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Compensatory effects of recruitment and survival when amphibian populations are perturbed by disease

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Summary

1. The need to increase our understanding of factors that regulate animal population dynamics has been catalysed by recent, observed declines in wildlife populations worldwide. Reliable estimates of demographic parameters are critical for addressing basic and applied ecological questions and understanding the response of parameters to perturbations (e.g. disease, habitat loss, climate change). However, to fully assess the impact of perturbation on population dynamics, all parameters contributing to the response of the target population must be estimated.

2. We applied the reverse-time model of Pradel in Program MARK to 6 years of capture–recapture data from two populations of *Anaxyrus boreas* (boreal toad) populations, one with disease and one without. We then assessed a priori hypotheses about differences in survival and recruitment relative to local environmental conditions and the presence of disease.

3. We further explored the relative contribution of survival probability and recruitment rate to population growth and investigated how shifts in these parameters can alter population dynamics when a population is perturbed.

4. High recruitment rates (0·41) are probably compensating for low survival probability (range 0·51–0·54) in the population challenged by an emerging pathogen, resulting in a relatively slow rate of decline. In contrast, the population with no evidence of disease had high survival probability (range 0·75–0·78) but lower recruitment rates (0·25).

5. *Synthesis and applications.* We suggest that the relationship between survival and recruitment may be compensatory, providing evidence that populations challenged with disease are not necessarily doomed to extinction. A better understanding of these interactions may help to explain, and be used to predict, population regulation and persistence for wildlife threatened with disease. Further, reliable estimates of population parameters such as recruitment and survival can guide the formulation and implementation of conservation actions such as repatriations or habitat management aimed to improve recruitment.

Key-words: amphibian decline, *Batrachochytrium dendrobatidis*, boreal toad, disease, host–pathogen dynamics, reverse-time Pradel model, United States

Introduction

Achieving an understanding of the immediate and the evolutionary responses of animal populations to perturbation depends on an appreciation of how demographic parameters interact and how they are affected by extrinsic factors. Survival receives the majority of attention in demographic studies, but other parameters such as recruitment can have significant influence on population dynamics. To fully assess the impact of perturbation on population dyuamics, studies must consider, and quautify, all parameters contributing to the response of the target population.

Emerging infectious disease is a perturbation that is increasingly named as a factor influencing human and wildlife populations (Daszak, Cunningham & Hyatt 2000; Reperant 2010). Disease can affect population demography (e.g. Lachish, McCallum & Jones 2009), population persistence (Woodworth *et al.* 2005), and potentially, organismal life histories (e.g. Choisy & Rohani 2006). Concern has escalated because of evidence for interactions between disease and environmental

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stressors (e.g. contaminants, habitat alteration and climate change) and changes in epidemiology (i.e. human-facilitated spread of disease). A better understanding of the role of disease in wildlife population dynamics is needed urgently given the onslaught of emerging and infectious diseases worldwide (Daszak, Cunningham & Hyatt 2000; Oli *et al.* 2006).

There is empirical evidence that emerging diseases affect wildlife population dynamics and increase the risk of extinction in a variety of taxonomic groups: White-nose syndrome in bats caused by the fungus *Geomyces destructans* (Wibbelt *et al.* 2010), facial tumour disease in Tasmanian devils *Sarcophilus harrisii* (Lachish, McCallum & Jones 2009), conjunctivitis in house finches *Carpodacus mexicanus* caused by *Mycoplasma gallisepticum* (Dhondt *et al.* 2005), upper respiratory disease in gopher tortoises *Gopherus polyphemus* caused by *Mycoplasma agassizii* (Ozgul *et al.* 2009) and chytridiomycosis caused by the fungus *Batrachochytrium dendrobatidis* (*Bd*) in amphibians (e.g.Vredenburg *et al.* 2010). Despite the clear link between emerging diseases and population persistence, *in situ* quantitative studies on the effects of disease on survival in wildlife populations are rare (but see Briggs *et al.* 2005; Lachish, McCallum & Jones 2009; Murray *et al.* 2009; Ozgul *et al.* 2009; Spencer & Janzen 2010) and few studies have explored relationships among demographic parameters to clarify how a disease is affecting a population.

Chytridiomycosis provides an example of a wildlife disease receiving considerable attention but lacking strong quantification of its effects on population demography in the wild. *Bd* is a pathogen impacting amphibian populations worldwide (e.g. Skerratt *et al.* 2007). The effects of *Bd* vary by amphibian species and by population, in some causing catastrophic extirpations and declines (e.g. Ryan, Lips & Eichholz 2008) and in others having minimal effects resulting in apparent coexistence (e.g. Retallick, McCallum & Speare 2004; Briggs *et al.* 2005; Pilliod *et al.* 2010). The profusion of research on this wildlife disease (at least 254 original research papers published since its discovery in 1997: Web of Science search on *Batrachochytrium dendrobatidis*) provides an excellent base of knowledge to develop and test hypotheses about the immediate effects of disease on demographic parameters and population dynamics. This opportunity is timely. Advances in molecular techniques now allow more accurate field assessments of disease presence and prevalence, providing opportunities to explore extrinsic factors affecting population dynamics. Advances in the analytical aspects of mark–recapture methodology have replaced traditional ad hoc measures (e.g. return rates) with models that account for imperfect detection and allow estimation of a broad range of demographic parameters.

Using advances in both fields, we assessed the population-level effects of *Bd* on a population of wild bufonids *Anaxyrus boreas* in western North America. In amphibians, the number of adults (reproductively mature individuals) at a breeding pond in a particular breeding season is a function of: (i) the number of adults that survived from the previous year and returned to breed, and (ii) the number of adults recruited into the population since the previous breeding season. Previous studies have evaluated the effect of *Bd* on the survival probability of adult amphibians (Murray *et al.* 2009; Briggs, Knapp & Vredenburg 2010; Pilliod *et al.* 2010). However, they have not estimated recruitment rates in populations infected by *Bd*. We assessed survival and recruitment as a way to evaluate population growth and persistence (e.g. Schmidt, Feldmann & Schaub 2005) and, in an approach that is atypical in wildlife disease studies, we compared demographic estimates from a diseased population to demographic estimates from a reference population that lacked evidence of disease in the same region. These components were also examined in relation to potential interactions with site-level environmental covariates.

Materials and methods

We collected capture–recapture data from male boreal toads in two populations, one in Colorado [Denny Creek (DC)] and, one in Wyoming [Blackrock (BR)]. The disease status of each population was based on PCR testing of > 100 individuals per site over 6 years (2003–2008, Table 2), yielding one population positive for disease (BR, elevation 2082 m, latitude = 43·8°) and one population negative for disease (DC, elevation 3360 m, latitude = 38·8°). Both populations are in typical rocky mountain habitat dominated by willow *Salix planifolia*, bog birch *Betula glandulosa*, aspen *Populus tremuloides* and evergreen trees (e.g. *Picea englemanii*, *Abies lasiocarpa*) and are similar ecologically. In the Rocky Mountains, toad populations at higher latitudes are found at lower elevations, and vice versa (Muths, Pilliod & Livo 2008) such that differences because of elevation (DC > BR) are partially balanced by differences in latitude (BR > DC). Naïve prevalence was determined by dividing the

Table 1. The hypotheses of spatial and temporal variation in survival probability (Φ) and recruitment rate (*f*) along with their associated model names

Hypothesis	Model name
Survival models	
Φ decreases as prevalence (PREV) increases in the *Batrachochytrium dendrobatidis* (*Bd*)-positive population while survival remains constant in the *Bd*-negative population	Φ (PREV + POP)
Φ is lower in the *Bd*-positive population (BR) than in the *Bd*-negative population (DC)	Φ (SITE)
Φ is positively related to TMAX/KFdays in the *Bd*-positive population, and the relationship is different in the *Bd*-negative population	Φ (TMAX*POP) Φ (KFdays*POP)
Φ is constant	Φ (.)
Recruitment models	
f is lower in the *Bd*-positive population (BR) than at the *Bd*-negative population (DC)	*f*(POP)
f is the same between populations	*f*(.)

number of *Bd*-positive individuals by the number of individuals tested. The accuracy of this estimate depends on a constant detection probability for both diseased and un-diseased individuals, an unlikely case (i.e. we suspect that diseased individuals are less likely to be detected because die-off events for this species are rarely observed). Thus, our prevalence estimate is likely to be low (Table 2). Animals were captured by hand, at night during the breeding season. Early season monitoring was problematic because sites were remote; therefore, we defined the breeding season as 1 week prior to the first observation of a toad plus 28 days after that observation (for details see Pilliod *et al.* 2010). We used the reverse-time model of Pradel (Williams, Nichols & Conroy 2002) in Program MARK (White & Burnham 1999) to analyse the capture–recapture data. Although we collected data for males and females, data on females were too sparse to be used in the analysis. Furthermore, female toads tend to show Markovian breeding probabilities (Muths, Scherer & Lambert 2010) which can cause bias in estimates of parameters in the Pradel model (Williams, Nichols & Conroy 2002; Schmidt, Feldmann & Schaub 2005). We used the parameterization of the Pradel model that contained the following parameters: apparent survival probability (Φ), recruitment rate (f) and capture probability (p) to elucidate the effects of *Bd* on Φ and f at the population level. In this model, the survival parameter, Φ, is 'apparent' because permanent emigration cannot be distinguished from death. However, permanent emigration in male boreal toads appears to be low (Muths *et al.* 2006); therefore, we consider Φ to represent true survival. Recruitment is the per capita number of individuals added to a breeding population each year (i.e. new breeders whether from *in situ* reproduction or immigration from other populations). In this study, as is often the case for bufonids, recruitment is not equivalent to reproduction because some animals spend multiple years as sexually immature juveniles prior to entering the breeding population (Schmidt, Feldmann & Schaub 2005). Capture probability, p, is defined as the probability that a marked individual in the population is captured during a particular sampling period. Adequate modelling of capture probability is necessary to avoid unnecessary bias and imprecision in estimates of survival probability and recruitment rate (Lebreton *et al.* 1992).

We developed a set of hypotheses regarding causes of the spatial (i.e. between populations) and temporal variation in the model parameters. For survival probability, Φ, we evaluated three general *a priori* hypotheses: (i) as the naïve prevalence of *Bd* in a population increases, Φ decreases, (ii) Φ is lower in the *Bd*-positive population (BR) vs. the *Bd*-negative population (DC) and (iii) the *Bd*-positive and *Bd*-negative populations are affected differently by environmental conditions (Table 1). We hypothesized relationships between Φ and two environmental covariates related to temperature because earlier studies highlight the importance of temperature in the *Bd*–host interaction (Muths, Pilliod & Livo 2008; Kilpatrick, Briggs & Daszak 2010). Average daily maximum air temperature (TMAX) represents a heat-related condition and the potential for amphibians to clear themselves of the disease behaviourally (Richards-Zawacki 2009). Therefore, we hypothesized that Φ would be positively associated with TMAX in the *Bd*-positive population but would have a different relationship to TMAX in the *Bd*-negative population. We defined TMAX as the temperature during the active season of July and August, the hottest months at the sites and the months after the breeding season in which toads were most active. The number of killing frost days during the breeding season (KFdays) represents a cold-related stressor and the potential for cold to limit growth and reproduction of *Bd* (i.e. fungal growth slows below 4 °C, Piotrowski, Annis & Longcore 2004). Therefore, we hypothesized that Φ would be positively associated with KFdays in the *Bd*-positive population and have a different relationship to KFdays in the *Bd*-negative population.

Table 2. For the *Batrachochytrium dendrobatidis* (*Bd*)-positive population (BR) and the *Bd*-negative population (DC): number of male toads captured; probability of capture (p); prevalence 1 (number of animals testing Bd-positive/no. of animals testing positive in previous years; this value is biased high because it includes animals that were targeted for testing because of previous positive results); prevalence 2 (number of animals testing Bd-positive/no. of animals tested; this value does not include animals that were targeted, i.e. it is a random sample from the captured population, but suffers from low sample numbers). The prevalence 2 data were used to derive the values for prevalence in the text.

Site	No. of animals caught	Capture probability (95% CI)	No. tested for *Bd*	Prevalence 1	Prevalence 2
BR					
2003	259	0·90 (0·47–0·99)	28	0·43	0·43
2004	249	0·47 (0·35–0·60)	41	0·41	0·39
2005	266	0·42 (0·31–0·55)	6	0·83	0
2006	216	0·51 (0·37–0·64)	28	0·57	0·36
2007	239	0·50 (0·34–0·65)	4	0·5	0
2008	170	0·32 (0·19–0·48)	22	0·45	0·45
DC					
2003	145	0·41 (0·30–0·53)	14	0	0
2004	80	0·42 (0·33–0·52)	24	0	0
2005	71	0·49 (0·39–0·60)	25	0	0
2006	88	0·44 (0·34–0·54)	37	0	0
2007	86	0·51 (0·37–0·65)	13	0	0
2008	55	0·39 (0·25–0·55)	10	0	0

We also evaluated two hypotheses for recruitment rate (f): (i) f is lower at the *Bd*-positive site, based on laboratory studies that report that *Bd* affects survival negatively in all post-metamorphic life stages of boreal toads (Carey *et al.* 2006; Murphy *et al.* 2009) such that there may be fewer juveniles to be recruited into the breeding population, and (ii) f is constant between the two populations (Table 1). We did not assess environmental covariates in relation to recruitment because each amphibian life stage that contributes to recruitment differs dramatically in its response to temperature and the inclusion of appropriately time-lagged covariates would have complicated the model set.

Finally, we assessed six hypotheses for p: (i) p constant [null model, $p(.)$]; (ii) p varies across years only [p(time)], (iii) p varies between populations only [p(POP)], (iv) p varies across years and between populations [p(POP*time)], (v) p is higher in years with higher temperatures during sampling occasions, (vi) p is higher in years with higher temperatures during sampling occasions, but the relationship differs between DC and BR (i.e. an interaction between temperature and population). For each parameter, we expressed the hypotheses as mathematical models (Table 1) and fit every possible combination of the models of Φ, f and p to the capture–recapture data.

Strength of evidence for each model was quantified using the difference in $QAIC_c$ of model i and the $QAIC_c$ of the top model, Δ_i, and Akaike weights, w_i; we used the information criterion, $QAIC_c$, because of the evidence of overdispersion in the data (Burnham & Anderson 2002). We used model averaging to derive estimates of all parameters (Burnham & Anderson 2002). The rate of population growth (λ) for each population was estimated as a derived parameter using Program MARK (White & Burnham 1999; Williams, Nichols & Conroy 2002). Because we hypothesized that Φ and f would be lower

in the *Bd*-positive population, we expected estimates of λ from the BR population to be much smaller.

Results

Over 6 years, we captured over 2000 male toads and tested over 250 for *Bd*. When averaged across years, prevalence was 27% at BR vs. 0% at DC (Table 2). A goodness-of-fit test is not available for the Pradel model but previous analyses using these data (Pilliod *et al.* 2010) indicated moderate over dispersion ($\hat{c} = 1.75$). We used the same $\hat{c}$ value in this analysis. We also ran the analysis using $\hat{c}$ of 1·00 and 2·50 and observed only minor changes in the model selection results and estimates of variance. Capture probability (p) varied across years and between populations such that the top eight models included an interactive effect of population and time in the structure on p. We found little evidence of a relationship between p and temperature. The highest ranked model that included this relationship had $\Delta_i > 12{\cdot}0$ and $w_i < 0{\cdot}01$.

There was strong evidence that Φ varied between populations and was compromised by the presence of *Bd* (Table 2 and Fig. 1). Specifically, Φ in the *Bd*-negative (DC) population was 0·77, whereas it was 0·52 in the *Bd*-positive (BR) population. An effect of population was present in the top six models (Table 3), and the estimate of the regression coefficient from the top model indicated higher survival probability in the *Bd*-negative population, DC [$\hat{\beta}_{DC} = 1{\cdot}11$ (95% CI: 0·63–1·58)]. There was also evidence that the effect of TMAX on Φ varied between populations. Estimates of regression coefficients from the second- and sixth-ranked model indicated that increases in TMAX were associated with decreases in Φ at BR [$\hat{\beta}_{TMAX*BR} = -12{\cdot}9$ (95% CI: −33 to 7·3)] but were associated with increases in Φ at DC [$\hat{\beta}_{TMAX*DC} = 40$ (95% CI: −24·3 to 104·3)]. These estimates were not consistent with our predictions (Table 1). There was little evidence for a relationship

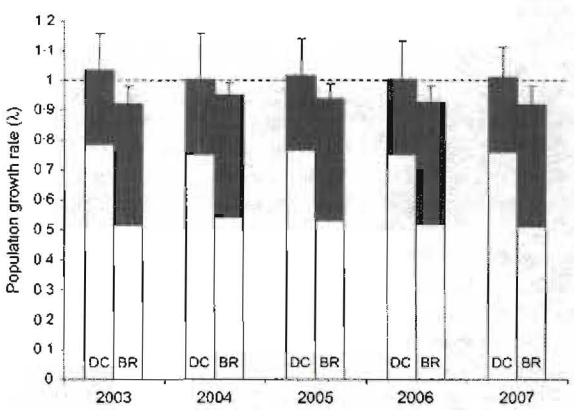

Fig. 1. Lambda, or rate of change in population size (λ) is composed of two parts, survival (open area of bars) and recruitment (solid area of bars). Values > 1 indicate growth and values < 1 indicate decline, error bars show 95% confidence interval of λ. Although survival is higher in the *Batrachochytrium dendrobatidis* (*Bd*)-negative population (DC), recruitment is higher in the *Bd*-positive population, thus probably compensating for reduced survival.

Table 3. Top six models of survival probability (Φ), recruitment rate (f) and capture probability (p). The model structure for p is not shown, because it was the same across all six models [p(POP*time)]

Model no.	Model name	$QAIC_c$	Δ_i	w_i	k	QDeviance
1	{Φ (POP), f(POP)}	4269·80	0·00	0·48	16	102·17
2	{Φ (TMAX*POP), f(POP)}	4271·38	1·58	0·22	18	99·67
3	{Φ (PREV + POP), f(POP)}	4271·68	1·88	0·19	17	102·00
4	{Φ (KFdays*POP), f(POP)}	4273·67	3·87	0·07	18	101·96
5	{Φ (POP) f(.)}	4276·28	6·48	0·02	15	110·68
6	{Φ (TMAX*POP), f(.)}	4277·45	7·65	0·01	17	107·78

$\Delta_i = \Delta QAIC_c$ of model i, w_i = Akaike weight of model i, k = number of parameters, QDeviance = −2 log likelihood/$\hat{c}$. POP denotes population (*Bd*-positive vs. *Bd*-negative), PREV denotes prevalence. See text for descriptions of covariates TMAX and KFdays.

between survival probability and KFdays or prevalence in either population. While models that included these covariates were supported by the data ($\Delta_i < 2$; Table 3), estimates of the regression coefficients in both cases were imprecise and clearly overlapped zero, precluding strong inference about these relationships.

The model selection results indicated strong support for differential recruitment between the two populations [the top four models all indicated an effect of population on recruitment rate (Table 3)]. Contrary to what we predicted, recruitment rates were higher in the *Bd*-positive population; recruitment rates were estimated as 0·41 (95% CI: 0·33–0·50) and 0·25 (95% CI: 0·17–0·68) for BR and DC, respectively (Fig. 1).

The population growth rates indicate very different fates for the two populations. At BR, the rate was consistently < 1, indicating a declining population, and at DC, the rate was consistently > 1 indicating an increasing population (Fig. 1).

Discussion

Although our study has limited inference, it offers important information on potential mechanisms underlying the dynamics of populations challenged by disease and how they may respond to perturbation. As predicted, survival rates were higher in the *Bd*-negative (77%, DC), compared to the *Bd*-positive (52%, BR), population. Previous work shows similar survival rates for male and female boreal toads (Scherer, Muths & Lambert 2008; Muths, Scherer & Lambert 2010), such that results from this study (male data only) may be applicable to other boreal toad populations.

Based on the following lines of evidence, we expected the relatively low survival probability of toads in the infected population to depress population growth (λ). First, boreal toads are long-lived (i.e. they have high survival rates once they attain adulthood; Muths & Nanjappa 2005), and thus, the death of sexually mature individuals should have consequences for population growth. Long-lived species typically have 'slow' life

histories characterized by delayed maturity and high survival, and thus, population growth (i.e. resiliency) is often heavily influenced by adult survival (Musick 1999; Sæther & Bakke 2000; Vonesh & De la Cruz 2002; Spencer & Janzen 2010). This pattern has been illustrated specifically for boreal toads (Bick *et al.* 2002). However, the λ estimates for the *Bd*-positive population (3–11% annual decline) were not as low as would be expected when examining the survival rates alone, suggesting that another parameter may be compensating for the low survival rates at BR. We provide evidence for higher rates of recruitment at BR than at DC such that estimates of λ in the *Bd*-positive population approach estimates of λ in the *Bd*-negative population (Fig. 1).

Our data indicate high recruitment rates compensate for low survival at BR such that the population is able to persist, rather than showing the catastrophic declines reported in other populations challenged by *Bd*. At DC, where recruitment is contributing relatively little to the persistence of the population, the data suggest that if challenged by disease and a resulting decline in adult survival, persistence would depend on an upward shift in recruitment rate. Simple geometric growth models, while not formal population viability analyses, can offer additional insight into our results. These models indicate a slow decline in the *Bd*-positive population (BR) (Fig. 2). Interestingly, if the recruitment rate from the *Bd*-negative population (DC) was substituted for f in modelling the population growth rate at BR, the BR population went extinct (< 2 individuals) within 20 years compared to persistence beyond 40 years at the current f (Fig. 2), highlighting the importance of recruitment in this system.

We present this idea of compensation by one demographic parameter for another, as one of many potential explanations

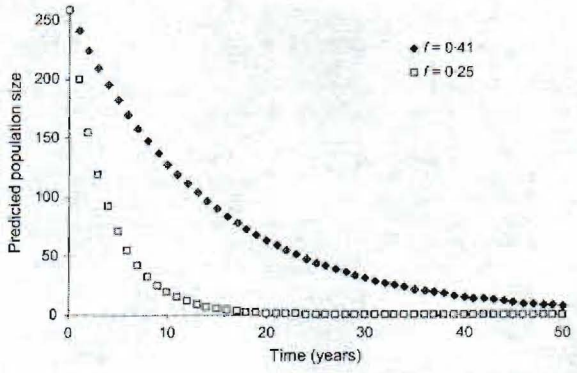

Fig. 2. We applied a simple geometric growth model to the data: $N_{t+1} = N_t + b^*N_t + d^*N_t$ where $b = f$ and $d = 1-\Phi$ to examine population persistence over 50 years. In this model, we used recruitment, instead of births, because we were interested in the addition of reproductively mature individuals to a population. The initial population size at year 0 was the number of captured adult male toads in 2003. We used a single value for b and d calculated from an average f and Φ from 2003 to 2007. The graph shows the geometric growth of the Bd-positive population (BR) using the observed recruitment rate for BR (f = 0·41, solid diamonds) and the observed recruitment rate for the *Bd*-negative population (DC, f = 0·25, open squares). Population size at time 0 = number of male toads observed in 2003 at BR.

of why some boreal toad populations are able to persist with disease while others are not. This idea may apply to other amphibians and wildlife species, although further testing of this hypothesis is warranted. We hypothesise that the differences in recruitment between our toad populations may reflect an elevation effect and contribute to an explanation of observed differences in vulnerability to *Bd* for high-vs. low-elevation amphibians (e.g. Walker *et al.* 2010), although this hypothesis remains to be tested.

Healthy high-elevation populations in general are 'on the edge' with limited recruitment because of short growing seasons and harsh climate. High-elevation amphibian populations are known to have slower growth rates, smaller clutch sizes and lower recruitment than lower elevation populations (e.g. Funk *et al.* 2005; Gibson, Van der Marel & Starzomski 2009), skip breeding opportunities (Muths, Scherer & Lambert 2010), and have less opportunity to behaviourally regulate their body temperature (potentially combating disease, Woodhams, Alford & Marantelli 2003). Recent work by McCaffery & Maxell (2010) reported increased survival and breeding probability with less severe winters, reminding us that even without disease, the environment can be stressful. When such populations are infected by disease, it is possible that their already low recruitment rate cannot compensate for decreasing survival probability and the population, without immigration, may be extirpated. Denny Creek is an example of such a site, within 280 m of the maximum elevation reported for boreal toads (Muths & Nanjappa 2005).

In contrast, low-elevation populations enjoy a less harsh environment, including longer growing seasons, potentially more productive habitats, shorter times to reproductive maturity and perhaps higher survival rates, particularly for earlier life stages. When disease perturbs a low-elevation system, flexibility exists, and recruitment, which may have been density limited, can compensate for lower survival because of disease. This is a key point because in some cases, survival is considered to be a fairly stable component for long-lived species and changes in the rate of population growth result largely from variability in the recruitment component (e.g. spotted owls (*Strix occidentalis caurina*) in the western U.S., Franklin *et al.* 2000).

Although the persistence of some populations (e.g. *Bufo calamita*, Sinsch 1992) has been ascribed to the 'rescue effect' (Brown & Kodric-Brown 1977), a phenomenon thought to be driven by immigration in either a metapopulation or source-sink context (Howe, Davis & Mosca 1991), extensive surveys in the area of this study have not revealed a source population and thus we suspect that high recruitment rates in the *Bd*-positive population are because of local reproduction.

Recruitment of first-time breeders is dependant on many inter-related factors, some of which affect juvenile animals disproportionately to adults. For example, local weather conditions (e.g. harsher winters at higher elevations) may reduce overwinter survival for smaller bodied juveniles; availability of suitable overwintering sites for recently metamorphosed toads may limit survival; and differences in site productivity (sites with higher productivity may provide more resources) can impact growth, fecundity and survival.

Our data suggest that DC is vulnerable to disease and that BR is able to persist with disease partly because of the demographic characteristics and responses of the populations. However, there are other factors that contribute to a population's response to such perturbation. Recent work by Briggs, Knapp & Vredenburg (2010) suggests that the fungal load (number of zoospores) carried by individual animals determines the level of persistence in a population, suggesting that after the initial exposure to *Bd* and consequent population decline, the population may persist with an endemic infection with little impact on survival rates. At BR, the fungal load ranged from 0 to 36·8 (SD 0·97) zoospore equivalents (based on a single year, E. Muths, D. S. Pilliod, R. D. Scherer, unpublished data), much lower values than reported for declining populations of yellow-legged frogs (Vredenburg *et al.* 2010) and similar values to those that Briggs, Knapp & Vredenburg (2010) report for populations that have putatively survived an initial onslaught of *Bd*, raising the possibility of a similar scenario for the BR population.

Population persistence is a subjective term, but any persistence, even short term, has the potential to benefit populations challenged with disease. Although the probability of survival of infected individuals was lower in our *Bd*-positive population than in the *Bd*-negative population, continued reproduction and a relatively high recruitment rate in the *Bd*-positive population appear to be slowing the rate of population decline and increasing chances of persistence as evidenced by our observations of the population for over 8 years coincident with *Bd* infection. This lag between putative initial infection and extirpation allows time for shifts in life-history strategies to occur (e.g. earlier sexual maturity sensu Lachish, McCallum & Jones 2009; Spencer & Janzen 2010) or acquired genetic immunity (Richmond *et al.* 2009), thus, short-term persistence may 'buy time' for changes that will allow long-term persistence with disease. In our case, it appears that environmental conditions appropriate to short-term persistence may exist only at lower elevation sites. While we have shown reduced survival in populations with *Bd* and the impact of differing recruitment rates in determining persistence, we are not able to evaluate the effects on age structure and the commensurate effects of *Bd* as a selection agent in altering life histories as was accomplished for Tasmanian devils with facial tumour disease (Lachish, McCallum & Jones 2009). Future efforts to assess age structure for bufonids might include an assessment of age (size) at breeding or clutch size.

Amphibian disease and the declines associated with it are multifactorial in cause and in effect. The global extent of declines and the panzootic nature of *Bd*, make amphibian decline a handy study system to investigate demographic response to disease, and potentially other perturbations. We provide evidence that populations challenged with *Bd* are not necessarily doomed to extinction because changes in other demographic parameters may compensate for depressed survival. Such information for amphibians, especially those species that may be able to persist with disease (e.g. Briggs, Knapp & Vredenburg 2010; Pilliod *et al.* 2010), is of critical importance – in the short term to facilitate conservation decision-making and action, and in the long term to increase understanding of evolutionary aspects of host–pathogen interactions. Although focused on a single taxa confronted by a single pathogen, this example from the field illustrates a quantitative approach to examining the underlying theory of population ecology and underscores the necessity of using quantitative techniques to acquire reliable estimates of multiple demographic parameters to unravel the complexities of disease ecology.

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References

Biek, R., Funk, W.C., Maxell, B.A. & Mills, L.S. (2002) What is missing in amphibian decline research: insights from ecological sensitivity analysis. *Conservation Biology*, **16**, 728–734.

Briggs, C.J., Knapp, R.A. & Vredenburg, V.T. (2010) Enzootic and epizootic dynamics of the chytrid fungal pathogen of amphibians. *Proceedings of the National Academy of Sciences, USA*, **107**, 9695–9700.

Briggs, C.J., Vredenburg, V.T., Knapp, R.A. & Rachowicz, L.J. (2005) Investigating the population-level effects of chytridiomycosis: an emerging infectious disease of amphibians. *Ecology*, **86**, 3149–3159.

Brown, J.H. & Kodric-Brown, A. (1977) Turnover rates in insular biogeography: effect of immigration on extinction. *Ecology*, **58**, 445–449.

Burnham, K.P. & Anderson, D.R. (2002) *Model Selection and Inference: A Practical Information – Theoretic Approach*. Springer-Verlag, New York, NY.

Carey, C., Bruzgul, J.E., Livo, L.J., Walling, M.L., Kuehl, K.A., Dixon, B.F., Pessier, A.P., Alford, R.A. & Rogers, K.B. (2006) Experimental exposures of boreal toads (*Bufo boreas*) to a pathogenic chytrid fungus. *EcoHealth*, **3**, 5–21.

Choisy, M. & Rohani, P. (2006) Harvesting can increase severity of wildlife disease epidemics. *Proceedings of the Royal Society of London B.*, **273**, 2025–2034.

Daszak, P., Cunningham, A.A. & Hyatt, A.D. (2000) Emerging infectious diseases of wildlife-threats to biodiversity and human health. *Science*, **287**, 443–449.

Dhondt, A.A., Altizer, S., Cooch, E.G., Davis, A.K., Dobson, A.P., Driscoll, M.J.L., Hartup, B.K., Hawley, D.M., Hochachka, W.M., Hosseini, P.R., Jennelle, C.S., Kollias, G.V., Ley, D.H., Swarthout, E.C.H. & Sydenstricker, K.V. (2005) Dynamics of a novel pathogen in an avian host: mycoplasmal conjunctivitis in house finches. *Acta Tropica*, **94**, 77–93.

Franklin, A.B., Anderson, D.R., Gutierrez, R.J. & Burnham, K.P. (2000) Climate, habitat quality and fitness in northern spotted owl population in northwestern California. *Ecological Monographs*, **70**, 539–590.

Funk, W.C., Blouin, M.S., Corn, P.S., Maxell, B.A., Pilliod, D.S., Amish, S. & Allendorf, F.W. (2005) Population structure of Columbia spotted frogs (*Rana luteiventris*) is strongly affected by the landscape. *Molecular Ecology*, **14**, 483–496.

Gibson, S.Y., Van der Marel, R.C. & Starzomski, B.M. (2009) Climate change and conservation of leading-edge peripheral populations. *Conservation Biology*, **23**, 1369–1373.

Howe, R.W., Davis, G.J. & Mosca, V. (1991) The demographic significance of "sink" populations. *Biological Conservation*, **57**, 239–255.

Kilpatrick, A.M., Briggs, C.J. & Daszak, P. (2010) The ecology and impact of chytridiomycosis: an emerging disease of amphibians. *Trends in Ecology and evolution*, **25**, 109–118.

Lachish, S., McCallum, H. & Jones, M.E. (2009) Demography, disease and the devil: life history changes in a disease-affected population of Tasmanian devils. *Journal of Animal Ecology*, **78**, 427–436.

Lebreton, J.D., Burnham, K.P., Clobert, J. & Anderson, D.R. (1992) Modeling survival and testing biological hypotheses using marked animals: a unified approach with case studies. *Ecological Monographs*, **62**, 67–118.

McCaffery, R.M. & Maxell, B.A. (2010) Decreased winter severity increases viability of a montane frog population. *Proceedings of the National Academy of Sciences, USA*, **107**, 8644–8649.

Murphy, P.J., St-Hilaire, S., Bruer, S., Corn, P.S. & Peterson, C.R. (2009) Distribution and pathogenicity of *Batrachochytrium dendrobatidis* in boreal toads from the Grand Teton area of western Wyoming. *EcoHealth*, **6**, 109–120.

Murray, K.A., Skerratt, L.F., Speare, R. & McCallum, H. (2009) Impact and dynamics of disease in species threatened by the amphibian chytrid fungus, *Batrachochytrium dendrobatidis*. *Conservation Biology*, **23**, 1242–1252.

Musick, J.A. (1999) Life in the slow lane: ecology and conservation of long-lived marine animals. American Fisheries Society Symposium 23, Bethesda, pp. 1–10.

Muths, E. & Nanjappa, P. (2005) Family Bufonidae: *Bufo boreas* Baird and Girard, 1852, western toad. *Amphibian Decline: The Conservation Status of United States Species* (ed. M. Lannoo), pp. 64–83. University of California Press, Berkeley, CA.

Muths, E., Pilliod, D.S. & Livo, L.J. (2008) Distribution and environmental limitations of an amphibian pathogen in the Rocky Mountains, USA. *Biological Conservation*, **141**, 1484–1492.

Muths, E., Scherer, R.D. & Lambert, B.A. (2010) Unbiased survival estimates and evidence for skipped breeding opportunities in females. *Methods in Ecology and Evolution*, **1**, 123–130.

Muths, E., Scherer, R.D., Corn, P.S. & Lambert, B.A. (2006) Estimation of temporary emigration in male toads. *Ecology*, **4**, 1048–1056.

Oli, M.K., Venkataraman, M., Klein, P.A., Wendland, L.D. & Brown, M.B. (2006) Population dynamics of infectious diseases: a discrete time model. *Ecological Modelling*, **198**, 183–194.

Ozgul, A., Oli, M.K., Bolker, B.M. & Perez-Heydrich, C. (2009) Upper respiratory tract disease, force of infection, and effects on survival of gopher tortoises. *Ecological Applications*, **19**, 786–798.

Pilliod, D.S., Muths, E., Scherer, R.D., Bartelt, P.E., Corn, P.S., Hossack, B.R., Lambert, B.A., McCaffery, R. & Gaughan, C. (2010) Impact of amphibian chytrid fungus (*Batrachochytrium dendrobatidis*) infection on individual survival probability of wild boreal toads. *Conservation Biology*, **24**, 1259–1267.

Piotrowski, J.S., Annis, S.L. & Longcore, J.E. (2004) Physiology of *Batrachochytrium dendrobatidis*, a chytrid pathogen of amphibians. *Mycologia*, **96**, 9–15.

Reperant, L.A. (2010) Applying the theory of island biogeography to emerging pathogens: toward predicting the sources of future emerging zoonotic and vector-borne diseases. *Vector-borne and Zoonotic Diseases*, **10**, 105–110.

Retallick, R.W.R., McCallum, H. & Speare, R. (2004) Endemic infection of the amphibian chytrid fungus in a frog community post-decline. *PLoS Biology*, **2**, e351, 1965–1971. Doi: 310.1371/journal.pbio.0020351.

Richards-Zawacki, C. (2009) Thermoregulatory behaviour affects prevalence of chytrid fungal infection in a wild population of Panamanian golden frogs. *Proceedings of the Royal Society of London B*, **277**, 519–528.

Richmond, J.Q., Savage, A.E., Zamudio, K.R. & Rosenblum, E.B. (2009) Toward immunogenetic studies of amphibian chytridiomycosis: linking innate and acquired immunity. *BioScience*, **59**, 311–320.

Ryan, M.J., Lips, K.R. & Eichholz, M.W. (2008) Decline and extirpation of an endangered Panamanian stream frog population (*Craugastor punctariolus*) due to an outbreak of chytridiomycosis. *Biological Conservation*, **141**, 1636–1647.

Sæther, B.-E. & Bakke, Ø. (2000) Avian life history variation and contribution of demographic traits to the population growth rate. *Ecology*, **81**, 642–653.

Scherer, R.D., Muths, E. & Lambert, B.A. (2008) Effects of weather on survival in populations of boreal toads in Colorado. *Journal of Herpetology*, **42**, 508–517.

Schmidt, B.R., Feldmann, R. & Schaub, M. (2005) Demographic processes underlying population growth and decline in *Salamandra salamandra*. *Conservation Biology*, **19**, 1149–1154.

Sinsch, U. (1992) Structure and dynamic of a natterjack toad metapopulation (*Bufo calamita*). *Oecologia*, **90**, 489–499.

Skerratt, L.F., Berger, L., Speare, R., Cashins, S., McDonald, K.R., Phillott, A.D., Hines, H.B. & Kenyon, N. (2007) Spread of chytridiomycosis has caused the rapid global decline and extinction of frogs. *EcoHealth*, **4**, 125–134.

Spencer, R.-J. & Janzen, F.J. (2010) Demographic consequences of adaptive growth and the ramifications for conservation of long-lived organisms. *Biological Conservation*, **143**, 1951–1959.

Vonesh, J.R. & De la Cruz, O. (2002) Complex life cycles and density dependence: assessing the contribution of egg mortality to amphibian declines. *Oecologia*, **133**, 325–333.

Vredenburg, V.T., Knapp, R.A., Tunstall, T.S. & Briggs, C.J. (2010) Dynamics of an emerging disease drive large-scale amphibian population extinctions. *Proceedings of the National Academy of Sciences, USA*, **107**, 9689–9694.

Walker, S.F., Bosch, J., Gomez, V., Garner, T.W.J., Cunningham, A.A., Schmeller, D.S., Ninyerola, M., Henk, D.A., Ginestet, C., Arthur, C.-P. & Fisher, M.C. (2010) Factors driving pathogenicity vs. prevalence of amphibian panzootic chytridiomycosis in Iberia. *Ecology Letters*, **13**, 372–382.

White, G.C. & Burnham, K.P. (1999) Program MARK: survival estimation from populations of marked animals. *Bird Study*, **46**(Suppl.), 120–130.

Wibbelt, G., Kurth, A., Hellmann, D., Weishaar, M., Barlow, A., Veith, M., Pruger, J., Gorfol, T., Grosche, L., Bontadina, F., Sophel, U., Seidl, H., Cryan, P.M. & Blehert, D.S. (2010) White-nose syndrome fungus (*Geomyces destructans*) in bats, Europe. *Emerging Infectious Diseases*, **16**, 1237–1242.

Williams, B.K., Nichols, J.D. & Conroy, M.J. (2002) *Analysis and Management of Animal Populations*. Academic Press, San Diego, CA.

Woodhams, D.C., Alford, R.A. & Marantelli, G. (2003) Emerging disease of amphibians cured by elevated body temperature. *Diseases of Aquatic Organisms*, **55**, 65–67.

Woodworth, B.L., Atkinson, C.T., LaPointe, D.A., Hart, P.J., Spiegel, C.S., Tweed, E.J., Henneman, C., LeBrun, J., Denette, T., DeMots, R., Kozar, K.L., Triglia, D., Lease, D., Gregor, A., Smith, T. & Duffy, D. (2005) Host population persistence in the faceoff introduced vector-borne diseases: Hawaii Amakihi and Avian malaria. *Proceedings of the National Academy of Sciences, USA*, **102**, 1531–1536.

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