Effects of habitat quality and size on extinction in experimental populations

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Stochastic population theory makes clear predictions about the effects of reproductive potential and carrying capacity on characteristic time-scales of extinction. At the same time, the effects of habitat size and quality on reproduction and regulation have been hotly debated. To trace the causal relationships among these factors, we looked at the effects of habitat size and quality on extinction time in experimental populations of Daphnia magna. Replicate model systems representative of a broad-spectrum consumer foraging on a continuously supplied resource were established under crossed treatments of habitat size (two levels) and habitat quality (three levels) and monitored until eventual extinction of all populations. Using statistically derived estimates of key parameters, we related experimental treatments to persistence time through their effect on carrying capacity and the population growth rate. We found that carrying capacity and the intrinsic rate of increase were each influenced similarly by habitat size and quality, and that carrying capacity and the intrinsic rate of increase were in turn both correlated with time to population extinction. We expected habitat quality to have a greater influence on extinction. However, owing to an unexpected effect of habitat size on reproductive potential, habitat size and quality were similarly important for population persistence. These results support the idea that improving the population growth rate or carrying capacity will reduce extinction risk and demonstrate that both are possible by improving habitat quality or increasing habitat size.

Keywords: carrying capacity; Daphnia magna; demographic stochasticity; extinction; population growth rate

1. INTRODUCTION

Habitat degradation is a major threat facing many populations (Sala et al. 2000). Degradation can come in two forms: decreased habitat size through habitat loss or fragmentation, and decreased habitat quality through loss of resources, pollution or other forms of habitat alteration. In consumer–resource systems, the abundance and nutrition of basal food resources are paradigmatic indicators of habitat quality. It is unknown whether the relative importance of habitat size and quality to persistence depends on system particularities or may be generalizable across consumer–resource systems.

Previous field and laboratory studies have demonstrated the effects of both. For example, habitat fragmentation in an Oklahoma shortgrass prairie reduced habitat size for prairie chickens and increased the risk of population extinction (Patten et al. 2005). By contrast, for collared pikas in alpine meadows in southwest Yukon, it was habitat quality (amount of vegetation), that most influenced extinction (Franken & Hik 2004). Similar results have been observed in laboratory populations: extinction rate was higher in fruitfly populations in smaller habitats than in larger habitats (Forney & Gilpin 1989), and decreased habitat quality via reduced food supply increased population extinction risk in fruitflies (Philippi et al. 1987), brine shrimp (Belovsky et al. 1999) and grain beetles (Bancroft & Turchin 2003).

Habitat size and quality are expected to influence extinction risk through different causal mechanistic pathways. Specifically, small habitat size is expected to influence extinction risk by reducing carrying capacity, thereby diminishing the buffer that exists between the long-run average population size and extinction. As a result, populations in smaller habitats are more susceptible to extinction from demographic stochasticity, which is strongest for small populations (Lande et al. 2003; Desharnais et al. 2006). Poor habitat quality, by contrast, is expected to influence extinction risk in two ways. First, as with small habitats, poor quality habitats can only support small populations that are prone to extinction from demographic stochasticity. Second, poor habitat quality diminishes a population’s growth rate, delaying the escape from vulnerability when populations are small. These implications of carrying capacity and reproductive potential for population extinction are theoretically well established (MacArthur 1972; Tier & Hanson 1981; Pimm et al. 1988; Hakoyama et al. 2000). The distinction between the mechanistic influence of habitat size and quality raises a fundamental question: which is more important to extinction risk?

Recent theory and empirical studies each come to different conclusions in answer to this question. For example, using a model for army ants, Partridge et al. (1996) argued that time to extinction depended more on habitat size than habitat quality. Alternatively, using a model for the common shrew, Klok & De Roos (1998) demonstrated that time to extinction increased exponentially with increasing population growth rate, while it only increased logarithmically with increasing carrying capacity. Because they expected population growth rate
2. MATERIAL AND METHODS

(a) Model system

Our model system consisted of a single clone of the freshwater cladoceran *Daphnia magna* reared on a food resource of blue-green algae (*Spirulina* sp.). *Daphnia* have previously been used to study extinction (Bengtsson & Milbrink 1995; Ebert et al. 2000; Drake & Lodge 2004; Drake et al. 2005) and are a model for ecological studies in general (Lampert 2006). This system enables us to achieve two conditions crucial to differentiating effects of habitat size and quality: (i) we isolate variation, while simultaneously manipulating habitat size and quality and (ii) our food resource (*Spirulina*) is inactivated, eliminating the tight coupling of consumer and resource populations, which leads to interesting complex dynamics, but is an unrealistic model of systems in which consumers prey on multiple species. Thus, despite the fact that the experimental *Daphnia–Spirulina* system is maintained on a single resource, it is representative of the broad-spectrum consumers common in nature.

(b) Laboratory experiment

We used a fully crossed experimental design with small (700 ml) or large (1400 ml) habitat size as one factor and low (200 μl solution d⁻¹), medium (400 μl solution d⁻¹) or high (800 μl solution d⁻¹) food abundance as a second factor. The experiment was conducted within small laboratory microcosms (31.5 × 21.7 × 1 cm thickness dependent on habitat size treatment). Microcosms were made of clear Plexiglas and filled with synthetic freshwater medium (USEPA 2002). We exchanged 50% of the medium monthly to prohibit build-up of nitrate and ammonium (metabolites) that can be toxic to *D. magna*. Each of the six treatments was replicated 16 times, yielding a total of 96 populations. Microcosms were located on a laboratory bench top, randomly ordered, in a room with 24 hours fluorescent light and the temperature held constant at 23.3 ± 1.4°C (mean ± s.d.). We fed *D. magna* a solution of processed *Spirulina* sp. (JEHM Co., Inc.). The food solution was prepared by mixing 0.05 g *Spirulina* (a blue-green alga, 10.16% N and 44.96% C) into 25 ml of deionized water. Using processed (i.e. dead) *Spirulina* rather than live algae eliminated the reproduction of food resources within experimental microcosms, allowing precise control of food resource available to the population.

Populations were inoculated with five individuals of a single *D. magna* clone, one non-gravid adult and four juveniles, on 28 February 2007 or 1 March 2007 (eight replicates of each treatment were randomly selected to start each day). The use of a single clone (no sexual reproduction was observed) did not address non-demographic factors such as inbreeding and loss of genetic diversity that can also influence extinction in small populations. Each population was censused weekly by counting the number of adults and the number of juveniles using a hand tally counter. Each count was repeated until duplicate estimates were obtained to ensure accuracy (generally only requiring two counts). The mobility of individuals decreased the accuracy of counting large populations (approximately more than 100 individuals). In these cases, replicate counts agreed within 2–3% and were averaged and rounded to the nearest whole number. Under the experimental conditions used here, generation times were 13.9 ± 4.0 days (mean ± s.d.; B. D. Griffen 2007, unpublished data). The sampling interval was thus approximately half the generation time. The experiment ended when all populations had gone extinct (49 weeks). Many populations produced resting eggs during our experiment. These were removed at each monthly water change to prevent confounding our test, which is intended to be a model for consumer-resource systems in general, with species adapted to persistence in fluctuating environments through long-term storage effects (Chesson & Warner 1981; Adler & Drake in press). While in natural populations, these resting eggs could have later hatched; for the purposes of this study, we considered populations to be extinct when no adult or juvenile individuals remained alive.

(c) Statistical analyses

The start of our experiment was blocked over 2 days. We initially included start day as a blocking factor in the analyses. However, it was not significant (*p* > 0.05 for all main effects and interaction terms for the effect of start day on carrying capacity, population growth rate or time to extinction), and so we pooled data across 2 days for all analyses reported below.
(i) Carrying capacity
We examined how carrying capacity was influenced by habitat size and quality. We estimated the carrying capacity for each population by fitting the Ricker model for population growth $\ln (N_{t+2}/N_t) = \lambda (1-N_t/K)$ to the time-series of census data for each population using nonlinear least squares, where $N_t$ is total population size and fixing population growth rate at $\lambda = N_2/N_0$. For 17 of the populations, extinction occurred in less than five weeks, providing insufficient data to obtain an estimate for carrying capacity from the Ricker model. In these cases, we determined the carrying capacity as the mean population size over all time periods preceding extinction. We also used other measures of carrying capacity, including the mean and median population sizes for each population. Analyses with these measures did not differ qualitatively from those obtained with carrying capacities estimated using the Ricker model. We therefore present only Ricker model estimates here.

We tested for effects of treatment on carrying capacity using a linear model with habitat size and food amount as factors and the $\ln$(carrying capacity) as the response. Because habitat size and resource volume have different units, we transformed experimental treatments (habitat size and food amount) to a continuous log2 scale so that analysis results could be interpreted as the effect of doubling habitat size and/or food abundance on carrying capacity, e.g. food levels were standardized as: 200 μl $\rightarrow$ log2 (200 μl/200 μl) = 0; 400 μl $\rightarrow$ log2 (400 μl/200 μl) = 1; and 800 μl $\rightarrow$ log2 (800 μl/200 μl) = 2. Thus, an increase in one unit was equivalent to a doubling of the experimental treatment.

(ii) Population growth rate
To determine how the population growth rate ($\lambda$) is influenced by habitat size and quality, we regressed $\ln (N_{t+2}/N_t)$ on log2 transformations of experimental treatment as described previously. We used $N_2$ rather than $N_1$ because our sampling period of one week was approximately half the generation time (discussed above). We further verified our results by repeating our comparisons with an estimate of population growth rate determined by regressing $N_{t+2}/N_t$ against $N_t$ for all sampling times and using the y-intercept as a model-independent estimate of $\lambda$ (see appendix I in the electronic supplementary material).

(iii) Time to extinction
We tested for effects of experimental treatment on time to extinction (in weeks) by regressing $\ln$(time to extinction) on log2 transformations of experimental treatment as described previously. We directly examined how time to extinction varied with changes in carrying capacity and population growth rate with multiple linear regression pooling populations across all treatments for analysis ($n = 96$). In doing so, we view population growth rate and carrying capacity as statistical descriptions of populations, rather than as intrinsic properties of species. Using these, statistical descriptors can therefore provide mechanistic insight into how changes to habitat size or quality influenced population dynamics.

3. RESULTS
(a) Carrying capacity
Population-carrying capacity increased with habitat size (consistent with our first hypothesis, $F_{1,92} = 21.24, p < 0.0001$; figure 1) and habitat quality (consistent with our second hypothesis, $F_{1,92} = 36.07, p < 0.0001$; figure 1). There was no evidence for an interaction between experimental treatments ($F_{1,92} = 0.23, p = 0.64$). Overall, doubling habitat size and doubling food each led approximately to an estimated near doubling of the log-carrying capacity (back transformed parameter estimates for habitat size: 1.95 ± 0.22 (mean ± s.e.) and for habitat quality: 1.66 ± 0.12; figure 1).

(b) Population growth rate
Increasing habitat quality by increasing food abundances led to higher population growth rates (consistent with our fourth hypothesis, $F_{1,92} = 45.09, p < 0.0001$; figure 2). Increasing habitat size also increased population growth rates (contrary to our third hypothesis, $F_{1,92} = 13.97$,
p = 0.0003). There was no evidence for an interaction of experimental treatments (F1,92 = 0.036, p = 0.85). Doubling habitat size and food also had similar effects on log population growth rate, leading to approximately a 1.4 times increase in growth rate (back transformed parameter estimates for habitat quality: 1.39 ± 1.07 and for habitat size: 1.33 ± 1.14; figure 2).

(c) Time to extinction

Individually, carrying capacity and population growth rates were each positively correlated with time to extinction (consistent with our fifth and sixth hypotheses; figure 3). However, when both factors were included in a multiple regression, changes in time to extinction were explained solely by changes in carrying capacity (carrying capacity: F1,92 = 294.45, p < 0.0001; population growth rate: F1,92 = 2.66, p = 0.11; overall R² = 0.79). Further, the effects of population growth rate and carrying capacity interacted compensatorily, such that increasing both simultaneously yielded benefits that were lower than the sum of increasing each individually (parameter estimate for multiple regression interaction term: −0.009 ± 0.001, F1,92 = 71.38, p < 0.0001). Overall, time to extinction increased both with increasing habitat size (F1,92 = 18.19, p < 0.0001; figure 4) and quality (F1,92 = 28.65, p < 0.0001; figure 4). We failed to reject the general linear (null) hypothesis for a difference in time to extinction between the two treatments (i.e. H0: quality–size = 0; t = 1.30, p = 0.20). Thus, there is strong evidence that both habitat quality and size increase persistence, but insufficient evidence to conclude that one is more important than the other overall. There was no evidence for an interaction between treatments in time to extinction (F1,92 = 1.41, p = 0.24).

4. DISCUSSION

We found that increasing habitat size and improving habitat quality both increased population-carrying capacity (figure 1). Similarly, we found that both factors also increased population growth rate (figure 2). Increased carrying capacity and population growth rate were both positively correlated with time to extinction (figure 3). While these effects of habitat quality are consistent with predictions (Klok & De Roos 1998; Hakoyama et al. 2000), the effect of habitat size on population growth rate was unexpected.

Theoretical studies, which have examined the influence of habitat size and quality on extinction risk, have assumed that population growth rates are influenced only by habitat quality (Klok & De Roos 1998; Hakoyama et al. 2000). Based on this assumption, theory predicting that population growth rates are greater determinants than carrying capacity of extinction risk leads to the conclusion that extinction risk can be lowered more by improving habitat quality than by increasing habitat size. Our results do not support this assumption and subsequent conclusion. Rather, we first found that population growth rates were similarly influenced by habitat size and quality, and second that carrying capacity explained more variation in time to extinction than did population growth rate.

The increase in population growth rate with habitat size was unexpected. In fact, if anything we expected the opposite, that is, a decrease in population growth rate with increasing habitat size would result from the decrease in food concentrations as habitat size increased. Consuming the same amount of food in each habitat size at a given food level would have therefore required twice the effort, and thus twice the energy expenditure, in large habitats. We examined the possibility that resting egg production may be responsible for lower growth rates in small habitats, but found evidence against this hypothesis (see appendix II in the electronic supplementary material). The reason for the increased growth rate in larger habitats is unclear.

While carrying capacity and population growth were both individually correlated with time to extinction, their effects were not strictly additive (indicated by the significant negative interaction term in multiple regressions). The significance of the interaction between these two parameters, even when we failed to detect an interaction in the factors causing those parameters (no significant interaction term in analysis of habitat size and quality) may be explained by the fact that both population parameters are influenced by both habitat factors. Thus, improving habitats by increasing their size and improving their quality can have additive impacts on population growth rate and carrying capacity. But the subsequent effects of these population parameters on the time to extinction were at least partially compensatory in our experiment. This explanation is further supported by the fact that the variances explained by regressing time to extinction on each parameter separately sum to more than one (figure 3), and that when time to extinction is regressed against both simultaneously, the variance explained is only slightly more than with carrying capacity alone, implying that the same benefit can to some extent be achieved by either increasing habitat size or by improving habitat quality.
and 1980s (Simberloff & Abele 1982). This historical debate continued among ecologists throughout the 1970s. As a result, the single large or several small (‘SLOSS’) populations tend to have higher growth rates.

Population growth rates provide a simpler way of decreasing extinction risk through both increased carrying capacity and increased habitat size. Wulff & Drake (2005) suggest that, in some instances, increasing habitat size may more effectively increase when rare. Aside from the initial population inoculations of five individuals, several instances occurred where population size decreased to very low numbers (five or less individuals) and then rebounded, avoiding extinction. However, the number of these events increased with both habitat size and quality. The numbers of events for each treatment were as follows: small size, low food = 1; small size, medium food = 1; small size, high food = 4; large size, low food = 2; large size, medium food = 5; and large size, high food = 5. In other words, twice as many events occurred overall in large habitats as in small (12 versus 6), and there was an incremental increase in the overall number of events with each jump in food level (3–6–9). Thus, increasing habitat size or quality both increase population growth rates (figure 2), allowing populations to escape extinction by increasing when rare.

The importance of habitat size was highlighted in the 1960s with the development of MacArthur & Wilson’s (1967) theory of island biogeography. This lead to the analyses described in electronic supplementary material, appendices I and II. This work was supported by the University of Georgia Odum School of Ecology and the University of Georgia Research Foundation.

REFERENCES


