High annual survival in infected wildlife populations may veil a persistent extinction risk from disease

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Abstract. Host response to emerging pathogens is variable, causing uncertainty about population-level impacts and challenging effective disease management. White-nose syndrome (WNS) has caused catastrophic declines in some bat species, while others appear less impacted. Developing predictive models based on observed survival patterns can generate testable hypotheses about mechanisms driving population dynamics and contribute to the development of targeted approaches to disease management. We conducted a mark–recapture study of federally endangered Indiana bats (Myotis sodalis) during 2011–2016. Annual survival decreased from 0.78 (95% CI: 0.59, 0.89) and 0.79 (95% CI: 0.70, 0.86) for females and males, respectively, in 2011 to 0.74 (95% CI: 0.33, 0.94) and 0.75 (95% CI: 0.53, 0.89) for females and males, respectively, in 2015. We then modeled two explanatory mechanisms potentially driving the observed patterns: (1) phased exposure to disease through the spatial spread of the pathogen within the hibernaculum; and (2) cumulative mortality risk from iterative yearly WNS infection. Under a phased exposure scenario, models suggest that infected individuals have an average survival probability of 0.68, and disease prevalence is predicted to reach 100% within 9 yr of disease emergence. Under the cumulative mortality risk hypothesis, survival probability of individuals decreases with each infection cycle. In either case, infected populations are predicted to stabilize at a negative growth rate. Results suggest that Indiana bats tolerate a pathogen load prior to onset of infection, leading to a less pronounced population decline than for other susceptible species. However, the long-term risk of WNS to Indiana bats may be more severe than current population trends suggest. To inform current conservation management, we performed a vital rate sensitivity analysis, which suggested that modest increases in survival (4–5%) through targeted intervention may return declining populations to stability (κ = 1.0). Demographic modeling approaches coupled with continued population monitoring can highlight important differences in disease response, and ultimately extinction risk, in host species allowing conservation practitioners to tailor intervention actions so that they will be most effective.

Key words: epidemiology; mark–recapture; Myotis sodalis; population viability analysis; Pseudogymnoascus destructans; white-nose syndrome; wildlife disease.

Received 6 July 2017; revised 21 September 2017; accepted 29 September 2017. Corresponding Editor: Debra P. C. Peters. Copyright © 2017 Maslo et al. This is an open access article under the terms of the Creative Commons Attribution License, which permits use, distribution and reproduction in any medium, provided the original work is properly cited.
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INTRODUCTION

Fungal pathogens are becoming increasingly prevalent as emerging disease agents and are responsible for several current major epizootics (Fisher et al. 2012). Notable examples include *Pseudogymnoascus destructans* (white-nose syndrome [WNS]) in bats (Gargas et al. 2009), *Batrachochytrium dendrobatidis* (chytridiomycosis) in frogs (Kilpatrick et al. 2010), *Batrachochytrium salamandrivorans* (chytridiomycosis) in salamanders (Martel et al. 2013), and *Ophidiomyces ophiodiicola* (snake fungal disease) in snakes (Lorch et al. 2015). Host susceptibility to emerging diseases is variable, with some species declining by >90% and others persisting with endemic infection (Daszak et al. 2003, Langwig et al. 2017).

Drivers of post-infection population dynamics likely include a combination of host physiological, genetic, and behavioral factors. For some species, evolutionary processes (Woodworth et al. 2005), compensatory effects (Muths et al. 2011), or changes in sociality (Langwig et al. 2012) can either reduce the rate of population decline or promote population persistence in the presence of the pathogen. Importantly, however, relatively moderate population declines may veil a persistent extinction risk. Host characteristics promoting disease resistance or tolerance may be negated by inter-host interactions that amplify pathogen prevalence or transmission (Mideo et al. 2008, Ellis et al. 2017). Alternatively, populations overcoming the acute mortality associated with the epidemic phase of pathogen invasion may experience more subtle, chronic negative impacts that maintain pronounced vulnerability to extinction.

Particularly for multi-host pathogens, understanding how each species responds to an emerging disease becomes critical to successful intervention. Conservation managers often face limited time and resources and thus must prioritize species, populations, and sites where intervention will have the greatest impact (Arponen 2012). Uncertainty in species-level impacts prevents such targeted disease management planning, diminishing the potential benefits of conservation actions (Possingham et al. 2001) and increasing the risk of unintended outcomes (i.e., exacerbating declines; Maslo et al. 2017). Combining population monitoring with predictive models aimed at generating testable hypotheses to explain observed dynamics, then investigating those hypotheses empirically, can guide conservation efforts through an adaptive management framework and ultimately increase the potential for success.

White-nose syndrome, caused by the fungal pathogen *Pseudogymnoascus destructans* (Gargas et al. 2009), is a disease of hibernating bats that has recently emerged in North America (Warnecke et al. 2012). Although WNS has been confirmed in multiple bat species (Blehert et al. 2009), there exists considerable variability in infection intensity and impact on host population dynamics (Frick et al. 2015, 2017). As an example, initial declines of little brown bats (*Myotis lucifugus*) averaged 73%, leading to predictions of regional extirpation in less than two decades (Frick et al. 2010a). In contrast, the closely related Indiana bat *Myotis sodalis* has experienced much less severe population-level impacts across most of its infected range (Frick et al. 2015). Counts of multiple hibernating colonies have estimated an ~6% reduction in average growth rate for WNS-infected populations (Langwig et al. 2012). These data, coupled with recent evidence of reduced pathogen loads on infected individuals (Frick et al. 2017), suggests that Indiana bats may be buffered against extirpation from WNS.

Here, we present the sex-specific and temporal trends in Indiana bat survival generated from five years of mark–recapture data from a WNS-infected hibernaculum. Based on these empirical estimates, we generate two hypotheses to potentially explain the mechanisms driving observed survival patterns: (1) spatial spread of *P. destructans* within a hibernaculum resulting in a phased exposure of Indiana bats to an infective dose (i.e., leading to disease); and (2) Indiana bats experiencing cumulative mortality risk from repeated exposure to the pathogen. Using mathematical modeling approaches, we predict the future dynamics of Indiana bat populations under each scenario.

METHODS

Mark–recapture methods

We conducted fieldwork from 2011 to 2016 at the western shaft of the Mt. Hope Mine complex located on privately owned land in Rockaway, New Jersey, USA. This gated 41-m vertical shaft is the roost site of the largest known hibernating...
colony of Indiana bats in New Jersey and was confirmed as a WNS-infected site in 2011 using UV fluorescence (Turner et al. 2014, Frick et al. 2017). In each project year, we captured Indiana bats during the fall swarm (August–October) over three separate three-night sampling events. We captured bats using two harp traps (Model G7; Bat Conservation and Management, Carlisle, Pennsylvania, USA) and three 38-mm mesh, 2.6-m mist nets (Avinet, Freeville, New York, USA) placed at 6, 9, and 12 m heights. We positioned the harp traps at the gate surrounding the shaft opening, and we stationed one mist net adjacent to the shaft opening. We deployed two mist nets within 200 m from the shaft opening, one on a nearby abandoned mining road and one at the entrance to an exposed rock gorge. We banded captured individuals with unique 2.9-mm (in 2011) or 2.4-mm (2012–2016) lipped alloy bands (Porzana, Ickleham, UK) and recorded their age (young-of-the-year or adult, >1 yr; Brunet-Rossini and Wilkinson 2009) and sex. Banding activities were conducted under annually issued NJ Division of Fish and Wildlife Scientific Collecting Permits and generally followed both the United States Fish and Wildlife Service Range-wide Indiana Bat Summer Survey Guidelines, as well as the methods of Kunz and Parsons (2009). All field personnel followed national White-Nose Syndrome Decontamination Protocols during all visits.

**Survival estimation**

We used the standard Cormack-Jolly-Seber model to estimate annual apparent survival, \( S \), and recapture probability, \( p \) (Lebreton et al. 1992). We generated 19 a priori candidate models containing combinations of constant, yearly, time trend, and sex-specific effects on annual survival and recapture probabilities (Appendix S1: Table S1). Because the sample size precluded meaningful age-specific inferences to be drawn, we excluded age-related data from all analyses. To test model fit, we used a parametric bootstrapping procedure with 500 simulations of our global model, which included fully time-dependent and sex-specific survival and recapture. We ranked candidate models by \( \Delta QAI C_C \), which represents the relative likelihood of the model, given the data (Johnson and Omland 2004). To reduce model selection bias and uncertainty, we calculated estimates by averaging parameters within all models returning a \( \Delta QAI C_C < 2 \) (Burnham and Anderson 2002, Burnham et al. 2011).

**Modeling population dynamics under a phased exposure to \( P. destructans \)**

Studies have shown that prevalence of \( P. destructans \) on Indiana bats within a hibernaculum nears 100% within three years of its arrival to a site (Frick et al. 2017), but individuals have moderate \( P. destructans \) loads relative to species that experienced massive initial declines (Hoyt et al. 2016). We hypothesized that the spatial spread of \( P. destructans \) within a hibernaculum could result in a phased exposure of Indiana bats to an infective dose (\( P. destructans \) load causing diagnostic signs) of WNS. We propose to differentiate between an inoculum that results in a positive \( P. destructans \) laboratory test and an “infective dose” sufficient to lead to active pathology in the host bat. In this case, survival probabilities derived from mark-recapture operations would reflect the population average. However, the population consists of both infected and uninfected individuals that are surviving at two different rates. To calculate the “true” survival probability of infected individuals, we disaggregated the Indiana bat population into two discrete compartments (uninfected and infected), where individuals from the uninfected compartment can contract WNS and join the infected compartment where they remain until death. We modeled the population growth of both compartments using the following set of exponential growth equations:

\[
U_{t+1} = \lambda^u U_t - \alpha_t \\
I_{t+1} = \lambda^i I_t + \alpha_t
\]

where \( I_t \) and \( U_t \) refer to the numbers of bats in the infected and uninfected compartments, respectively, \( \lambda \) is the compartment-specific growth rate (specified by the superscripts \( i \) and \( u \)), \( \alpha_t \) is the number of bats newly infected by transmission in year \( t \), and the subscript \( t \) denotes time in years.

To simulate population growth, we calculated \( \lambda_u \) as the dominant eigenvalue for the uninfected compartment using the following two-stage Lefkovitch matrix (Morris and Doak 2002):

\[
\lambda^u = \left[ \frac{S_j \times B_i \times m_j}{S_j} \times \frac{S_a \times B_a \times m_a}{S_a} \right] v
\]
where $v$ is the eigenvector associated with the unique largest positive eigenvalue; $S$ represents survival of uninfected Indiana bats; $B$ represents the proportion of uninfected females breeding; $m$ is fecundity; and the subscripts $j$ and $a$ indicate values for juvenile (i.e., first-year) or adult bats, respectively. Because the estimated survival probability for 2011 generated from the mark-recapture analysis was consistent with literature describing Indiana bat dynamics just prior to the emergence of WNS (Thogmartin et al. 2012), we used this value to parameterize adult survival in the matrix ($S_a$). We set first-year bat survival as a constant proportion (0.47) of adult survival (Frick et al. 2010b, Maslo et al. 2015), and we held fecundity constant at $m = 1$, as Indiana bats typically produce a single pup per year (Barclay and Harder 2003). We assigned breeding proportion values for first-year bats and adults as $B_j = 0.38$ and $B_a = 0.85$, respectively, based on estimates reported in the closely related little brown bat because no current empirically derived data for Indiana bats exist (Reichard and Kunz 2009, Frick et al. 2010b, Maslo et al. 2015).

We calculated the growth rate of the infected compartment, $\lambda_i$, by using the exponential growth equation subdivided into the two compartments and weighted by the proportion of infected individuals in a given year, $p_i$:

$$\Lambda_i^w = (1 - p_i)\lambda^w + p_i\lambda^j$$  

(4)

where $\Lambda_i^w$ represents the growth rate of the entire population in any given year, $t$, calculated by populating the above matrix with the empirically derived survival probabilities for 2012. Initially, we assumed that 5–15% of the population became infected by the second year.

To calculate $I_t$, we first determined the proportion of individuals who are in the infected compartment during a given year, $p_i$, by solving for $p_i\lambda^j$ in Eq. 4. We then calculated the number of infected bats in a specified year:

$$I_t = p_iN_t$$

where $N_t$ is the projected total population size at time $t$ given exponential growth with $\Lambda_i^w$, and an initial population size of 409 (USFWS 2017). We then solve for the number uninfected bats that become infected during a specified year, $x_t$, according to an assumed Type I functional response curve (i.e., linear):

$$x_t = kI_t + \pi$$  

(5)

where $k$ and $\pi$ are the slope and intercept of a linear regression estimating the relationship between the number of uninfected bats that became infected ($x_t$) and the total number of bats ($I_t$). A Type I functional response is similar to how transmission is modeled in a basic susceptible-infected-recovered epidemiological model and assumes that the contact rate between individuals in either compartment is proportional to population density (McCallum et al. 2001).

To determine the long-term dynamics of the population, we projected the initial population 20 yr into the future using Eqs. 1 and 2 and tracking $U, I$, and $N$. We added stochastic effects by running 1000 iterations of the model, drawing from normal distributions with a standard deviation of 0.1 of each mean $\lambda^w$ and $\lambda^j$. Because the Indiana bat population is low, there are likely no density-dependent effects; we ignored potential Allee effects because no data are available for bat populations reduced by WNS.

**Modeling cumulative mortality risk to Indiana bats from iterative WNS infection**

Growth of *P. destructans* is temperature dependent (Verant et al. 2012), and infected bats surviving the hibernation period can clear fungal loads during the summer season. Bats are then reinfection upon their return to hibernation sites, which serve as environmental reservoirs (Lorch et al. 2013). We hypothesized that Indiana bats could be experiencing systemic physiological degradation from WNS such that survival probability decreases with each repeated exposure to *P. destructans*. We thus calculated the impact of each accumulation of years of disease exposure as a mixed rates problem. We assumed that in the absence of the pathogen all vital rates were constant, and we used the mean observed 2011 survival probability generated from the mark-recapture analysis as the uninfected baseline. We then assumed that the observed annual decrease in survival was the impact solely of disease introduction into the population (Thogmartin et al. 2012). Using 2012 as the first year under disease influence, we assumed that all adults surviving in the population were living with the burden of one year of disease exposure. In 2013, first-year bats born in 2012 would then be exposed to...
WNS for a single year, but all other surviving adults now carried the burden of two years of exposure. In 2014, first-year bats experienced a single year of exposure, 2-yr-old bats had two years of exposure, and all older surviving adults existed with the burden of three years of exposure. Based upon longevity records for Indiana bats (Paradiso and Greenhall 1967, Humphrey and Cope 1977), we used a maximum life expectancy of 11 yr. Therefore, interaction of post-exposure demography was stable only after 10 yr of disease presence in the population. At this time, there would no longer be multiple age classes experiencing the same number of years of exposure; rather, number of years of exposure would simply be determined by an individual’s age.

Based upon these assumptions, we calculated the survival penalty Indiana bats incur with each cumulative WNS infection cycle. We define \( s_k \) as the average survival probability for each age \( k \) adult class in the years prior to WNS emergence (as estimated by the mark–recapture analysis). We then define \( s^*_k \) as the modified survival in each age class \( k \), based on the duration of exposure in each year \( t \) (in this case, from 2012 to 2015). Because we know the average annual survival probability over all age classes during these years, \( S_t \), from the mark–recapture data, we can solve for the penalty associated with each accumulation of years of exposure, \( e_k \), for each year according to the equation

\[
\bar{S}_t = \frac{\sum_k s^*_k x_k}{k}
\]

where \( x_k \) is the proportion of individuals in age class \( k \), and

\[
s^*_k = \begin{cases} 
  s_k p_{t-2011} & \text{if } t - 2011 \leq k \\
  s_k e_k & \text{if } t - 2011 > k
\end{cases}
\]

(6)

Because the observational window includes only four years of data following WNS introduction, we hypothesized two potential functional forms for the shape of the exposure penalty lasting more than four years in duration: linear and logarithmic progression. To define these, we employed a standard curve fitting algorithm for linear and logarithmic functions (minimizing sum of squared differences) on the four penalties: \( e_1, \ldots, e_4 \). We then used those two fit functions to project the progression of the penalty for years 5 through 11. Using the penalty modifiers to the survival probabilities (i.e., the \( s^*_k \) for each subsequent year), we then projected the population over the first 10 yr following WNS emergence using standard Leslie matrix calculations and calculated the expected population growth rate at stability (i.e., the associated dominant eigenvalue for the matrix using the \( s^*_k \) for \( k > 11 \)).

**Vital rate sensitivity analysis**

To determine how best to rapidly increase population size of an infected Indiana bat colony in a short timeframe, we used a vital rate sensitivity analysis (VRSA). This approach is more advantageous than traditional sensitivity analyses (e.g., Morris and Doak 2002) because the stable age distribution assumption can be relaxed (Fefferman and Reed 2006), which is beneficial when populations are undergoing rapid declines (e.g., Fefferman and Reed 2006, Reed et al. 2009). Further, vital rate sensitivity analyses focus on maximizing population size over a short timeframe, which can be critical for conservation action (Field et al. 2007). To be conservative, we populated the VRSA matrix with the lowest survival estimate generated from our data.

Based on the status of ongoing research projects on WNS management, published literature on potential management actions (Cornelison et al. 2014, Wilcox and Willis 2016, Maslo et al. 2017), and the results of our survival analysis, we developed three management strategies for returning infected Indiana bat colonies to stable population growth (\( \lambda = 1 \)): (1) increasing adult annual survival; (2) increasing adult and juvenile annual survival; and (3) increasing adult and juvenile reproduction. For each strategy, we calculated the 10-yr cumulative growth rate (\( \lambda_{10} \)) and final population size of the colony resulting from a given percentage increase in the relevant vital rate(s). We projected the infected population vector through the matrix and began vital rate perturbations in year 2 of WNS infection. To account for uncertainty, we ran 10,000 iterations of the VRSA projections for each population structure in a Monte Carlo simulation, incorporating stochasticity (as above) by drawing adult and juvenile survival from beta distributions where the standard deviation was set to 0.1.
RESULTS

Survival of Indiana bats
From 2011 to 2016, we captured 670 Indiana bats (617 males, 53 females); of these, 328 individuals were recaptured in subsequent years. The goodness-of-fit test returned a variance inflation factor of \( \hat{\sigma} = 1.26 \) for our global model, indicating modest overdispersion in our data; thus, we adjusted our small sample corrected quasi-Akaike’s Information Criterion (QAIC\(_c\)) by this value (Burnham and Anderson 2002). Model results showed strong support for a linear decreasing trend in survival since arrival of WNS (Table 1). Of the 19 candidate models tested, 68% of the Akaike weights were captured in the top two models, which returned a \( \Delta\text{QAIC}_c < 2 \). Model-averaged survival decreased from 0.78 (95% CI: 0.59, 0.89) and 0.79 (95% CI: 0.70, 0.86) for females and males, respectively, in 2011 to 0.74 (95% CI: 0.33, 0.94) and 0.75 (95% CI: 0.53, 0.89) for females and males, respectively, in 2015 (Fig. 1). The analysis suggested no significant differences in survival between the sexes, which both decreased at a rate of ~1% per year (Fig. 1). Recapture rates did vary significantly between the sexes, ranging from 0.04 (95% CI: 0.00, 0.30) to 0.06 (95% CI: 0.01, 0.39) for females and 0.22 (95% CI: 0.16, 0.28) and 0.51 (95% CI: 0.43, 0.59) for males, likely because banding operations occurred during the fall swarm (Fenton 1969, Cope 1976).

Table 1. Top eight a priori candidate models used for estimating survival and recapture probabilities for Indiana bats (\textit{Myotis sodalis}; \( N = 670 \)) at Mt. Hope Mine, Rockaway, New Jersey, USA.

<table>
<thead>
<tr>
<th>Model</th>
<th>QAIC(_c)</th>
<th>( \Delta\text{QAIC}_c )</th>
<th>( w )</th>
</tr>
</thead>
<tbody>
<tr>
<td>( S(\text{time}), p(\text{year, sex}) )</td>
<td>1797.06</td>
<td>0.00</td>
<td>0.36</td>
</tr>
<tr>
<td>( S(\text{time, sex}), p(\text{year, sex}) )</td>
<td>1797.28</td>
<td>0.21</td>
<td>0.32</td>
</tr>
<tr>
<td>( S(\text{constant, sex}), p(\text{year}) )</td>
<td>1799.31</td>
<td>2.25</td>
<td>0.12</td>
</tr>
<tr>
<td>( S(\text{constant}), p(\text{year, sex}) )</td>
<td>1799.35</td>
<td>2.29</td>
<td>0.11</td>
</tr>
<tr>
<td>( S(\text{constant, sex}), p(\text{year, sex}) )</td>
<td>1801.00</td>
<td>3.94</td>
<td>0.05</td>
</tr>
<tr>
<td>( S(\text{time, sex}), p(\text{year}) )</td>
<td>1802.62</td>
<td>5.56</td>
<td>0.02</td>
</tr>
<tr>
<td>( S(\text{year}), p(\text{year, sex}) )</td>
<td>1804.56</td>
<td>7.50</td>
<td>0.01</td>
</tr>
<tr>
<td>( S(\text{year}), p(\text{year, sex}) )</td>
<td>1807.54</td>
<td>10.5</td>
<td>0.00</td>
</tr>
</tbody>
</table>

Notes: Top two models in boldface (\( \Delta\text{QAIC}_c \)) were used for model-averaged survival parameter estimation. QAIC\(_c\), quasi-Akaike’s Information Criterion corrected for small sample size; \( \Delta\text{QAIC}_c \), difference between the QAIC\(_c\) value between each model and the top model; \( w \), quasi-Akaike weight; \( S \), survival; \( p \), recapture probability; \( \text{time} \), linear time trend; \( \text{year} \), yearly variation.

Within hibernaculum spatial spread of \textit{P. destructans}
The uninfected Indiana bat compartment growth rate was positive, at \( k = 1.05 \). We calculated survival of infected individuals as 0.68 (95% CI: 0.58, 0.71). Model results indicate an infective dose of \textit{P. destructans} spreads within the population at a rate of ~12.5% annually (Fig. 2). While population size of Indiana bats initially appears to be increasing in the year of disease emergence, the proportion of infected bats gradually increases to 100% within nine years. In this model, when the proportion of infected bats become \( >36\% \), the overall growth rate of the population becomes negative and eventually stabilizes at \( k = 0.91 \) (9% annual decline) when the proportion of infected bats is 100%.

Cumulative mortality risk of iterative WNS infection
Based on the empirical data generated through the mark–recapture analysis, the exposure penalty increases from 0.987 in the first year of infection to 0.845 after 11 yr under a linear progression (Table 2), increasing in severity by ~1.4% with each WNS infection cycle. After 11 yr, the population stabilizes at a survival probability of \( s^* = 0.71 \) and a growth rate of \( \lambda = 0.993 \) (Fig. 3). Under a logarithmic progression, survival stabilizes at \( s^* = 0.73 \) resulting from an exposure penalty that gradually lessens in magnitude with each WNS infection cycle (Table 2). Population growth rate stabilizes at \( \lambda = 0.9991 \) (Fig. 3).
Overall, increasing adult and juvenile survival (Management Strategy 2) has the greatest cumulative impact on population growth of infected Indiana bat colonies (Tables 3–5). A 4% increase in these vital rates returns an infected population to stable growth ($\lambda^{10} = 1$) in 10 yr. Outcomes of increasing adult survival (Management Strategy 1) are only marginally lower, with population growth reaching $\lambda^{10} = 1$ after a 5% increase in annual survival. In contrast, both adult and juvenile reproduction must be increased (Management Strategy 3) by 28% to achieve stability.

**DISCUSSION**

Our work provides the first empirically derived survival estimates for Indiana bats after the arrival of WNS. Estimates are consistent with observations that the Indiana bat population was experiencing average annual growth of 1.4% in the two decades prior to disease emergence (Thogmartin et al. 2012). While some hibernating colonies have decreased by $>80\%$ after WNS (Thogmartin et al. 2012), there have been limited observations of massive mortality events. Indeed, population declines for Indiana bats have been markedly less severe than for other susceptible species (Frick et al. 2015). Our results support these observations and show that annual survival remains high even when *Pseudogymnoascus destructans* has been documented at a site.

Under a phased exposure scenario, Indiana bats may be escaping significant population-level impacts through a slowed transmission rate, as model results indicate it takes nine years for the entire population to become infected. Prevalence of *P. destructans* on substrates small distances

Fig. 2. Predicted population dynamics for Indiana bats (*Myotis sodalis*) experiencing a phased exposure to an infective dose of *Pseudogymnoascus destructans* resulting from increasing spatial spread of the pathogen within the hibernaculum. Solid blue and red lines indicate the change in the number of uninfected and infected bats, respectively, over the years for which empirical data on survival exist (2011–2015). Dotted lines illustrate population projections along with 95% confidence intervals.

**Vital rate sensitivity analysis**

Overall, increasing adult and juvenile survival (Management Strategy 2) has the greatest cumulative impact on population growth of infected Indiana bats (*Myotis sodalis*) experiencing a phased exposure to an infective dose of *Pseudogymnoascus destructans* resulting from increasing spatial spread of the pathogen within the hibernaculum. Solid blue and red lines indicate the change in the number of uninfected and infected bats, respectively, over the years for which empirical data on survival exist (2011–2015). Dotted lines illustrate population projections along with 95% confidence intervals.

**Table 2.** Predicted survival, $s_i$, of Indiana bats (*Myotis sodalis*) and penalty, $e_i$, incurred with each year of infection under a cumulative risk of iterative exposure to *Pseudogymnoascus destructans* hypothesis.

<table>
<thead>
<tr>
<th>Survival</th>
<th>Linear</th>
<th>Logarithmic</th>
<th>Exposure penalty</th>
<th>Linear</th>
<th>Logarithmic</th>
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<tr>
<td>$s_0$</td>
<td>0.78</td>
<td>0.78</td>
<td>$c_1$</td>
<td>0.987</td>
<td>0.987</td>
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<tr>
<td>$s_1$</td>
<td>0.77</td>
<td>0.77</td>
<td>$c_2$</td>
<td>0.974</td>
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<tr>
<td>$s_2$</td>
<td>0.76</td>
<td>0.76</td>
<td>$c_3$</td>
<td>0.960</td>
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<tr>
<td>$s_3$</td>
<td>0.75</td>
<td>0.75</td>
<td>$c_4$</td>
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<tr>
<td>$s_4$</td>
<td>0.74</td>
<td>0.74</td>
<td>$c_5$</td>
<td>0.931</td>
<td>0.942</td>
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<tr>
<td>$s_5$</td>
<td>0.73</td>
<td>0.74</td>
<td>$c_6$</td>
<td>0.916</td>
<td>0.936</td>
</tr>
<tr>
<td>$s_6$</td>
<td>0.72</td>
<td>0.73</td>
<td>$c_7$</td>
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<td>0.932</td>
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<tr>
<td>$s_7$</td>
<td>0.72</td>
<td>0.73</td>
<td>$c_8$</td>
<td>0.888</td>
<td>0.928</td>
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<tr>
<td>$s_8$</td>
<td>0.71</td>
<td>0.73</td>
<td>$c_9$</td>
<td>0.873</td>
<td>0.924</td>
</tr>
<tr>
<td>$s_9$</td>
<td>0.71</td>
<td>0.73</td>
<td>$c_{10}$</td>
<td>0.859</td>
<td>0.921</td>
</tr>
<tr>
<td>$s_{10}$</td>
<td>0.71</td>
<td>0.73</td>
<td>$c_{11}$</td>
<td>0.845</td>
<td>0.918</td>
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</table>

*Note:* Estimates are reported for both linear and logarithmic decay functions.
from roosting bats is less than under roosting bats (Langwig et al. 2015), suggesting that at least during the early years of disease presence transmission is primarily bat-to-bat (e.g., Indiana bats occupying a hibernaculum with infected little brown bats may not be immediately exposed unless roosting in the same cluster). While similar in timescale, the possibility of increasing cumulative impacts from ongoing exposure yields a different trajectory for population decline. Under this scenario, it is not until the duration of pathogen presence in the population approaches the life expectancy of the hosts that the full negative impact of disease exposure will be observable in the age-specific vital rates of all ages and, therefore, for the entire population.

In either case, the generally high survival estimates derived from the mark–recapture data indicate that Indiana bats have a higher tolerance of *P. destructans* relative to other affected species. Our results are consistent with other studies examining WNS impacts, which found Indiana bats are less impacted than those from other species. Further research is needed to understand the factors influencing this variation and to develop effective mitigation strategies.
bats sustain *P. destructans* loads similar to less affected species both in its invasive range as well as its endemic range where WNS causes little mortality (Puechmaille et al. 2011, Hoyt et al. 2016, Frick et al. 2017). Reduced mortality may be driven by physiological mechanisms (i.e., interactions with the cutaneous microbiome, increased fat reserves), epidemiology (delayed or decreased transmission rates leading to lower infection intensity), or suboptimal habitat suitability for *P. destructans* where Indiana bats roost. Given that some hibernating colonies have declined dramatically (Thogmartin et al. 2012), it is not likely that the species possesses an inherent genetic resistance.

Despite high survival rates, however, we found strong evidence for a declining trend in this vital rate over time since disease emergence, and both population models stabilize at negative growth. Therefore, the apparent tolerance of *P. destructans* by Indiana bats may not be indicative of reduced long-term extinction risk. Subtle cumulative costs, aggregating over time, may insidiously compromise population persistence in ways that take a decade or more to reach their full impact (due to baseline host life expectancy). In fact, the threat to Indiana bat population viability may be as severe as that of bat species that have experienced high population-level impacts over the 10 yr that WNS has been present in North America (e.g., little brown bats). In areas where *P. destructans* first established in North America, some populations of little brown bats and the highly impacted tricolored bat (*Perimyotis subflavus*) are persisting (Frick et al. 2017). Studies have shown that little brown bat survival rebounds and exhibits gradual improvement each year following the initial mass mortality event, suggesting that resistance to WNS infection may exist in some populations (Maslo et al. 2015). For this species, WNS may have imposed a strong selective pressure such that a surviving phenotype is becomingly increasingly represented in these populations and returning them to positive growth (Maslo and Fefferman 2015). The selective forces acting on Indiana bats appear to be considerably weaker, as evidenced by the gradual population decline and the lack of significant mortality in most sites. Therefore, evolutionary processes are unlikely to rescue populations from extirpation even if resistant genotypes are present.

Less pronounced population-level impacts, however, likely render proposed conservation actions more feasible. As shown with little brown bats (Maslo et al. 2015, 2017), actively improving reproduction has little impact on population recovery relative to increasing survival; therefore, captive breeding programs are not likely cost-effective. Slight increases in adult and juvenile Indiana bat reproduction (Management Strategy 3), as predicted by vital rate sensitivity analysis (VRSA).

<table>
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<tr>
<th>Increase (%)</th>
<th>$B_a$</th>
<th>$B_j$</th>
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<th>$k_{10}$‡</th>
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† $\Delta N$ values represent the change in population size from (year 0) to (year 10) of the VRSA.
‡ $k_{10}$ represents the cumulative annual growth rate derived from the VRSA.
juvenile survival could help Indiana bat populations to persist over the short term. Currently, several alternatives for chemical and biological control of P. destructans have shown promising inhibitory properties in the laboratory ( Cornelison et al. 2014, Hoyt et al. 2015, Raudabaugh and Miller 2015, Padhi et al. 2016); however, these strategies face considerable challenges before the treatment of infected populations can be successful. Treatment efficacy in the wild is not yet known, and economic and logistical constraints significantly reduce the percent of the infected population that feasibly can be treated. Habitat modification approaches (i.e., heated roost boxes; Wilcox and Willis 2016) may positively impact both vital rates by directly improving survival (through reduced energy costs) and indirectly improving reproduction (births earlier in the summer). For species like Indiana bats declining at slower timescales, targeted intervention even with modest results may return infected populations to stationary dynamics.

The opposite survival patterns exhibited by Indiana bats and the closely related little brown bats (Maslo et al. 2015) reinforce that variation exists in the impacts emerging pathogens have on host species. Slight differences in physiology or behavior may result in markedly different responses, leading to noticeable variation in demography early in the disease chronology. Modest declines may lead to a false sense of assurance that a population can persist. In the case of multi-host pathogens, a one-size-fits-all disease strategy is likely ineffective in reaching conservation goals. Demographic modeling approaches coupled with continued population monitoring can highlight important differences in disease response, and ultimately extinction risk, in host species, allowing conservation practitioners to tailor intervention actions so that they will be most effective.

ACKNOWLEDGMENTS

We thank multiple field technicians who assisted in the collection of mark–recapture data at Mt. Hope Mine, as well as J. Chenger, B. Hines, and C. Herzog for fruitful discussions on the observed dynamics of Indiana bat populations across the WNS-infected range. The United States Fish and Wildlife Service White-nose Syndrome Research Small Grants Program, administered through the Wildlife Management Institute, provided funding for this work (Award number 69-2B29-12-176).

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Supporting information

Additional Supporting Information may be found online at: http://onlinelibrary.wiley.com/doi/10.1002/ecs2.2001/full