - p_2)I_{Aa} - (d_a + h_a + \mu_C + \gamma)I_{Ca} \tag{14}$$

$$\frac{dR_a}{dt} = \Omega R_j + \delta p_2 I_{Aa} + \gamma I_{Ca} - (d_a + h_a + v)R_a \tag{15}$$

Basic reproduction number (R_0)

To calculate R_0 for our systems of ODEs, we used the next-generation method (NGM) [108]. See [S2 file](#) for the full derivations of R_0 . For model 1 the derived expression for R_0 (Eq A1) was:

$$R_0 = R_{0A} + R_{0C}.$$

The terms R_{0A} and R_{0C} represent the average number of secondary infections resulting from

interactions between susceptible and acutely- and chronically-infected hosts, respectively. For model 2 the derived expression for R_0 (Eq A2) was:

$$R_0 = \frac{1}{2} [R_{01} + R_{02} + \sqrt{(R_{01} - R_{02})^2 + 4R_{03}R_{04}}].$$

The terms R_{01} and R_{02} represent the average number of secondary juvenile or adult infections, respectively, produced by introduction of one exposed juvenile E_j during its entire infectious period. The terms R_{03} and R_{04} represent the average number of secondary juvenile or adult infections produced by introduction of one exposed adult E_a during its entire infectious period, respectively.

Parameter estimates

Parameter notations, definitions, mean values, distributions for sensitivity analyses, and sources of information are described below and in [Table 1](#). All demographic parameters except population size were constant and disease parameters were allowed to vary around their corresponding mean (baseline) values.

Model 1

Host demography and harvest. We defined the initial population of parakeets as ($N(0) = 200$), a commonly reported flock size [\[100,109\]](#), which included those birds roosting together at a communal site throughout the year, except during the breeding season when pairs separate for nesting. We set population recruitment (Λ) equal to the sum of natural mortality (d) and the current (baseline) harvest (h_b) to maintain a stable population in absence of additional harvest (h_l), so that $\Lambda = N(d+h_b)$ [\[53\]](#). Daily natural mortality was calculated as the inverse of the mean life expectancy ($D_d = 5$ years) from captive white-winged parakeets, so that $d = 1/D_d$ [\[110\]](#). Baseline harvest of white-winged parakeets was set at a conservatively low constant daily rate for a cumulative total annual harvest of 1% ($h_b = 0.01/365$).

Transmission rate. Transmission coefficients (β) for ND in psittacines have not been published; therefore, we adapted a transmission probability published for backyard poultry that was considered to reasonably represent ND dynamics in wild white-winged parakeets. In a mathematical model describing density-dependent ND dynamics in a backyard chicken flock, Johnston [\[119\]](#) estimated 3% infection probability during a 14-day period. We prorated this probability to a per day rate for our population size and used it as a baseline transmission rate for acutely-infectious individuals, so that $\beta_A = (3/14)200$ individual⁻¹ day⁻¹ ([Table 1](#)). Chronic ND transmission rate was assumed to be a fraction ($I_r = 10\%$) of the acute transmission rate, so that $\beta_C = \beta_A I_r$.

Infection stages. According to the standard assumption of exponentially-distributed periods of infection [\[98\]](#), the reciprocal values of the mean durations in days that a parakeet spends in the exposed (E), acutely-infected (I_A), chronically-infected (I_C), and recovered (R) stages were used as the daily rates α , δ , γ , and ν , respectively ([Table 1](#); [Fig 1](#)). The daily rate of becoming infectious, therefore, was the inverse of the duration of an average latent period reported for most studied avian species ($\alpha = 1/5.5$) [\[56\]](#). We based the duration of the acute-infectious stage on the study by Erickson et al. [\[99\]](#) where individual Neotropical psittacines ($n = 48$) shed ND virus on average for 30 days so that $\delta = 1/30$. Although some parrots continued to chronically shed for over a year, on average individuals had stopped shedding by 39 days post infection [\[69\]](#), which represented our baseline recovery rate from chronic infection $\gamma = 1/39$ and was consistent with shedding duration observed in other avian species [\[122,128,129\]](#). We used this same duration for individuals that became chronically infected directly following

Table 1. Definitions and values of parameters for the model of Newcastle disease (ND) transmission in a homogeneous (model 1) and age-structured (model 2) population of wild white-winged parakeets.

Notation	Definition (unit)	Baseline value	Sensitivity analysis	5 th and 95 th percentiles	Model	Source
<i>Population Dynamics Parameters</i>						
$N(0)$	Initial number of individuals of white-winged parakeets in a typical flock in Ucayali, Peru	200	Log-normal (ln(189), ln(1.4))	109, 329	1	[100,109]
N_j	Initial number of individual juvenile (N_j) and adult (N_a) white-winged parakeets in a typical post-breeding flock in Ucayali, Peru	$N_j = 0.08N$ $N_a = 0.92N$	-	-	2	Estimated for $D_d = 5$ years ^a
D_d	Life expectancy (year)	5.0 ^b	-	-	1, 2	[110]
d	Natural mortality rate (day ⁻¹)	$1/D_d$	-	-	1, 2	-
D_Ω	Duration of juvenile stage (day)	135	-	-	2	Informed from: [102,111–116]
Ω	Rate of leaving juvenile stage (day ⁻¹)	$1/D_\Omega$	-	-	2	-
d_j	Natural juvenile mortality rate (day ⁻¹)	$1/D_\Omega/\ln(0.6)^c$	-	-	2	[115,117]
d_a	Natural adult mortality rate (day ⁻¹)	$1/(1/d-1/d_j)$	-	-	2	-
h_b	Current (baseline) harvest rate (year ⁻¹) ^d	1%	-	-	1, 2	[53]
$h1$	Additional harvest rate (year ⁻¹) ^d	0 ^e	-	-	1, 2	-
h	Total harvest rate (year ⁻¹) ^d	h_b+h1	-	-	1, 2	-
h_j	Total juvenile harvest rate (year ⁻¹) ^d	$0.4h$	-	-	2	Informed from: [118]
h_a	Total adult harvest rate (year ⁻¹) ^d	$0.6h$	-	-	2	Informed from: [118]
<i>Infection Parameters</i>						
β_A	Transmission rate from an acutely-infected parakeet (individual ⁻¹ day ⁻¹)	0.00107	Uniform (0.00107*0.5, 0.00107*1.5)	0.000601, 0.0016	1, 2	[119]
I_r	Infectiousness reduction coefficient for chronically-infected parakeets	0.1	Uniform (0.05, 0.15)	0.055, 0.145	1, 2	Assumed
β_C	Transmission rate from a chronically-infected parakeet (individual ⁻¹ day ⁻¹)	$\beta_A I_r$	-	-	1, 2	-
c_{β_A}	Age-dependent acute transmission factor	0.25	Uniform (0.25*0.5, 0.25*1.5)	0.1375, 0.3625	2	Informed from: [120–122]
β_{Aj}	Transmission rate for acutely-infected juvenile parakeet (individual ⁻¹ day ⁻¹)	$(1+c_{\beta_A})\beta_A$	-	-	2	-
β_{Aa}	Transmission rate for acutely-infected adult parakeet (individual ⁻¹ day ⁻¹)	$(1-c_{\beta_A})\beta_A$	-	-	2	-
D_α	Duration of infected but not yet infectious stage (day)	5.5	Exponential (1/5.5)	0.3, 16.5	1, 2	[56]
α	Rate of becoming infectious (day ⁻¹)	$1/D_\alpha$	-	-	1, 2	-
D_δ	Duration of acute-infectious stage (day)	30	Exponential (1/30)	1.5, 89.9	1, 2	[99]
δ	Rate of leaving acute-infectious stage (day ⁻¹)	$1/D_\delta$	-	-	1, 2	-
D_γ	Duration of chronic-infectious stage (day)	39	Exponential (1/39)	2.0, 116.8	1, 2	[69]
γ	Rate of leaving chronic-infectious stage (day ⁻¹)	$1/D_\gamma$	-	-	1, 2	-
D_ν	Duration of immunity (day)	243	Uniform (120, 365)	132.25, 352.75	1, 2	[69]
ν	Rate of losing immunity (day ⁻¹)	$1/D_\nu$	-	-	1, 2	-
p_1	Probability of acute infection	0.625	Uniform (0.5, 0.75)	0.5125, 0.7375	1	[69]

(Continued)

Table 1. (Continued)

Notation	Definition (unit)	Baseline value	Sensitivity analysis	5 th and 95 th percentiles	Model	Source
p_2	Probability of recovery from acute infection	0.5	Uniform (0.375, 0.625)	0.3875, 0.6125	1, 2	Informed from: [123–125]
p_{1j}	Probability of acute juvenile infection	0.75	Uniform (0.625, 0.875)	0.6375, 0.8625	2	Informed from: [65,68,126,127]
p_{1a}	Probability of acute adult infection	0.5	Uniform (0.375, 0.625)	0.3875, 0.6125	2	Informed from: [65,68,126,127]
pd_A	Probability of acute disease-related death	0.25	Uniform (0.1,0.4)	0.115, 0.385	1, 2	Informed from: [73]
μ_A	Disease-related mortality rate for acutely-infected parakeet (day ⁻¹)	pd_A/D_δ	-	-	1, 2	-
pd_C	Probability of chronic disease-related death	0.075	Uniform (0.025, 0.125)	0.03, 0.12	1, 2	[69,99]
μ_C	Disease-related mortality rate for chronically-infected parakeet (day ⁻¹)	pd_C/D_ν	-	-	1, 2	-
c_m	Age-dependent acute mortality factor	0.25	Uniform (0.25*0.5, 0.25*1.5)	0.1375, 0.3625	2	Informed from: [65,68,126,127]
μ_{Aj}	Disease-related mortality rate for acutely-infected juvenile parakeet (day ⁻¹)	$(1+c_m)\mu_A$	-	-	2	-
μ_{Aa}	Disease-related mortality rate for acutely-infected adult parakeet (day ⁻¹)	$(1-c_m)\mu_A$	-	-	2	-

^a Proportions of N_j and N_a were fixed according to disease-free equilibrium (DFE) conditions and remained constant for all iterations. When life expectancy $D_d = 2$, $N_j = 0.22N$, $N_a = 0.78N$; and when $D_d = 9$, $N_j = 0.05N$, $N_a = 0.95N$.

^b Scenario analysis was conducted at 2, 5, and 9 years.

^c 0.6 represents 40% mortality in the juvenile stage.

^d Annual rates were prorated to daily rates.

^e Scenario analysis for hI was conducted at 0, 2, 5, and 10%.

doi:10.1371/journal.pone.0147517.t001

exposure and for those individuals transitioning from an acute infection (Fig 1). We based the immunity-loss rate on the average duration of effective ND antibody titers ($> 1.2 \log_{10}$) [121] in experimental infections so that the average daily rate of immunity loss was $\nu = 1/243$ [69].

Acute and chronic infections. According to ND infection rates in psittacines in captive and experimental conditions, we assumed that all effectively contacted white-winged parakeets would become either acutely or chronically infected, with acutely-infected parakeets shedding more ND virus [69,70,72,130]. Following Erickson et al.’s [69] experimental study, we considered that 63% (p_1) of newly infected white-winged parakeets would become acutely infected, and $1-p_1$ would become chronically infected (Fig 1). After acute infection, parakeets could recover or become chronically infected where individuals continued to shed virus but at lower levels. Little has been published regarding recovery or progression to a chronic ND state; however, a chronic-like state is common following live ND vaccination and has been considered a concern for environmental contamination and infection spread [123–125]. Therefore, we considered that 50% of acutely-infected individuals would recover (p_2), while the remainder ($1-p_2$) would become chronically infected.

Disease-related mortality. Mortality from ND was $> 50\%$ in captive white-winged parakeets [73]. Under free-ranging conditions, we assumed that mortality would be lower and considered that 25% of individuals would die from ND during the acute-infectious period (30

days) making our daily rate, $\mu_A = 0.25/30$ (Table 1). To obtain disease-related mortality in chronically-infected parakeets, we averaged mortality recorded in experimentally-infected psittacines during the nine days following the acute-infection period so that $\mu_C = 0.075/39$ [69,99].

Model 2

Host demography and harvest. We set the initial population size of the two age classes according to their distribution at the disease-free equilibrium with life expectancy (D_d), so that $N(0) = N_j + N_a$. For instance, when $D_d = 5$ years and $N(0) = 200$, $N_j = 16$ (8%) and $N_a = 184$ (92%) (Table 1). We estimated the duration of the juvenile stage based on two factors: (1) the age when juveniles develop immune-competence, and (2) when their survival rate starts to increase. Development of immuno-competence in psittacines and other altricial species is poorly understood [131,132], but appears to occur between six weeks [102,111] and five months [112]. Considering juvenile survival, it is widely recognized that juveniles of many avian species suffer their highest mortality during the first several months after fledging [113–116], which for white-winged parakeets occurs at roughly six weeks of age [109]. Combining these two factors we considered that juveniles transition to adults at $D_\Omega = 135$ days (4.5 months), meaning that the maturation rate was $\Omega = 1/135$.

We fixed juvenile mortality at 40% during the 135-day long juvenile period [115,117]. The corresponding juvenile population was modeled as an exponential decay $S_j(t) = S_j(0)e^{(-d_j \cdot t)}$ so that 60% of the population remained after the juvenile period. The daily natural mortality was then estimated as $d_j = 1/(-D_\Omega/\ln(0.6)) = 1/264$. Adult daily natural mortality d_a was calculated from the difference between the natural mortality already defined for model 1 (d) and the juvenile mortality (d_j), so that $d_a = 1/(1/d - 1/d_j)$ (Table 1). According to market survey data in Peru, roughly 20% more adult white-winged parakeets were harvested annually than juveniles [118], therefore, we set 60% of the total harvest (h) to represent adults ($h_a = 0.6h$) and 40% juveniles ($h_j = 0.4h$).

Transmission rate. Transmission coefficients (β) for ND in juvenile or adult psittacines have not been published; however, acutely-infected juvenile chickens, pigeons, and other species were considered more infectious than adults [120–122,126,133,134]. It is reasonable to assume that the same would be true for juvenile parakeets. For simplicity, we parameterized an acute-transmission factor ($c_{\beta A}$) to represent a 25% increase for acutely-infected juveniles over the baseline rate of transmission (β_A) in model 1, so that $\beta_{Aj} = (1+c_{\beta A})\beta_A$, and a 25% decrease for adult transmission below baseline so that $\beta_{Aa} = (1-c_{\beta A})\beta_A$ (Table 1; Fig 2). We used the same chronic transmission rate (β_C) for juveniles and adults as in model 1. The transition rates for leaving infectious states, α , δ , γ , and ν , used in model 2 were previously defined for model 1 (Table 1).

Acute and chronic infections and disease-related mortality. In natural outbreaks and experimental studies, juvenile birds frequently disproportionately suffered acute clinical signs and high mortality following ND exposure compared to adults [65,68,126,127]. Therefore, we considered that 75% of juvenile white-winged parakeets would become acutely infected following exposure ($p_{Ij} = 0.75$), while $1-p_{Ij}$ would become chronically infected (Table 1; Fig 2). For adults, we considered a 50% probability of becoming acutely or chronically infected, p_{Ia} and $1-p_{Ia} = 0.5$. The proportion of acutely-infected individuals that would recover (p_2) versus becoming chronically infected was defined as in model 1. We used an acute-mortality factor (c_m) to estimate a $\pm 25\%$ age-related difference so that disease-related mortality for acutely-infected juveniles was $\mu_{Aj} = (1+c_m)\mu_A$ and for adults, $\mu_{Aa} = (1-c_m)\mu_A$. Little is known about age-related mortality from chronic ND infection. We assumed that little difference existed among juveniles and adults and, therefore, used the parameter μ_C from model 1 for both.

Scenario analysis

Because life expectancy (D_d) is unknown in the wild, we conducted a scenario analysis to compare the baseline scenario ($D_d = 5$ years) with two extreme scenarios ($D_d = 2$ and $D_d = 9$ years) for both models. Also, we evaluated the effect of additional harvest (hl) at 2, 5, and 10% along with the 1% annual baseline (h_b) harvest (so that the total harvest $h = h_b + hl$) through two scenarios for each model. In the first scenario, population recruitment was fully compensated to include baseline and additional harvest to maintain a stable population in absence of infection, so that $\Lambda = N(d+h)$ in model 1 and $\Lambda_2 = N(d_j+h_j+\Omega)(d_a+h_a)/(d_a+h_a+\Omega)$ in model 2 (Table 1). In the second scenario, additional harvest and infection were introduced simultaneously into the host population, but additional harvest was uncompensated by recruitment and both additional harvest and infection-induced mortality caused the population to decline so that $\Lambda_u = N(d+h_b)$ in model 1 and $\Lambda_{2u} = N(d_j+h_{bj}+\Omega)(d_a+h_{ba})/(d_a+h_{ba}+\Omega)$ in model 2, where $h_{bj} = 0.4h_b$ and $h_{ba} = 0.6h_b$. These two scenarios provided a means to evaluate the interaction between harvest and disease under two different host population reproductive responses to additional harvest (i.e., compensated additional harvest by increased natality in scenario 1 and uncompensated additional harvest with stable natality in scenario 2).

Sensitivity analysis

We examined the sensitivity of the predicted disease-related population decline and R_0 to parameters' uncertainties (Table 1). Selected parameter distributions were supported by the literature; population size was Log-normally distributed [135], values for D_{α} , D_{δ} , and D_{γ} were exponentially distributed [98], and the remaining parameters with unknown distribution were uniformly distributed (Table 1). We performed a sensitivity analysis by simulating 10,000 iterations of the (1) R_0 expressions (Eq A1 and A2) and (2) ODE models (Eqs 1–5 and 6–15) run up to 10,000 days. Parameters were varied simultaneously using the Monte Carlo method and Latin Hypercube sampling that randomly-selected parameter values from their respective distributions [136]. With this number of iterations, R_0 was estimated with a precision of +/- 0.08.

Spearman's correlation coefficient (r_s) was used to calculate and test correlation between each uncertain parameter and R_0 (Table 1). We used $\alpha = 0.05$ as the statistical significance, which was adjusted for multiple testing by implementing the Bonferroni correction. The corrected significance level was $\alpha = 0.0045$ for model 1 and $\alpha = 0.0028$ for model 2. To explore how parameters interacted to create conditions of disease-free ($R_0 < 1$) or endemic ($R_0 \geq 1$) states, we used the parameter values and the associated R_0 estimates from 10,000 iterations per scenario to construct classification trees—binary decision trees that identify the most influential parameters in predicting disease-free or endemic conditions for our homogeneous and age-structured populations [137]. We combined results from all of our scenario analyses on life expectancy (D_d) and additional harvest (hl), for a total of 120,000 data points (iterations) to determine whether these two, and other, model parameters were important predictors of disease-free or endemic states. Classification trees were built using the rpart package in R software [138] and we used the gini index as a measure of node impurity along with a 10-fold cross-validation to select the tree with the smallest misclassification error [139].

In a separate analysis, we compared differences in R_0 estimates produced by models 1 and 2, by starting with equal parameters so that each model produced the same R_0 (i.e., we set the age-structured parameters to be equal and to match those in model 1). We then varied the age-structured parameters (e.g., $c_{\beta A}$, c_m , p_{1j} , and p_{1a}) one at a time to evaluate their individual influence on R_0 . This provided a method to compare results between the two models and to identify which parameters made the biggest difference in R_0 estimates.

Table 2. Scenario analysis of the effect of uncompensated additional harvest (h_1) on the basic reproduction number (R_0) and population size following introduction of Newcastle disease into a homogeneous (model 1) and age-structured (model 2) populations of white-winged parakeets. ^a

Additional harvest, h_1 (%)	Baseline R_0	Mean R_0 (5 th , 95 th percentiles)	Proportion of simulations where $R_0 < 1$ (%)	$N(730)^b$ (5 th , 95 th percentiles)
Model 1				
0 ^c	3.63	3.65 (0.35, 11.30)	21	154.8 (84.9, 254.5)
2	3.31	3.32 (0.31, 10.29)	24	149.3 (81.9, 245.0)
5	2.92	2.93 (0.28, 9.05)	27	141.4 (77.6, 231.6)
10	2.44	2.44 (0.23, 7.52)	33	129.1 (71.1, 211.2)
Model 2				
0 ^c	2.66	2.62 (0.28, 7.53)	28	168.6 (94.9, 272.2)
2	2.54	2.50 (0.27, 7.18)	29	165.0 (92.9, 266.3)
5	2.39	2.34 (0.26, 6.71)	31	159.7 (89.9, 257.6)
10	2.17	2.12 (0.23, 6.07)	34	151.2 (85.2, 243.9)

^a For each scenario, results were based on 10,000 simulations.

^b Population size at day 730 post infection introduction was chosen to capture the short-term effect of harvest on the population size (the initial population size Log-normally distributed with mean of 200 and the 5th and 95th percentiles of 109 and 329, respectively).

^c In all scenarios, the uncompensated additional harvest (h_1) was added to the 1% baseline harvest (h_b). Here uncompensated additional harvest means that population natality did not increase to compensate the population decline due to additional harvest.

doi:10.1371/journal.pone.0147517.t002

Results

Dynamics of ND transmission, R_0 , and harvest

Our baseline R_0 estimates without additional harvest were 3.63 and 2.66 for models 1 and 2, respectively (Table 2). These baseline values were similar to mean R_0 estimates obtained from 10,000 simulations. The distribution range of the R_0 estimates (represented by the 5th and 95th percentiles) were slightly wider in model 1 compared to model 2 (Table 2). With just baseline harvest (i.e., no additional harvest), 21 and 28% of R_0 simulated values were < 1 for models 1 and 2, respectively, meaning that ND failed to become established in the host population (Table 2). The majority of simulated R_0 estimates were < 5 for both models (77% and 87%, respectively). The baseline outbreak dynamics peaked at approximately 120 days for the homogeneous population of white-winged parakeets (Fig 3A), and closer to 150 days with less infected individuals and longer duration in the age-structured population (Fig 3B). Short-term ND dynamics oscillated in both models, but the second infection cycle was slightly delayed in model 2 compared to model 1 (Fig 3; See S1 Fig for separate juvenile and adult trajectories).

As shown in both panels in Fig 4, population size was relatively stable for approximately the first 20 days, after which parakeet abundance decreased sharply and similarly for all tested harvest rates until approximately 180 days in model 1 and 200 days in model 2. This sharp decline, related to the disease outbreak and disease-induced mortality shown in Fig 3, was followed by further, but slower, decline in population size. By two years ($t = 730$ days) post ND introduction with baseline compensated harvest, ND caused the initial population of 200 individuals to decrease by 33% in the homogeneous population ($N(730) = 135$; Fig 4A, blue line), but only by 24% ($N(730) = 153$) when age structure was considered (Fig 4B, blue line).

By 20 years post ND introduction, homogeneous and age-structured populations decreased $> 50\%$ before stabilizing in an endemic state when no additional harvest was considered (Fig 5, blue lines). The distribution of the estimated number of parakeets from 10,000 ODE simulations was slightly wider for model 2 than model 1, which likely reflected additional uncertain parameters in model 2 (Table 2). When we fixed the initial population size to the baseline

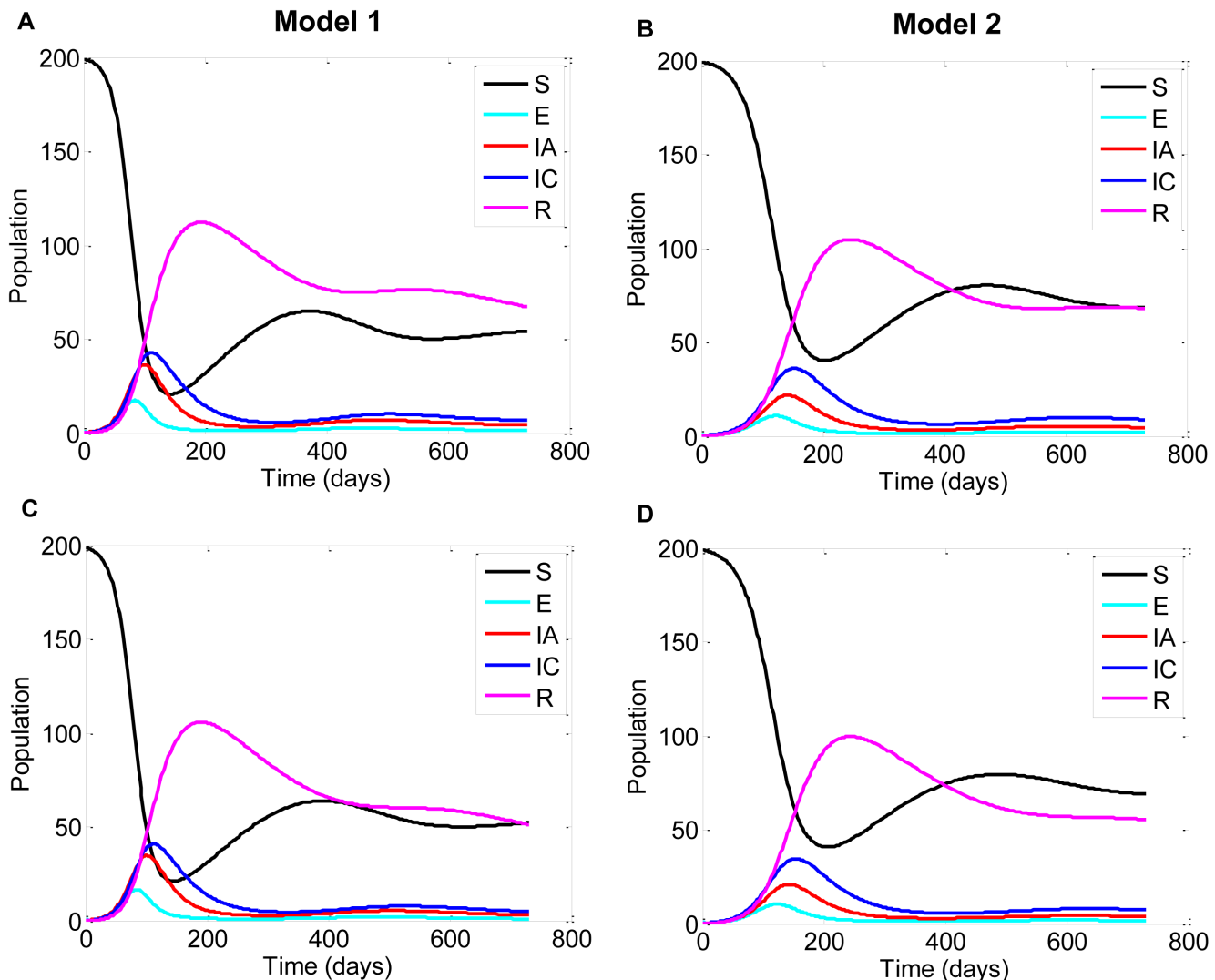


Fig 3. Deterministic two-year time trajectories for Newcastle disease transmission. Simulated outbreak dynamics from homogeneous (model 1) and age-structured (model 2) populations of white-winged parakeets with (A-B) no additional harvest ($hI = 0\%$) and (C-D) 10% additional (uncompensated) harvest ($hI = 10\%$). Here uncompensated additional harvest means that the population natality did not increase to compensate population decline due to additional harvest. Depicted states of infection are: susceptible (S), exposed (E), acutely-infected (IA), chronically-infected (IC) and recovered (R). Age-structured panels (B, D) show summed juvenile and adult parakeets for each infection state. See [S1 Fig](#) for separate juvenile and adult trajectories for model 2.

doi:10.1371/journal.pone.0147517.g003

value ($N = 200$) so that we could evaluate the effects of harvest and disease-related mortality on population decline without the influence of the uncertain initial population size, the width of the 5th and 95th percentiles decreased by over half for both models (45–60%; [S2 Fig](#)). Uncertainty in the initial population size had much less influence on predicted R_0 values ([S3 Fig](#)).

Scenario analyses

Varying life expectancy where $D_d = 2$ or 9 years produced minimal effect in ND transmission represented by mean R_0 estimates and host population size for either homogeneous or age-structured populations of white-winged parakeets ([S4](#) and [S5 Figs](#)). Uncompensated additional harvest dampened R_0 in both homogeneous and age-structured populations ([Table 2](#)).

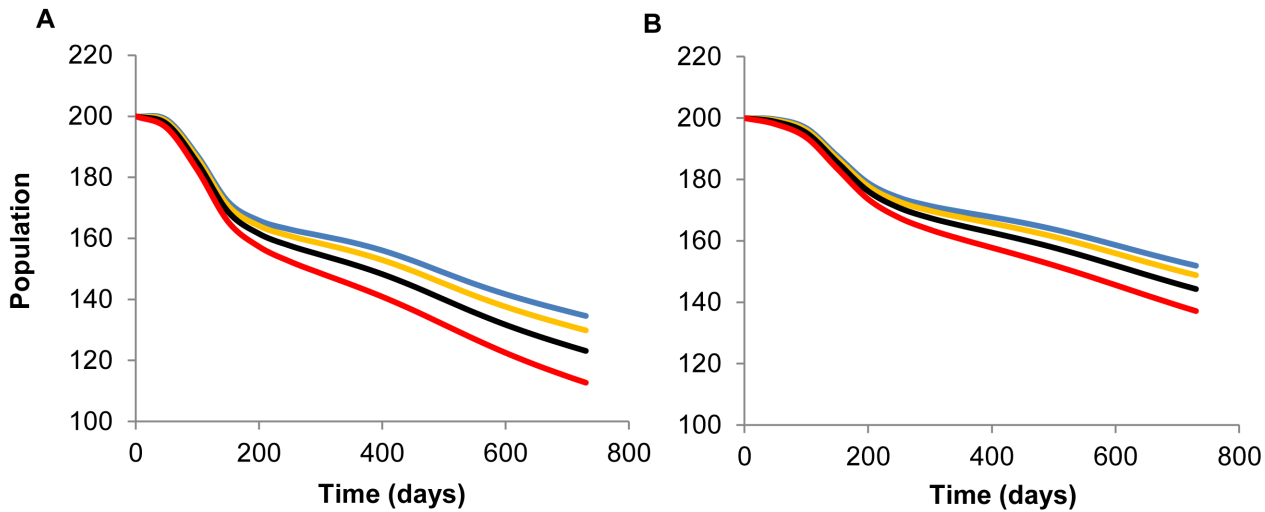


Fig 4. Population decline during two years post Newcastle disease introduction. Population decline in (A) homogeneous (model 1) and (B) age-structured (model 2) populations of white-winged parakeets with no additional harvest ($hI = 0$) and three additional, uncompensated harvest rates (hI ; 0-blue, 2%-orange, 5%-black, and 10%-red). Here uncompensated additional harvest means that population natality did not increase to compensate the population decline due to additional harvest.

doi:10.1371/journal.pone.0147517.g004

Increasing the additional harvest rate from 0 to 10% increased the probability of infection fade out (Table 2). As expected, compensating for additional harvest, where natality (λ) increased in response to higher harvest, caused R_0 to remain stable with increasing additional harvest in both models (Fig 6); however, the confidence intervals for R_0 estimates with compensated and uncompensated harvest largely overlapped. Overall, disease dynamics were minimally affected by higher harvest (Fig 3). The recovered class, which was the longest stage in our compartmental models, was the most affected by higher harvest rates in both models as noted

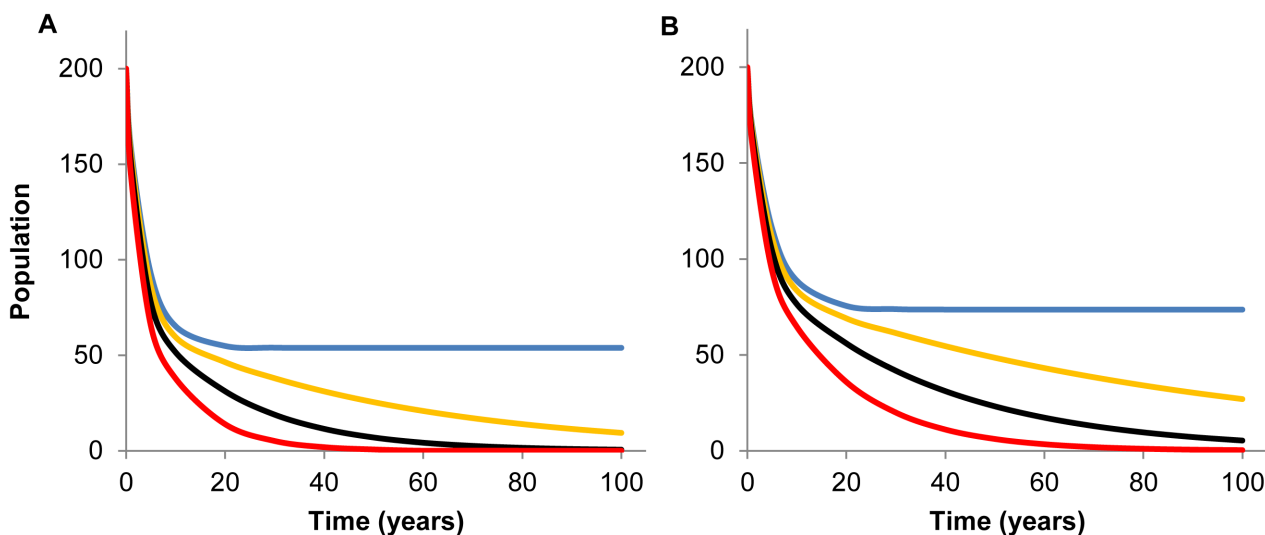


Fig 5. Population decline during 100 years post Newcastle disease introduction. Population decline in (A) homogeneous (model 1) and (B) age-structured (model 2) populations of white-winged parakeets with no additional harvest ($hI = 0$) and three additional uncompensated harvest rates (hI ; 0-blue, 2%-orange, 5%-black, and 10%-red). Here uncompensated additional harvest means that population natality did not increase to compensate the population decline due to additional harvest.

doi:10.1371/journal.pone.0147517.g005

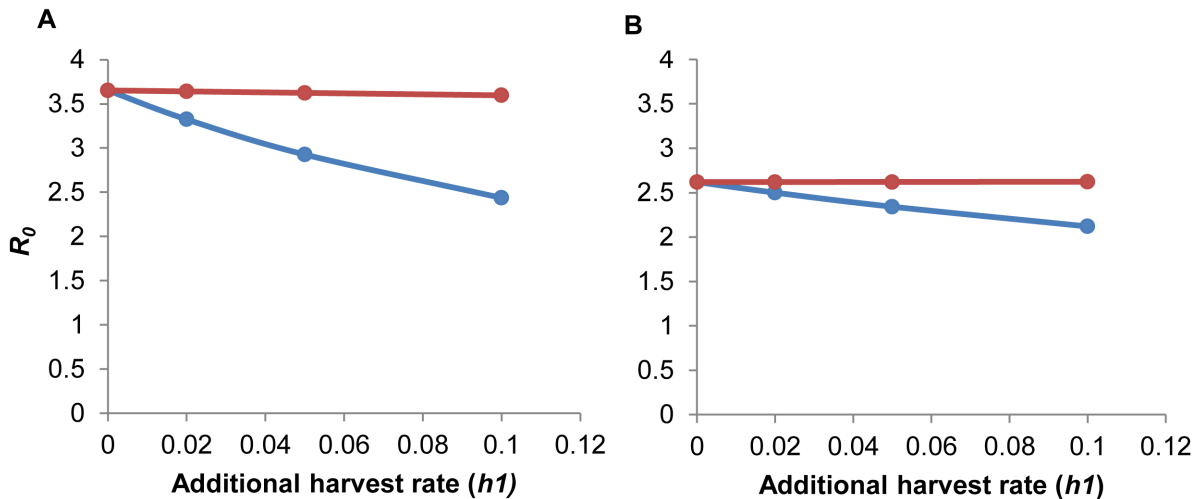


Fig 6. Influence of additional harvest ($h1$) on mean estimates of the basic reproduction number, R_0 . Comparison of mean R_0 estimates under assumptions of compensated (red) and uncompensated (blue) additional harvest rates for (A) homogeneous and (B) age-structured populations of white-winged parakeets following introduction of Newcastle disease. Here compensated and uncompensated additional harvest means respectively that population natality did and did not increase to compensate the population decline due to additional harvest.

doi:10.1371/journal.pone.0147517.g006

by the lower number of individuals with 10% additional harvest versus baseline harvest (Fig 3, panels C–D versus panels A–B, respectively).

Higher uncompensated harvest decreased the size of both homogeneous and age-structured populations of white-winged parakeets, although the decrease was more notable in model 1 versus model 2 and the decline was steeper with higher harvest rates (Fig 4). At two years post ND introduction, 10% additional harvest decreased the homogeneous population of 200 individuals by 35%, but only by 24% in model 2 for the age-structured population (Table 2). The combination of ND and 10% uncompensated additional harvest (without density-dependent population regulation) caused the population to steadily decline, approaching zero within 35 and 60 years for models 1 and 2, respectively (Fig 5).

Sensitivity analysis

The duration of the acute-infectious stage (D_δ) was the most influential (positively-correlated) parameter in determining R_0 for both homogeneous and age-structured populations of white-winged parakeets (Fig 7). Initial population size and the transmission rate for acutely-infected individuals were also positively correlated with R_0 . The probability of acute disease-related death (pd_A) was negatively correlated with R_0 in both models, but had a much stronger influence in model 2 (Fig 7B). Along with pd_A , disease-related mortalities for acutely-infected juvenile (μ_{Aj}) and adult (μ_{Ad}) parakeets, both of which were partially derived from pd_A (Table 1), were highly negatively correlated with R_0 in model 2. The probability of chronic disease-related death (pd_c) was likewise negatively correlated with R_0 , but only significant in model 2.

In the correlation analyses, parameters were evaluated individually; in the multivariable classification trees analyses all uncertain parameters as well as life expectancy (D_d) and additional harvest ($h1$) were evaluated simultaneously. Parameters D_d and $h1$ were not identified as influential in predicting disease-free or endemic conditions. As with correlation analyses, the most optimal classification trees identified the duration of the acute-infectious stage (D_δ) as the most important factor determining whether the infection would persist or undergo fade-out, indicated by its position closest to the root of the tree (Fig 8). By following the rule for branches

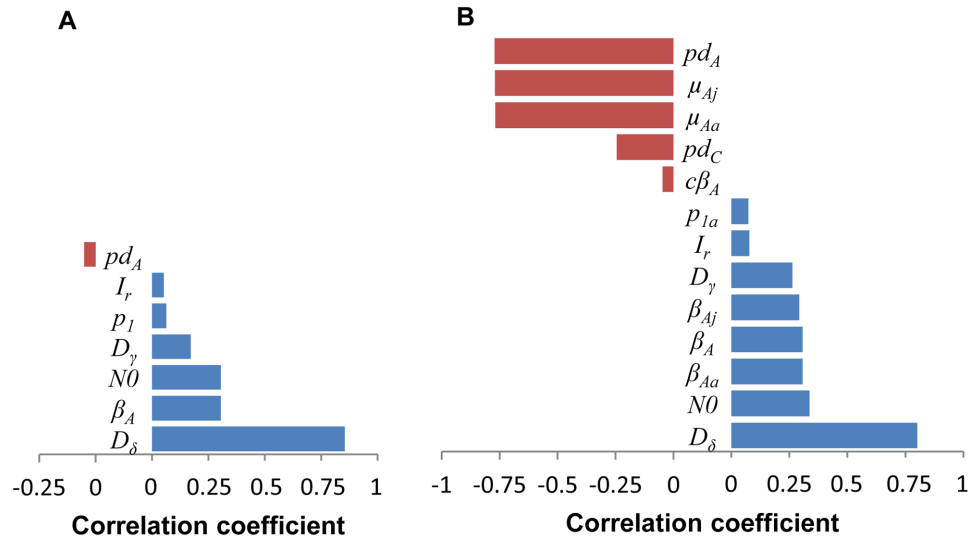


Fig 7. Spearman's correlation coefficient values for models 1 and 2. Spearman's coefficients indicating the strength of the relationship between parameters of the (A) homogeneous (model 1) and (B) age-structured (model 2) models and the basic reproduction number (R_0) with 10% additional harvest from 10,000 simulations. Only parameters with statistically significant coefficients are shown. See Table 1 for parameter descriptions.

doi:10.1371/journal.pone.0147517.g007

to the right, endemic conditions ($R_0 \geq 1$) were predicted to occur under two scenarios in model 1: (1) when $D_\delta \geq 11$ days, and (2) when $D_\delta < 11$ days, but with a duration of chronic-infectious stage $D_\gamma \geq 48$ days, and a transmission rate for chronically-infected parakeets $\beta_c \geq 0.0001071$ (Fig 8A). In model 2, the most optimal classification tree indicated that an endemic state was predicted by four parameters following three different pathways (Fig 8B). As with

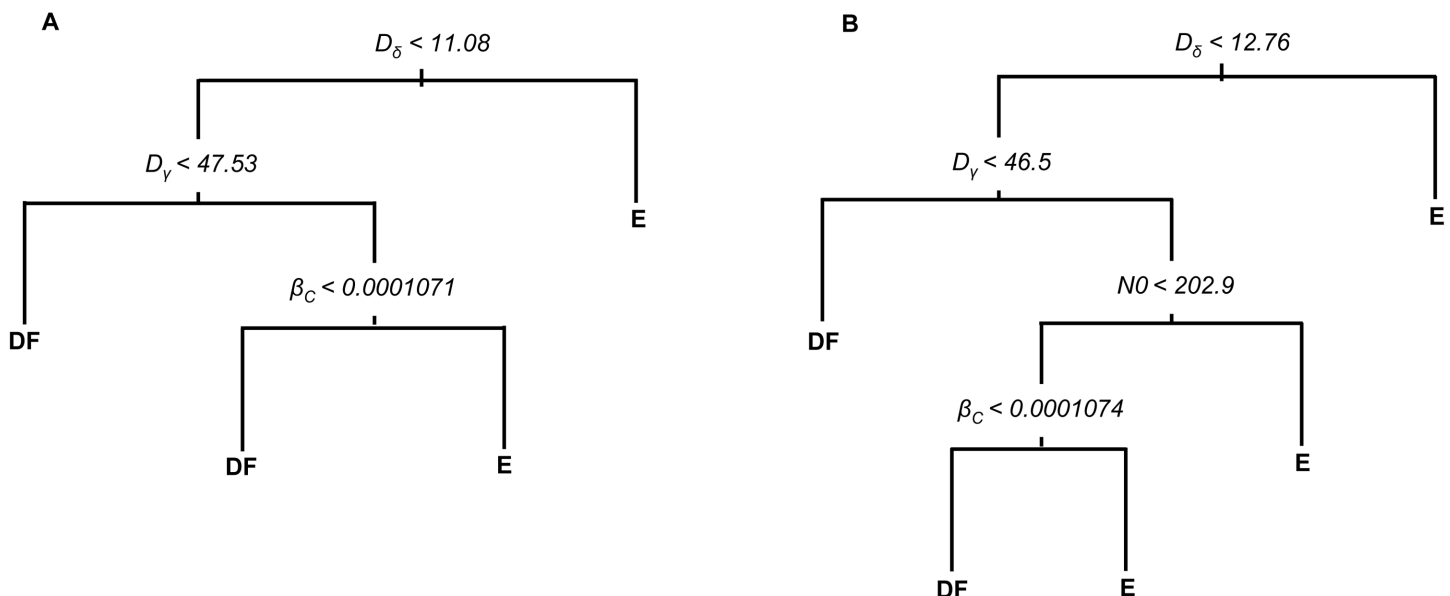


Fig 8. Classification trees for Newcastle disease. Classification tree for disease-free (basic reproduction number, $R_0 < 1$) or endemic ($R_0 \geq 1$) conditions of Newcastle disease (ND) in (A) homogeneous and (B) age-structured populations of white-winged parakeets. The rule for data partitioning is on top of each node. For example, in panel (A), the root node rule is the duration of the acute infectious stage (D_δ) less than 11.08 days; the subset of simulations satisfying this rule partitioned to the left daughter node and consecutively down the nodes. The terminal nodes represent disease-free (DF) or endemic (E) conditions for ND. See Table 1 for parameter descriptions.

doi:10.1371/journal.pone.0147517.g008

model 1, D_δ , D_γ , and β_c were influential parameters in predicting endemic ND, along with the initial population size, $N(0)$. Cross-validation indicated that the predictive error rates were low and similar for models 1 and 2 (12.5% and 12.7%, respectively).

By directly comparing models 1 and 2 and the age-related parameters that differed between the two, we determined that the age-dependent acute transmission factor ($c_{\beta A}$), which increased or decreased the transmission rate by 25% for acutely-infected juveniles and adults, respectively, made the biggest difference in R_0 between the two models. Specifically, lowering the transmission rate for acutely-infected adult parakeets (β_{Aa}) decreased R_0 by 21% from 3.63 in model 1 to 2.88 in model 2. Lowering the probability of acute adult infection (p_{Ia}) in model 2 compared to the homogenous value (p_I) in model 1 decreased R_0 by 15%. The acute mortality factor (c_m), which lowered the disease-related mortality rate for acutely-infected adult parakeets (μ_{Aa}), resulted in increasing R_0 slightly by 4% compared to model 1. The corresponding increase in juvenile parameter values associated with $c_{\beta A}$, p_{Ij} and c_m , in model 2 minimally influenced R_0 compared to model 1.

Discussion

Our results demonstrate that illegal harvest could play an important role in virus transmission during a ND outbreak. We developed two mathematical models to compare the influence of harvest on ND dynamics in a homogeneous and an arguably more realistic age-structured population of white-winged parakeets. We determined that introduction of ND would likely provoke considerable disease-related mortality, but the magnitude of the outbreak would be dampened by high harvest rates. Incorporating age structure into the model produced moderate differences in both R_0 and disease dynamics, primarily due to lower adult disease transmission, compared to our homogeneous population model. Most importantly, the homogenous model likely overestimated the severity of an ND outbreak and highlighted the importance of incorporating even simplistic age structure in disease modeling despite increased complexity and reduced tractability.

R_0 and ND outbreak

Our compartmental models demonstrated that introducing just one ND-infected individual would provoke an outbreak ($R_0 \geq 1$) in susceptible populations of white-winged parakeets with roughly 75% probability (Table 2). In the short term, without population compensation through density-dependent recruitment, population size decreased by 24–44% depending on the harvest rate and host population structure (Fig 4). Even the low end of this range, observed in the age-structured population (model 2) with baseline harvest, is a conservation concern (Fig 4B, blue line). The fact that some psittacine species can chronically shed the ND virus for extended periods [69], implies this disease has the potential to become endemic and remain in the population causing low-level disease-related mortality with significant population decline (Fig 5). In a case of another infectious disease affecting wild avian populations, Hochachka & Dhondt [140] found that epizootic *Mycoplasma* sp. conjunctivitis caused significant population decline in house finches along the eastern United States.

Our results indicate that, in the long term, ND alone would not cause population extinction; however, the combination of ND and consistently high annual harvest (10%) would provoke > 75% population decline in 20 years without a density-dependent response in fecundity (Fig 5). While we did not investigate the interaction between density-dependent effects and illegal harvest on the host population, it would be reasonable to expect that density-dependent regulation would help replenish the population by increasing reproduction [141]. Higher recruitment could potentially increase the supply of ND susceptible parakeets, and thereby exacerbate and/or prolong the outbreak [13,142]. The combination of illegal harvest and ND-

related mortality could have potential devastating population-level effects, but without better demographic data for the white-winged parakeet, it is unclear from what level of decline the population could recover, or possibly stabilize at a new carrying capacity [140,143].

Age structure

The importance of including realistic population demographic factors in disease modeling is well established [144]. In the case of ND, age structure is an important factor to consider because the disease disproportionately affects juveniles. Our modeling results demonstrated that without including age structure (model 1), the severity of a potential ND outbreak was up to 28% higher based on mean R_0 estimates (Table 2) and disease-related population decline was 9–13% higher, depending on the harvest rate, in model 1 versus model 2 at two years post ND introduction (Fig 4). To put this in perspective, a higher R_0 observed in model 1 compared to model 2 suggests that a higher effort is needed to control the infection, which may lead to suboptimal control strategies [145,146].

The dynamics of an ND outbreak were similar in homogeneous and age-structured populations of white-winged parakeets (Fig 3); however, the mildly prolonged infectious periods (I_A , I_C) in model 2 (Fig 3B–3D) would likely provide more opportunities for contact and cross-species exposure. Psittacine species, especially species of similar size, often interact at fruiting trees or clay licks [147,148]. Up to 17 species and hundreds of individuals have been identified at clay licks in Peru [149], which would provide favorable conditions for ND transmission and spread among species [150]. Expanding our age-structured model to include spatial connectivity for meta-population and interspecies interactions would provide valuable insight regarding ND dynamics on a larger scale [151,152] and should be a focus of future research.

Comparison of models 1 and 2 demonstrated that the lower R_0 estimates predicted from model 2 primarily reflect disease dynamics in adult parakeets, specifically a lower transmission rate for acutely-infected adults (determined by $c_{\beta A}$) and, to a lesser degree, a lower probability of acute adult infection. Juvenile parakeets, despite being more infectious than adults, made little impact on R_0 estimates. This was due, in part, to the fact that 40% of juveniles were removed by natural mortality from the population by day 135, and the remaining would then “mature” to adults (Table 1). Adult parakeets, which comprised a much larger proportion of the population than juveniles (Table 1), naturally survived longer and had more time to influence ND transmission and R_0 . As such, lower acute adult mortality in model 2 (determined by c_m) slightly increased R_0 compared to model 1. This suggested that maintaining infected adults for longer time in the population (i.e., not dying from the disease), would exacerbate an ND outbreak even though adults were less infectious compared to juveniles.

We should emphasize that the parameters responsible for the differences in R_0 predictions between models 1 and 2 ($c_{\beta A}$, c_m , and p_{1a}) were uncertain and should be prioritized for future investigation, especially the transmission rate and probability for acutely-infected adult parakeets. However, because model 2 more closely reflects the biological system, it is reasonable to assume that it also more accurately predicts ND dynamics. By extension, it is reasonable to conclude that model 1 overestimated disease transmission and the magnitude of the ND outbreak. In a similar situation, Brooks-Pollock et al. [96] found that more realistic age-specific mortality versus constant mortality rates in mathematical models of human tuberculosis decreased R_0 estimates and the effort required for disease control.

Role of harvest

Through scenario analysis we determined that increasing uncompensated harvest rates (i.e., those not compensated for by natality), had a modest dampening influence on R_0 , meaning

that higher uncompensated harvest increased the probability of ND fadeout ($R_0 < 1$; [Table 2](#)). Although the increase in the proportions of fadeout with increased additional harvest was less dramatic in model 2 versus model 1 (6% compared to 12%; [Table 2](#)), this increase must be considered along with the already lower R_0 estimates in model 2. Compensating additional harvest ($h1$) caused R_0 to remain high in both models (> 2.5), indicating that an influx of susceptible individuals (i.e., offspring) may help sustain higher potential for an ND outbreak ([Fig 6](#)); however, because the confidence intervals for R_0 estimates with compensated and uncompensated harvest largely overlapped (results not shown), largely because of the wide uniform distributions used in our models ([Table 1](#)), the influence of host population density-dependent response on R_0 should be interpreted cautiously.

Higher harvest rates produced slower population decline in the age-structured population compared to the homogeneous population ([Fig 4](#)). This dampened effect in model 2 reflected, in part, the 40% removal of juveniles through natural mortality ([Table 1](#)). High juvenile mortality is often exploited in harvested populations because their lower survival rates and lower reproductive value increase the probability of compensation [143]. For this reason, Choisy & Rohani [13] predicted that shifting harvest to younger age classes would decrease the risk of disease-related mortality in hypothetical scenarios, which is supported by our results.

Overall, higher harvest rates had minimal effect on ND dynamics ([Fig 3](#)) and population size ([Fig 4](#)). This is partly due to the way we prorated the annual harvest rate for the one-day time step in our model simulations, which diluted the effect of harvest, particularly over short time periods such as the 135-day juvenile period. In addition, because we do not know the actual harvest rate, we set our baseline harvest (h_b) conservatively and even compensated for it with natality ([Table 1](#)). It is likely that even our upper limit of 10% additional annual harvest ($h1$) was conservatively low. In model 2, we fixed the proportion of juvenile (40%) and adult (60%) harvest as the average of what was recorded in market surveys throughout the year. In reality, we know that there are seasonal harvest differences, which would likely influence ND dynamics as they do for other diseases [153,154]. For example in model 2, when we changed the fixed harvest proportions to reflect harvest rates during a nesting period (juveniles 90%; adults 10%), R_0 was 20% higher, but the population decline at two years post ND introduction was 12% less compared to the opposite proportions (juveniles 10%, adults 90%; results not shown).

Key parameters

The duration of the acute (D_δ) and chronic (D_γ) infectious stages were most influential in determining R_0 , as identified in both the univariate correlation ([Fig 7](#)) and multivariable classification-tree analyses ([Fig 8](#)). The positive Spearman correlations indicated that longer duration of the infectious stages resulted in higher R_0 estimates. In poultry operations, decreasing the duration of infectious stages (i.e., the length of time of ND viral shedding), is one of the primary goals for improving vaccination programs [145,155]. With captive pet birds, preventive measures such as vaccination could help diminish clinical signs and the duration and amount of viral shedding. The efficacy of ND vaccination in white-winged parakeets is unknown, although Denadai et al. [112] determined that Australian parakeets could be safely and effectively vaccinated against ND. The negative correlation between the probability of acute disease-related death (pd_A) and R_0 in both models ([Fig 7](#)) reflects the importance of removing acutely-infected individuals from the population. During an outbreak, this could be achieved by quarantining or culling acutely-infected individuals [95]. During outbreaks of ND in commercial flocks, quickly culling all infected and potentially-infected individuals has been a critical component of typical management strategies to prevent expansion of the outbreak [156].

Neither vaccination nor quarantine, however, would be feasible to control ND in wild populations of white-winged parakeets. This highlights the vulnerability of wild populations.

The classification-tree analyses provided a broad perspective of the key parameters and their interactions to produce disease-free or endemic ND states (Fig 8). In addition to the duration of the acute (D_a) and chronic (D_c) infectious stages, the most optimal classification trees identified the transmission rate for chronically-infected parakeets (β_c) as influencing R_0 , rather than the transmission rate for acutely-infected parakeets (β_a) as in our correlation analysis. When the interaction of transmission rates was evaluated along with other parameters, it became clear that the rate of virus transmission from chronically-infected birds (β_c) to susceptible individuals would become critical in determining whether ND would die out or persist, specifically when chronic infection period (D_c) lasted longer than approximately 47 days (Fig 8). Such insight was impossible evaluating correlation alone, and demonstrates the value of multi-variable analyses. Even though the most optimal classification trees retained only three or four parameters, the predictive error rates were relatively low for both models, indicating that the identified optimal classification trees correctly predicted infection fade out or an endemic state in almost 90% of independent simulations.

Limitations

Our findings are dependent on several modeling assumptions. For instance, we assumed that ND transmission was density dependent in white-winged parakeets. Density-dependent transmission is commonly assumed for wildlife diseases [103,157]. In most cases of wildlife diseases, empirical data are difficult to obtain to confirm transmission, but Hochachka & Dhondt [140] used pre- and post-enzootic data to conclude that mycoplasma conjunctivitis transmission in house finches was density dependent. In some situations, the mode of pathogen transmission may not be constant throughout the year as demographic seasonal traits affect social behavior and spatial structure of the host population [158]. For example, Hosseini et al. [154] combined frequency- and density-dependent transmission for *Mycoplasma gallisepticum* to represent seasonal variation in social structure of house finches.

A similar situation could occur with ND transmission in white-winged parakeets as flock size fluctuates throughout the year [109]. During the roughly 4-month breeding season, when pairs separate for nesting and flock size decreases, transmission may be more consistent with frequency dependence. During the post-breeding period, when adults along with their fledglings rejoin the flock, transmission is more likely to be density dependent. Addressing the role of chance in pathogen transmission could be evaluated by incorporating demographic stochasticity into the model. A stochastic approach could also assess the influence of changing the initial conditions, (e.g., number of ND-infectious individuals released into the population). For instance, we assumed that just one infectious individual would be introduced into a susceptible population of white-winged parakeets. In reality, it is common for authorities to release dozens to hundreds of potentially exposed individuals confiscated from markets [80]. While this was beyond the scope of the work presented here, including demographic stochasticity and density-dependent population natality and mortality processes in a seasonal age-structured model would provide a method to more thoroughly investigate the influence of harvest on ND dynamics [151].

Recommendations

Few realistic options exist to control an ND outbreak in wild white-winged parakeets or other parrots. Assuring that *criollo* chickens and fighting cocks in the Amazon region are vaccinated against ND would reduce the probability of cross infection to psittacine species in animal

markets. In our models, we used a non-threatened psittacine species; however, many parrots are threatened in Peru—often by illegal trade—and could be seriously affected by an introduced infectious disease [46]. The most effective preventive measure would be to avoid releasing confiscated parakeets without prior health screening and, ideally, preventing illegal harvest in the first place. Authorities should coordinate more effectively with non-governmental organizations in Peru working to decrease the illegal wildlife trade including wildlife rescue centers and zoological parks [54], to assist with quarantine and rehabilitation of confiscated individuals. Similarly, authorities could increase collaboration with veterinary colleges to assist with physical exams and diagnostic testing of confiscated animals. Finally, combined efforts to increase enforcement of Peru's wildlife legislation and to decrease demand for wild-caught native birds for the domestic pet market would help mitigate the risk of introducing infectious diseases.

In conclusion, our study improved understanding of ND dynamics in a wild population of harvested psittacines. We demonstrated that the hypothetical release of a confiscated individual infected with ND would provoke considerable population decline in a wild population of white-winged parakeets. To our knowledge, this is the first study to use infectious disease modeling to link illegal wildlife trade and disease introduction in a native wildlife population. The differences we observed in both R_0 and disease dynamics between our homogeneous and age-structured populations highlight the importance of incorporating even simplistic age structure in disease modeling. While we recognize that further enhancements, such as including density-dependent regulation and demographic stochasticity, could contribute to the understanding of ND dynamics, our initial models provide a baseline for future evaluation. We encourage the conservation community to examine other disease risks associated with illegal wildlife trade, particularly in endangered species where disease may contribute to species extinctions.

Supporting Information

S1 Fig. Deterministic two-year time trajectories for Newcastle disease transmission for juvenile and adult white-winged parakeets.

(PDF)

S2 Fig. Comparison of the effect of fixed and uncertain population size on population size.

(PDF)

S3 Fig. Comparison of the effect of fixed and uncertain population size on R_0 .

(PDF)

S4 Fig. Mean basic reproductive number (R_0) estimates.

(PDF)

S5 Fig. Population size following Newcastle disease (ND) introduction.

(PDF)

S1 File. Matlab codes for Model 1 and Model 2.

(PDF)

S2 File. Analytic derivation of the basic reproduction number (R_0).

(PDF)

S3 File. Photographs of parrots and chickens at animal markets in three Peruvian cities.

(PDF)

Acknowledgments

We are grateful to D. Brightsmith (Texas A&M University) and P. Mendoza (Wildlife Conservation Society, Peru) for their insights regarding parrot trade in Peru. E. Daut is grateful for the financial and academic support provided by Applied Biodiversity Science NSF-IGERT program, the Department of Veterinary Pathobiology, and the Schubot Exotic Bird Health Center at Texas A&M University. We also thank two anonymous reviewers who provided valuable comments that helped improve the manuscript.

Author Contributions

Conceived and designed the experiments: EFD GL RI. Performed the experiments: EFD GL RI. Analyzed the data: EFD RI. Wrote the paper: EFD RI MJP.

References

1. Daszak P, Cunningham AA, Hyatt AD (2000) Emerging infectious diseases of wildlife—threats to biodiversity and human health. *Science* 287: 443–449. PMID: [10642539](#)
2. Fèvre EM, Bronsvoort BMdC, Hamilton KA, Cleaveland S (2006) Animal movements and the spread of infectious diseases. *Trends in Microbiology* 14: 125–131. PMID: [16460942](#)
3. Rosen GE, Smith KF (2010) Summarizing the evidence on the international trade in illegal wildlife. *Ecohealth* 7: 24–32. doi: [10.1007/s10393-010-0317-y](#) PMID: [20524140](#)
4. Nekaris KAI, Shepherd CR, Starr CR, Nijman V (2010) Exploring cultural drivers for wildlife trade via an ethnoprimate approach: a case study of slender and slow lorises (*Loris* and *Nycticebus*) in South and Southeast Asia. *American Journal of Primatology* 72: 877–886. doi: [10.1002/ajp.20842](#) PMID: [20806336](#)
5. Nijman V, Shepherd CR, Mumpuni, Sanders KL (2012) Over-exploitation and illegal trade of reptiles in Indonesia. *The Herpetological Journal* 22: 83–89.
6. Shepherd CR (2012) Illegal primate trade in Indonesia exemplified by surveys carried out over a decade in North Sumatra. *Endangered Species Research* 11: 201–205.
7. Sung Y-H, Karraker NE, Hau BCH (2013) Demographic evidence of illegal harvesting of an endangered Asian turtle. *Conservation Biology* 27: 1421–1428. doi: [10.1111/cobi.12102](#) PMID: [23869813](#)
8. Daszak P, Cunningham AA, Hyatt AD (2003) Infectious disease and amphibian population declines. *Diversity and Distributions* 9: 141–150.
9. Frick WF, Pollock JF, Hicks AC, Langwig KE, Reynolds DS, Turner GG, et al. (2010) An emerging disease causes regional population collapse of a common North American bat species. *Science* 329: 679–682. doi: [10.1126/science.1188594](#) PMID: [20689016](#)
10. Smith KF, Sax DF, Lafferty KD (2006) Evidence for the role of infectious disease in species extinction and endangerment. *Conservation Biology* 20: 1349–1357. PMID: [17002752](#)
11. Warner RE (1968) The role of introduced diseases in the extinction of the endemic Hawaiian avifauna. *Condor* 70: 101–120.
12. Wyatt KB, Campos PF, Gilbert MTP, Kolokotronis S-O, Hynes WH, DeSalle R, et al. (2008) Historical mammal extinction on Christmas Island (Indian Ocean) correlates with introduced infectious disease. *PLoS ONE* 3: e3602. doi: [10.1371/journal.pone.0003602](#) PMID: [18985148](#)
13. Choisy M, Rohani P (2006) Harvesting can increase severity of wildlife disease epidemics. *Proceedings of the Royal Society B: Biological Sciences* 273: 2025–2034. PMID: [16846909](#)
14. Woodroffe R, Donnelly CA, Cox DR, Bourne FJ, Cheeseman CL, Delahay RJ, et al. (2006) Effects of culling on badger (*Meles meles*) spatial organization: Implications for the control of bovine tuberculosis. *Journal of Applied Ecology* 43: 1–10.
15. Wonham MJ, de Camino-Beck T, Lewis MA (2004) An epidemiological model for West Nile virus: Invasion analysis and control applications. *Proceedings of the Royal Society of London Series B: Biological Sciences* 271: 501–507. PMID: [15129960](#)
16. van den Berg T (2009) The role of the legal and illegal trade of live birds and avian products in the spread of avian influenza. *Revue Scientifique et Technique—Office International des Epizooties* 28: 93–111.
17. Brooks-Moizer F, Robertson SI, Edmunds K, Bell D (2009) Avian influenza H5N1 and the wild bird trade in Hanoi, Vietnam. *Ecology and Society*. Available: <http://www.ecologyandsociety.org/vol14/iss1/art28/>.

18. Gilbert M, Sokha C, Joyner PH, Thomson RL, Poole C (2012) Characterizing the trade of wild birds for merit release in Phnom Penh, Cambodia and associated risks to health and ecology. *Biological Conservation* 153: 10–16.
19. Karesh WB, Smith KM, Asmussen MV (2012) The unregulated and informal trade in wildlife: implications for biodiversity and health. *Compendium of the OIE Global Conference on Wildlife*. Paris, France: OIE (World Organisation for Animal Health). pp. 51–57.
20. Karesh WB (1995) Wildlife rehabilitation: Additional considerations for developing countries. *Journal of Zoo and Wildlife Medicine* 26: 2–9.
21. Bennett EL (2011) Another inconvenient truth: the failure of enforcement systems to save charismatic species. *Oryx* 45: 476–479.
22. Keane A, Jones JPG, Edwards-Jones G, Milner-Gulland EJ (2008) The sleeping policeman: understanding issues of enforcement and compliance in conservation. *Animal Conservation* 11: 75–82.
23. Chng SCL, Eaton JA, Krishnasamy K, Shepherd CR, Nijman V (2015) In the market for extinction: an inventory of Jakarta's bird markets. *Petaling Jaya, Malaysia: TRAFFIC*.
24. Shepherd CR (2006) The bird trade in Medan, North Sumatra: An overview. *BirdingASIA* 5: 16–24.
25. Tieguhong JC, Ndoye O, Mpele TO (2006) Commodity-chain analysis for the capture and trade in the African grey parrots (*Psittacus erithacus erithacus*) in Cameroon. Washington, DC: World Resources Institute.
26. RSPCA (2006) It's time to stop winging it. West Sussex, England: Royal Society for the Prevention of Cruelty to Animals.
27. Eniang EA, Akpan CE, Eniang ME (2008) A survey of African grey parrots (*Psittacus erithacus*) trade and trafficking in Ekonganaku are of Ikpan forest block, Nigeria. *Ethiopian Journal of Environmental Studies and Management* 1: 68–73.
28. Pain DJ, Martins TLF, Boussekey M, Diaz SH, Downs CT, Ekstrom JMM, et al. (2006) Impact of protection on nest take and nesting success of parrots in Africa, Asia and Australasia. *Animal Conservation* 9: 322–330.
29. Daut EF, Brightsmith DJ, Mendoza AP, Puhakka L, Peterson MJ (2015) Illegal domestic bird trade and the role of export quotas in Peru. *Journal for Nature Conservation* 27: 44–53.
30. Herrera M, Hennessey B (2007) Quantifying the illegal parrot trade in Santa Cruz de la Sierra, Bolivia, with emphasis on threatened species. *Bird Conservation International* 17: 295–300.
31. Alves RRN, Lima JR, Araujo HFP (2012) The live bird trade in Brazil and its conservation implications: An overview. *Bird Conservation International* 23: 53–65.
32. Cantú JC, Saldaña MES, Grosselet M, Gamez JS (2007) The illegal parrot trade in Mexico: a comprehensive assessment. Washington, DC: Defenders of Wildlife.
33. Drews C (2001) Wild animals and other pets kept in Costa Rican households: Incidence, species and numbers. *Society & Animals* 9: 107–126.
34. Regueira RFS, Bernard E (2012) Wildlife sinks: Quantifying the impact of illegal bird trade in street markets in Brazil. *Biological Conservation* 149: 16–22.
35. Roldán-Clarà B, Lopez-Medellín X, Espejel I, Arellano E (2014) Literature review of the use of birds as pets in Latin America, with a detailed perspective on Mexico. *Ethnobiology and Conservation*. Available: <http://www.ethnobiococonservation.com/index.php/ebc/article/view/43>.
36. BirdLife International (2012) Overexploitation is a threat to many large and conspicuous bird species. Available: <http://www.birdlife.org/datazone/sowb/casestudy/162>.
37. Levinson M (1987) In Brazil, bird songs aren't just cheap trills. *International Wildlife Magazine* 17: 48–53.
38. Jepson P, Ladle RJ (2009) Governing bird-keeping in Java and Bali: Evidence from a household survey. *Oryx* 43: 364–374.
39. Alves R, Nogueira E, Araujo H, Brooks S (2010) Bird-keeping in the Caatinga, NE Brazil. *Human Ecology* 38: 147–156.
40. Thomsen JB, Edwards SR, Mulliken TA, editors (1992) Perceptions, conservation and management of wild birds in trade. Cambridge, U.K.: TRAFFIC International.
41. Beissinger SR, Snyder NFR, Derrickson SR, James FC, Lanyon SM (1991) The conservation crisis—international-trade in live exotic birds creates a vast movement that must be halted. *Auk* 108: 982–984.
42. Collar NJ (2000) Globally threatened parrots: Criteria, characteristics and cures. *International Zoo Yearbook* 37: 21–35.

43. Butchart SHM, Stattersfield AJ, Bennun LA, Shutes SM, Akçakaya HR, Baillie JEM, et al. (2004) Measuring global trends in the status of biodiversity: Red list indices for birds. *PLoS Biology* 2: e383. PMID: [15510230](#)
44. BirdLife International (2015) BirdLife Data Zone. Available: www.birdlife.org/datazone/species/search.
45. Clarke R, de By R (2013) Poaching, habitat loss and the decline of Neotropical parrots: A comparative spatial analysis. *Journal of Experimental Criminology* 9: 333–353.
46. BirdLife International (2014) Country profiles. Available: <http://www.birdlife.org/datazone/country/peru>.
47. González JA (2003) Harvesting, local trade, and conservation of parrots in the northeastern Peruvian Amazon. *Biological Conservation* 114: 437–446.
48. Gastañaga M, Hennessey B, Ugarte Núñez J, Puse E, Arrascue A, Hoyos J, et al. (2010) A study of the parrot trade in Peru and the potential importance of internal trade for threatened species. *Bird Conservation International* 21: 76–85.
49. Begazo AJ (1989) La comercialización de animales silvestres vivos en Lima con énfasis en el mercado central. Lima, Perú: Thesis, Universidad Nacional Agraria La Molina, Lima, Perú.
50. Ríos L, Riva F, Canaquire L (2008) Reporte situacional del tráfico ilegal de fauna silvestre en la región nororiental del Perú. Lima, Peru: ProNaturaleza. 1–74 p.
51. Myers N, Mittermeier RA, Mittermeier CG, da Fonseca GAB, Kent J (2000) Biodiversity hotspots for conservation priorities. *Nature* 403: 853–858. PMID: [10706275](#)
52. Schulenberg TS, Stotz DF, Lane DF, O'Neill JP, Parker TT III (2007) *Birds of Peru*. Princeton: Princeton University Press.
53. Birdlife International (2014) Species factsheet: *Brotogeris versicolorus*. Available: <http://www.birdlife.org/datazone/speciesfactsheet.php?id=1622&m=1>.
54. Daut EF, Brightsmith DJ, Peterson MJ (2015) Role of non-governmental organizations in combating illegal wildlife-pet trade in Peru. *Journal for Nature Conservation* 24: 72–82.
55. Beissinger SR, Bucher EH (1992) Sustainable harvesting of parrots for conservation. In: Beissinger SR, Snyder NFR, editors. *New world parrots in crisis: Solutions from conservation biology*. Washington, DC: Smithsonian Institution Press. pp. 73–115.
56. Leighton FA, Heckert RA (2007) Newcastle disease and related avian paramyxoviruses. In: Thomas NJ, Hunter B, editors. *Infectious diseases of wild birds*. Ames, IA: Blackwell Publishing. pp. 3–16.
57. Kinde H, Hullinger PJ, Charlton B, McFarland M, Hietala SK, Velez V, et al. (2005) The isolation of exotic Newcastle disease (END) virus from nonpoultry avian species associated with the epidemic of END in chickens in southern California: 2002–2003. *Avian Diseases* 49: 195–198. PMID: [16094822](#)
58. Walker JW, Heron BR, Mixson MA (1973) Exotic Newcastle disease eradication program in the United States. *Avian Diseases* 17: 486–503. PMID: [4748340](#)
59. Alexander DJ, Lister SA, Wilson GWC (1986) Avian paramyxovirus type 1 infection of racing pigeons: 5 continued spread in 1984. *The Veterinary Record* 118: 424–427. PMID: [3705396](#)
60. Kuiken T (1999) Review of Newcastle disease in cormorants. *Waterbirds: The International Journal of Waterbird Biology* 22: 333–347.
61. Spradbrow P (2005) Appropriate vaccination and therapies for rural poultry flocks in developing countries and their relevance to developed countries. *World's Poultry Science Journal* 61: 47–54.
62. Jibril AH, Umoh JU, Kabir J, Saidu L, Magaji AA, Bello MB, et al. (2014) Newcastle disease in local chickens of live bird markets and households in Zamfara state, Nigeria. *ISRN Epidemiology*. doi: [10.1155/2014/513961](#)
63. Awan MA, Otte MJ, James AD (1994) The epidemiology of Newcastle disease in rural poultry: A review. *Avian Pathology* 23: 405–423. PMID: [18671109](#)
64. World Parrot Trust (2004) Deadly Newcastle disease discovered in parrots and other birds imported from Pakistan to Italy. Available: http://www.parrots.org/pdfs/press_releases/newcastle_disease.pdf.
65. Alexander DJ (2009) Ecology and epidemiology of Newcastle disease. In: Capua I, Alexander DJ, editors. *Avian influenza and Newcastle disease*. Milan, Italy: Springer-Verlag. pp. 19–26.
66. Hines NL, Miller CL (2012) Avian paramyxovirus serotype-1: A review of disease distribution, clinical symptoms, and laboratory diagnostics. *Veterinary Medicine International*: 1–17. doi: [10.1155/2012/708216](#)
67. Kaleta E, Baldauf C (1988) Newcastle disease in free-living and pet birds. In: Alexander DJ, editor. *Newcastle disease*. New York: Springer. pp. 197–246.

68. Panigrahy B, Senne DA, Pearson JE, Mixson MA, Cassidy DR (1993) Occurrence of velogenic viscerotropic Newcastle disease in pet and exotic birds in 1991. *Avian Diseases* 37: 254–258. PMID: [8452504](#)
69. Erickson GA, Maré CJ, Gustafson GA, Miller LD, Proctor SJ, Carbrey EA (1977) Interactions between viscerotropic velogenic Newcastle disease virus and pet birds of six species. I. Clinical and serologic responses, and viral excretion. *Avian Diseases* 21: 642–654. PMID: [606222](#)
70. Clubb SL, Levine BM, Graham DL. An outbreak of viscerotropic velogenic Newcastle disease in pet birds; 1980; Washington, DC. *American Association of Zoo Veterinarians*. pp. 105–109.
71. Hirai K (1981) An occurrence of Newcastle disease in other psittacine birds exposed to imported cockatoos (*Kakatoe sulphurea*). *Japanese Journal of Veterinary Science* 43: 557–559. PMID: [7339142](#)
72. Bruning Fann C (1992) Investigation of an outbreak of velogenic viscerotropic Newcastle disease in pet birds in Michigan, Indiana, Illinois, and Texas. *Journal of the American Veterinary Medical Association* 201: 1709–1714. PMID: [1293111](#)
73. Grausgruber VW (1972) Newcastle-krankheit bei papagein. *Wiener tierärztliche monatschrift* 59: 353–356. PMID: [4679543](#)
74. Pearson GL (1975) The role of indigenous wild, semidomestic, and exotic birds in the epizootiology of velogenic viscerotropic Newcastle disease in Southern California, 1972–1973. *Journal of the American Veterinary Medical Association* 167: 610–614. PMID: [1176357](#)
75. Nilsson G (1985) Importation of birds into the United States 1980–1984, volume 2. Washington, DC: Animal Welfare Institute.
76. OIE (2015) World Animal Health Information Database (WAHID) Interface; Event summary: Newcastle disease, Peru. Available: http://www.oie.int/wahis_2/public/wahid.php/Diseaseinformation/statusdetail.
77. Ferrer M, Icochea E, Salas A, Alba M (2008) Prevalencia de anticuerpos contra el virus de la enfermedad de Newcastle en *Gallus gallus* de Lima: Estudio de caso-control. *Revista de investigaciones veterinarias del Perú* 19: 67–74.
78. The Poultry Site (2009) Newcastle disease back in Peru. Available: <http://www.thepoultrysite.com/poultrynews/18057/newcastle-disease-back-in-peru>.
79. Diel DG, Susta L, Cardenas Garcia S, Killian ML, Brown CC, Miller PJ, et al. (2012) Complete genome and clinicopathological characterization of a virulent Newcastle disease virus isolate from South America. *Journal of Clinical Microbiology* 50: 378–387. doi: [10.1128/JCM.06018-11](https://doi.org/10.1128/JCM.06018-11) PMID: [22135263](#)
80. González Medina OE (2004) Ecología de aves urbanas en un parque de la ciudad de Lima. Lima, Peru: Universidad Nacional Mayor de San Marcos.
81. Jiménez I, Cadena CD (2004) Por qué no liberar animales silvestres decomisados. *Ornitología Colombiana* 2: 53–57.
82. Godoy SN, Matushima ER (2010) A survey of diseases in passeriform birds obtained from illegal wildlife trade in São Paulo City, Brazil. *Journal of Avian Medicine and Surgery* 24: 199–209. PMID: [21046940](#)
83. Marini MA, Garcia FI (2005) Bird conservation in Brazil. *Conservation Biology* 19: 665–671.
84. Zhang L, Hua N, Sun S (2008) Wildlife trade, consumption and conservation awareness in southwest China. *Biodiversity and Conservation* 17: 1493–1516.
85. Gomes GF, Pimentel TL, Monti R, Cabral R, Barreto R (2012) Efforts to combat wildlife animals trafficking in Brazil. Available: doi: [10.5772/48351](https://doi.org/10.5772/48351)
86. Goodman BB, Hanson RP (1988) Isolation of avian paramyxovirus-2 from domestic and wild birds in Costa Rica. *Avian Diseases* 32: 713–717. PMID: [3202769](#)
87. Deem SL, Noss AJ, Cuéllar RL, Karesh WB (2005) Health evaluation of free-ranging and captive blue-fronted Amazon parrots (*Amazona aestiva*) in the Gran Chaco, Bolivia. *Journal of Zoo and Wildlife Medicine* 36: 598–605. PMID: [17312715](#)
88. Gilardi KVK, Lowenstine LJ, Gilardi JD, Munn CA (1995) A survey for selected viral, chlamydial, and parasitic diseases in wild dusky-headed parakeets (*Aratinga weddellii*) and tui parakeets (*Brotogeris sanctithomae*) in Peru. *Journal of Wildlife Diseases* 31: 523–528. PMID: [8592384](#)
89. Johnson DC, Couvillion CE, Pearson JE (1986) Failure to demonstrate viscerotropic velogenic Newcastle disease in psittacine birds in the Republic of the Philippines. *Avian Diseases* 30: 813–815. PMID: [3814018](#)
90. Karesh WB, Campo AD, Braselton WE, Puche H, Cook RA (1997) Health evaluation of free-ranging and hand-reared macaws (*Ara spp.*) in Peru. *Journal of Zoo and Wildlife Medicine* 28: 368–377. PMID: [9523629](#)

91. Dubé C, Garner G, Stevenson M, Sanson R, Estrada C, Willeberg P. The use of epidemiological models for the management of animal diseases; 2007; Washington, DC. World Organisation for Animal Health (OIE). pp. 13–23.
92. Keeling MJ, Rohani P (2007) Modeling infectious diseases in humans and animals. Princeton: Princeton University Press.
93. Heesterbeek JAP (2002) A brief history of R_0 and a recipe for its calculation. *Acta Biotheoretica* 50: 189–204. PMID: [12211331](#)
94. Haydon DT, Kao RR, Kitching RP (2004) The U.K. foot-and-mouth disease outbreak—the aftermath. *Nature Reviews Microbiology* 2: 675–681. PMID: [15263902](#)
95. Wobeser G (2002) Disease management strategies for wildlife. *Revue Scientifique et Technique—Office International des Epizooties* 21: 159–178.
96. Brooks-Pollock E, Cohen T, Murray M (2010) The impact of realistic age structure in simple models of tuberculosis transmission. *PLoS ONE* 5: e8479. doi: [10.1371/journal.pone.0008479](#) PMID: [20062531](#)
97. Heisey DM, Joly DO, Messier F (2006) The fitting of general force-of-infection models to wildlife disease prevalence data. *Ecology* 87: 2356–2365. PMID: [16995636](#)
98. Anderson RM, May RM (1991) *Infectious diseases of humans: Dynamics and control*. New York: Oxford University Press.
99. Erickson GA, Maré CJ, Beran GW, Carbrey EA (1978) Epizootiologic aspects of viscerotropic velogenic Newcastle disease in six pet bird species. *American Journal of Veterinary Research* 39: 105–107. PMID: [629430](#)
100. Forshaw JM (1989) *Canary-winged parakeet. Parrots of the world*, 3rd Edition. Willoughby, Australia: Lansdowne Editions. pp. 528–531.
101. Tossas AG, Colón YM, Sanders I (2012) Seasonal changes in roost counts of the non-native white-winged parakeet (*Brotogeris versicolurus*) in San Germán, Puerto Rico. *Journal of Caribbean Ornithology* 25: 1–6.
102. Lung NP (1996) Maternal immunoglobulin G antibody transfer and development of immunoglobulin G antibody responses in blue and gold macaw (*Ara ararauna*) chicks. *American Journal of Veterinary Research* 57: 1162–1167. PMID: [8836368](#)
103. McCallum H, Barlow N, Hone J (2001) How should pathogen transmission be modelled? *Trends in Ecology and Evolution* 16: 295–300. PMID: [11369107](#)
104. Anderson RM, May RM (1981) The population dynamics of microparasites and their invertebrate hosts. *Philosophical Transactions of the Royal Society of London Series B, Biological Sciences* 291: 451–524.
105. May RM, Anderson RM (1979) Population biology of infectious diseases: Part II. *Nature* 280: 455–461. PMID: [460424](#)
106. Li X, Chai T, Wang Z, Song C, Cao H, Liu J, et al. (2009) Occurrence and transmission of Newcastle disease virus aerosol originating from infected chickens under experimental conditions. *Veterinary Microbiology* 136: 226–232. doi: [10.1016/j.vetmic.2008.11.002](#) PMID: [19091492](#)
107. Hugh-Jones M, Allan WH, Dark FA, Harper GJ (1973) The evidence for the airborne spread of Newcastle disease. *The Journal of Hygiene* 71: 325–339. PMID: [4515881](#)
108. van den Driessche P, Watmough J (2002) Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission. *Mathematical Biosciences* 180: 29–48. PMID: [12387915](#)
109. Shroads CV (1974) *Studies on a population of the canary-winged parakeet Brotogeris versicolurus (P. L. S. Muller) in Dade County, Florida (Aves: psittacidae)*. Miami, FL: The University of Miami.
110. Young AM, Hobson EA, Lackey LB, Wright TF (2012) Survival on the ark: Life-history trends in captive parrots. *Animal Conservation* 15: 28–43. PMID: [22389582](#)
111. Ritchie BW, Niagro FD, Latimer KS, Steffens WL, Pesti D, Campagnoli RP, et al. (1992) Antibody response to and maternal immunity from an experimental psittacine beak and feather disease vaccine. *American Journal of Veterinary Research* 53: 1512–1518. PMID: [1416348](#)
112. Denadai J, Carlos Paulillo A, dos Santos Schmidt EM, Martins GRV, Lapera IM, de Oliveira Tories Carrasco A (2010) Clinical and immunological aspects of Newcastle disease vaccination in budgerigars (*Melopsittacus undulatus*). *International Journal of Poultry Science* 9: 756–766.
113. Rowley I (1983) Mortality and dispersal of juvenile galahs, *Cacatua roseicapilla*, in the western Australian wheatbelt. *Wildlife Research* 10: 329–342.
114. Lindsey GD, Arendt WJ, Kalina J (1994) Survival and causes of mortality in juvenile Puerto Rican parrots. *Journal of Field Ornithology* 65: 76–82.

115. Salinas-Melgoza A, Renton K (2007) Postfledging survival and development of juvenile lilac-crowned parrots. *Journal of Wildlife Management* 71: 43–50.
116. Cox WA, Thompson F, Cox A, Faaborg J (2014) Post-fledging survival in passerine birds and the value of post-fledging studies to conservation. *Journal of Wildlife Management* 78: 183–193.
117. Sandercock BK, Beissinger SR (2002) Estimating rates of population change for a neotropical parrot with ratio, mark-recapture and matrix methods. *Journal of Applied Statistics* 29: 589–607.
118. Daut EF (2015) Conservation implications of illegal bird trade and disease risk in Peru. Ph.D. Thesis: Texas A&M University.
119. Johnston J. Computer modelling to expand our understanding of disease interactions in village chickens. In: Spradbrow PB, editor; 1992; Kuala Lumpur, Malaysia. Australian Centre for International Agricultural Research. pp. 46–55.
120. Erickson GA, Brugh M, Beard CW (1980) Viscerotropic velogenic Newcastle disease in pigeons: Clinical disease and immunization. *Avian Diseases* 24: 257–267.
121. Alexander DJ, Bell JG, Alders RG (2004) Technology review: Newcastle disease. Rome, Italy: Food and Agriculture Organization of the United Nations.
122. Kapczynski DR, Wise MG, King DJ (2006) Susceptibility and protection of naïve and vaccinated racing pigeons (*Columbia livia*) against exotic Newcastle disease virus from the California 2002–2003 outbreak. *Avian Diseases* 50: 336–341. PMID: [17039831](#)
123. Parede L, Young PL (1990) The pathogenesis of velogenic Newcastle disease virus infection of chickens of different ages and different levels of immunity. *Avian Diseases* 34: 803–808. PMID: [2282010](#)
124. Carrasco AT, Seki M, de Sousa RM, Raso T, Pinto A (2009) Protection levels of vaccinated pigeons (*Columba livia*) against a highly pathogenic Newcastle disease virus strain. *Tropical Animal Health and Production* 41: 1325–1333. doi: [10.1007/s11250-009-9318-7](#) PMID: [19253000](#)
125. Chukwudi OE, Chukwuemeka ED, Mary U (2012) Newcastle disease virus shedding among healthy commercial chickens and its epidemiological importance. *Pakistan Veterinary Journal* 32: 354–356.
126. Kuiken T, Leighton FA, Wobeser G, Danesik KL, Riva J, Heckert RA (1998) An epidemic of Newcastle disease in double-crested cormorants from Saskatchewan. *Journal of Wildlife Diseases* 34: 457–471. PMID: [9706555](#)
127. Barman LR, Flensburg MF, Permin A, Madsen M, Islam MR (2010) A controlled study to assess the effects of vaccination against Newcastle disease in village chickens. *The Bangladesh Veterinarian* 27: 56–61.
128. Vickers ML, Hanson RP (1979) Experimental Newcastle disease virus infections in three species of wild birds. *Avian Diseases* 23: 70–79. PMID: [486009](#)
129. Kuiken T, Heckert RA, Riva J, Leighton FA, Wobeser G (1998) Excretion of pathogenic Newcastle disease virus by double-crested cormorants (*Phalacrocorax auritus*) in absence of mortality or clinical signs of disease. *Avian Pathology* 27: 541. doi: [10.1080/03079459808419381](#) PMID: [18484041](#)
130. Pearson GL. Techniques for the isolation of viruses from exotic birds submitted for importation into the United States; 1976; Madison, Wisconsin. pp. 61–68.
131. Schat K, Kaspers B, Kaiser P, editors (2013) *Avian immunology*. New York: Elsevier Science.
132. Apanius V (1980) Ontogeny of immune function. In: Starck J, Ricklefs RE, editors. *Avian growth and development: Evolution within the altricial-precocial spectrum*. New York: Oxford University Press. pp. 203–222.
133. Rupiper DJ (1998) Diseases that effect race performance of homing pigeons. Part I: Husbandry, diagnostic strategies, and viral diseases. *Journal of Avian Medicine and Surgery* 12: 70–77.
134. Hoque MA, Burgess GW, Karo-Karo D, Cheam AL, Skerratt LF (2012) Monitoring of wild birds for Newcastle disease virus in north Queensland, Australia. *Preventive Veterinary Medicine* 103: 49–62. doi: [10.1016/j.prevetmed.2011.08.013](#) PMID: [21945812](#)
135. Limpert E, Stahel WA, Abbt M (2001) Log-normal distributions across the sciences: Keys and clues. *BioScience* 51: 341–352.
136. Helton JC, Davis FJ (2003) Latin hypercube sampling and the propagation of uncertainty in analyses of complex systems. *Reliability Engineering & System Safety* 81: 23–69.
137. Ivanek R, Gröhn YT, Wells MT, Lembo AJ, Sauders BD, Wiedmann M (2009) Modeling of spatially referenced environmental and meteorological factors influencing the probability of *Listeria* species isolation from natural environments. *Applied and Environmental Microbiology* 75: 5893–5909. doi: [10.1128/AEM.02757-08](#) PMID: [19648372](#)
138. Therneau T, Atkinson B, Ripley B (2014) Package 'rpart'. Available: <http://cran.r-project.org/web/packages/rpart/rpart.pdf>.

139. Vayssières MP, Plant RE, Allen-Diaz BH (2000) Classification trees: An alternative non-parametric approach for predicting species distributions. *Journal of Vegetation Science* 11: 679–694.
140. Hochachka WM, Dhondt AA (2000) Density-dependent decline of host abundance resulting from a new infectious disease. *Proceedings of the National Academy of Sciences* 97: 5303–5306.
141. Ricklefs RE (2000) Density dependence, evolutionary optimization, and the diversification of avian life histories. *The Condor* 102: 9–22.
142. Woodroffe R, Cleaveland S, Courtenay O, Laurenson MK, Artois M (2004) Infectious diseases in the management and conservation of wild canids. In: Macdonald D. W., Sillero-Zubiri C, editors. *Biology and conservation of wild canids*. Oxford, UK: Oxford University Press. pp. 123–142.
143. Beissinger SR (2001) Trade of live wild birds: Potentials, principles and practices of sustainable use. In: Reynolds JD, Mace GM, Redford KH, Robinson JG, editors. *Conservation of exploited species*. Cambridge: Cambridge University Press. pp. 182–202.
144. Hudson PJ, Rizzoli AP, Grenfell BT, Heesterbeek H, Dobson AP, editors (2002) *The ecology of wild-life diseases*. New York: Oxford University Press.
145. van Boven M, Bouma A, Fabri THF, Katsma E, Hartog L, Koch G (2008) Herd immunity to Newcastle disease virus in poultry by vaccination. *Avian Pathology* 37: 1–5. doi: [10.1080/03079450701772391](https://doi.org/10.1080/03079450701772391) PMID: [18202943](https://pubmed.ncbi.nlm.nih.gov/18202943/)
146. John TJ, Samuel R (2000) Herd immunity and herd effect: New insights and definitions. *European Journal of Epidemiology* 16: 601–606. PMID: [11078115](https://pubmed.ncbi.nlm.nih.gov/11078115/)
147. Burger J, Gochfeld M (2003) Parrot behavior at a Rio Manu (Peru) clay lick: Temporal patterns, associations, and antipredator responses. *Acta Ethologica* 6: 23–34.
148. Galetti M (1997) Seasonal abundance and feeding ecology of parrots and parakeets in a lowland Atlantic forest of Brazil. *Ararajuba* 5: 115–126.
149. Powell LL, Powell TU, Powell GVN, Brightsmith DJ (2009) Parrots take it with a grain of salt: Available sodium content may drive collpa (clay lick) selection in southeastern Peru. *Biotropica* 41: 279–282.
150. Butron O, Brightsmith DJ (2010) Testing for *Salmonella* spp. in released parrots, wild parrots, and domestic fowl in lowland Peru. *Journal of Wildlife Diseases* 46: 718–723. PMID: [20688677](https://pubmed.ncbi.nlm.nih.gov/20688677/)
151. Lande R, Saether BE, Engen S (2001) Sustainable exploitation of fluctuating populations. In: Reynolds JD, Mace GM, Redford KH, Robinson JG, editors. *Conservation of exploited species*. Cambridge, UK: Cambridge University Press. pp. 67–86.
152. Hess G (1996) Disease in metapopulation models: Implications for conservation. *Ecology* 77: 1617–1632.
153. Altizer S, Dobson A, Hosseini P, Hudson P, Pascual M, Rohani P (2006) Seasonality and the dynamics of infectious diseases. *Ecology Letters* 9: 467–484. PMID: [16623732](https://pubmed.ncbi.nlm.nih.gov/16623732/)
154. Hosseini PR, Dhondt AA, Dobson A (2004) Seasonality and wildlife disease: How seasonal birth, aggregation and variation in immunity affect the dynamics of *Mycoplasma gallisepticum* in house finches. *Proceedings of the Royal Society of London Series B: Biological Sciences* 271: 2569–2577. PMID: [15615682](https://pubmed.ncbi.nlm.nih.gov/15615682/)
155. Miller PJ, Estevez C, Yu Q, Suarez DL, King DJ (2009) Comparison of viral shedding following vaccination with inactivated and Live Newcastle disease vaccines formulated with wild-type and recombinant viruses. *Avian Diseases* 53: 39–49. PMID: [19432002](https://pubmed.ncbi.nlm.nih.gov/19432002/)
156. United States Department of Agriculture (2014) *Newcastle disease response plan—the red book*. Washington, DC: USDA.
157. Lloyd-Smith JO, Cross PC, Briggs CJ, Daugherty M, Getz WM, Latto J, et al. (2005) Should we expect population thresholds for wildlife disease? *Trends in Ecology and Evolution* 20: 511–519. PMID: [16701428](https://pubmed.ncbi.nlm.nih.gov/16701428/)
158. Oraby T, Vasilyeva O, Krewski D, Lutscher F (2014) Modeling seasonal behavior changes and disease transmission with application to chronic wasting disease. *Journal of Theoretical Biology* 340: 50–59. doi: [10.1016/j.jtbi.2013.09.003](https://doi.org/10.1016/j.jtbi.2013.09.003) PMID: [24035840](https://pubmed.ncbi.nlm.nih.gov/24035840/)