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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).

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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

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to increase with habitat quality but not with habitat size, Klok & De Roos (1998) argued that extinction risk should be influenced more by habitat quality than habitat size. Results of empirical studies are similarly inconclusive, showing that extinction risk can be influenced most by habitat size (e.g. Johansson & Ehrlen 2003), habitat quality (e.g. Franken & Hik 2004) or a combination of the two (e.g. Dennis & Eales 1997; Fleishman *et al.* 2002).

The expectations from the preceding two paragraphs may be summarized in six hypotheses. The following four hypotheses derive directly from the preceding arguments.

(i) Larger habitats support populations with higher carrying capacities.
(ii) Higher quality habitats support populations with higher carrying capacities.
(iii) There is no difference in population growth rate between large and small habitats.
(iv) Population growth rates are higher in high-quality habitats than low-quality habitats.

The last two hypotheses are drawn from the standard theory of stochastic population growth (e.g. Tier & Hanson 1981).

(v) Population persistence increases with population growth rate.
(vi) Population persistence increases with carrying capacity.

From (i), (iii) and (vi), we predicted that population persistence would increase with habitat size through its effects on carrying capacity. From (ii), (iv), (v) and (vi), we predicted that population persistence would increase with habitat quality through its effect on both carrying capacity and the intrinsic rate of increase.

We experimentally tested these hypotheses using a laboratory system. Our experiment was devised to partition the effects of habitat quality and size. We used model-based estimates of key population parameters (carrying capacity and population growth rate) to assign effects to different causal pathways of population regulation and to quantify their relative importance.

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 alga (*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 the effects of demographic stochasticity from environmental variation, while simultaneously manipulating habitat size and quality and (ii) our food resource (pulverized *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 ($31.5 \times 21.7 \times 1$ or 2 cm thickness depended 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 $\pm$ 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 (approx. 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 $\pm$ 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 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 $\log_2$ 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 $\rightarrow \log_2$ (200 µl/200 µl) = 0; 400 µl $\rightarrow \log_2$ (400 µl/200 µl) = 1; and 800 µl $\rightarrow \log_2$ (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 (λ) is influenced by habitat size and quality, we regressed $\lambda = N_2/N_0$ ($N_0 = 5$) on $\log_2$ 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 λ (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 $\log_2$ 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, $F_{1,92} = 21.24$, $p \ll 0.0001$; figure 1) and habitat quality (consistent with our second hypothesis, $F_{1,92} = 36.07$, $p \ll 0.0001$; 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 ± 1.22 (mean $\pm$ s.e.) and for habitat quality: 1.66 ± 1.12; figure 1).

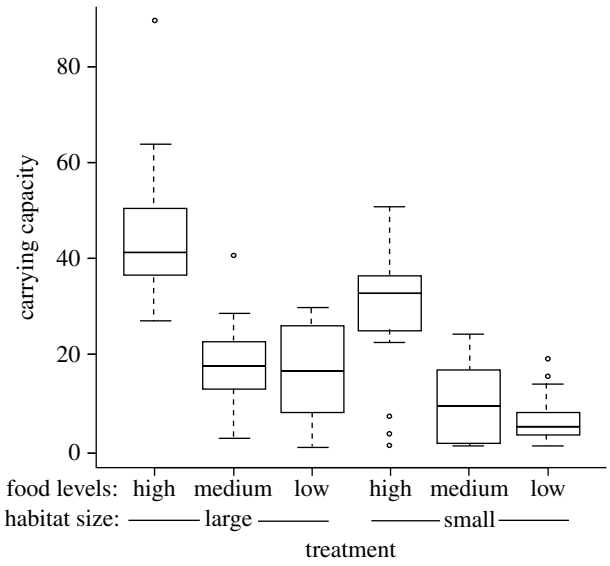

Figure 1. Effect of habitat size and quality (food abundance) on population-carrying capacity. Carrying capacity was determined for each population by fitting the Ricker model to the time-series of population sizes.

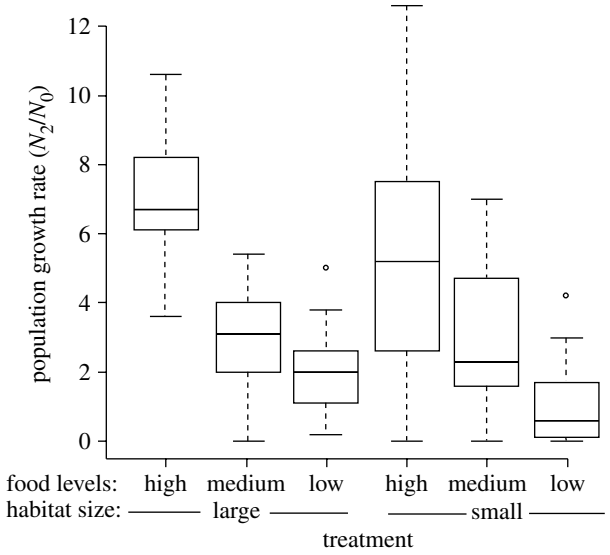

Figure 2. Effect of habitat size and quality (food abundance) on population growth rate (λ). Population growth rates were determined as N_2/N_0, where N_0 is the initial population size of five individuals. We used N_2 because generation times were approximately twice the sampling period.

(b) *Population growth rate*

Increasing habitat quality by increasing food abundances led to higher population growth rates (consistent with our fourth hypothesis, $F_{1,92} = 45.09$, $p \ll 0.0001$; figure 2). Increasing habitat size also increased population growth rates (contrary to our third hypothesis, $F_{1,92} = 13.97$,

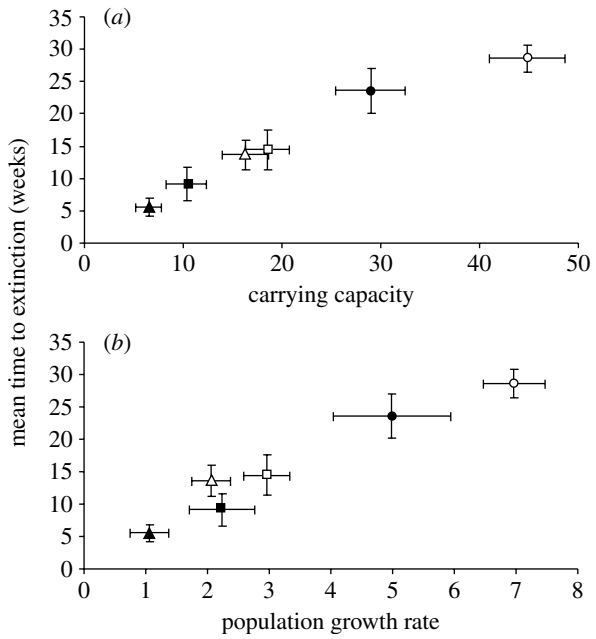

Figure 3. Correlation between time to extinction and (*a*) carrying capacity ($\ln(y)=0.05x+1.28$; $R^2=0.64$; $p\ll0.0001$) and (*b*) population growth rate ($\ln(y)=0.23x+1.56$; $R^2=0.41$; $p\ll0.0001$). Values are means$\pm$ s.e. ($n=16$). However, regressions used populations as replicate ($n=96$), rather than using means.

$p=0.0003$). There was no evidence for an interaction of experimental treatments ($F_{1,92}=0.036$, $p=0.85$). 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 rate 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: $F_{1,92}=294.45$, $p\ll0.0001$; population growth rate: $F_{1,92}=2.66$, $p=0.11$; overall $R^2=0.79$). 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, $F_{1,92}=71.38$, $p\ll0.0001$). Overall, time to extinction increased both with increasing habitat size ($F_{1,92}=18.19$, $p\ll0.0001$; figure 4) and quality ($F_{1,92}=28.65$, $p\ll0.0001$; figure 4). We failed to reject the general linear (null) hypothesis for a difference in time to extinction between the two treatments (i.e. H_0: 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 ($F_{1,92}=1.41$, $p=0.24$).

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.

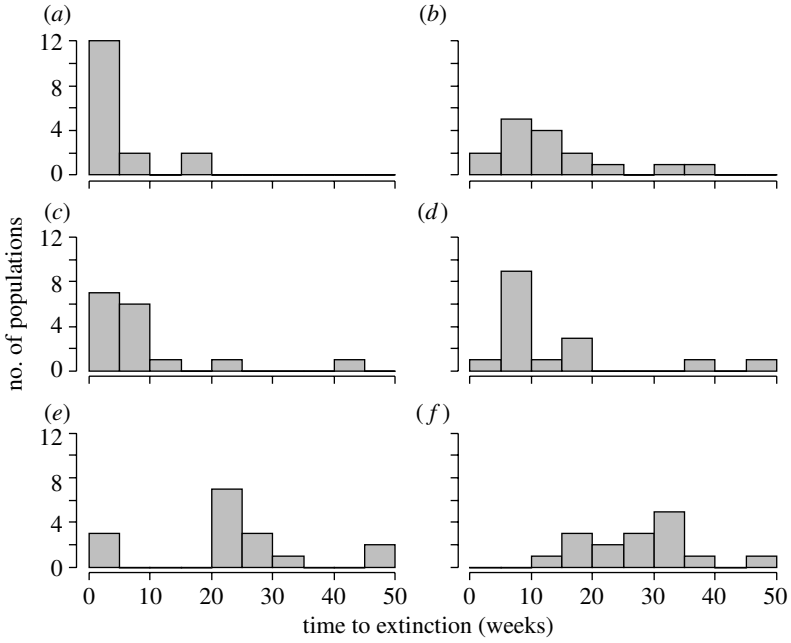

Figure 4. Population extinctions by treatment. (*a*) Low/small, (*b*) low/large, (*c*) medium/small, (*d*) medium/large, (*e*) high/small and (*f*) high/large.

Our experiment also provided additional evidence that improving habitat size and quality can both reduce extinction risk by increasing population growth rates (figure 2) and thus allowing populations to 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 suggestion that large habitats may be preferable to several small habitats for species conservation (Diamond 1975). As a result, the single large or several small ('SLOSS') debate continued among ecologists throughout the 1970s and 1980s (Simberloff & Abele 1982). This historical focus on habitat size has largely overlooked the importance of habitat quality. However, habitat quality is difficult to determine because different species may 'measure' habitat quality differently. Results from this simplified system suggest that, in some instances, increasing habitat size may provide a simpler way of decreasing extinction risk through both increased carrying capacity and increased population growth rate.

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APPENDIX I – Alternative statistical analyses

The reproductive ratio λ is defined as the maximum per capita population growth rate. In typical populations (i.e., populations without an Allee effect) per capita population growth rate increases as population size goes to zero (but is undefined at zero). We use the ratio N_2/N_0 as a model-independent estimator of reproductive ratio. The advantage of this estimator is that it makes no assumptions about the form of density dependence other than that reproductive ratio at N_0=5 is close to the true value. The disadvantage of this approach is that this estimator is biased by the deceleration of growth by time t=2 due to the effect of carrying capacity. Particularly, if N_2 is no longer small, relative to carrying capacity, the true growth rate may be severely underestimated. In our system, because carrying capacity is expected to increase with food level, the differences between the ratio N_2/N_0 and the true value might vary between food level treatments, and similarly for habitat size treatments. Our analysis assumes that this effect would not bias our overall conclusion.

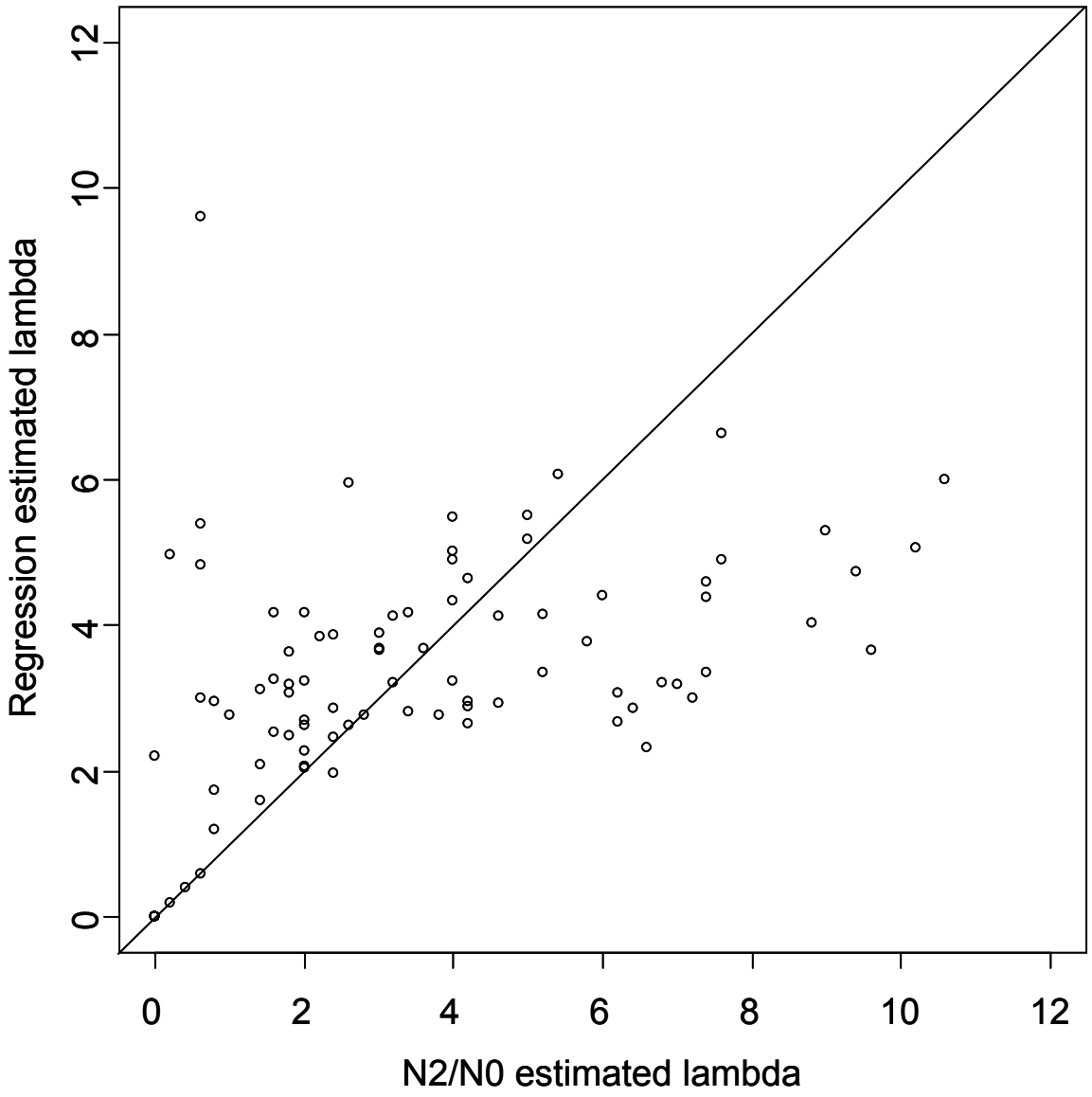
As a check on this assumption, we also performed analyses using a model-dependent estimator of reproductive ratio as follows. We first calculated realized inter-interval population growth rates according to N_{t+2}/N_t for each sampling period. Then we regressed these values against population size (N_t). If the effect of density dependence is linear (i.e., logistic population growth), then the intercept of the regression is an estimator of the reproductive ratio near zero. This method is inferior to the first method in that it makes the unlikely assumption of linearity. It is superior in that it corrects for the deceleration due to carrying capacity. We note that we could not simultaneously estimate λ and K with high precision due to the relatively short time series of the experiment.

Figure A1.1 plots the model-dependent estimates of λ (y-axis) against the model independent estimates obtained from the ratio N_2/N_0. The negative deviation from the one-to-one line at high values of the model-independent estimator suggests that the linearity assumption inherent in the model-based estimator is indeed flawed. (If the linear model were correct, the model-independent estimates should all lie above the one-to-one line.) Despite these differences, results of our analyses were qualitatively identical using the two estimates. Specifically, increasing habitat quality still led to higher population growth rates ($F_{1,92}$=4.29, p=0.04), as did increasing habitat size ($F_{1,92}$=16.66, p<<0.0001). Additionally, increasing population growth rate still increased the log time to extinction (multiple regression, $F_{1,92}$=17.34, p<<0.0001), as did increasing carrying capacity (multiple regression, $F_{1,92}$=270.81, p<<0.0001; overall R^2=0.77).

Figure A1.1

Comparison of lambda values estimated as N_2/N_0 and as the intercept of a regression analysis of N_{t+2}/N_t on population size (N_t).

Fig. A1.1


Regression estimated lambda
N2/N0 estimated lambda
0
2
4
6
8
10
12

APPENDIX II – Resting egg production

A plausible confounding explanation for the increase in population growth rate with habitat size is the sexual production of resting eggs. Population density is among the factors that can induce this reproductive mode in *Daphnia* (Kleiven *et al*. 1992). When population density increases, *Daphnia* can switch from asexual to sexual reproduction. However, since the brood size of resting eggs (one or two) is typically smaller than in asexual reproduction (typically 4-10, occasionally up to 15), resting egg reproduction could decrease total population growth rate, compared with asexual reproduction. If this explanation were correct, population growth rate in our experiment would have decreased when resting eggs were produced, an effect that would have been even more pronounced in the data as resting eggs were removed from the population. Further, the above mechanism would be confounded with the habitat size treatments , if smaller habitats had higher population densities that stimulated greater resting egg production.

To exclude this explanation, we (i) tested for effects of habitat size and food on population density with two-way ANOVA; (ii) tested for effects of habitat size and food amount on total resting egg production over the course of the experiment with two-way ANOVA.

These analyses indicated that population density was indeed higher in smaller habitats than in larger habitats (ANOVA, habitat size: $F_{1,90}$=7.16, p=0.009; food amount: $F_{2,90}$=58.04, p<<0.0001; interaction term: $F_{2,90}$=2.83, p=0.06; Fig. A2.1). However, resting egg production was also influenced by habitat size and food amount, but it was highest in larger habitats (ANOVA, habitat size: $F_{1,90}$=8.87, p=0.004; food amount:

$F_{2,90}$=38.61, p<<0.0001; interaction term: $F_{2,90}$=2.78, p=0.07; Fig. A2.2). Resting egg production could only have been responsible for increased population growth rates in larger habitats within our experiment if more resting eggs had been produced in smaller habitats. However, these results demonstrate the opposite trend. Additionally, while asexual reproduction can produce large numbers of eggs/brood under natural conditions, brood sizes were typically only 1-3 individuals under our experimental conditions.

These factors suggest that resting egg production was likely not the reason for the effect of habitat size on population growth rate in our experiment.

References:

Kleiven, O. T., Larsson, P. & Hobaek, A. 1992 Sexual reproduction in *Daphnia magna* requires three stimuli. *Oikos* **65**, 197–206.

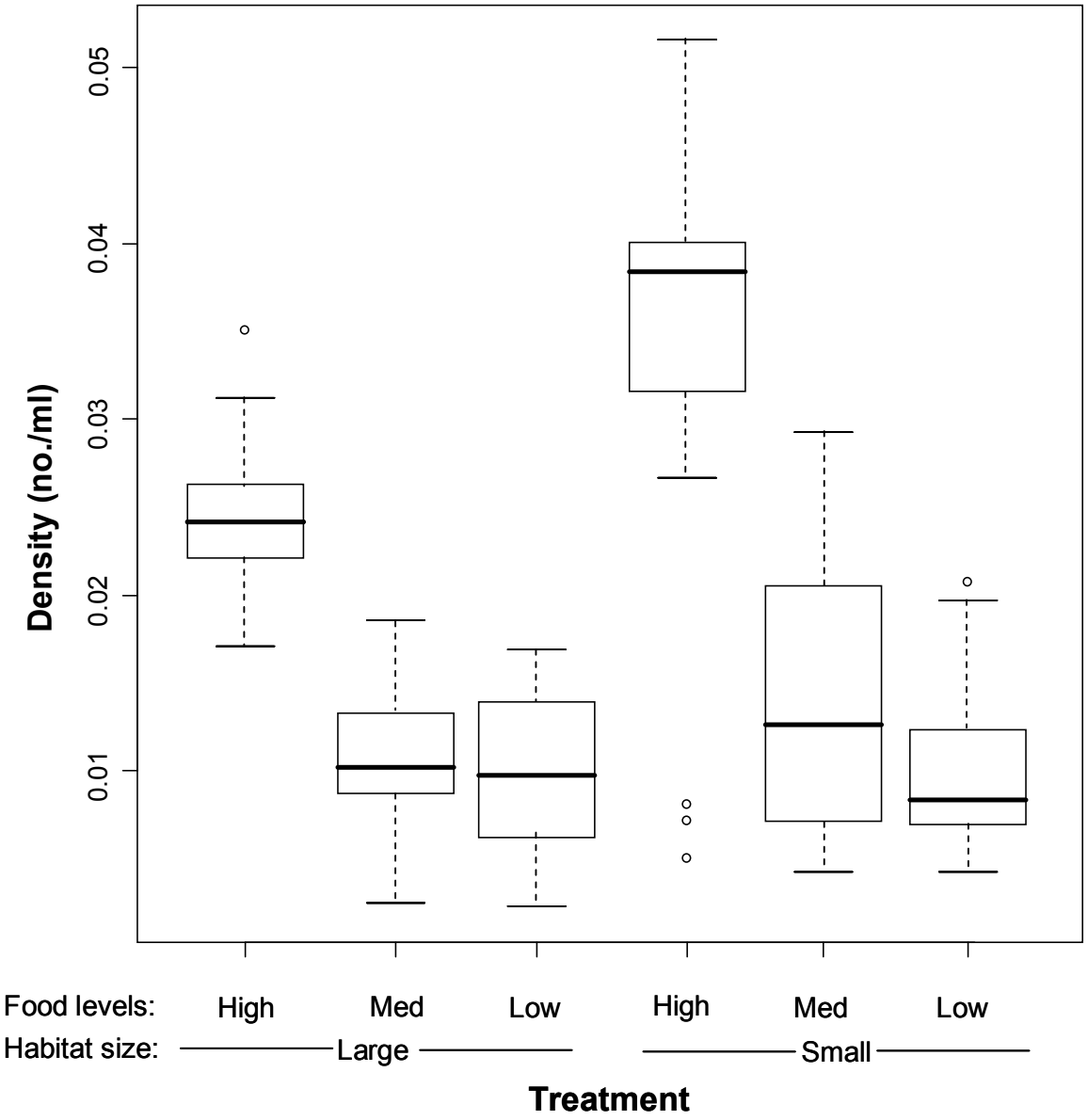
Figure A2.1

Population density as a function of experimental treatment.

Figure A2.2

Total resting egg production over the course of the experiment as a function of experimental treatment.

Figure A2.1


Density (no./ml)
0.01
0.02
0.03
0.04
0.05
Food levels:
High
Med
Low
High
Med
Low
Habitat size:
Large
Small
Treatment

70 Figure A2.2

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