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Drivers of population dynamics of at-risk populations change with pathogen arrival

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ABSTRACT

Successful wildlife conservation in an era of global change requires understanding determinants of species population growth. However, when populations are faced with novel stressors, factors associated with healthy populations can change, necessitating shifting conservation strategies. For example, emerging infectious diseases can cause conditions previously beneficial to host populations to increase disease impacts. Here, we paired a population dataset of 265 colonies of the federally endangered Indiana bat (*Myotis sodalis*) with 50.7 logger-years of environmental data to explore factors that affected colony response to white-nose syndrome (WNS), an emerging fungal disease. We found variation in colony responses to WNS, ranging from extirpation to stabilization. The severity of WNS impacts was associated with hibernaculum temperature, as colonies of cold hibernacula declined more severely than those in relatively warm hibernacula, an association that arose following pathogen emergence. Interestingly, this association was opposite that of a sympatric bat species, the little brown bat (*Myotis lucifugus*), illustrating that environmental dependence of disease can vary by species in a multi-host community. Simulating future colony dynamics suggests that most extirpations have already occurred, as the pathogen has been present for several years in most colonies, and that relatively small colonies are more susceptible to extirpation. Overall, this study illustrates that emerging infectious diseases can change the factors associated with host population growth, including through novel environmental associations that vary by host species. Consideration of these shifting associations and differences between impacted species will be essential to the conservation of host communities challenged by emerging infectious disease.

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1. Introduction

Conserving global biodiversity in the Anthropocene requires a deep understanding of the factors that contribute to the resilience and growth of wildlife populations. The suite of biotic and abiotic conditions that can contribute to population growth, from ambient conditions like temperature (Woodworth et al., 2017), humidity (Lu and Wu, 2011), or precipitation (Maso et al., 2020´), to community-level factors like predator density (Salo et al., 2010), competitor density (Gamelon et al., 2019), or pathogen prevalence (Hoyt et al., 2020), can further serve as targets for conservation and management. However, novel stressors such as climate change (Acevedo et al., 2020; S¸ekercioglu et al., 2012ˆ ; Van Dyck et al., 2015), invasive species introduction (Hagman et al., 2009), and habitat fragmentation and loss (Haddad et al., 2015; Mantyka-Pringle et al., 2015) can alter associations between biotic and abiotic conditions and population growth or stability. The form and magnitude of these altered associations is often ambiguous or difficult to characterize and can create the potential for a mismatch between conservation interventions and intended outcomes, possibly with negative effects on the targeted population (Sutherland et al., 2004). Therefore, understanding how novel stressors change the way the resilience and growth of wildlife populations respond to biotic and abiotic conditions will be essential to their successful conservation in a rapidly changing world.

Emerging infectious diseases are one of many stressors that threaten wildlife populations and can result in host species declines or population extirpations (Altizer et al., 2013; Anderson et al., 2004; Daszak et al., 2001, 2000; Fisher et al., 2012; Rogalski et al., 2017), as evidenced by West Nile Virus and avian malaria in birds (Kilpatrick, 2011; Van Riper III et al., 1986), chytridiomycosis in amphibians (Skerratt et al., 2007), snake fungal disease (Lorch et al., 2016), sarcoptic mange in wombats (Martin et al., 2018), facial tumor disease in Tasmanian devils (Mccallum, 2008), and white-nose syndrome in bats (Hoyt et al., 2021). Early conservation measures to bolster the resilience and growth of host populations might dampen the impact of disease following its arrival (Grant et al., 2017; Hodgson et al., 2015). However, it is often unclear whether those same conservation measures will similarly promote the growth of host populations during pathogen invasion when pathogen transmission dynamics (Huang et al., 2021; Leach et al., 2016) or environmental dependence of disease (Langwig et al., 2012; McNew et al., 2019; Samuel et al., 2015) can significantly shift the fitness landscape of the host. This information is particularly important for rare and endangered host species with limited habitat availability or population sizes before pathogen invasion, as their sensitivity to novel stressors and management interventions necessitates unequivocal conservation best practices. Therefore, in this study, we explore the role of two potential correlates of host population growth, population size and ambient environmental conditions, in driving host population growth of an endangered species before, during, and following the epidemic of a novel and emerging infectious disease. We hypothesized that associations between these biotic and abiotic factors and host population growth would change over epidemic progression, illustrating the need to adapt conservation strategies to changing conditions.

The size of a host population can contribute to its response to pathogen invasion and ultimately its risk of extirpation. Greater standing genetic diversity in large host populations may promote evolutionary adaptation to an invading pathogen before total population collapse occurs (Barrett and Schluter, 2008; Bell, 2013; Carlson et al., 2014; Gomulkiewicz and Holt, 1995; Maslo and Fefferman, 2015). Large populations also have less risk of stochastic fadeout, which can be exacerbated in populations declining from disease (de Castro and Bolker, 2005; Frick et al., 2015; Langwig et al., 2012). However, if large populations also have high host densities, pathogen transmission may be elevated, leading to more rapid declines and greater extinction risk (Brunner et al., 2017; Cross et al., 2010; Cunningham et al., 2021; Rachowicz and Briggs, 2007; Smith et al., 2009; Storm et al., 2013).

While there are clear theoretical mechanisms of how host population size can contribute to its response to an emerging infectious disease, this association is often left uncharacterized due to the difficulty of obtaining sufficient data, despite being essential to mitigating impacts to host populations.

Environmental dependence of pathogen transmission or disease severity can create both hotspots (Lambin et al., 2010; Ostfeld et al., 2005; Paull et al., 2012) or refugia from (Heard et al., 2015; Mosher et al., 2018; Puschendorf et al., 2011; Scheele et al., 2017; Spitzen-Van Der Sluijs et al., 2017) infection and disease. Temperature, for example, can have complex effects on disease by influencing pathogen growth rates (Hopkins et al., 2021), pathogen transmission (Mordecai et al., 2019), host susceptibility to infection (Cohen et al., 2017), or disease severity following infection (Debes et al., 2017), processes that scale up to variation in population-level impacts on the host. These environmental effects can further exacerbate impacts to host populations if high-quality habitat prior to pathogen invasion becomes population sinks or ecological traps of high pathogen transmission and disease severity following disease emergence (Hopkins et al., 2021; Leach et al., 2016). Understanding the contribution of environmental conditions to host population resilience and growth and how this relationship changes following disease emergence will be essential to mitigation efforts, as context-appropriate protection and enhancement of critical environmental conditions is fundamental to the conservation of threatened host species.

White-nose syndrome (WNS) is an emerging infectious disease of hibernating bats caused by the fungal pathogen *Pseudogymnoascus destructans* (Lorch et al., 2011; Minnis and Lindner, 2013; Warnecke et al., 2012). The pathogen was introduced to North America in the early 2000s from its origin in Eurasia and has since spread widely through naïve hibernating bat populations (Blehert et al., 2009; Drees et al., 2017; Leopardi et al., 2015), causing severe population declines (Frick et al., 2015; Hoyt et al., 2021; Langwig et al., 2016, 2012). *Pseudogymnoascus destructans* establishes an environmental pathogen reservoir within winter hibernacula (Hoyt et al., 2020, 2015; Laggan et al., 2023), the caves and mines where bats hibernate. This results in seasonal infection dynamics in which WNS prevalence increases as bats arrive to hibernacula in late fall but declines during spring and summer months when bats do not primarily use hibernacula for roosting and bats are often euthermic (Langwig et al., 2015a). Infection severity increases over the winter hibernation period when body temperatures drop to approximately ambient conditions (Langwig et al., 2015a, 2017), allowing the cold-adapted fungus to invade the epidermal tissue (Lorch et al., 2011; Warnecke et al., 2013). Infection with *P. destructans* is accompanied by a suite of physiological changes that lead to dehydration (Cryan et al., 2013; Mcguire et al., 2017; Verant et al., 2014), frequent arousals from torpor (Lilley et al., 2016; Reeder et al., 2012; Warnecke et al., 2012), emaciation, and ultimately mortality (Cryan et al., 2010; Warnecke et al., 2013).

The Indiana bat (*Myotis sodalis*) is a North American species that has experienced severe but variable population declines due to WNS (Langwig et al., 2012; Thogmartin et al., 2012). Despite its status as endangered under the U.S. Endangered Species Act, little is known about drivers of variation in Indiana bat declines and persistence with disease, information important for its conservation. Environmental correlates of disease severity and population response have been previously reported for other species, particularly the little brown bat (*Myotis lucifugus*), where warmer hibernacula are associated with severe disease and declines (Grieneisen et al., 2015; Grimaudo et al., 2022; Hopkins et al., 2021; Langwig et al., 2016, 2012; Lilley et al., 2018), likely due to enhanced pathogen growth under those conditions (Marroquin et al., 2017; Verant et al., 2012). However, previous work has not found a clear association between temperature conditions of hibernacula and the response of Indiana bat colonies (Langwig et al., 2012). Evidence additionally suggests that for many species impacted by WNS, colony size may be related to the initial population response to the disease (Frick et al., 2015; Langwig et al., 2012). However, the particularly gregarious nature and large cluster sizes of Indiana bats (Clawson et al., 1980; Hardin and Hassell, 1970; Thomson, 1982) may result in constant density across colony sizes and time, keeping pathogen transmission and colony declines high regardless of colony size.

In this study, we pair an extensive bat population dataset curated by the U.S. Fish and Wildlife Service with a hibernacula environmental dataset to identify potential contributions of host population size and environmental conditions to Indiana bat population trends prior, during, and after pathogen arrival. Our data provide information on how population-level processes and variation in environmental conditions can generate heterogeneous host population responses to novel infectious diseases, lending insights into how to successfully manage their impacts.

2. Material and methods

2.1. Data collection

2.1.1. Population data

We analyzed data on population sizes of 265 colonies of hibernating Indiana bats from 15 U.S. states collected between 2003—2022 (Supplemental Table 1). Population data were collected by various federal and state agencies and were compiled by the U.S. Fish and Wildlife Service. Surveillance for *P. destructans* infection was conducted during regular visits to hibernacula, which occur on a bi-annual basis following federal recommendations due to the endangered status of the Indiana bat. Detection of *P. destructans* infection in colonies of Indiana bats was typically visual, as WNS is associated with visible external fungal growth, and later confirmed with lab-based protocols to detect the presence of *P. destructans* on swab samples via PCR. For each colony, the year of detection of *P. destructans* infection within their hibernaculum was recorded and used to assign 'epidemic year,' which is the year since detection (e.g., epidemic year 0 corresponds to the year of *P. destructans* detection). Due to the bi-annual schedule of WNS surveillance surveys of Indiana bat colonies, it is possible *P. destructans* arrived between survey years, which could have resulted in the classification of epidemic year 1 as epidemic year 0 in some cases. We estimated the annual population growth rates, λ_i, for each Indiana bat colony for each winter with an available colony census. λ_i was derived by dividing the census value taken in a winter (N_i) to the next most recent winter census value (N_{i-T}), separated by T years, with a constant of 1 added to all N_i and N_{i-T} values to avoid division by 0 (Eq. (1)):

$$\lambda_i = \sqrt[T]{\frac{N_i}{N_{i-T}}} \quad (1)$$

We classified census data by epidemic phase, with the 'Pre-Invasion' phase as epidemic years less than or equal to 0, 'Epidemic' being epidemic years 1 through 5, and 'Established' being epidemic years 6 or greater. To avoid making comparisons across wide swaths of time and ecological contexts, no values of N_i collected earlier than epidemic year − 5 were used when calculating λ_i or in subsequent analyses. Including epidemic year 0 in the "Pre-invasion" phase reflects the lack of population declines in the year of WNS arrival to a colony, driven by low infection prevalence and severity in the population (Frick et al., 2017; Langwig et al., 2015b), with declines typically beginning in epidemic year 1 (Hoyt et al., 2020; Laggan et al., 2023; Langwig et al., 2015a, 2015b). To identify the transition from the 'Epidemic' to 'Established' phase, we used a segmented regression with annual colony growth rates, λ_i, as the response and epidemic year as the explanatory variable using R package *segmented* (Muggeo, 2017). The segmented regression indicated breakpoints in annual colony growth rates at epidemic year 3.29 (+/− 1.26 SE) and 6.00 (+/− 1.18 SE). The breakpoint at 3.29 reflected the apex of the U-shaped transition between increasingly more negative average population growth rates and a trend towards more positive population growth rates. The breakpoint at 6 corresponded to the point at which colony declines begin to ameliorate and then reach population stability, respectively. We therefore defined the 'Established' phase as epidemic years greater than or equal to epidemic year 6, reflecting the point at which extant colonies have generally stabilized. We identified a single phase for the Epidemic period as mean population growth rates remained below stability during this entire period. Mean population growth rates, λ_m, for each colony in each epidemic phase were calculated by averaging all available annual lambda values for a colony within each epidemic phase.

We assigned a classification to each colony based on their overall response to WNS, referred to as 'colony status.' 'Extirpated' colonies were those with a most recent post-WNS arrival (epidemic year greater than or equal to 0) census value of 0, indicating total collapse of the colony. 'Declining' colonies were those that never exhibited a year of stable or positive annual colony growth (λ_i greater than or equal to 0) after epidemic year 1. Finally, 'persisting' colonies were those that had at least one year of stable or positive annual colony growth after epidemic year 1. These classifications were possible for 231 of the 265 colonies in the population dataset. The remaining 34 colonies did not have sufficient post-WNS detection census data to accurately assign a classification, as they did not have census data available following epidemic year 0.

Indiana bats are directly surveyed by photographing or counting clusters of bats during single biannual trips to avoid excess disturbance to the colony. Previous work has found that deviation between surveyors counting the same Indiana bat colonies is typically <1.5 % (Meretstky et al., 2010), so we used this value to determine how potential observer error in count values could affect our results by randomly drawing from a uniform distribution centered on the original census value with a range of 1.5 % of the original value, rounded to the nearest integer value. We then used the new, randomly drawn dataset to re-calculate annual (λ_i) and mean (λ_m) population growth rates. We used the new dataset from randomly drawn census values to re-run all statistical models described below, testing for potential effects of observer error on model results and conclusions.

2.1.2. Environmental data

Data on the temperature and humidity conditions of Indiana bat roosting locations within hibernacula were collected overwinter in a total of 48 hibernacula between 1994 and 2022, 53.9 % of which was collected since 2015. While environmental data were collected from Indiana bat roosting locations and largely representative of the conditions experienced by hibernating bats, hibernacula can be environmentally variable and additional microclimates may have been available to hibernating bats. In total, the environmental dataset we used in this study constitutes 50.7 logger-years of data on the environmental conditions within hibernacula (summarized in Supplemental Table 2). Environmental data were recorded by a variety of data loggers, including Onset HOBO H8 Pro, Onset HOBO Pro V2, Onset HOBO Pendant, Onset HOBO XT, Madgetech TransiTempII-RH, Maxim Integrade iButton, and modified onset HOBO MX2303 data loggers (Supplemental Table 2). HOBO MX2303 units were modified into wet bulb psychrometers to estimate both temperature and humidity conditions within hibernacula (Supplemental Fig. 1). Humidity was measured as vapor pressure deficit (VPD; kilopascals), which measures the difference between the amount of moisture in the air and how much moisture the air could hold at the same temperature when saturated. Higher VPD values correspond to drier conditions, and is directly calculable from the relative humidity (*RH*) and temperature (*Temp*) data recorded by environmental data loggers using the below equation (Eq. (2)):

$$VPD = \left(0.6108 \times e^{\left(\frac{Temp}{Temp+237.3}\right)} \times 17.2694\right) \times \left(1 - \frac{RH}{100}\right) \quad (2)$$

For each hibernaculum, we calculated the mean early hibernation (November and December) temperature and VPD across all available years of data. Mean values were calculated so that they could be directly compared to mean annual colony growth values, λ_m, as there were few cases in which annual colony growth rates, λ_i, were directly comparable to environmental data collected in the same year. We quantified the environmental conditions present early in hibernation because they are most associated with initial infection severity and ultimately survival probability (Hopkins et al., 2021; Langwig et al., 2016). Mean VPD values were skewed towards zero with a long right tail, so we $\log_{10}$-transformed the mean values before further analysis. To include untransformed VPD values of zero, a constant of 1×10^{-4} (same order of magnitude as the next-lowest non-zero VPD value) was added to all VPD values before transformation. Mean early hibernation temperature values were available for all 48 hibernacula and mean early hibernation VPD values were available for 34, capturing wide variation in temperature and humidity conditions across hibernacula (Supplemental Fig. 2). Sites with environmental monitoring tended to be larger in size as USFWS guidance on environmental monitoring generally encompassed priority I hibernation sites which tended to contain larger colonies (Supplemental Fig. 3).

2.2. Statistical analyses

2.2.1. Effect of population size on declines

To characterize variation in colony growth rates over epidemic time, we summarized the median and interquartile range of annual colony growth rate, λ_i, by epidemic year and colony status (Fig. 1, Supplemental Table 3). We

explored associations between λ_i (response variable) and $\log_{10}$ colony size ($N_{i\text{-}T}$ in Eq. (1)) interacting with epidemic phase (predictors) using a generalized linear mixed model with gamma error distribution and a log link with colony ID as a random effect (Model 1). Colony size was confounded with colony status and was therefore used in a different model exploring λ_i. We additionally assessed whether colony status was related to the size of the colony prior to the arrival of WNS by using a negative binomial model with log link function, the pre- WNS detection census value for each colony as the response variable, and colony status as the explanatory variable (Model 2). To test for potential spatial autocorrelation in rates of colony growth, we used a Moran's I test in R package *spdep* with the county centroids of hibernacula and the average annual colony growth rate of each epidemic phase as the response (Bivand et al., 2024). The weight matrix of the Moran's I test was generated using a k-nearest neighbor (k = 10) approach.

2.2.2. *Effects of environmental conditions on declines*

To explore associations between environmental conditions within hibernacula and the response of Indiana bat colonies to WNS, we used a series of linear and generalized linear models. First, to test for the effect of mean early hibernation temperature, epidemic phase, and their interaction on the average annual colony growth value of a colony in an epidemic phase, λ_m, we used a generalized linear model with a gamma error distribution and log link function (Model 3). The same model structure was used for a separate model to determine whether mean

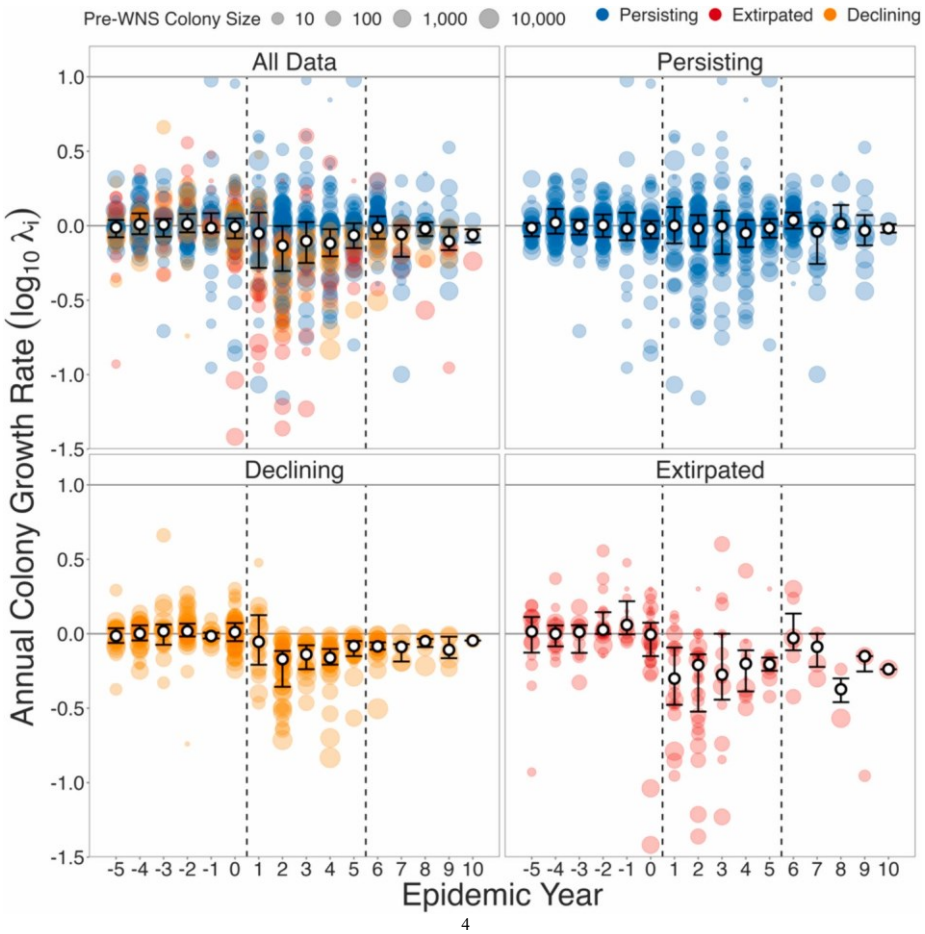


Fig. 1. Population trends of Indiana bat colonies over time since detection of WNS. Epidemic year 0 on the x-axis corresponds to the year in which WNS was detected within a hibernaculum. Vertical dashed lines separate epidemic phases (left to right: pre-invasion, epidemic, established). Annual population growth rates of 0 on the y-axis correspond to population stability, and values below or above 0 indicate declining or growing colonies, respectively. Points are colored by colony status and size corresponds to pre-WNS arrival colony size. White points and error bars correspond to median values and interquartile range of data.

early hibernation VPD, epidemic phase, and their interaction influenced colony growth, λ_m (Model 4). Because environmental conditions may have influenced colony size prior to WNS arrival, we also examined the relationship between environmental conditions of hibernacula and pre- WNS detection colony size by using two negative binomial generalized linear models with log link functions and mean early hibernation temperature (Model 5) or VPD (Model 6) as the predictor and the latest pre- WNS colony size for each colony as the response variable. Separate models were constructed with temperature and VPD as explanatory variables because they were correlated (output of generalized linear model with gamma error distribution and log link function: $\beta = 0.038 +/- 0.012$ SE, $p = 0.001$) and their inclusion in the same model would violate model assumptions of multicollinearity. Finally, to explore potential associations between temperature and VPD conditions within hibernacula with colony status, we constructed two linear models with colony status as the explanatory variable and mean early hibernation temperature (Model 7) or VPD (Model 8) as the response variables. All linear models using normal distributions satisfied assumptions of Gaussian error distributions and homoscedastic error. All analyses were performed in R packages *stats* (R Core Team 2022), *glmmTMB* (Magnusson et al., 2017), and *MASS* (Ripley et al., 2013) in R v4.2.1.

2.2.3. Effect of little brown bat abundance on Indiana bat declines

Little brown bats (*Myotis lucifugus*) are a relatively abundant species in WNS-impacted communities and may contribute to community-wide disease dynamics (Laggan et al., 2023). However, because colony declines in little brown bats are also associated with the temperature and humidity of hibernacula (Grieneisen et al., 2015; Grimaudo et al., 2022; Hopkins et al., 2021; Langwig et al., 2016, 2012; Lilley et al., 2018), it is possible that any apparent associations between environmental conditions and Indiana bat declines could arise instead from the contribution of little brown bat abundance to Indiana bat survival and colony response. To identify a potential effect of little brown bat abundance on Indiana bat colony declines, we constructed a generalized linear mixed model with gamma error distribution and log link function with λ_i of Indiana bat colonies as the response variable, the $\log_{10}$-transformed colony size of little brown bats ($N_{i\text{-}T}$ of little brown bats) interacting with epidemic phase as the explanatory variables, and colony ID as a random effect (Model 9). An association between annual Indiana bat and little brown bat census values (result of negative binomial regression: $\beta = 0.820 +/- 0.188$ SE, $p < 0.001$) suggested that hibernacula that contained large colonies of one species typically contained large colonies of the other, thus necessitating their inclusion as explanatory variables in separate models (Models 1 and 9) to satisfy model assumptions of collinearity. Census data from sympatric Indiana and little brown bat colonies were available for 45 hibernacula and used in Model 9. Being sympatric with the Indiana bat colonies, little brown bat colonies experienced the same timeframe of WNS arrival and epidemic progression. Little brown bat census data were collected by various state and federal agencies following systematic counting protocols, performed by trained observers. All statistical models reported in this study are described in Table 1.

2.2.4. Colony extirpation simulation

We performed a simulation to estimate the proportion of the 231 colonies with known population status that might be extirpated by year 2030. We first classified each colony as "small" (≤ 68 individuals) or "large" (>68 individuals) based on its most recent colony census value prior to the detection of WNS within its hibernaculum, using the median colony size of 68 as the cut-off. If a pre-WNS census value was not available due to survey gaps, then we used the earliest census value post- WNS detection instead to classify the size of the colony. We constructed cumulative extirpation curves for each of these two datasets using colonies that were WNS-positive for at least seven years because the probability of becoming extirpated appeared to plateau following epidemic year 6. We fit logistic models to the two cumulative extirpation curves (Supplemental Fig. 4, Supplemental Table 4) using the *stats* package in R (R Core Team 2022) and estimated the probability a colony was extirpated in epidemic year *T* if extant in epidemic year *T-1* as the difference between the epidemic year model estimates for both small and large colonies. We then simulated the annual probability of extirpation by 2030 (binomial variable, 1 = extirpated 0 = extant) using the most recent known status of each colony as well as the epidemic year of its most recent census. To simulate colony extirpation, each small or large colony that was extant in year *T-1* was assigned a new extirpation status in year *T* by drawing from a binomial probability distribution with the probability of extirpation corresponding to that calculated for each epidemic year using the cumulative extirpation curves for small and large colonies. The simulation was run for 1000 iterations and summarized. To account for potential uncertainty in the logistic models of cumulative extirpation probability, we re-ran the simulations using the original logistic model parameter estimates +/− one standard error and summarized the output. The re-parameterized logistic models (Supplemental Fig. 4) accounted for uncertainty in observed rates of colony extirpations, as unrecorded extirpations could result in underestimated rates of cumulative extirpation probability.

Table 1
Names and descriptions of models reported in this study.

Model name	Response variable	Explanatory variable(s)	Model type
Model 1	Annual colony growth (λ_i)	$\log_{10}$(colony size, $N_{i\text{-}T}$) + epidemic phase + $\log_{10}$(colony size, $N_{i\text{-}T}$) * epidemic phase + (1|site)	Generalized linear mixed model with gamma error distribution and log link function
Model 2	Pre-WNS colony size	Colony status	Negative binomial generalized linear model
Model 3	Average annual colony growth (λ_m)	Mean early hibernation temperature + epidemic phase + mean early hibernation temperature * epidemic phase	Generalized linear model with gamma error distribution and log link function
Model 4	Average annual colony growth (λ_m)	Mean early hibernation VPD + epidemic phase + mean early hibernation VPD * epidemic phase	Generalized linear model with gamma error distribution and log link function
Model 5	Pre-WNS colony size	Mean early hibernation temperature	Negative binomial generalized linear model
Model 6	Pre-WNS colony size	Mean early hibernation VPD	Negative binomial generalized linear model
Model 7	Mean early hibernation temperature	Colony status	Linear model
Model 8	Mean early hibernation VPD	Colony status	Linear model
Model 9	Annual colony growth (λ_i)	$\log_{10}$(colony size of MYLU, $N_{i\text{-}T}$) + epidemic phase + $\log_{10}$(colony size of MYLU, $N_{i\text{-}T}$) * epidemic phase + (1|site)	Generalized linear mixed model with gamma error distribution and log link function

3. Results

3.1. General population trends

Population trends of Indiana bat colonies varied over time since pathogen arrival to their hibernacula (Fig. 1, Supplemental Figs. 5, 6). Prior to the detection of WNS, the median annual colony growth rate (λ_i) across all colonies in the data set was 1.000 (IQR range: 0.866–1.168), indicating overall population stability. Following WNS detection within hibernacula, however, colonies declined, with the largest declines in epidemic year two (epidemic year 2 median λ_i (IQR): 0.734 (0.496—0.994)). Of the 231 colonies in our dataset, 56 (24.24 %) were extirpated following the detection of WNS within their hibernacula and 53.57 % of these extirpations occurred between epidemic years zero and two (Supplemental Fig. 7). Additionally, 72 of the 231 colonies (31.16 %) continued to decline following WNS arrival and never achieved an annual lambda, λ_i, greater than or equal to 1.0 (stability) following WNS detection. The remaining 103 colonies (44.58 %) were stable or growing for at least one year following the second year of the epidemic, and 47.57 % of

these had their first stable year by epidemic year three (Supplemental Fig. 7). Population trends during the epidemic phase varied between extirpated, declining, and persisting colonies, with lower annual declines during the epidemic phase in persisting colonies (median λ_i (IQR): 0.948 (0.748—1.123)) compared to declining (median λ_i (IQR): 0.780 (0.633—0.944)) or extirpated (median λ_i (IQR): 0.707 (0.420—1.000)). Additionally, we found that extirpated colonies were, on average, significantly smaller than persisting ($\beta = -3.168 \pm 0.358$ SE, $p < 0.001$) or declining ($\beta = -2.517 \pm 0.389$ SE, $p < 0.001$) colonies (Model 2; Supplemental Fig. 8, Supplemental Table 5). Pre-WNS arrival colony size in declining colonies was significantly lower than those in persisting colonies, but this effect was small ($\beta = -0.651 \pm 0.326$ SE, $p = 0.046$). We detected a significant negative association between Indiana bat census values and annual colony growth during all three epidemic phases (Model 1; Supplemental Fig. 9, Supplemental Table 6), though this association appears to be driven by inflated values of λ_i in small colonies (Supplemental Fig. 10). After removing colonies of <10 bats in size from the dataset, we were unable to detect the negative association between Indiana bat colony size and annual colony growth (all epidemic phase p-values >0.05). We did not detect any statistically significant association between little brown bat colony size and annual Indiana bat colony growth, λ_i, regardless of epidemic phase (Model 9; Supplemental Fig. 11, Supplemental Table 7). All statistical model outputs were qualitatively unchanged (re-sampled model outputs available in Supplemental Table 10) when analyzed using the dataset produced by re-drawing census values from a uniform distribution to simulate observer error, indicating results are robust to sampling error in the population dataset. The Moran's I test of spatial autocorrelation did not detect an effect of spatial proximity on rates of colony growth, regardless of epidemic phase ($p > 0.406$ in all cases).

3.2. Environmental associations

Associations between early winter temperature conditions within hibernacula and population growth rates varied with epidemic phase (Model 3; Fig. 2, Supplemental Table 8). We did not detect a statistically clear association between average early hibernation temperature and annual population growth of colonies prior to WNS arrival within hibernacula (temperature coefficient during Pre-invasion phase $\beta = -0.021 \pm 0.022$ SE, $p = 0.350$). However, following the detection of WNS (Epidemic phase), we found that colonies roosting at colder early hibernation temperatures experienced more severe declines (temperature coefficient during Epidemic phase: $\beta = 0.054 \pm 0.022$ SE, $p = 0.016$), a significantly different association than that observed in the Pre-invasion phase (interaction coefficient: $\beta = -0.075 \pm 0.031$ SE, $p = 0.018$). Correspondingly, the estimated mean early hibernation temperature of extirpated hibernacula was 5.282 °C (± 0.437 SE, $p \leq 0.001$), significantly colder than both persisting and declining colonies with estimates of 8.021 °C (± 0.189 SE, $p < 0.001$) and 7.975 °C (± 0.275 SE, $p \leq 0.001$), respectively (Model 7; Fig. 3A). Early winter temperature conditions of hibernacula used by persisting colonies did not significantly differ from those used by declining colonies ($\beta = -0.046 \pm 0.334$ SE, $p = 0.890$). Following the establishment of *P. destructans* within hibernacula, the positive association between average early hibernation temperature and population growth rates weakened towards the neutral association observed prior to the WNS epidemic (temperature coefficient during Established phase: $\beta = 0.016 \pm 0.024$ SE, $p = 0.514$), such that the association was not statistically different than pre-invasion ($\beta = -0.036 \pm 0.033$ SE, $p = 0.267$) or epidemic phases ($\beta = 0.039 \pm 0.033$ SE, $p = 0.241$). We found no statistically significant association between early hibernation temperature and pre-WNS Indiana bat colony sizes (Model 5; $\beta = -0.082 \pm 0.054$ SE, $p = 0.126$; Supplemental Fig. 12A).

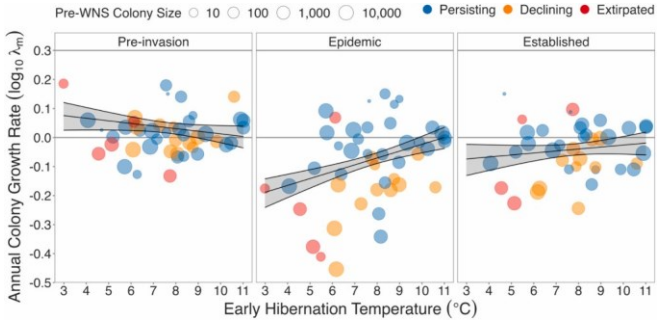


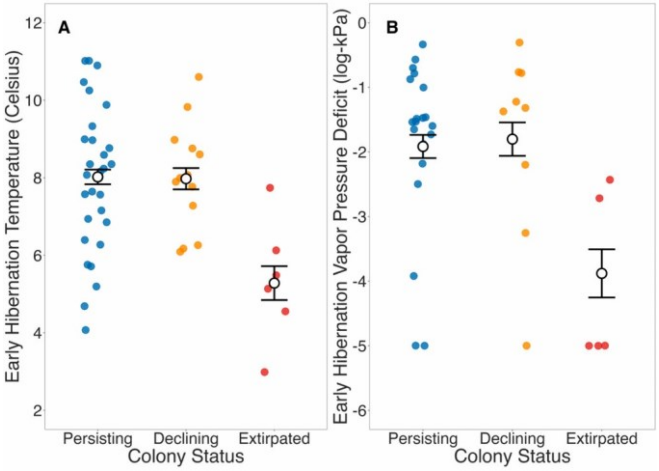
Fig. 2. Annual colony growth rate over mean early hibernation temperature. Data are broken into separate panels by epidemic phase. A colony growth rate value of 0 corresponds to population stability, with values below or above 0 corresponding to colony decline or growth, respectively. Points are colored by colony status and point size corresponds to the most recent pre-WNS colony size. Solid black lines and shaded regions correspond to model estimates +/− one standard error of the model predictions.

Fig. 3. Effect of temperature and VPD on colony status. A) Mean early hibernation temperature and B) $\log_{10}$ vapor pressure deficit across persisting, declining, and extirpated colonies. Higher values of vapor pressure deficit correspond to drier conditions. Points are jittered and colored by colony status. White points and error bars correspond to model estimates +/− one standard error of predicted mean.

For vapor pressure deficit (VPD), we found no statistically clear relationships with colony growth, regardless of epidemic phase (Model 4; Supplemental Fig. 13, Supplemental Table 9). However, we found that VPD varied with site status, as extirpated colonies occurred in significantly more humid hibernacula than declining ($\beta = 2.078 \pm 0.454$ SE, $p < 0.001$) or persisting colonies (Model 8; $\beta = 1.964 \pm 0.414$ SE, $p < 0.001$; Fig. 3B). We detected a slight negative association between early hibernation VPD and pre-WNS Indiana bat colony sizes indicating that, prior to the arrival of WNS, drier hibernacula were used by relatively smaller colonies on average compared to humid hibernacula (Model 6; $\beta = -0.210 \pm 0.093$ SE, $p = 0.025$; Supplemental Fig. 12B).

3.3. Colony extirpation simulation

Our simulation of future Indiana bat colony extirpations suggests that the rate of colony extirpation will slow in coming years (Fig. 4). Most extirpations occur early in the epidemic and the probability of



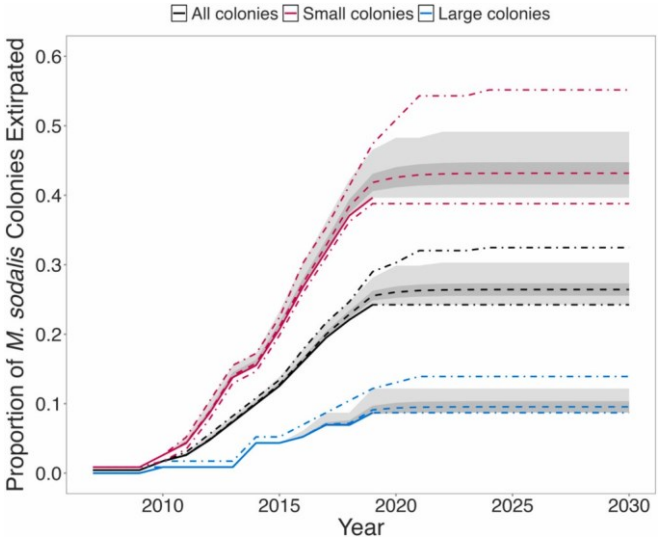


Fig. 4. Simulated and empirical accumulation of extirpated Indiana bat colonies over time as a proportion of small, large, or all colonies. Solid lines show observed data and bold dashed lines show average simulated values for each year. The dark gray shaded region corresponds to one standard deviation from the mean simulated value at each year. The light gray shaded region corresponds to the range of simulated values at each year. The dash-dotted lines correspond to the range of simulated values when the parameters of the logistic regressions describing the cumulative probability of extirpation by epidemic year are varied by one standard error. The color of lines corresponds to dataset (black = all colonies, red = small colonies (≤ 68 individuals), blue = large colonies (> 68 individuals)). (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

colony extirpation declines in later epidemic years (Supplemental Figs. 4, 7). Therefore, our simulation predicts a decay in the rate of new extirpations, with 26.41 % of total colonies (simulated range: 24.24–30.30 %), or 61 of the 231 colonies in the dataset (simulated range: 56–70), extirpated by 2028, the earliest year after which no simulation predicted additional extirpations. Because most extirpations occur early in the epidemic and most of the Indiana bat range has been affected by WNS for at least 5 years, this constitutes only an additional five extirpations from those recorded as of 2019. However, the number of predicted extirpations varied between small and large colonies. On average, 43.17 % (range: 39.66–49.14 %) of simulated small colonies were extirpated by 2028, corresponding to approximately 50 of the 116 small colonies in the dataset (range: 46–57), an increase of four extirpations from 2019. Conversely, only 9.55 % (range: 8.70–12.17 %) of simulated large colonies were extirpated, or approximately 11 of the 115 large colonies in the dataset (range: 10–14), an addition of a single extirpation from 2019. Allowing the parameters of the logistic model of colony extirpation probability to vary produced higher maximum estimates of future extirpations, stabilizing at 32.47 %, 55.17 %, and 13.91 % for all colonies, small colonies, and large colonies, respectively. While the exact rates of simulated future extirpations could be sensitive to the chosen threshold defining “small” and “large” colonies (68 bats, the median colony size), the qualitative finding that relatively smaller colonies will make up most future extirpations is robust to the chosen threshold.

4. Discussion

We found that the factors associated with Indiana bat population dynamics shifted over epidemic time. Prior to the arrival of WNS, we did not detect an effect of colony size, hibernaculum temperature, or humidity on colony growth. Following the invasion of *P. destructans*, however, responses of Indiana bat colonies varied from complete extirpation to persistence, with smaller colonies being more prone to extirpation. In addition, colonies in colder hibernacula experienced more severe declines than those in relatively warm hibernacula and were more likely to be extirpated, a novel association that arose during the WNS epidemic. Colonies in relatively humid hibernacula were also at elevated risk of extirpation, potentially another association that arose following disease emergence. Once the pathogen became established in colonies, however, we found that the effects of environmental conditions weakened slightly, suggesting a possible return to the more neutral association observed prior to WNS arrival. Together, these data illustrate the potential of novel population stressors to shift the biotic and abiotic conditions optimal for population growth. The successful conservation of wildlife populations threatened by infectious diseases emerging around the globe will therefore require flexible management strategies that reflect the shifting, context-dependent nature of population growth dynamics and resilience.

Although population responses of Indiana bats were highly variable, many colonies in our dataset eventually stabilized in the presence of WNS. Colony persistence has been observed in other species of hibernating bats impacted by WNS in North America, such as the little brown bat (Dobony et al., 2011; Frick et al., 2015; Hoyt et al., 2021; Langwig et al., 2017, 2012), potentially the result of an interaction between evolutionary forces and favorable environmental conditions that allow for relatively high survival (Grimaudo et al., 2022; Hopkins et al., 2021; Langwig et al., 2017). Persisting Indiana bat colonies had less severe declines during the initial WNS epidemic than declining or extirpated colonies, potentially due to their larger colony sizes and warmer hibernaculum environments which buffered against unsustainable rates of mortality. This illustrates that for emerging infectious disease systems that threaten at-risk host populations, identification of factors associated with population persistence could direct strategies to promote persistence in populations not yet challenged by the disease, such as those ahead of the pathogen invasion front. However, stabilized colonies are not necessarily completely protected from extirpation, as they may be declining on average despite having a single year of colony stability or growth, so the continued monitoring and conservation of remnant host populations following initial epidemic stages is essential.

In accordance with prevailing theory and corroborating previous work (Frick et al., 2015; Langwig et al., 2012), we found that extirpation risk was elevated in small colonies, potentially due to susceptibility to stochastic fadeout (Lande, 1993; Lande et al., 2003; Melbourne and Hastings, 2008) or demographic Allee effects (Gascoigne et al., 2009; Kramer et al., 2009; Stephens et al., 1999). Indiana bats are a highly gregarious species, historically having formed large clusters during hibernation (Clawson et al., 1980; Hardin and Hassell, 1970; Thomson, 1982), a behavior potentially beneficial for limiting energy expenditure during arousals from torpor (Boyles et al., 2008). Sufficiently low colony sizes may prevent conspecifics from forming clusters, increasing per- arousal energy expenditure already exacerbated by infection with *P. destructans*, causing down-stream effects on survival and ultimately leading to colony decline and extirpation. Further, the inability to form sufficiently large clusters may elevate emigration from reduced colonies, additionally contributing to their decline. If immigration to colonies occurs through individuals being led to hibernacula by conspecifics, then reduced colony sizes will further receive little immigration input, potentially slowing population growth in small colonies. Together, the consequences of reduced colony size may operate synergistically to elevate the risk of complete colony extirpation. More generally, our results suggest that pre-stressor population size, often a factor used to classify population risk, was positively associated with population resilience following pathogen arrival despite the potential for greater host density to amplify pathogen transmission. However, given the diversity of emerging infectious disease systems threatening host populations globally, the association between host population size, pathogen transmission, and population declines will be variable. For disease systems like WNS in which host infection is largely acquired through an environmental pathogen reservoir (Hoyt et al., 2020, 2015; Laggan et al., 2023), similar levels of host infection prevalence are observed across the range of host densities (de Castro and Bolker, 2005; Hoyt et al., 2020). In such cases, as illustrated by this study, the benefit of large host population sizes to lowering extirpation risk may outweigh the potential enhancement of pathogen transmission at high host densities. Conversely, for systems in which pathogen transmission is more strongly a function of host density, we may expect greater disease impacts in large initial host populations with greater rates of pathogen transmission. Ultimately, consideration of the mode of pathogen transmission in its relation to host population impacts will be important to the successful conservation of host populations following disease emergence.

Although small colonies were more likely to go extinct, we find that most colonies that will become extirpated have likely already done so due to the spread of WNS across the Indiana bat range. As the *P. destructans* invasion front advanced from its origin near Albany, New York, pathogen arrival was detected in Indiana bat colonies in every year between 2007 and 2017 in our dataset. However, all extant colonies are currently in later stages of the epidemic, when mortality is reduced and extirpation is less likely, resulting in a steep decline in the extirpation rate as the most recently infected colonies are either lost or stabilize. In the near future, declining colonies that have not yet been extirpated will either entirely collapse or settle into a stable population trajectory, ultimately determining the overall impact of WNS on Indiana bat colonies across their range. Our results further illustrate that identifying drivers of extirpation risk in the earliest-impacted populations can provide insights useful to prioritizing conservation actions for populations not yet or only recently impacted by pathogen arrival. As novel infectious diseases continue to emerge in diverse wildlife populations, gaining an early understanding of extirpation risk will be essential to their successful conservation.

We found that environmental conditions of hibernacula influenced Indiana bat colony response to WNS, an association that varied over epidemic time. In the years of high mortality and colony decline immediately following *P. destructans* arrival (epidemic phase), colonies using warmer hibernacula experienced lower declines than those in colder hibernacula. The role of temperature appeared to have arisen due to the emergence of WNS, as we did not detect the association prior to detection of the disease within hibernacula, illustrating the ability of novel infectious diseases to alter associations between environmental conditions and population growth. The effect of temperature extended to extirpation risk, as extirpated colonies used significantly colder hibernacula than extant colonies. Furthermore, while we did not detect an association between VPD within hibernacula and colony growth rates, we did find that extirpated colonies typically used more humid hibernacula than extant colonies, suggesting that humidity may additionally impact colony growth dynamics following WNS emergence. Importantly, we detected no association between pre-WNS colony size and temperature conditions within hibernacula, and a weak negative one for VPD, indicating that environmental conditions are unconfounded with colony size and operate independently to drive colony responses to WNS. Finally, while we did not investigate the role of temporal or spatial variation in temperature or humidity conditions within hibernacula on population response to WNS, it represents an additional potential driver of disease processes in this system. Future research is warranted to explore how variation in environmental conditions might influence disease processes from the individual- to population-level impacts on bat communities with WNS.

The positive association between hibernaculum temperature and colony response to WNS is opposite of that reported for the little brown bat, in which warmer hibernacula have higher disease severity and declines (Grieneisen et al., 2015; Hopkins et al., 2021; Langwig et al., 2016, 2012; Lilley et al., 2018), likely due to increased pathogen growth under those conditions (Grieneisen et al., 2015; Hopkins et al., 2021; Johnson et al., 2014; Langwig et al., 2016; Verant et al., 2012). We did not detect an association between little brown bat abundance within hibernacula and Indiana bat colony growth, suggesting that Indiana bat population declines at colder temperatures are not due to larger little brown bat colonies in cold hibernacula increasing environmental pathogen contamination (Laggan et al., 2023) or disturbance from torpor (Turner et al., 2014). Instead, this discrepancy might arise if the energy expended during arousals from torpor, which are more frequent in bats with WNS (Lilley et al., 2016; Reeder et al., 2012; Warnecke et al., 2012), is more important for surviving the disease for Indiana bats than little brown bats. The gregarious nature of Indiana bats and their propensity to form large clusters, which may further reduce cost of arousals from torpor (Boyles et al., 2008), lend possible credence to this explanation. Energy expended during arousals from torpor accounts for the majority of that spent during hibernation and is reduced under warmer conditions (Boyles and Willis, 2010; Thomas et al., 1990). The reduced cost of arousals in warm hibernacula potentially allowed Indiana bats to conserve enough energy to survive WNS over winter, buffering their overall declines. In cold hibernacula, however, the cost of additional arousals due to WNS may have been too great and resulted in premature energy depletion and mortality. For little brown bats, the disruption of water balance may instead be the physiological pathway that contributes most to mortality (Ben-Hamo et al., 2013; Cryan et al., 2010; Grimaudo et al., 2022; Thomas and Cloutier, 1992; Webb et al., 1995), which hypothetically scales positively with pathogen load. Given that the growth of *P. destructans* increases with hibernaculum temperature (Marroquin et al., 2017; Verant et al., 2012), little brown bat colonies in warm hibernacula could experience greater and unsustainable rates of water loss, resulting in their positive association between hibernaculum temperature and colony decline whereas the opposite exists for Indiana bats. However, this hypothesis necessitates a morphological or physiological mechanism by which little brown bats are more susceptible to disruptions to water balance and dehydration than Indiana bats, potentially including differences in respiratory rate or diffusion of water across the wing membrane (Cryan et al., 2010; Warnecke et al., 2013; Verant et al., 2014). These potential differences remain unexplored and future research is warranted in identifying the drivers of the opposite responses of Indiana and little brown bat colonies to hibernaculum temperatures when challenged by WNS.

The difference between Indiana and little brown bats in their colony response to WNS illustrates that in novel infectious disease systems with multiple host species there may be species-specific differences in how environmental conditions influence population response to pathogen invasion. To mitigate the impact of invading pathogens on host populations, management strategies that consider inherent differences among host species are essential. For example, habitat manipulations meant to cool or warm hibernacula and benefit a target bat species could inadvertently harm another, so careful consideration of the bat communities present is essential (Boyles et al., 2023). For example, artificially cooling hibernacula is a common technique to promote the survival of little brown bats challenged by WNS, including creating additional site entrances to promote airflow or pumping in cool air from the surface. However, our results suggest that if Indiana bat colonies are present in the same hibernacula, cooling them to augment little brown bat survival may inadvertently drive the decline of sympatric Indiana bat colonies. Furthermore, Indiana and little brown bats are only two members of a larger community of hibernating bat species in North America for which the environmental dependence of WNS impacts have not been thoroughly explored. Additional variation among these species in the form of environmental dependence of WNS dynamics may exist, further complicating the efficacy of environmental manipulation of hibernacula as a conservation strategy. Therefore, an alternative conservation strategy may be the acquisition and conservation of diverse hibernacula critical to each species, such as those that offer protective microclimates or where colony populations are stabilizing. Given that many recently emerged infectious diseases of wildlife impact more than a single host species (Cleaveland et al., 2001; Pedersen and Davies, 2010; Woolhouse, 2002) mitigating their impacts will require an understanding of how each species responds and caution is needed when considering intervention strategies that impact entire host communities. Ultimately, the appropriate management strategy will be dependent on the species community, environment, and disease conditions present, and management flexibility should reflect this potential for variation.

This study highlights how existing associations between biotic and abiotic conditions and population growth can be altered by novel stressors, including emerging infectious diseases. To protect vulnerable host populations from disease-driven extirpation, conservation measures should reflect these changing associations and consider differences among host species in shared habitat. Furthermore, it is crucial to conserve diverse habitats available to host species, as novel associations may arise and offer refugia from severe disease impacts. However, determining how to balance the conservation of habitat that is beneficial to one species while potentially detrimental to another (i.e. ecological traps), such as the case with thermal conditions of Indiana and little brown bat hibernacula, deserves additional research. As novel host—pathogen interactions continue to arise, the successful mitigation of their impacts will be dependent on our ability to characterize and respond to how they interact with the unique ecological context in which they arise.

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CRediT authorship contribution statement

Alexander T. Grimaudo: Writing – review & editing, Writing – original draft, Visualization, Project administration, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **R. Andrew King:** Project administration, Methodology, Data curation. **Chris Simpson:** Investigation. **Cory Holliday:** Investigation. **Alexander Silvis:** Investigation. **Rick T. Doyle:** Investigation. **Joseph A. Kath:** Writing – review & editing, Supervision, Resources, Project administration, Methodology, Investigation, Funding acquisition, Data curation, Conceptualization. **Mike P. Armstrong:** Investigation. **Virgil Brack:** Investigation. **Richard J. Reynolds:** Investigation. **Ryan H. Williamson:** Investigation. **Gregory G. Turner:** Investigation. **Vona Kuczynska:** Investigation. **Jordan J. Meyer:** Investigation. **Kyle Jansky:** Investigation. **Carl J. Herzog:** Methodology, Investigation. **Skylar R. Hopkins:** Project administration, Methodology, Investigation, Funding acquisition, Data curation, Conceptualization. **Kate E. Langwig:** Writing – review & editing, Supervision, Resources, Project administration, Methodology, Investigation, Funding acquisition, Data curation, Conceptualization. **Declaration of competing interest**

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Data accessibility statement

The data used in this study is available on Dryad (doi: 10.5061/dryad.3xsj3txqb).

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.biocon.2024.110693.

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