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Disease as a selective force precluding widespread cannibalism: a case study of an iridovirus of tiger salamanders, *Ambystoma tigrinum*

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ABSTRACT

Question: Do realistic models predict that infectious disease will select for altered life histories? Specifically, under what conditions can trophic disease transmission influence life-history evolution in tiger salamanders by selecting against cannibalistic morphs?

Data: Previous information from laboratory and field studies on *Ambystoma tigrinum nebulosum* populations from the Kaibab Plateau and Mogollon Rim regions of northern Arizona.

Features of model: Differential equation model incorporating ecological, epidemiological, and genetic structure of tiger salamander populations.

Conclusions: The model can replicate observed patterns of density, phenotypic and genotypic frequency of cannibal morphs, but only by assuming very high disease levels. Disease-induced mortality of both aquatic and terrestrial adults is necessary to reduce the genotypic frequency of cannibalism to observed levels. Given the high forces of infection required to reduce genetic propensity towards cannibalism, other life-history trade-offs may also constrain the genotypic frequency of cannibalism in tiger salamanders. More generally, cannibalism and infectious disease may interfere with each other by reducing population densities, limiting disease-induced selection against cannibalism.

Keywords: *Ambystoma tigrinum*, cannibalism, disease, iridovirus, selection.

INTRODUCTION

As ubiquitous drivers of population and community dynamics (McCallum and Dobson, 1995), parasites can also shape host life histories (Galvani, 2003). The presence of lethal parasites should generally select hosts for earlier reproduction and (potentially) earlier senescence

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(Hochberg *et al.*, 1992; Forbes, 1993). General theoretical explorations of host–parasite life-history co-evolution (Castillo-Chavez and Yakubu, 2001; Gandon *et al.*, 2002), studies of parasite life-history evolution (Day, 2003), and studies of the effects of infection on individual hosts (e.g. Guegan *et al.*, 2001; Kristan, 2004) are common. However, empirical or system-specific studies of the evolutionary *effects* of parasites are much rarer and are largely confined to mollusc, insect, and plant hosts (Agnew *et al.*, 2000; Koskela, 2002; Branson, 2003; Fredensborg and Poulin, 2006).

Cannibalism, a behavioural trait that interacts with life history (Elgar and Crespi, 1992; Wildy *et al.*, 1999), is common in ecological communities. However, given its advantages (e.g. availability – conspecifics are often the most abundant prey – and stoichiometric matching – conspecifics all have approximately the same proportion of limiting nutrients, so cannibals gain the benefit of a perfectly balanced diet), cannibalism is arguably not as widespread as these advantages might predict (Pfennig *et al.*, 1991; Denno and Fagan, 2003). Set against these advantages are the disadvantages of preying on a similar-sized individual, the risk of accidentally consuming relatives (reducing inclusive fitness), and the risk of disease transmission among cannibals (Polis, 1981; Pfennig *et al.*, 1991, 1998; Pfennig and Collins, 1993). This last risk has been exemplified in the last few decades by epidemics of transmissible spongiform encephalopathies amplified by cannibalism (e.g. kuru, BSE), although Rudolf and Antonovics (2007) point out that disease is unlikely to be transmitted solely by cannibalism unless cannibals share meals. However, there are few detailed ecological explorations of the role of infectious disease in limiting cannibalism.

Tiger salamanders and iridoviruses are an ideal system for testing hypotheses on the relationship between disease and cannibalism. Tiger salamanders breed annually in fishless ponds (that are often ephemeral) and have a diverse life history that includes phenotypic plasticity in development (Collins, 1981). A hatchling may develop either into a typical larva that feeds on invertebrates or into a cannibal that eats invertebrates but specializes on conspecifics (Collins, 1981). This phenotypic shift to cannibalism is facilitated by high conspecific densities and low heterospecific prey density (Collins and Cheek, 1983; Wildy and Blaustein, 2001). Cannibals have a performance advantage, probably facilitated by large vomerine teeth, for preying on conspecifics (Reilly *et al.*, 1992; Loeb *et al.*, 1994). Cannibalistic larvae eat a wider range of prey than typical larvae (Collins *et al.*, 1993) and can metamorphose earlier, an advantage in ephemeral habitats (Brunkow and Collins, 1996). However, cannibalism carries an enhanced risk of acquiring pathogens from conspecifics (Pfennig *et al.*, 1991, 1998).

Crowding of tiger salamander larvae into small ponds exposes them to a variety of infectious diseases, both bacterial (Worthylake and Hovingh, 1989; Pfennig *et al.*, 1991, 1998) and viral (Brunner *et al.*, 2004; Collins *et al.*, 2004). Bacterial outbreaks were originally thought to be the proximate cause of die-offs; however, the ranavirus, *Ambystoma tigrinum* virus (ATV) (Jancovich *et al.*, 1997, 2001, 2003; Docherty *et al.*, 2003), is likely the primary pathogen causing rapid local die-offs of *A. tigrinum* populations (Brunner *et al.*, 2004; Jancovich *et al.*, 2005). Since the discovery of ATV, bacterial infections are thought to be opportunistic infections of stressed and dying virus-infected salamanders (Jancovich *et al.*, 1997; Brunner *et al.*, 2004; Parris *et al.*, 2005). Thus, we focus here on ranaviruses that have generally been implicated in global amphibian epizootics and may consequently affect the life-history dynamics of a number of amphibian species (Chinchar, 2002; Daszak *et al.*, 2003; Collins *et al.*, 2004).

A negative correlation between disease frequency and cannibal frequency among salamander populations (and subspecies) throughout Arizona suggests that salamander life history may have evolved in response to iridovirus outbreaks. In the San Rafael Valley (*A. t. stebbinsi*), where historically iridoviral epizootics are most common, cannibals are

almost never observed. On the Kaibab Plateau, larval epizootics from bacterial (Pfennig *et al.*, 1991) or viral (Jancovich *et al.*, 1997) pathogens are common and cannibals are rare (<1%). On the Mogollon Plateau in north-central Arizona, cannibals are common (up to 23%) and disease is rare. Finally, in the White Mountains, both cannibals (10%) and disease are found at intermediate frequencies between sites in the Kaibab and Mogollon Plateaus. Experimental data suggest that these patterns may be a result of past selection by ATV against cannibalism. As predicted from the above geographic pattern, laboratory-reared Kaibab Plateau larvae were less likely than Mogollon Plateau larvae to become cannibals when reared in the laboratory, implying a genetic component to geographic differences in cannibal frequencies (Pfennig *et al.*, 1991). In a more recent experiment, significantly fewer salamanders from San Rafael Valley than from the White Mountains became cannibalistic (Parris *et al.*, 2005). However, despite overall differences in cannibal frequencies between regions, there were no differences within regions in percent cannibals of control versus virus-exposed salamanders (Parris *et al.*, 2005). Thus, regional variation in cannibalism frequency is a product of genetic differences and not solely of plastic responses to environmental differences.

Here we use a model that combines ecological, epidemiological, and genetic dynamics to explore the hypothesis that the observed geographic variation in cannibal frequency is driven by differing strengths of disease-transmission-induced selection against cannibalism in different areas. To unravel the reasons behind this population-genetic pattern (i.e. to determine whether the phenomenon was consistent with selection or could alternately be explained by genetic drift), we would need much more detailed genetic data on the determinants of the cannibal phenotype and natural history information (e.g. to understand differences in the seasonal abundance of the prey community and the fitness of cannibals under different conditions). However, a model does allow us to ask several key questions about the system. In light of our long-term data about the life history and natural history of the *A. tigrinum*–ATV system, how plausible is the disease selection hypothesis? Under what conditions do pathogen-specific parameters, such as force of infection, result in the range of cannibalism frequencies found in the field? What range of plausible pathogen and host parameter values result in genetic differentiation in cannibal frequencies among populations? To which parameters are the patterns most sensitive?

METHODS

Description of the model and parameter values

Model structure

We model the deterministic population dynamics of state variables that track life-history stage, cannibalism status (i.e. non-cannibal or cannibal morph), disease status, and genotype in continuous time. We implement a standard ordinary differential equation (ODE) model, with the exception that we run the model only during the active larval growth season (150 days per year based on data from tiger salamander subspecies in Arizona; the breeding season is set to the first 15 days of the growing season) and allow the model to jump from the end of one growing season to the beginning of the next [allowing for overwintering mortality (Dugaw *et al.*, 2004)]. Table 1 shows a complete list of parameters and default values: the Appendix gives details of the numerical methods and parameter estimates.

Table 1. Parameter definition and default values (parameters fitted in the sensitivity analysis are in italics)

Parameter	Symbol	Baseline value (units)	Meaning
growth0	α_0	0.35 (mm/day)	Baseline growth rate
growth1	α_1	0.0146 (mm/day/prey/m^3)	Dependence of growth rate on heterospecific prey density
growth2	α_2	0.085 (mm/day/prey/m^3)	Dependence of growth on cannibalism rate
matsize	S_m	40 (mm)	Size at maturation
mort1	μ_l	0.029 (/day)	Larval mortality rate
mort2	μ_a	0.002 (/day)	Aquatic adult (cannibal and non-cannibal) mortality rate
mort3	μ_t	0.0074 (/day)	Terrestrial adult mortality rate
mort4	μ_{cl}	0.005 (/day)	Cannibal larvae mortality rate
cannib	c_1	1 (/day/(uninf, noncannib. larvae/m^3))	Per capita predation rate on uninfected larvae
caninf	c_2	3 (/day)	Proportional per capita predation rate on infected larvae
canhand	c_h	1 (days)	Handling time of conspecific prey (cannibals)
candev0	c_0	0	Base rate of transition to cannibalism
canprey	c_4	1/24 (/prey/m^3)	Decrease in cannibalism development with heterospecific prey density
cancon	c_c	1/conspecific larvae/m^3	Increase in cannibalism development with conspecific prey density
canscale	c_{max}	0.01(/day)	Maximum rate of cannibalism development
candom	d	1.0	Dominance of cannibal allele
eggrate1	e_1	10 (/day breeding season)	Per capita larval production of terrestrial adults
eggrate2	e_2	1	Proportional per capita larval production of aquatic adults, relative to terrestrial adults
move	m	0.001–0.05 (/year)	Annual probability of movement between sites
foi	Λ	0.08 (/day)	Force of infection: infection probability per day
cantrans	c_t	1	Infection probability of a cannibal that eats an infected conspecific
dismort1	M_1	0.021(/day)	Disease-induced death rate of larvae
dismort2	M_2	0.021(/day)	Disease-induced death rate of aquatic adults
dismort3	M_3	0 (/day) (initially)	Disease-induced death rate of terrestrial adults
terrprob	p_t	0.95	Probability that maturing larvae become terrestrial
breedlen		15 (days)	Length of the breeding season
seaslen		150 (days)	Length of the growing season
preydens	d_p,$P(S)$	24 (ind/m^3)	Density of conspecific prey, per m^3

Life history

The basic life cycle incorporated in the model includes larvae, aquatic adults – also referred to in the literature as branchiates or gilled adults (Collins and Cheek, 1983; Brunkow and Collins, 1996, 1998; Maret and Collins, 1997; Whiteman *et al.*, 2003) – and terrestrial adults (Fig. 1). The egg stage is implicit: adults produce a fixed number of larvae per capita at the beginning of each season.

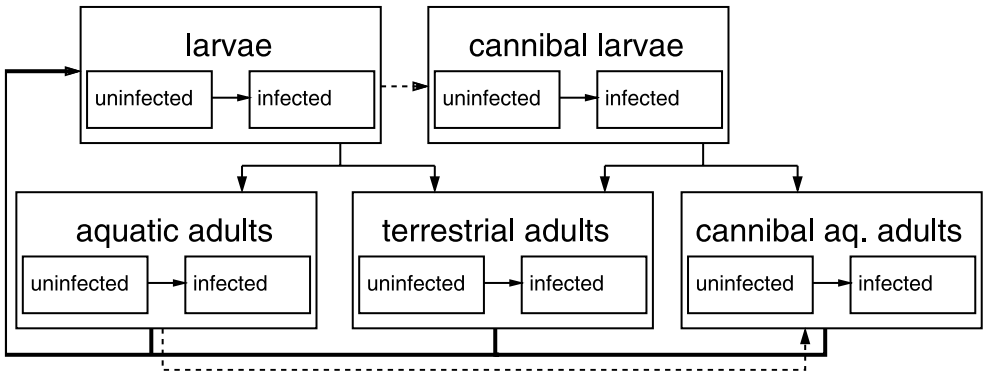

Fig. 1. Flowchart of the model. Thick lines represent reproduction, thin lines represent life-history transitions, dotted lines indicate transition to cannibalism. Infection carries over through life-history and cannibalism transitions (e.g. an infected larva transforms into an infected cannibal larva).

The model tracks the densities of (non-cannibal) larvae, cannibal larvae, (non-cannibal) aquatic adults, cannibal aquatic adults, and terrestrial adults. All classes are susceptible to disease.

Larvae mature at a rate inversely proportional to their growth rate, which is in turn a linear function of heterospecific prey density (Collins *et al.*, 1993; Brunkow and Collins, 1996) and (for cannibals) the rate of consumption of conspecifics. The model assumes that the time spent as a larva is exponentially distributed, with the mean development time equal to the time it would take to grow linearly from 0 to the typical size at maturity [set at 40 mm (Lannoo *et al.*, 1989; Whiteman *et al.*, 2003)]. Cannibals thus develop faster the more conspecifics they eat (Reilly *et al.*, 1992). Any larva that fails to mature by the end of the growing season dies and is removed from the model (this assumption does not qualitatively affect our results).

'Natural' mortality rates (i.e. from causes other than cannibalism and disease) are constant over time but differ among life-history classes. Because of their larger size, aquatic adults survive slightly better than cannibal larvae during the growing season. Mortality of terrestrial adults is difficult to estimate, but over the full year it must be much lower than that of larvae, and because of the risks of migration (e.g. due to predators) we assume it is slightly higher than that of aquatic adults.

Aquatic and terrestrial adults produce larvae at a constant rate over the course of the breeding season (the first 15 days of the growing season). Fecundity is density-dependent, a decreasing logistic function of combined adult density. Newly produced larvae are assumed to be non-cannibals and free of infection, as there is currently no evidence of vertical transmission of ATV (Brunner *et al.*, 2004). Larval genotype distributions follow Hardy-Weinberg proportions based on the genotypes of all adults present (after migration) at a site.

When an individual matures, it 'chooses' whether to become an aquatic or a terrestrial adult with a constant probability (set to P(terrestrial) = 0.95 by default), independent of growth trajectory, cannibal status, and prevailing conditions. This parameter mostly influences the distribution of adults among life-history stages. Since all classes can become infected, the proportion of terrestrial adults does not strongly influence the effects of disease on population dynamics.

Cannibalism

The rate at which individuals develop into the cannibal morph depends on conspecific prey density – and potentially on heterospecific prey density as well (Collins and Cheek, 1983; Maret and Collins, 1994, 1997; Brunkow and Collins, 1996, 1998; Whiteman *et al.*, 2003) – although the model assumes a constant heterospecific prey density of 24 individuals per cubic metre (E. Harvey, unpublished). The transition to cannibalism occurs at a rate proportional to a logistic function of heterospecific prey and conspecific prey densities, and a genetic factor ($g = 0$ for recessive homozygotes, 1 for dominant homozygotes, and an intermediate value for heterozygotes: see 'Genetics' below). Thus the equation for the per capita rate of transition to cannibalism is

$$g \cdot c_{\mathbf{max}} \cdot \exp(c_0 + c_c \cdot \text{density})/(1 + \exp(c_0 + c_c \cdot \text{density})), \quad (1)$$

where $c_{\mathbf{max}}$ is the maximum rate of transition, c_0 governs the rate when the population is sparse, and c_c governs the dependence on conspecific density. Cannibal larvae consume only non-cannibal larvae, while cannibal adults consume both cannibal and non-cannibal larvae. Because cannibal larvae have a higher growth rate than non-cannibal larvae (the difference being proportional to the cannibalism rate), they are more likely to reach maturity. Non-cannibalistic adults cannot become cannibals.

Cannibals attack, kill, and eat conspecifics according to a Holling type II functional response with a handling time of 1 day (Lannoo *et al.*, 1989). They have different attack rates (although not handling times) for uninfected and infected conspecifics, and so their actual predation rates on uninfected and infected conspecifics depends on a weighted average of the densities of these two classes. While in general cannibals could preferentially attack either uninfected or infected prey (and the selective argument we make here would suggest the evolution of a preference for uninfected prey), the balance between the ease of predation on moribund conspecifics and avoidance of disease in the field is unknown. In laboratory experiments with bacterial wasting disease, cannibals ate twice as many infected as uninfected conspecific larvae (Pfennig *et al.*, 1999). In contrast, Pfennig and colleagues (1998) interpreted cannibals' preference for healthy heterospecific prey – rather than infected conspecific prey that would transmit bacterial disease more effectively if infected – as a disease-avoidance mechanism. The effects of ATV on prey preference are unknown. However, cannibals that eat infected conspecifics are highly likely to contract ATV (Jancovich *et al.*, 1997, 2001) (see below).

Genetics

Data supporting the genetic basis of cannibalism come from two common-garden experiments (Pfennig *et al.*, 1991; Parris *et al.*, 2005). In the absence of any information on the genetic architecture underlying cannibalism, we assume that the ability to develop into a cannibal is based on a one-locus, two-allele Mendelian genetic system. We further assume that salamanders mate at random within sites, leading to newborn larvae that are in Hardy-Weinberg proportions based on adult genetics in each site after movement. We assume also that the cannibal allele is dominant. We have also implemented and run a haploid genetic system for the ability to develop into a cannibal.

Infection dynamics

Disease is modelled as a constant, external force of infection (probability per day of becoming infected), where the concentration of the virus in the environment remains

constant rather than increasing as more individuals become infected. Infected larvae can still go through standard life-history transitions (metamorphosing or turning into cannibals), but they die at a higher rate than the background due to disease.

Non-cannibal adults acquire the infection from infected adults (cannibal or otherwise), while cannibal adults can also become infected trophically, at a rate proportional to their consumption of infected conspecifics. Infected individuals remain infected until death, and we assume that there is substantial disease-induced mortality. The default model assumes perfect transmission of the disease – every cannibal that eats an infected conspecific becomes infected. This value is based on observed 100% transmission through injection and a 50% transmission by simply touching a sick individual (Brunner *et al.*, 2004; J.L. Brunner, unpublished). Disease-induced mortality rates are different for each life-history stage but do not differ by cannibalism status.

Movement

The model allows for the movement of terrestrial adults, who can carry both disease and cannibalism alleles, between otherwise isolated populations (Brunner *et al.*, 2004). The model incorporates the possibility of structured movement among sites on a one-dimensional transect (and the possibility that environmental conditions such as force of infection and heterospecific prey density have gradients along this transect). We coupled five sites along a linear gradient of force of infection from 0 to 0.75, with high-disease and low-disease sites at either end analogous to the Kaibab and Mogollon regions. We then modified the level of movement over several orders of magnitude, from 0.1% to 30% mixing between neighbouring sites per year, to establish how much movement is necessary to drive significant changes in density, frequency of the cannibal morph, and frequency of the cannibal allele. In the model, terrestrial adults move after the end of the growing season, after all disease and cannibalism dynamics have occurred for the season and before breeding begins in the next year.

Parameter estimation

Existing laboratory and field studies provide direct estimates of many of the parameters (see above). Further effort is required to estimate the allele frequencies for the putative Mendelian 'cannibalism locus' and some of the disease-related parameters (Table 2).

Estimate of cannibal allele frequency

We have little detailed information on the genetic basis of the propensity to develop a cannibalistic morphology. Within the framework of our assumptions (a one-locus,

Table 2. Observed values of population parameters (Pfennig *et al.*, 1991; E. Harvey, unpublished data)

	Cannibal frequency			Salamander density (ind./m^3)		
Location	mean	range	standard error	mean	range	standard error
Mogollon Plateau	11.7	0–22.8	3.3	27.2	1.6–75.3	11.5
Kaibab Plateau	0.7	0–5.6	0.7	14.4	0.4–42.6	7.0
Grand Canyon Nat. Park	3.6	0–3.6	0.3	15.2	–	4.7

two-allele Mendelian system), however, we can estimate the difference in the propensity towards cannibalism in the two (Kaibab and Mogollon) populations, and ascribe it to a difference in gene frequencies. Assuming that the 'cannibal allele' is fixed or nearly fixed in the Mogollon Rim (low-disease, high-cannibalism) population, the estimated relative propensity to cannibalism in the Kaibab population is equivalent to the allele frequency in that population. We used the aggregated data shown in Figure 2 of Pfennig *et al.* (1991) to find the least-squares fit, weighted by the estimated binomial variance ($1/(np(1-p))$), to a logistic function with different maximum values for each population (Fig. 2). (Because the data were aggregated, we could not fit a standard binomial or quasi-binomial generalized linear model.) We estimated the relative propensity of becoming a cannibal, and hence the frequency of the cannibal allele in the Kaibab population, as 0.29; the other parameters of the fit (which must be interpreted in terms of laboratory larval densities per 20-litre tank) were $c_0 = -16.7$, $c_c = 5.91$, and $c_{max} = 0.00996$ (see equation 1).

Calibration

For those parameters about which we do not have direct information or where the applicability of laboratory estimates to the field is uncertain, we calibrated the model parameters to fit the field-observed values of salamander density (individuals per m^3) and fraction of cannibal alleles (%). Pfennig *et al.* (1991) provide field estimates of both variables in a set of lakes from the Mogollon Rim (low disease, high cannibalism) and a set of lakes from the Kaibab Plateau (high disease, low cannibalism). We use the estimates from both groups of lakes to fit the model parameters.

We first performed a limited sensitivity test of the model, keeping fixed those parameters estimated from field data or laboratory experiments, and varying baseline fecundity and the maximum rate of cannibalism development across reasonable ranges to obtain values of density of aquatic individuals and fraction of cannibals as close as possible to the observed values. We then used these values of baseline fecundity and cannibalism rate as starting values to find the best estimates of these two parameters and others that determine the development of cannibals and their effect on the populations, namely the baseline rate of cannibal development, logistic dependence of cannibal development on conspecific density, attack rate of cannibals on conspecifics, and the relative attack rate of cannibals on infected conspecifics.

Movement

The values of migration probability (proportional exchange between neighbouring sites per year) range from 0.001 to 0.3; these likely correspond to realistic salamander migration rates, which are generally low (Semlitsch and Bodie, 2003).

RESULTS

Fecundity and cannibalism

The values obtained from the calibration of fecundity, cannibal development, and the effect of cannibalism on the population closely match the observed values from field studies (Table 3); the parameter values required to achieve this calibration are reasonable. The maximum rate of transformation into cannibal is 2.2% per day, the attack rate of cannibals on conspecifics is 0.59 individual per day, and fecundity is 11 larvae per adult per day.

Table 3. Parameter values from the optimization routine

Parameter	Value	Variable	Field value	Simulated value
No disease (Mogollon)				
eggrate1	11.37600	Population density	27.2	27.2145
canscale	0.02210	Percentage of phenotypic cannibals	11.7	11.7257
cannib	0.59280	Percentage of genotypic cannibals	1?	0.9968
cancon	0.99610			
candev0	–0.00069			
With disease (0 < dismort < 1: Kaibab)				
dismort1	1.00000	Population density	14.4	13.6543
dismort2	1.00000	Percentage of phenotypic cannibals	0.7	1.9754
dismort3	1.00000	Percentage of genotypic cannibals	0.29 (estimated)	0.7015
foi	0.63390			
With disease (0 < dismort < ∞: Kaibab)				
dismort1	1.97	Population density	14.4	14.4731
dismort2	0.45050	Percentage of phenotypic cannibals	0.7	0.7104
dismort3	0.66880	Percentage of genotypic cannibals	0.29 (estimated)	0.2901
foi	0.77680			

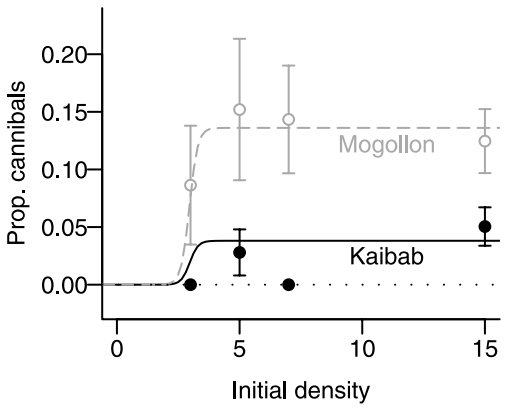

Fig. 2. Observed proportions of cannibal-morph larvae from two locations in Arizona after 14 days of laboratory rearing, and the fit of a logistic model (data from Pfennig *et al.*, 1991).

Disease

Introduction of disease reduces both population density and fraction of cannibal morphs (Figs. 3 and 4). As a first approximation, we assumed that terrestrial adults were not affected by the disease. Under this assumption, only moderate forces of infection and disease mortalities are required to reduce the density and *phenotypic* incidence of cannibalism. For example, total population density decreases from approximately 20 to 7 for forces of infection of approximately 0.2 as disease-induced mortality rates increase

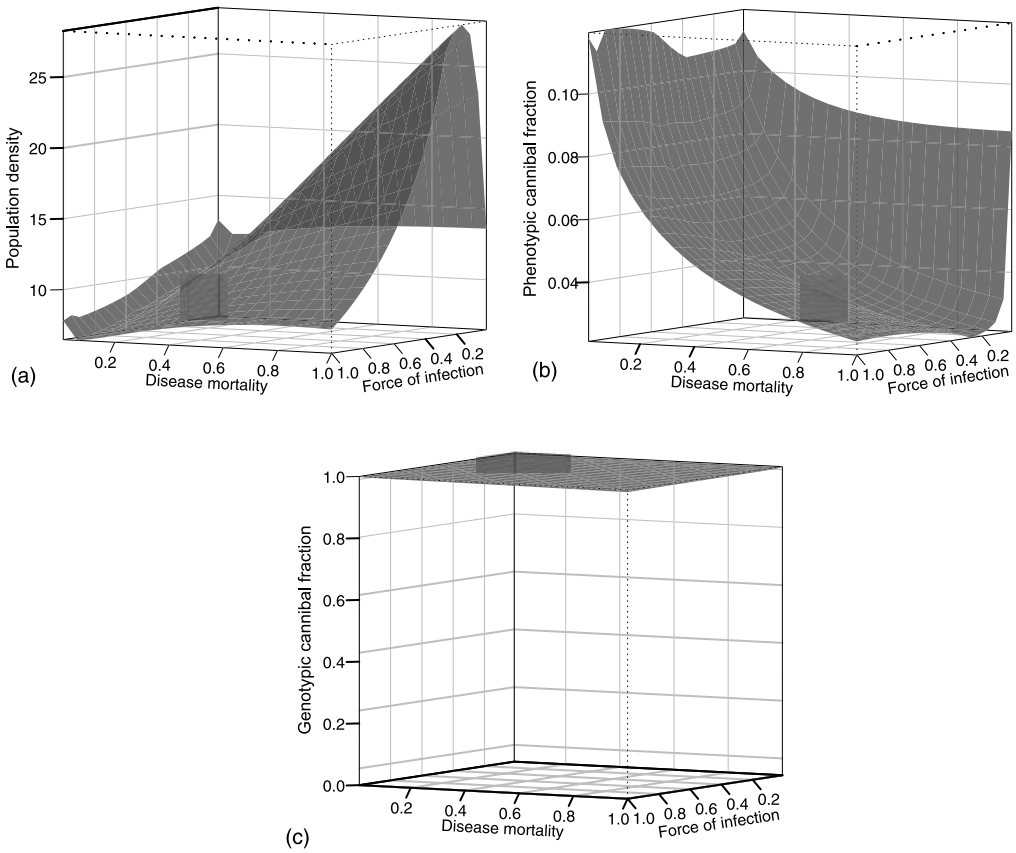

Fig. 3. Effect of disease on total population density (a), phenotypic cannibal fraction (b), and genotypic cannibal fraction (c), assuming no disease-induced mortality for terrestrial adults.

from 0 to 1 (Fig. 3a), while cannibal morphs drop sharply from 10% to 2% as soon as either disease-induced mortality rates or force of infection increase above their minimum values (Fig. 3b). At high forces of infection and low levels of disease-induced mortality, increasing disease-induced mortality *increases* the equilibrium population density, by preferentially removing cannibals from the population. Parameter combinations do exist that can significantly decrease the fraction of the population carrying cannibal alleles – but only when the forces of infection are so high as to cause the localized extinction of a whole population. For values of larval and aquatic adult disease-induced mortality rates, and forces of infection, up to 1 per day, the frequency of the cannibal allele remains above 0.98 (Fig. 3C).

On the other hand, by allowing disease to affect terrestrial adults, which is realistic (Brunner *et al.*, 2004), the level of disease required to reduce the fraction of cannibal alleles is much lower (Fig. 4). Figures 4a and 4b, which show the effects of disease-induced mortality and force of infection on total population density and fraction of phenotypic cannibals when adults are affected by disease, are qualitatively similar to Figs. 3a and 3b. In contrast to Fig. 3c, Fig. 4c shows that moderately high forces of infection (see Discussion), in combination

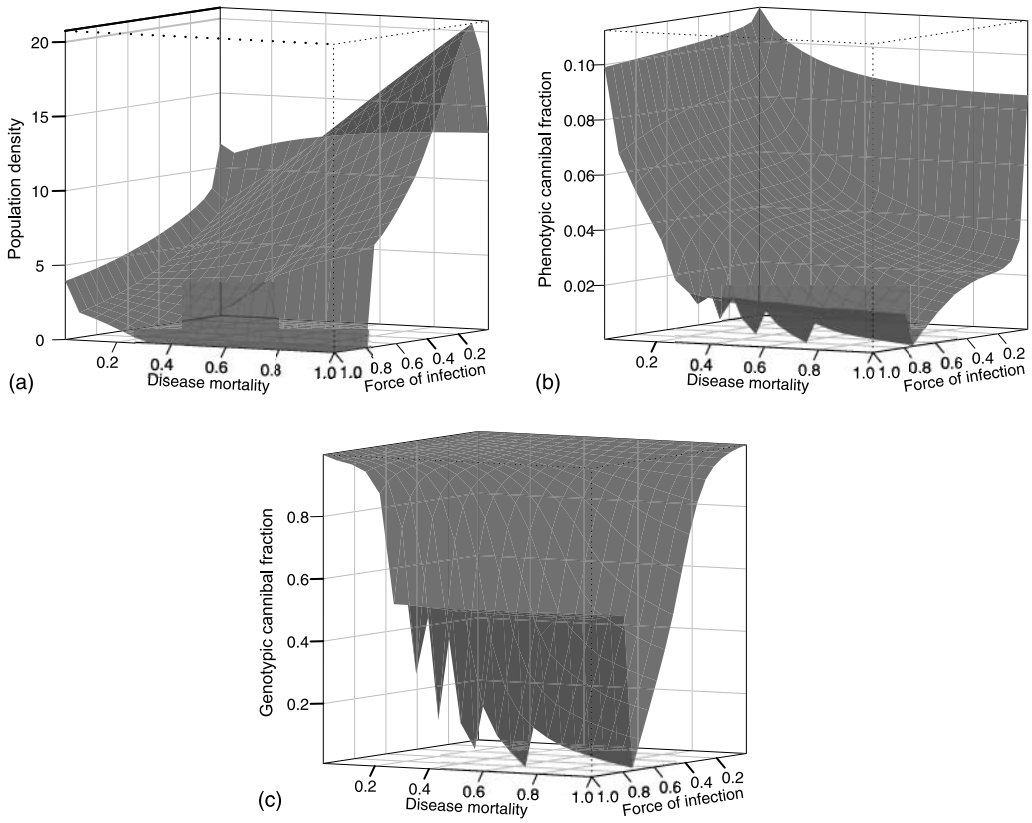

Fig. 4. Effect of disease on total population density (a), phenotypic cannibal fraction (b), and genotypic cannibal fraction (c). Disease-induced mortality rates are assumed equal for all life-history stages.

with high disease-induced mortality rates for all life-history stages, can indeed reduce the cannibal allele frequency from near 100% down to levels of approximately 20%.

Movement

The results of the simulations including several populations along a geographic gradient in force of infection (Fig. 5) suggest that very little migration among locations is required to homogenize the population and fix the cannibal allele everywhere. At the zero-infection site, the cannibal allele always fixed and the proportion of phenotypic cannibals remained around 13%. At all other sites, the effects of disease on the population dynamics decreased the proportion of phenotypic cannibals to about 3%, but the phenotypic proportion did not drop further at high-infection sites unless movement rates were extremely low (<0.003). The fraction of cannibal alleles showed a similar pattern: the cannibal allele fixed everywhere unless $m < 0.01$, showed a very slight decline at high-infection sites if $m = 0.003$, and only declined substantially at high-infection sites if $m < 0.003$. Essentially, the difficulty of selecting against cannibal alleles along with the high population density source in the low-infection sites guarantees in this case that individuals with the propensity to become cannibals will persist everywhere.

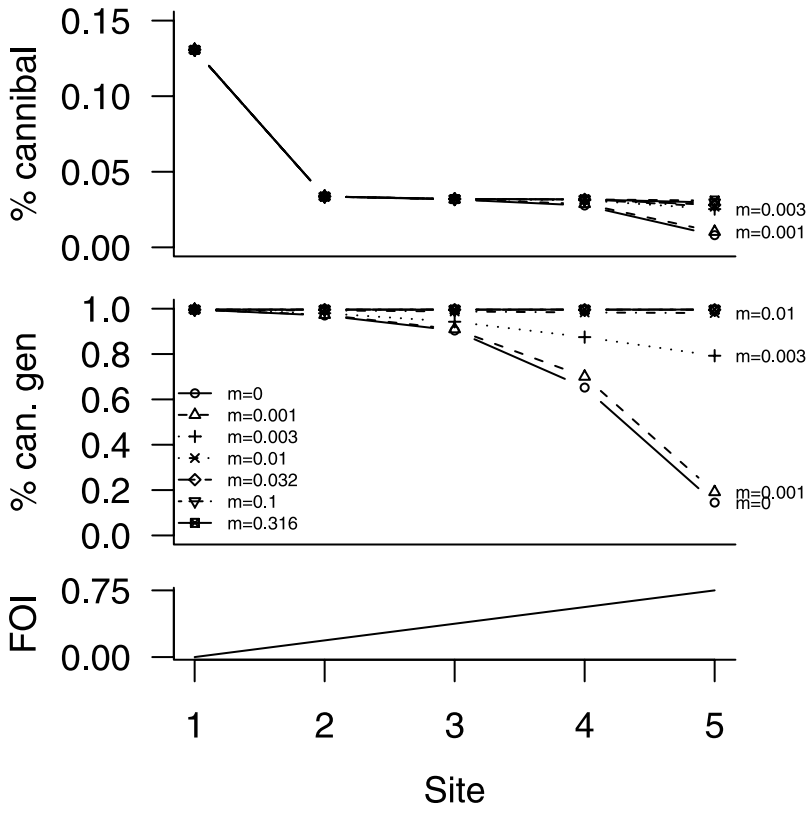

Fig. 5. Phenotypic and genotypic fractions of cannibals at different points along a spatial gradient in force of infection (FOI), for different movement rates ranging from 0 to 0.316 (probability of movement per year). Except at the zero-infection site (FOI = 0), phenotypic and genotypic cannibal fractions converge on a constant equilibrium unless movement is less than 0.003.

DISCUSSION

We gained four main insights from modelling disease-induced selection: (1) we can replicate observed patterns of density, phenotypic and genotypic cannibalism from northern Arizona by manipulating disease frequency; (2) disease-induced mortality on adults is necessary to select against cannibalism; (3) very high forces of infection are necessary to have strong negative effects on cannibal allele frequencies; and (4) very low migration results in fixation of the cannibal allele in a metapopulation structure. Disease frequency must remain high to reproduce natural patterns. This constraint is realistic, given that disease is consistently present on the Kaibab Plateau where long-term monitoring has occurred (Brunner *et al.*, 2004; A. Storfer, unpublished data; A. Greer, unpublished data) and epizootics from ranaviruses can be extreme, sometimes leading to apparent localized larval extirpation within a breeding season (Jancovich *et al.*, 1997, 2005; Brunner *et al.*, 2004; Collins *et al.*, 2004).

Values of tuned model parameters

It was easy to calibrate our model by adjusting cannibalism development rate and fecundity to mimic the observed overall and (phenotypic) cannibal densities on the Mogollon Rim. The estimated fecundity of 11.7 translates into approximately 170 larvae per adult per season, which is plausible given that adults can lay at least 1000 eggs per season (Petranka, 1998), unadjusted for post-hatching mortality. The maximum cannibalism development rate is estimated as 0.022 per individual per day, close to our baseline value of 0.01 and the estimate of 0.00995 from the common-garden experiment (note that these values are inferred rates per day, not overall frequencies of the cannibal morph).

Once we allowed adults to die from disease, bringing genotypic and phenotypic cannibal frequencies down to observed levels while maintaining overall populations at reasonable densities was also straightforward. When we capped disease-induced mortality at 1 per day (this is an exponential rate, rather than a probability, corresponding to an average survival time of 1 day with many individuals dying faster or slower rather than 100% mortality in 1 day), we could bring the phenotypic cannibal frequency within reasonable bounds (0.7% vs. 2% observed, with a wide range), but the cannibal genotype could only be brought down to 70%. We could have explored genetic models other than our simple one-locus, two-allele Mendelian system, but we had already tried to choose the genetic system that would make the cannibal allele most sensitive to selection by disease. For example, making the cannibalism allele additive or recessive, as we did during our initial model development, made it less rather than more sensitive to disease. Among the other possible variations in the genetic model (e.g. incorporating drift, multiple loci or quantative traits), strong epistasis – which can drastically change the strength of selection for sexual reproduction (Kondrashov, 1994) – is the most likely candidate for strengthening disease-induced selection, but in the absence of any information on the genetics of cannibalism we chose not to experiment with such a complicated model.

Allowing more rapid disease-induced mortality for all life-history stages brought the simulated values of density and cannibal frequencies to near-perfect agreement with field observations, and led to lower mortality rates for adults while doubling the larval mortality rates to approximately 2 per day (average survival time of half a day). Based on laboratory experiments, the required values of disease-induced mortality are unrealistically high. However, we note that: (1) observed population die-offs due to iridovirus are rapid, with most individuals having died within 2–3 weeks based on laboratory experiments (Jancovich *et al.*, 1997, 2001; Brunner *et al.*, 2004; Forson and Storfer, 2006a, 2006b); (2) the low values used as our initial estimates are derived from bacterial diseases [experimental data from Pfennig *et al.* (1998) suggest a mortality rate due to disease of 0.011 per day]; and (3) estimates for iridovirus survival in laboratory experiments, while they may represent very high initial doses, also represent mortality in the absence of various stressors found in the field [e.g. temperature (Rojas *et al.*, 2005; Bosch and Martinez-Solano, 2006; Pounds *et al.*, 2006) or pesticides (Forson and Storfer, 2006a, 2006b)].

Force of infection was the most difficult variable to test for sensitivity. The model suggests that forces of infection of around 0.6–0.7 (randomly chosen individuals become infected in less than 2 days on average) are necessary to reduce the genotypic and phenotypic frequencies of cannibalism, in contrast to our starting estimate of 0.02. Disease and die-off rates are the natural phenomena for which we have the poorest data, with both pond observations and die-off observations occurring sporadically. Our model also assumes a

constant force of infection while disease outbreaks are in fact density-dependent, becoming intense shortly after peak larval densities are achieved in mid- to late breeding season (Brunner *et al.*, 2004). We do not know what frequency of severe disease outbreaks would produce selection against cannibalism of the same magnitude as a constant force of infection of 0.6. Nevertheless, the large force of infection required to bring down the frequency of the cannibal allele suggests that disease frequency is probably not a complete explanation of the observed patterns, and that other explanations (e.g. involving abiotic conditions, population size structure, or heterospecific prey availability) are still plausible. Thus, ATV alone is likely not sufficient to explain differences in cannibalism rates among regions in Arizona.

We have taken the observed phenotypic and genotypic correlations between ATV and cannibalism as evidence that ATV may select against cannibalism. Since our model gives at best weak support for a causal link in this direction, future studies should consider alternative hypotheses. For simplicity in testing the hypothesis that ATV drives cannibalism, our model assumed (1) similar abiotic and biotic conditions (other than disease prevalence) across environments, and (2) a constant force of infection from disease. In reality, of course, variation in biotic factors such as prey availability will generate differences in conspecific density, which could in turn select for different levels of cannibalism. At the simplest level, one might expect that increased density would drive increases in both cannibalism and disease, leading to a *positive* correlation. However, if we allowed disease to vary dynamically – and especially if we incorporated seasonal dynamics in outbreak probability interacting with seasonal variation in size and the size hierarchy – we might see how increases in cannibalism levels driven by prey availability or other factors could decrease the force of infection, reversing the arrow of causality from our study.

Need for terrestrial adult mortality

We initially assumed that terrestrial adults were a small part of the evolutionary process within a pond, and that their main evolutionary function in the disease system was to carry genes and possibly act as intraspecific disease reservoirs within and between different sites (Brunner *et al.*, 2004). To our surprise, disease-induced mortality of terrestrial adults is necessary for disease-induced selection against the cannibal genotype (Fig. 6). Assuming that adults die from disease is reasonable. Catastrophic die-offs in terrestrial adults have been observed in Utah (Worthylake and Hovingh, 1989), where the presence of iridovirus has since been confirmed (Jancovich *et al.*, 2005). Without allowing terrestrial adults to die from disease (even with very high mortality of aquatic adults), the terrestrial adults can apparently act as a reservoir for both disease (Brunner *et al.*, 2004) and the cannibal allele in the population. Brunner and colleagues (2004) suggested that terrestrial adults may act as an ontogenetic reservoir for disease, carrying disease between ponds and allowing persistence of disease across years within a pond after localized host extirpation and consequent pathogen extinction. This finding represents one more example where the terrestrial life stage of amphibians, which is often understudied relative to the aquatic stages that are far more convenient to sample in the field and experiment on in the laboratory, turns out to be surprisingly important in population dynamics [for example, Vonesh and de la Cruz (2002) pointed out that density-dependence in larval stages makes amphibian populations less sensitive to changes in egg mortality].

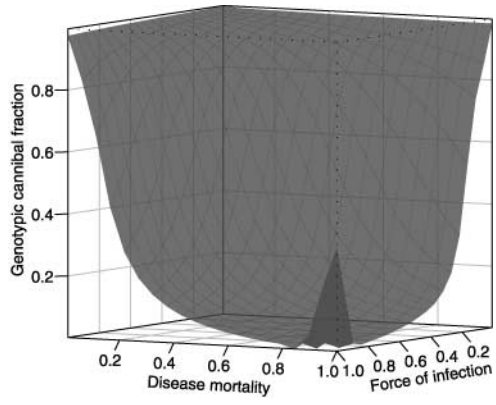

Fig. 6. Genotypic fraction of cannibals (gfrac) as a function of larval/aquatic adult and terrestrial adult mortality (force of infection = 0.75). Genotypic fraction of cannibals does not decrease, even for large values of larval and aquatic adult mortality, unless there is some degree of mortality of terrestrial adults. [Peak at (1, 1) represents a collapsed population where genotype fraction is poorly defined.] This is a revised figure with a corrected label for the y axis.

Movement

The model suggests that very little migration between different sub-populations is required to quickly fix the cannibal allele in a meta-population structure (Fig. 5). This counter-intuitive effect is influenced by the proportion of adults that become terrestrial, since they are the only stage that can migrate between ponds. Since we allowed terrestrial adults to be affected by the disease, they could also act as vectors and transmit the virus between locations.

CONCLUSION

While we were able to calibrate the model to replicate observed patterns of population density, cannibal morphs, and cannibal alleles, we needed to introduce extremely high forces of infection to regulate the genetic frequency of cannibalism. In retrospect, this result is not surprising, since cannibalism responds plastically to conspecific density. Under conditions of high disease-induced mortality, population densities decrease (Fig. 4a), the cannibal phenotype remains unexpressed even in genetic cannibals (Fig. 2), and selection becomes powerless to remove the cannibal genotype from the population. The need for a high force of infection supports the general phenomenon of plasticity impeding the evolution of specialization, but it is a special case based on the particular interaction among disease, population density, and cannibalism in the tiger salamander–ATV system.

More generally, our results suggest that cannibalism and infectious disease may interact less strongly, or at least less directly, than previously suggested. Even in the absence of the extreme plasticity displayed by tiger salamanders, cannibalistic behaviour and morphology are likely to be plastic in the sense that they will decline as densities decrease. Transmission and prevalence of infectious disease are also likely to be density-dependent (although they are not in our model), and both cannibalism and disease tend to reduce population density. Thus cannibalism and infectious disease would tend to exclude each other from populations

through ecological, rather than evolutionary, mechanisms. Rather than the presence of infectious disease directly selecting against cannibalism, it may simply regulate the population size to levels where cannibalism is inefficient. Ecologists have begun to realize that the interactions between predation and disease are ubiquitous (Packer *et al.*, 2003; Duffy *et al.*, 2005; Hall *et al.*, 2007) and often non-intuitive [for example, Holt and Roy (2007) show that predation can sometimes *increase* the prevalence of infection]. Cannibalism is a special case where the 'predators' are always present.

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APPENDIX

Parameter estimates

Larval growth

Default value of 0.35 mm/day is based on the observed mean growth rate at a macroinvertebrate density of 24/m^3 (E. Harvey, unpublished). The default enhancement of cannibal growth per conspecific consumed is 0.085 mm/day/conspecific, based on an observed 0.52 mm/day growth rate for cannibals and an average conspecific consumption rate of 2/day/cannibal at experimental densities.

Natural mortality

Based on exponential estimates from a mark–recapture study (Collins and Cheek 1983), the default values of death rates of larvae, aquatic adults, terrestrial adults, and cannibal larvae are 0.029, 0.002, 0.01, and 0.02 respectively.

Fecundity

The default value of 10 larvae/adult/day during the 15-day breeding season (leading to a maximum fecundity of 150 larvae/adult/year) incorporates all sources of pre-hatching egg mortality (non-viability, predation, etc.) and was chosen based on natural history. We assume there is no delay between egg-laying and hatching. The density-dependence parameters make fecundity essentially constant over the observed range of adult densities (i.e. a logistic function that remains basically constant up to a density of 30 adults/m^3 and declines rapidly to zero thereafter). This density-dependence is included simply to make the model behave sensibly in parameter ranges where other sources of density-dependence (cannibalism and trophically transmitted disease) are missing. The model structure allows for aquatic adults' fecundity to differ from that of terrestrial adults, but in the absence of field data we set their fecundities equal.

Cannibalism development parameters

The values used in the model for these parameters are: cancon ($c_\mathbf{c}$): 0.5, based on experimental conditions of 2 larvae/m^3; candev0 ($c_\mathbf{0}$): 0, which gives 0.5/day transition rate to cannibalism with zero densities of heterospecific and conspecific prey [Whiteman *et al.* (2003) report a minimum density of 7 individuals/m^3 below which cannibalism does not develop]; canscale ($c_{\mathbf{max}}$): the values of this parameter were estimated through sensitivity analysis (see 'Simulations' below).

Cannibal attack rates

The default values used in the model for attack rate on non-infected (cannib, $c_\mathbf{1}$) and infected conspecifics (caninf, $c_\mathbf{2}$) are 1 and 3 respectively. These values are based on a 2 individuals/day predation rate at experimental densities of 2 larvae/m^3 and on estimates that cannibals ate nearly twice as many infected as infected conspecifics (Pfennig *et al.*, 1999): we initially increased the $c_\mathbf{2}$ parameter in hopes of strengthening the effects of trophic transmission. However, the sensitivity analysis (see below) allowed these values to vary.

Force of infection

The initial value of force of infection (foi) is 0.02, based on observations of five locations across 3 years where approximately 70% of larvae showed symptoms (unpublished results), although the final value obtained in the sensitivity analysis of the model was much higher.

Disease-induced mortality rates

The initial values for these parameters are based on density estimates before and after an outbreak in two locations: approximately 80–90% of those showing symptoms died before the onset of winter (unpublished results). Initial default values were: larvae 0.021, aquatic adults 0.021, terrestrial adults 0, assuming that terrestrial adults, being mostly out of the water, remain uninfected. Results obtained from the test of the model changed this assumption. Pfennig *et al.* (1998) provided experimental estimates of mortality due to what they identified as a different pathogen (*Clostridium*) in *A. tigrinum*; subsequent research suggests the pathogen was likely ATV. In laboratory experiments, 42% of cannibals that ate diseased conspecifics died before metamorphosis (7 weeks). Assuming that all cannibals that ate diseased conspecifics became infected, this would correspond to a daily mortality rate of 0.011.

Parameter calibration

We first ran a set of simulations changing the two main parameters that control population growth and percent of cannibals – baseline fecundity and maximum cannibalism rate – assuming there is no disease present. The baseline fecundity (eggrate1) determines the growth rate of the population, while the maximum rate of cannibalism development (canscale) influences both the percentage of cannibals and also the population growth rate (the density-dependence of fecundity in the model only acts for adult densities higher than those presented here). The results (not shown) indicate that for higher values of maximum cannibalism rate (0.1), the fraction of cannibals is always too high (>20%). For a lower maximum cannibalism rate (0.01), the fraction of cannibals is too low, and the population growth is also too high. At a maximum cannibalism rate of 0.025 and baseline fecundity between 15 and 20, the population density is comparable to the field average (27.1) and the fraction of cannibals is slightly above 10% for most of the range of eggrate1, very close to the field average (11.7). Higher cannibalism rates produce too high a fraction of cannibals and too low densities.

We then use these values of baseline fecundity and cannibalism rate as starting values to find the best estimates of these two parameters and others that determine the development of cannibals and their effect on the populations; namely, the baseline rate of cannibal development (candev0), logistic dependence of cannibal development on conspecific density (cancon), attack rate of cannibals on conspecifics (cannib), and the relative attack rate of cannibals on infected conspecifics (caninf). The optimization procedure allows us to specify both the response variable to be minimized and 'box constraints' (independent minimum and maximum values) on the independent variables. The response variable is the sum of the squared difference between the predicted and observed values of each variable (density, phenotypic fraction of cannibals, and genotypic fraction of cannibals), weighted by the potential range of each variable.

With the values of the above parameters fixed at the best estimates, we then introduce disease, and use the same optimization algorithm to find the best estimates for the

disease-related parameters: disease-induced mortality of larvae, aquatic and terrestrial adults, and force of infection. The response variable is the same as before, but the target values of overall and cannibal densities are those observed in high-disease, low-cannibalism lakes on the Kaibab Plateau (Pfennig *et al.*, 1991). All four parameters are constrained to be positive.

Simulation methods and model equations

We numerically integrated the model for various parameter sets using the LSODA differential equation solver built into the odesolve package in R (R Development Core Team, 2005).

The simulations start at the beginning of the growing season. For convenience, we assume that terrestrial adults move and lay eggs at the beginning of the season (although in reality this corresponds to moving, mating, and laying eggs anywhere between the end of the previous season and the beginning of the present one):

$$T(G, S, 0) = (1 - m)T(G, S, \tau) + \sum_{i \in N} \frac{m}{|N|} T(G, i, \tau)$$

where $T(G, S, t)$ is the density of terrestrial adults of genotype G at site S and time t, m is the movement probability, τ represents the end of the growing season, N is the set of neighbouring sites, and $|N|$ is the number of neighbouring sites. In general, the full set of indices for a state variable is (I, G, S, t) referring to infection status 0 (uninfected) or 1 (infected), genotype (cc, cC, or CC), site (1 to N), and time within the season. Where indices are suppressed, it means that we are summing over categories: for example, $L(0, S, t)$ would refer to uninfected larvae of all genotypes, while $L(G, S, t)$ would refer to larvae of a particular genotype regardless of status.

Aquatic adults survive perfectly between seasons:

$$A(I, G, S, 0) = A(I, G, S, \tau)$$

$$CA(I, G, S, 0) = CA(I, G, S, \tau)$$

where $A(I, G, S, t)$ is the density of (non-cannibal) aquatic adults of infection status I and genotype G in site S at time t; and $CA(I, G, S, t)$ is the same for cannibalistic aquatic adults. All larvae die if they fail to metamorphose by the end of the season.

Larvae are generated in Hardy-Weinberg proportions according to the adults present (after movement) at a site. For simplicity, we say that eggs hatch at a constant rate over the breeding season (the initial 15 days of the growing season), and that e incorporates whatever kinds of egg mortality (non-viability, predation, etc.) happen before hatching, so this number of eggs translates directly into larvae at the beginning of the season. All larvae are uninfected and non-cannibalistic at birth.

$$L(0, G, S, 0) = e \cdot HW(A(G, S, 0) + CA(G, S, 0) + T(G, S, 0))$$

$$L(1, G, S, 0) = C(I, G, S, 0) = 0$$

Here, I denotes arbitrary infection status: 0 refers to uninfected and 1 to infected individuals.

Non-cannibalistic, non-infected larvae can leave this category by: (1) dying from 'background' mortality (desiccation, predation other than by cannibals, etc.); (2) being infected; (3) being eaten by a cannibal; (4) changing into a cannibal; (5) metamorphosing into an adult (aquatic adult or terrestrial).

Because cannibal consumption of larvae is governed by Holling type II functional responses, and because the attack rate on infected and uninfected larvae is potentially different, we have to be careful in defining consumption rates. The attack-rate-weighted density of larvae at site S, time t is $W(S, t) = c_1 L(S, 0, t) + c_2 L(S, 1, t)$; it is this weighted density that determines the saturation of cannibal predation. The per-cannibal consumption rate of uninfected larvae is $c_1 L(S, 0, t)/(1 + c_h W(S, t))$, and of infected larvae is $c_2 L(S, 0, t)/(1 + c_h W(S, t))$.

To specify the rate of developing into a cannibal, we define the logistic function $\text{Logist}(x) = e^x/(1 + e^x)$, where x is typically a linear function of some other parameter(s). The intercept of the linear function determines where $\text{Logist}(x) = 0.5$ and the slope(s) determine the effects of the parameters and the steepness of the transition between 0 and 1. The genetic predisposition towards cannibalism, D, is 0 for cc (homozygous non-cannibal) individuals, d for heterozygotes, and 1 for CC (homozygous cannibal) individuals.

$$\frac{dL(0, G, S, t)}{dt} = -L(0, G, S, t)\left(\begin{array}{l}\mu_1 + \Lambda(S) + \dfrac{c_1(C(S, t) + AC(S, t))}{1 + c_h W(S, t)} \\ + D(G)\text{Logist}(c_3 - c_4 P(S) + c_c L(G, s, t)) \\ + \alpha_0 + \alpha_1 P(S)\end{array}\right)$$

where μ_l is the background mortality rate; $\Lambda(S)$ is the force of infection at site S; c_1 is the cannibal attack rate; $P(S)$ is the prey density at site S (held constant across all sites in all of our simulations); and c_3, c_4, and c_c govern the density- and prey-dependent transition to cannibalism. The parameters α_0 and α_1 govern the maturation rate; here, they are implicitly scaled by the size and maturity S_m, to convert from effects on growth rates to effects on rate of maturity.

Infected (non-cannibalistic) larvae are produced by infection; they can in principle metamorphose or turn into cannibals, if they survive, but they die at a much higher rate than the background, and they do get eaten by cannibals.

$$\frac{dL(1, G, S, t)}{dt} = \Lambda(S)L(0, G, S, t) - L(1, G, S, t)\left(\begin{array}{l}\mu_1 + M_1 + \dfrac{c_2(C(S, t) + AC(S, t))}{1 + c_h W(S, t)} \\ + D(G)\text{Logist}(c_3 - c_4 P(S) + c_c L(G, s, t)) \\ + \alpha_0 + \alpha_1 P(S)\end{array}\right)$$

where M_1 is the disease-induced mortality rate of larvae.

Uninfected, cannibalistic larvae are produced by metamorphosis of (uninfected) non-cannibalistic larvae; they do most of the same things, but grow faster (proportional to their consumption of conspecifics), and also get infected (in addition to the baseline rate) proportional to their consumption of conspecifics:

$$\frac{dC(0,\,G,\,S,\,t)}{dt} = L(0,\,G,\,S,\,t) - D(G)\text{Logist}\left(c_3 - c_4P(S) + \frac{c_c(c_1L(0,\,S,\,t) + c_2L(1,\,S,\,t))}{1 + c_hW(S,\,t)}\right) -$$

$$C(0,\,G,\,S,\,t)\left(\begin{array}{l}\mu_1 + \Lambda(S) + \beta\dfrac{c_2L(1,\,S,\,t)}{1 + c_hW(S,\,t)} + \\ \alpha_0 + \alpha_1P(S) + \alpha_2\dfrac{c_1L(0,\,S,\,t) + c_2L(1,\,S,\,t)}{1 + c_hW(S,\,t)}\end{array}\right)$$

Infected cannibal larvae die at the same rate as infected non-cannibals and, like them, they can in principle survive to maturity.

$$\frac{dC(1,\,G,\,S,\,t)}{dt} = (\Lambda(S) + \beta\frac{c_2L(1,\,S,\,t)}{1 + c_hW(S,\,t)}C(0,\,G,\,S,\,t)) -$$

$$C(1,\,G,\,S,\,t)\left(\begin{array}{l}\mu_1 + M_1 \\ + D(G)\text{Logist}\left(c_3 - c_4P(S) + \dfrac{c_c(c_1L(0,\,S,\,t) + c_2L(1,\,S,\,t))}{1 + c_hW(S,\,t)}\right) \\ + \alpha_0 + \alpha_1P(S) + \alpha_2\dfrac{c_1L(0,\,S,\,t) + c_2L(1,\,S,\,t)}{1 + c_hW(S,\,t)}\end{array}\right)$$

The equations for adults are simpler because they neither develop into cannibals nor are they eaten by cannibals. Aquatic adults emerge by maturation (a fraction $(1 - p_t)$ of maturing larvae become aquatic adults), may be infected by environmental or trophic transmission, and die from background and disease-induced mortality.

Non-cannibal aquatic adults:

$$\frac{dA(0,\,G,\,S,\,t)}{dt} = L(0,\,G,\,S,\,t)(1 - p_t)(\alpha_0 + \alpha_1P(S)) - A(0,\,G,\,S,\,t)(\mu_a + \Lambda(S))$$

$$\frac{dA(1,\,G,\,S,\,t)}{dt} = L(1,\,G,\,S,\,t)(1 - p_t)(\alpha_0 + \alpha_1P(S)) + A(0,\,G,\,S,\,t)\Lambda(S) - A(1,\,G,\,S,\,t)(\mu_a + M_2)$$

Cannibal aquatic adults:

$$\frac{dCA(0,\,G,\,S,\,t)}{dt} = C(0,\,G,\,S,\,t)(1 - p_t)\left(\alpha_0 + \alpha_1P(S) + \alpha_2\frac{c_1L(0,\,S,\,t) + c_2L(1,\,S,\,t)}{1 + c_hW(S,\,t)}\right) -$$

$$CA(0,\,G,\,S,\,t)\left(\mu_a + \Lambda(S) + \beta c_2\frac{c_2L(1,\,S,\,t)}{1 + c_hW(S,\,t)}\right)$$

$$\frac{dCA(1,\,G,\,S,\,t)}{dt} = C(1,\,G,\,S,\,t)(1 - p_t)(\alpha_0 + \alpha_1P(S) + \alpha_2(c_1L(0,\,G,\,S,\,t) + c_2L(1,\,G,\,S,\,t))) +$$

$$CA(0,\,G,\,S,\,t)\left(\Lambda(S) + \beta c_2\frac{c_2L(1,\,S,\,t)}{1 + c_hW(S,\,t)}\right) -$$

$$CA(1,\,G,\,S,\,t)(\mu_a + M_2)$$

Terrestrial adults do not acquire infection through trophic transmission nor become cannibals.

$$\frac{dT(I, G, S, t)}{dt} = p_t\Big(\begin{matrix}L(I, G, S, t)(\alpha_0 + \alpha_1 P(S)) + \\ C(I, G, S, t)(\alpha_0 + \alpha_1 P(S) + \alpha_2(c_1 L(0, G, S, t) + c_2 L(1, G, S, t))\end{matrix}\Big) - T(I, G, S, t)(\mu_t + [M_t])$$

where the disease-induced mortality term $[M_t]$ applies, obviously enough, only to diseased individuals ($I = 1$).

Note: R model code is available on request from the first author.