The Relative Importance of Competition and Predation Varies with Productivity in a Model Community

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abstract: Recent theory predicts that productivity can influence the relative importance of predation and competition in determining patterns in abundance, diversity, and community structure. In low-productivity systems, competition is predicted to be the major influence on community patterns, while at high productivity, the major influence is predicted to be predation. We directly tested this theory using a laboratory model community. Our model community consisted of the bacteriophage T2 (a virus that feeds on *Escherichia coli*) and two populations of *E. coli*, in glucose-limited chemostats. One *E. coli* population consisted of individuals that were sensitive to predation by T2 ("vulnerable" *E. coli*), and the other population consisted of individuals that were partially resistant to predation by T2 ("less vulnerable" *E. coli*). We manipulated productivity in this experiment by running replicate chemostats with different input concentrations of glucose. Our observations were consistent with theoretical predictions. We observed the decline of the more vulnerable prey population at higher productivity but not at lower productivity, and the decline of the less vulnerable prey population at lower productivity but not at higher productivity. However, the rate of decline in some replicates was slower than predicted, and extinctions were not observed during the experiments, contrary to theoretical predictions. We present some testable hypotheses that might explain the slow rate of decline observed.

Keywords: bacteria, bacteriophage, productivity, competition, predation, community structure.

One of the great challenges facing ecologists is to link pattern to process. This is especially difficult in community ecology, where multiple processes can interact to produce patterns in abundance, diversity, and community structure. Historically, ecologists have focused on single processes such as competition or predation that may explain variation in these patterns (e.g., Brooks and Dodson 1965; Cody 1974). More recently, ecologists have recognized the importance of studying interactions between these processes and have begun to identify factors that may determine the relative role each process plays (Leibold 1989; Power 1992). For example, recent theory predicts that productivity can influence the relative importance of predation and competition in determining community patterns (Holt et al. 1994; Leibold 1996). In low-productivity systems, competition is predicted to be the major influence on community patterns, and organisms that are successful at competing for resources are predicted to be dominant. In contrast, in high-productivity systems, predation is predicted to be the major influence, and organisms that are successful at avoiding predation are predicted to be dominant.

These predictions come from theoretical studies of simple, abstract communities. For example, consider a simple community that consists of two prey species that share a common resource and predator (fig. 1). Let us assume that there is a trade-off between being successful at competing for resources ("exploitation ability") and being successful at avoiding predation ("predator resistance"), such that the better exploiter is least resistant to predation. There is evidence that such trade-offs exist (Lenski 1988; Simms 1992; Grover 1995; Kraaijeveld and Godfray 1997). At low levels of productivity (e.g., region I in fig. 2*A*), theory predicts that the better exploiter (species A) will exclude the more resistant prey species (species B), through a combination of resource and "apparent" competition (sensu Holt 1977). At high levels of productivity (e.g., region III in fig. 2*A*), theory predicts that the more resistant prey (species B) will exclude the better exploiter (species A), again through a combination of resource and apparent competition. At intermediate levels of productivity (e.g., region II in fig. 2*A*), theory predicts that both prey species may coexist due to the trade-off between exploitation abil-

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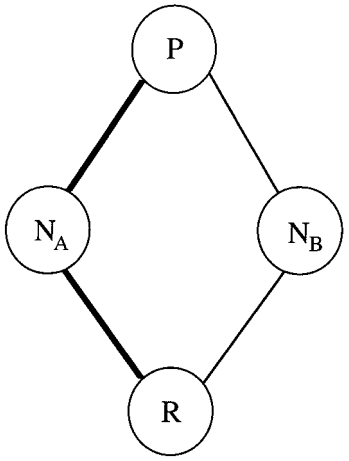

Figure 1: Simple community in which two prey types (N_A, N_B) share a common resource (R) and a common predator (P). A trade-off between competitive ability for the resource and resistance to predation is assumed. The thick lines linking N_A to R and P indicate that N_A is a better competitor for resources and more susceptible to predation. Conversely, the thin lines linking N_B to R and P indicate that N_B is an inferior competitor for resources and less susceptible to predation. This simple community assumes that interference competition does not exist between prey types or among predators and that higher-order predators (i.e., consumers of P) do not exist.

ity and predator resistance. The conditions for equilibrium coexistence are shown graphically in figure 2*B*.

The general patterns predicted by this theory are consistent with observations of some natural communities. For example, this theory predicts the following three general patterns (fig. 2*A*). First, the biomass of total prey and predator will both increase, in a steplike manner, in response to increased productivity. Second, the relative biomass of predator-resistant species will increase as productivity is increased. Third, the diversity of prey species will first increase then decrease as productivity is increased, resulting in a hump-shaped relationship between diversity and productivity. These patterns are predicted even if the models are expanded to include multiple prey types and spatial patchiness (Leibold 1996). These general patterns have been observed in some natural communities, particularly freshwater lentic communities. Positive correlations between biomass at adjacent trophic levels in response to changes in productivity have often been observed (Leibold 1989, 1996). The biomass of predator-resistant species has also been observed to increase as productivity increases (Watson et al. 1992). Hump-shaped relationships between diversity and productivity have been observed in a number of studies (reviewed in Rosenzweig 1995).

However, this theory has not been tested directly. In this article, we report a direct test of this theory using a labo-

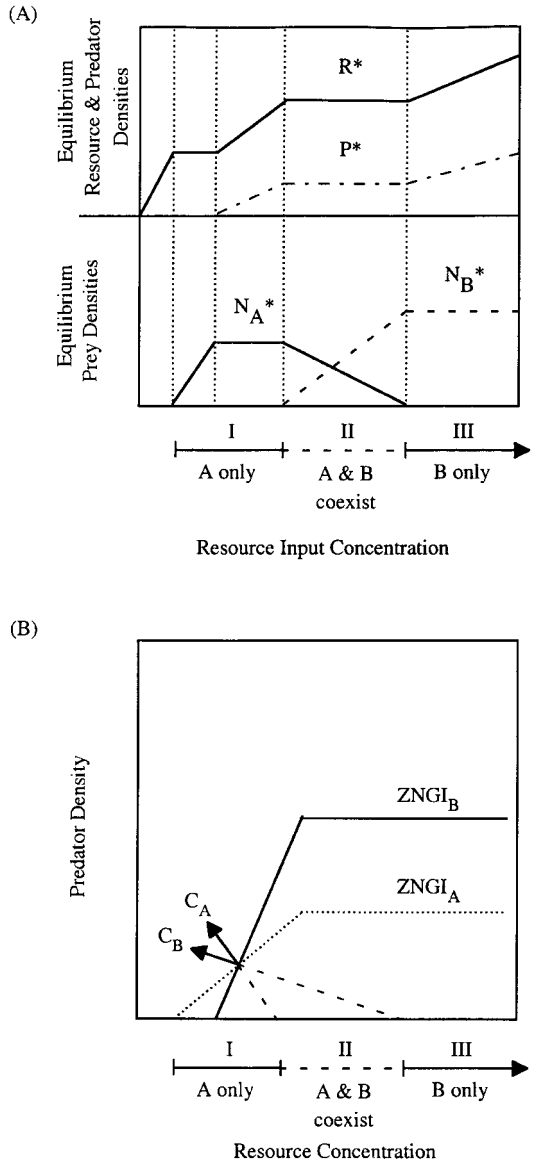

Figure 2: Effect of productivity on the density of two prey types, a shared predator, and a shared resource. Productivity is assumed to be proportional to resource input concentration. *A*, Predicted pattern of equilibrium population densities across a gradient of productivity (modified from Leibold 1996); R^* = equilibrium resource concentration; P^* = equilibrium predator density; N_A^* = equilibrium density of more vulnerable prey; N_B^* = equilibrium density of less vulnerable prey. *B*, Graphical analysis of an idealized community model (modified from Leibold 1996) showing zero net growth isoclines (*ZNGI*) and net consumer-prey impact vectors (*C*) for two prey types (*A* and *B*). Coexistence is possible because the ZNGIs intersect. The zone of coexistence is determined by the slope of the impact vectors and their intersection with the resource axis.

ratory model community. We assembled communities that consisted of bacteriophage T2 and two *Escherichia coli* strains that differed in their susceptibility to this bacteriophage. We manipulated the productivity of this model community and compared the observed responses to the predictions of a mathematical model that includes both competition for resources and avoidance of a shared predator.

Chemostat communities of *E. coli* and bacteriophage T2 are excellent model systems with which to test shared predator/shared resource models for a number of reasons. First, T2 is one of the few bacteriophages against which *E. coli* has been observed to evolve partial resistance (i.e., reduced vulnerability; Lenski 1984). *Escherichia coli* can evolve complete resistance (i.e., invulnerability) to many bacteriophages; however, partial resistance is rarely observed. Because partial resistance against T2 is possible, model communities that consist of prey populations that differ quantitatively in susceptibility to a shared predator can be constructed using *E. coli* and bacteriophage T2. Second, *E. coli* mutants that are partially resistant to bacteriophage T2 have the correlated trait of complete resistance to bacteriophage T4 (Lenski 1984). This relationship allows the partially resistant mutants to be easily obtained (by selecting for T4-resistant mutants) and easily tracked in a model community (by screening for T4-resistant bacteria). Third, a trade-off between exploitation ability for glucose and partial resistance to T2 has been demonstrated in *E. coli* (Lenski and Levin 1985; Lenski 1988). Such trade-offs are assumed in most shared predator/shared resource models (e.g., Leibold 1996). Fourth, chemostat communities of *E. coli* and bacteriophage T2 share the advantages of other microbial model systems (Bohannan and Lenski 1997, 1999). These advantages include the ease with which experimental variables such as productivity can be manipulated and the relatively short time period necessary to observe steady state responses to changes such as increased productivity.

Resource competition between microorganisms has been demonstrated in numerous laboratory studies (reviewed in Grover 1997), and the exclusion of vulnerable prey by more predator-resistant prey has been shown to occur in laboratory communities of bacteriophage and bacteria (Levin et al. 1977; Lenski 1984), protist predators and prey bacteria (Nakajima and Kurihara 1994*a*, 1994*b*), and protist predators and prey (Lawler 1993). However, the effect of changes in productivity on the relative importance of competition and predation has not previously been demonstrated in laboratory model communities. The research we report here expands on two previous studies, in which we used laboratory model communities of *E. coli* and bacteriophage to determine the effect of changes in productivity on the population dynamics in simple food chains (Bohannan and Lenski 1997) and in communities with vulnerable and invulnerable prey (Bohannan and Lenski 1999).

Methods

Model Community

Our model community consisted of *Escherichia coli* B strain REL607 (Lenski et al. 1991), *E. coli* B strain REL6584 (Bohannan and Lenski 1999), and the virulent bacteriophage T2 (provided by L. Snyder) in glucose-limited chemostats. Strain REL6584 is identical to REL607 with the exceptions that it cannot utilize the sugar arabinose and it is invulnerable to predation by bacteriophage T4. We used the ability to utilize arabinose as a neutral marker to distinguish the two *E. coli* strains. We previously checked the neutrality of the arabinose-utilization marker, and we detected no effect of the arabinose-utilization marker on competitive ability in our strains (Bohannan and Lenski 1999). Invulnerability to predation by T4 has been shown to result in a competitive disadvantage in a glucose-limited environment when phage is not present (Lenski and Levin 1985; Lenski 1988). This disadvantage is approximately 35% for REL6584 relative to REL607 (Bohannan and Lenski 1999). Mutants of *E. coli* that are invulnerable to predation by T4 have also been shown to be less vulnerable to predation by bacteriophage T2 (Lenski 1984). This effect occurs because T4-invulnerable mutants of *E. coli* achieve invulnerability through the loss of the cell surface receptor to which T4 initially attaches (Lenski 1988). This cell surface receptor is also one of two distinct receptors involved with attachment by T2 (Lenski 1984). The loss of this receptor reduces the rate at which T2 infects *E. coli* by approximately 50% (Lenski 1984). Thus, mutants of *E. coli* that have reduced vulnerability to T2 can be detected by screening for the correlated trait of invulnerability to T4.

Our chemostat vessels and culture conditions are identical to those described previously (Bohannan and Lenski 1997, 1999). The media consisted of Davis minimal broth (Carlton and Brown 1981) supplemented with 2×10^{-3} μg thiamine hydrochloride/mL and various concentrations of glucose (see next paragraph). The volume of the chemostats was maintained at approximately 30 mL, the flow rate at approximately 0.2 turnovers/h, and the temperature at 37°C. We maintained replicate chemostats at each glucose concentration. The treatment chemostats were inoculated with the vulnerable prey (*E. coli* strain REL607), the less vulnerable prey (*E. coli* strain REL6584), and the predator (bacteriophage T2) simultaneously. Control chemostats containing each of the *E. coli* strains alone with the predator were established at each glucose concentration and maintained simultaneously with the treatment

chemostats. Control chemostats containing both *E. coli* strains together without the predator were also run at high and low glucose input levels.

We manipulated productivity in this experiment by running replicate chemostats at different input concentrations of glucose. Each glucose input concentration represented a different level of productivity. Three groups of chemostats were run. The first group consisted of two replicate treatment chemostats with a glucose input concentration of 0.1 μg/mL, two replicate treatment chemostats with a glucose input concentration of 0.5 μg/mL, and six control chemostats. These resource levels were chosen so that our results could be compared to previous experiments at these concentrations (Bohannan and Lenski 1997, 1999). The second group consisted of two replicate treatment chemostats with a glucose input concentration of 0.09 μg/mL, two replicate treatment chemostats with a glucose input concentration of 0.5 μg/mL, and six control chemostats. The third group consisted of two replicate treatment chemostats with each of the following glucose input concentrations: 0.07, 0.08, 0.09, 0.10, 0.11, and 0.12 μg/mL and four control chemostats. The productivity levels in groups two and three were chosen to explore productivity levels slightly lower and slightly higher than 0.1 μg/mL, in an effort to determine the relationship between productivity and the rate of decline. We ran groups 1 and 2 for 200 h and group 3 for 100 h.

The population densities of the *E. coli* strains and phage T2 were estimated twice daily in groups 1 and 2 and daily in group 3. Population densities were estimated by dilution and plating. REL607 cells were plated on Davis minimal agar supplemented with 2×10^{-3} μg thiamine hydrochloride/mL and 4×10^{3} μg/mL arabinose (this media allows growth of REL607 but not REL6584, since REL6584 cannot utilize arabinose). Bacteriophage T2 was plated on a lawn of REL607 using Davis minimal agar and the plate count technique described by Carlson and Miller (1994). REL6584 cells were plated on Davis minimal agar supplemented with 2×10^{-3} μg thiamine hydrochloride/mL and 4×10^{3} μg/mL glucose; a concentrated phage T4 lysate was mixed with each sample to kill REL607 cells before plating.

The relative success of the two prey populations was compared by calculating the fitness of the less vulnerable prey relative to the more vulnerable prey in each chemostat. The relative fitness was defined as the slope of $\ln(N_B/N_A)$ over time, where N_B is the population density of the less vulnerable prey and N_A is the population density of the more vulnerable prey.

In past laboratory studies, the vulnerability of *E. coli* to predation by T2 has been observed to change due to evolution (Lenski 1984). Therefore, in each chemostat we tracked the evolution of mutants with reduced vulnerability and complete invulnerability to T2. We estimated the total population density of mutants invulnerable to T2 in each chemostat by mixing concentrated phage T2 lysate with an aliquot of each chemostat sample and plating on minimal glucose. The fraction of these mutants derived from REL607 was estimated by mixing concentrated phage T2 lysate with an aliquot of each sample and plating on minimal arabinose (REL6584 cannot grow on minimal arabinose media). The fraction of the invulnerable mutants derived from REL6584 was then determined by subtraction of the REL607-derived mutants from the total. As described above, invulnerability to predation by phage T4 can be used as a marker for detecting partial invulnerability to phage T2. We estimated the density of mutants of REL607 that were partially invulnerable to T2 by mixing concentrated phage T4 lysate with an aliquot of each sample and plating on minimal arabinose. This media allows the growth of both partially invulnerable and completely invulnerable mutants; we estimated the density of partially invulnerable mutants by subtracting the density of invulnerable mutants (estimated as described above) from the total.

Mathematical Model

We modeled our experimental system using a modification of the model developed by Levin et al. (1977). We analyzed this model graphically and examined the behavior of the model numerically using STELLA II simulation software (High Performance Systems 1994).

Mathematical Model. The model consisted of four coupled differential equations,

$$\frac{dR}{dt} = (R_0 - R)\omega - \frac{\epsilon N_A \psi_A R}{K_A + R} - \frac{\epsilon N_B \psi_B R}{K_B + R},$$

$$\frac{dN_A}{dt} = \frac{N_A \psi_A R}{K_A + R} - \alpha_A N_A P - \omega N_A,$$

$$\frac{dN_B}{dt} = \frac{N_B \psi_B R}{K_B + R} - \alpha_B N_B P - \omega N_B,$$

$$\frac{dP}{dt} = \beta e^{-\tau\omega} \alpha_A N_A' P' + \beta e^{-\tau\omega} \alpha_B N_B' P' - \alpha_A N_A P - \alpha_B N_B P - \omega P.$$

In these equations, R is the concentration of glucose in the chemostat, N_A and N_B are the population densities of more vulnerable and less vulnerable bacteria, respectively, and P is the population density of the bacteriophage. The model parameters are defined in table 1. Those parameters

Table 1: Symbols used in the mathematical model

Symbol	Definition
R	Concentration of glucose in the chemostat
N_A	Population density of more vulnerable bacteria
N_B	Population density of less vulnerable bacteria
P	Population density of bacteriophage
R_0	Concentration of glucose in the reservoir
ω	Flow rate
ϵ	Growth efficiency (reciprocal of the bacterial yield)
ψ_A	Maximum specific growth rate of more vulnerable bacteria
ψ_B	Maximum specific growth rate of less vulnerable bacteria
K_A	Resource concentration at which the more vulnerable bacteria grow at one-half ψ_A
K_B	Resource concentration at which the less vulnerable bacteria grow at one-half ψ_B
α_A	Attack (i.e., adsorption) rate of bacteriophage on more vulnerable bacteria
α_B	Attack (i.e., adsorption) rate of bacteriophage on less vulnerable bacteria
β	Burst size of bacteriophage
τ	Latent period of bacteriophage
$e^{-\tau\omega}$	Fraction of bacteria infected at time t $-$ τ that has not washed out before lysing
N_A'	Population density of more vulnerable bacteria at time $t - \tau$
N_B'	Population density of less vulnerable bacteria at time $t - \tau$
P'	Population density of bacteriophage at time $t - \tau$

that are specific to the more vulnerable population are followed by the subscript *A*, and those that are specific to the less vulnerable population are followed by the subscript *B*. Note that equations describing the dynamics of infected bacteria could also be written. Such equations were not included in this model because infected bacteria cannot be easily tracked in the chemostats. The inclusion of infected cell populations has very little effect on the dynamics of the other populations. The parameter values we used for this model are listed in table 2.

This model assumes an open and completely mixed environment. Growth-limiting resource flows into the environment at concentration R_0 and rate ω. Bacteria, phage, and unutilized resource flow out of the habitat at this same rate. Bacteria multiply at a per capita rate that is a hyperbolic function of the resource concentration in the environment ($\psi R/[K + R]$; Monod 1949) and each cell replication consumes ϵ of the resource. Phage attack and kill bacteria at a per capita rate that is a linear function of the bacterial density (αN; nearly all models of phage-bacteria interactions assume a linear rather than saturating functional response because saturation occurs at very high prey densities that are seldom reached in the laboratory or the field; Stent and Wollman 1952; Bohannan and Lenski 1999). Each attack is lethal and results in the release of β phage progeny after a time lag of τ. Phage reproduce at a rate that is a function of the number of progeny produced from each *E. coli* attacked (β), the fraction of bacteria attacked at time $t - \tau$ that has not flowed out of the chemostat before the phage progeny are released ($e^{-\tau\omega}$), the attack rate at time $t - \tau$ (αN) and the phage density at time $t - \tau(P')$.

Table 2: Parameter values used in the mathematical model

Symbol	Parameter value	Source of estimate
R_0	Either .1 or .5 μg/mL	Manipulated experimentally
ω	.2/h	Manipulated experimentally
ϵ	2×10^{-6} μg	Bohannan and Lenski 1997
ψ_A	.7726/h	Vasi et al. 1994
ψ_B	.7027/h	Bohannan and Lenski 1999
K_A	.0727 μg/mL	Vasi et al. 1994
K_B	.123 μg/mL	Bohannan and Lenski 1999
α_A	2×10^{-7} mL/h	Lenski 1984
α_B	1×10^{-7} mL/h	Lenski 1984
β	98 viruses per bacterial cell infected	Levin et al. 1977
τ	.5 h	Levin et al. 1977

Graphical Analysis. We analyzed the model graphically as described by Leibold (1996). This approach consists of plotting zero net growth isoclines and net consumer-prey impact vectors for both prey types. The relationship between the impact vectors and the isoclines determines the overall relationship between community structure and productivity (fig. 2*B*). We calculated the net consumer-prey impact vectors for each prey type in our experimental system by taking the vector sum of the per capita feeding rate of each prey type (calculated as $\epsilon N_A \psi_A R/[K_A + R]$ or $\epsilon N_B \psi_B R/[K_B + R]$) and the per capita contribution of each prey type to predator growth (calculated as $\beta[\alpha_A P]$ or $\beta[\alpha_B P]$) as described by Leibold (1996).

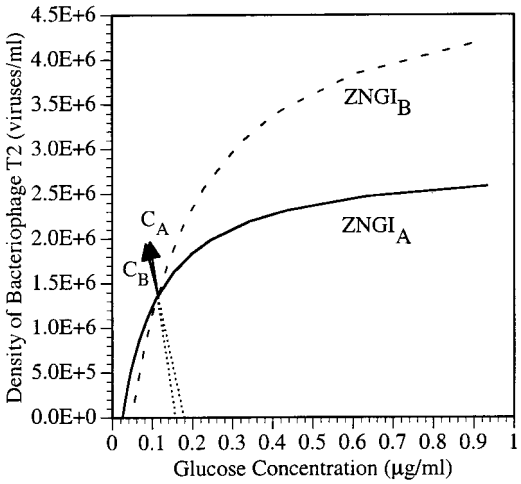

Figure 3: Graphical analysis of the community model for T2, prey bacteria, and glucose. The zero net growth isoclines and the consumer impact vectors for the more vulnerable prey (strain REL607) and the less vulnerable prey (strain REL6584) are shown. Key: zero net growth isocline for strain REL607 = $ZNGI_A$, zero net growth isocline for strain REL6584 = $ZNGI_B$; consumer impact vector for strain REL607 = C_A; consumer impact vector for strain REL6584 = C_B.

Numerical Simulations. We ran all numerical simulations using a time step of 0.05 h. We tested the sensitivity of the simulations to time step size by running replicate simulations at step sizes of 0.1, 0.05, and 0.025 h. Varying the size of the time steps had no detectable effect on the results of the simulations. We "sampled" the output of each simulation every 12 h (the approximate sampling interval of our experiments) to produce the predictions depicted graphically.

Results

Model Predictions

The graphical analysis of the model revealed that coexistence of both prey types would occur only within a very small range of productivity levels (fig. 3). The net consumer-prey impact vectors for the two types were very similar in slope (2.85×10^7 viruses/μg glucose for the more vulnerable prey; 1.99×10^7 viruses/μg glucose for the less vulnerable prey), resulting in predicted coexistence only within the range of 0.157 μg/mL glucose to 0.178 μg/mL glucose. Below this range, the more vulnerable prey were predicted to displace the less vulnerable prey. Above this range, the less vulnerable prey were predicted to displace the more vulnerable prey. In both cases, displacement of the inferior competitor is predicted to be due to a combination of resource and apparent competition.

Numerical simulations of the model are presented in figure 4. The two input concentrations we initially chose for this experiment are predicted to lie on either side of the narrow range of coexistence as described above. In chemostats with an input glucose concentration of 0.1 μg/mL, the more vulnerable prey is predicted to displace the less vulnerable prey (fig. 4*A*). In chemostats with an input glucose concentration of 0.5 μg/mL, the less vulnerable prey is predicted to displace the more vulnerable prey (fig. 4*B*). Both *E. coli* strains are predicted to persist for the duration of the experiment at both glucose input concentrations when the other competitor is absent (fig. 5).

Empirical Observations

The dynamics of T2 and *E. coli* prey populations in representative treatment chemostats are shown in figure 6. In

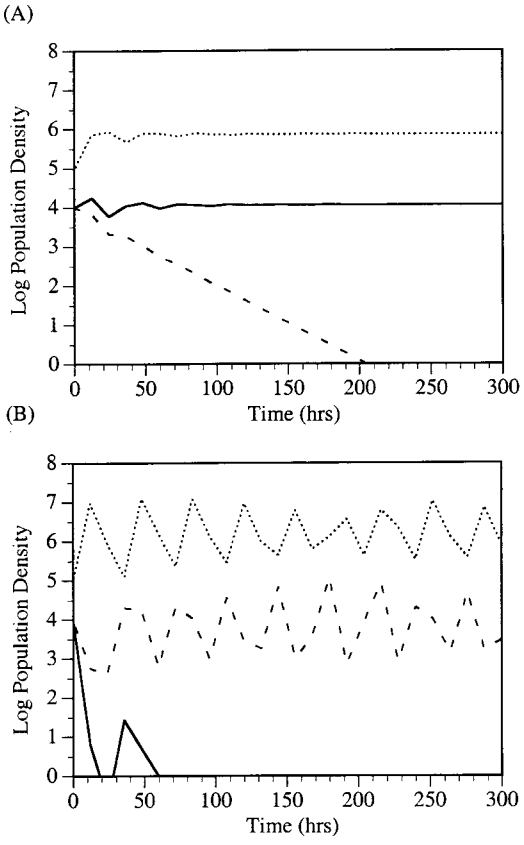

Figure 4: Population equilibria and dynamics predicted by the model for the treatment chemostats. Dynamics are from numerical simulations of the model, "sampled" at 12-h intervals. The population densities (viruses/mL or bacteria/mL) have been log-transformed. *A*, Model with a glucose input concentration of 0.1 μg/mL. *B*, Model with a glucose input concentration of 0.5 μg/mL. *Solid line* = more vulnerable *Escherichia coli* dynamics. *Dashed line* = less vulnerable *E. coli* dynamics. *Dotted line* = T2 dynamics.

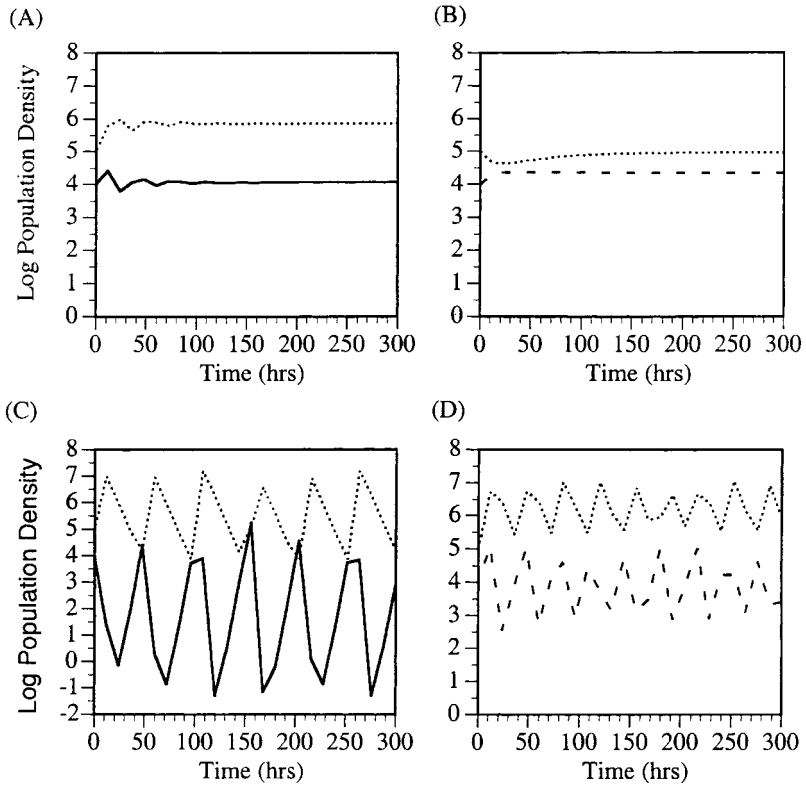

Figure 5: Population equilibria and dynamics predicted by the model for the control chemostats. Dynamics are from numerical simulations of the model, "sampled" at 12-h intervals. The population densities (viruses/mL or bacteria/mL) have been log-transformed. *A*, T2 and more vulnerable prey with a glucose input concentration of 0.1 μg/mL. *B*, T2 and less vulnerable prey with a glucose input concentration of 0.1 μg/mL. *C*, T2 and more vulnerable prey with a glucose input concentration of 0.5 μg/mL. *D*, T2 and less vulnerable prey with a glucose input concentration of 0.5 μg/mL. *Solid line* = more vulnerable *E. coli* dynamics. *Dashed line* = less vulnerable *E. coli* dynamics. *Dotted line* = T2 dynamics.

chemostats with an input glucose concentration of 0.1 μg/mL, the less vulnerable population declined in all four replicates (fig. 6*A*), as predicted by the model (fig. 4*A*). However, the rate of decline in several replicates was slower than predicted (e.g., cf. figs. 4*A* to 6*A*). Invulnerable *E. coli* mutants were not detected in any of the replicates of this treatment. In chemostats with an input glucose concentration of 0.5 μg/mL, the more vulnerable prey initially declined in all four replicates (fig. 6*B*), as predicted by the model (fig. 4*B*). The evolution of invulnerable *E. coli* (from a "less vulnerable" ancestor) occurred in all four replicates of this higher glucose treatment. The invasion of the chemostats by these invulnerable mutants initially halted the decline in density of the more vulnerable *E. coli*. However, once the invulnerable *E. coli* population reached its equilibrium, the more vulnerable *E. coli* resumed their population decline in three of the four replicates. The phage persisted in all four replicates of the higher glucose treatment, including the three replicates where the invulnerable *E. coli* population reached its equilibrium and the more vulnerable *E. coli* declined. The persistence of phage in these three chemostats indicates that a minority population of the less vulnerable *E. coli* persisted in these chemostats as well.

Fitness estimates of the less vulnerable prey relative to the vulnerable prey for all treatment chemostats are presented in figure 7. The relative fitness of the less vulnerable prey was positive in all four of the higher productivity chemostats (0.5 μg/mL) and negative in all 16 lower-productivity chemostats (glucose input concentrations of 0.07–0.12 μg/mL). This result is highly unlikely to occur due to chance (one-tailed $P = .00021$, by Fisher's exact test), and it is consistent with the theoretical predictions (fig. 3).

Control chemostats with the viral predator T2 and each of the *E. coli* prey strains were run simultaneously with the treatment chemostats. The dynamics of the T2 and *E. coli* populations in representative chemostats are shown in figure 8*A*–8*D*. All populations persisted in all chemostats. Neither less vulnerable nor invulnerable *E. coli* mutants

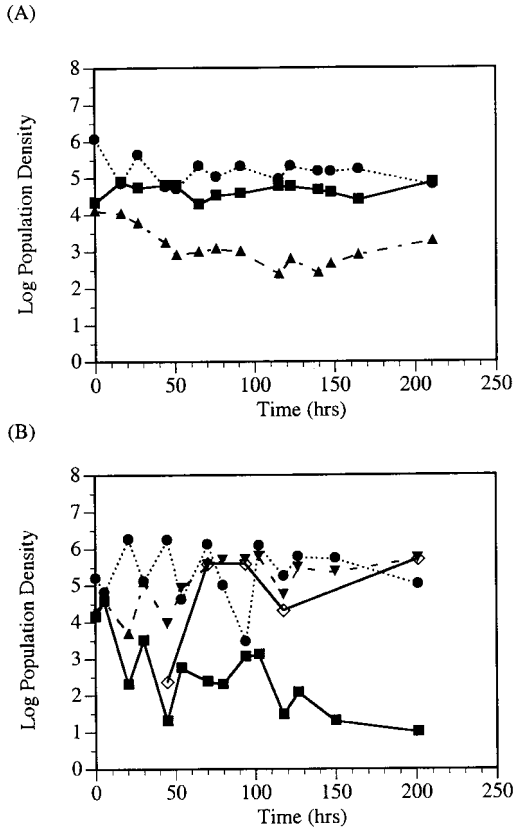

Figure 6: Dynamics in treatment chemostats containing more vulnerable *Escherichia coli* (*squares*), less vulnerable *E. coli* (*triangles*), and bacteriophage T2 (*circles*) in chemostats supplied with media containing different amounts of glucose. The dynamics of invulnerable mutants derived from the less vulnerable strain are indicated with open diamonds. Once these mutants invaded the chemostats, we were no longer able to track the less vulnerable *E. coli* directly; only the sum of the less vulnerable and the invulnerable *E. coli* (*inverted triangles*) could be quantified. The population densities (viruses/mL or bacteria/mL) have been log-transformed. *A*, 0.1 μg/mL glucose. *B*, 0.5 μg/mL glucose.

were detected in the lower glucose controls (0.1 μg/mL and below); however, both kinds of mutants were eventually detected in the higher glucose controls. Control chemostats containing the two *E. coli* strains together without the predator were also run. The dynamics of the *E. coli* populations in representative chemostats are shown in figure 8*E* and 8*F*. The less vulnerable strain declined in all replicates. The rate of decline at low glucose input concentrations was not significantly different from the rate of decline at high glucose input concentrations ($t = 1.3459$, df = 3, $P = .2710$).

Discussion

Ecologists have demonstrated theoretically that productivity can determine the relative importance of competition and predation in determining community structure. The overall patterns predicted by this theory are consistent with some field observations; however the theory has not previously been tested directly. We tested this theory using chemostat communities of bacteria and bacteriophage. We assembled communities with two prey populations that shared a common resource and a common predator. Productivity was manipulated in this experiment by running replicate chemostats with different input concentrations of resource. Our observations of these communities were consistent with the theoretical predictions. At lower productivity, we observed that the population density of the inferior competitor for resources declined, even though it was less vulnerable to predation. At high productivity, we observed that the population density of the more vulnerable prey declined, even though it was the superior competitor for resources. It is likely that in both treatments the decline of the inferior competitor was due to a combination of resource and apparent competition; however we were unable to distinguish between these two mechanisms experimentally because we did not estimate resource concentration in the chemostats.

The rate at which the less vulnerable population de-

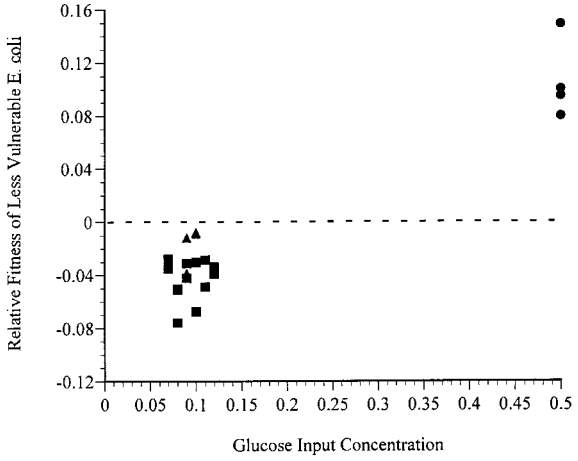

Figure 7: Fitness of less vulnerable *Escherichia coli* relative to vulnerable *E. coli* in chemostats that contain phage T2. Relative fitness is defined as the slope of $\ln(N_B/N_A)$ over time, where N_B is the population density of the less vulnerable prey and N_A is the population density of the more vulnerable prey. Values will be negative if N_A is competitively dominant, positive if N_B is competitively dominant. Relative fitness was estimated over the first 45 h of competition for the higher-productivity treatments because invulnerable mutants arose in these treatments and changed the dynamics of the populations after hour 45. Relative fitness was estimated over the entire duration of the lower-productivity treatments (200 h for groups 1 and 2 and 100 h for group 3) because invulnerable mutants did not arise in these chemostats. Key: group 1 and 2 high-productivity treatments (*circles*), group 1 and 2 lower-productivity treatments (*triangles*),and group 3 lower-productivity treatments (*squares*).

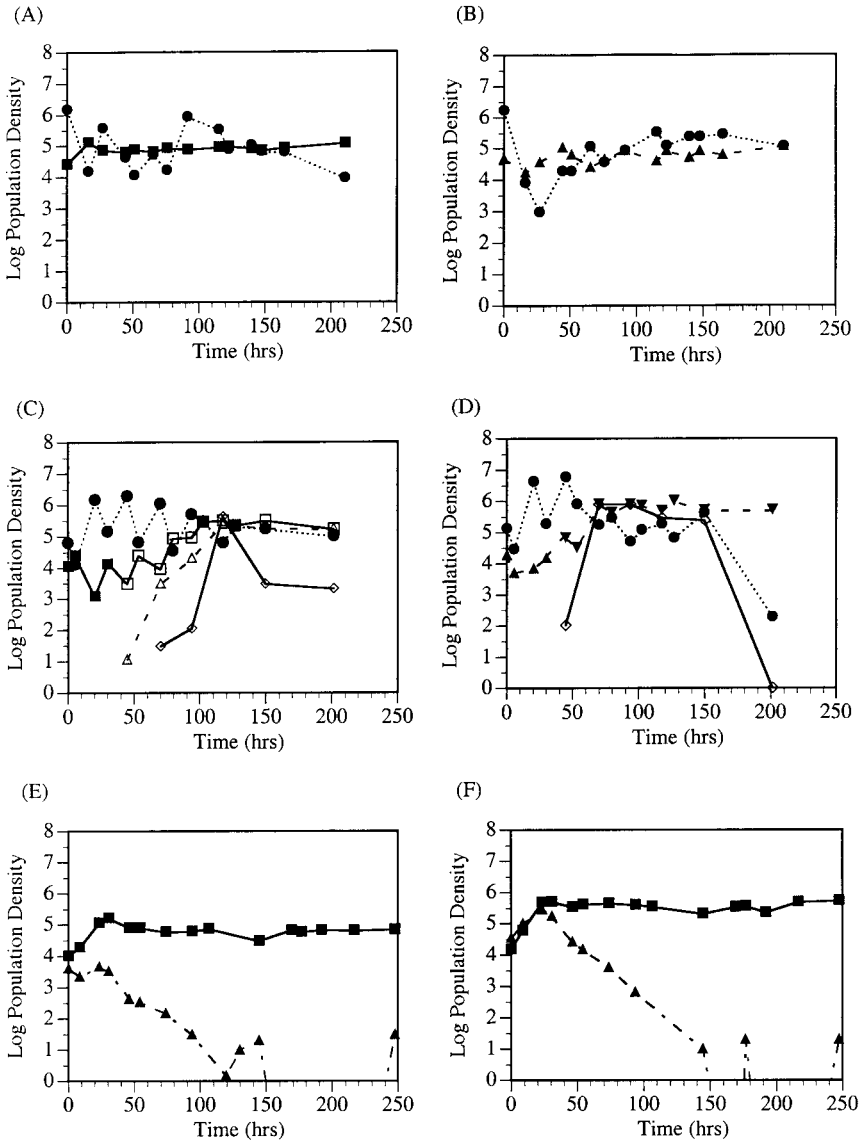

Figure 8: Dynamics in control chemostats supplied with media containing either 0.1 μg/mL or 0.5 μg/mL of glucose. The population densities (viruses/mL or bacteria/mL) have been log-transformed. *A*, Bacteriophage T2 (*circles*) and more vulnerable *Escherichia coli* (*squares*) at 0.1 μg/mL. *B*, Bacteriophage T2 (*circles*) and less vulnerable *E. coli* (*triangles*) at 0.1 μg/mL. *C*, Bacteriophage T2 (*circles*) and more vulnerable *E. coli* (*squares*) at 0.5 μg/mL. Less vulnerable *E. coli* mutants (*open triangles*) and invulnerable *E. coli* mutants (*open diamonds*) arose through spontaneous evolution in these chemostats. Once these mutants invaded the chemostats, we were no longer able to track the more vulnerable *E. coli* directly; only the total *E. coli* (*open squares*) could be quantified. *D*, Bacteriophage T2 (*circles*) and less vulnerable *E. coli* (*triangles*) at 0.5 μg/mL. Invulnerable *E. coli* mutants (*open diamonds*) arose through spontaneous evolution in these chemostats. Once these mutants invaded the chemostats, we were no longer able to track the less vulnerable *E. coli* directly; only the total *E. coli* (*inverted triangles*) could be quantified. *E*, More vulnerable *E. coli* (*squares*) and less vulnerable *E. coli* (*triangles*) at 0.1 μg/mL. *F*, More vulnerable *E. coli* (*squares*) and less vulnerable *E. coli* (*triangles*) at 0.5 μg/mL.

clined in our initial low productivity treatments (the 0.1 μg/mL treatments in group 1) was slower than predicted, and the less vulnerable prey did not go extinct during the experiment, contrary to predictions (e.g., fig. 6*A*). The persistence of all three populations in these chemostats led us initially to suspect that the range of glucose concentrations that allowed coexistence might actually be lower and/or broader than originally predicted and that our low productivity treatment might actually fall within the coexistence range. This might occur, for example, if our estimates of model parameters were inaccurate or if these parameters varied with productivity in our system. To de-

termine if the range of coexistence was indeed broader (as well as lower) than predicted, we repeated our experiment using input concentrations of glucose that were slightly lower and slightly higher than 0.1 μg/mL. Concentrations of 0.07, 0.08, 0.09, 0.10, 0.11, and 0.12 μg/mL glucose were used. However, we observed that the less vulnerable prey declined relative to the more vulnerable prey in all of these treatments, and at rates faster than the initial low productivity treatment (fig. 7), suggesting that our original predictions were indeed accurate.

Why then did we see such slow rates of decline in our initial low productivity treatments? One possible explanation emerges from inspection of figure 7. Of the lower productivity chemostats (i.e., those with glucose input concentrations of 0.07–0.12 μg/mL), the chemostats in which the less vulnerable prey had the highest relative fitness (i.e., the slowest decline) are all chemostats run for the longest duration (200 h). In these chemostats, the rate of decline appeared to decrease with time (e.g., fig 6*A*). In fact, if we calculate the relative fitness of the less vulnerable prey in these chemostats over the first 100 hours only, it is much lower than when estimated over the entire 200 h (data not shown). What might be causing this decrease in the rate of decline? We can think of three possibilities. First, growth of prey on the chemostat wall may have prevented complete exclusion of the less vulnerable prey. Other researchers have observed that wall growth can interfere with competitive exclusion (Chao and Ramsdell 1985). We did not observe growth on the chemostat walls in these experiments, but growth need not be visible to have an effect. Migration of prey from wall populations into the chemostat vessel could prevent the less vulnerable prey from declining below a certain level. Such an effect might not be noticeable until prey density approached this minimum and, thus, might not be obvious in the shorter duration experiments. A second possible explanation is that evolutionary adaptation within the less vulnerable prey population reduced the magnitude of the trade-off between reduced vulnerability and competitive ability. As the magnitude of the trade-off decreased, the rate of decline would also decrease. Such adaptations might take considerable time to arise and to sweep through the less vulnerable prey population and, thus, would be noticeable only in the longer duration experiments. A third possibility is that complex dynamics may have slowed competitive exclusion. The analysis of our model assumes a system at equilibrium; it is possible that transient departures from equilibrium (due, e.g., to minor environmental changes) could have slowed competitive exclusion.

In the high glucose treatment (0.5 μg/mL glucose), invulnerable *Escherichia coli* evolved and invaded all four replicates (e.g., fig. 6*B*). The invasion of the chemostats by the invulnerable mutants had a substantial effect on population dynamics, initially halting the decline of the more vulnerable *E. coli* population. However, this invasion had only a temporary effect on the outcome in the majority of the replicate chemostats; once the invulnerable *E. coli* population reached its equilibrium density, the more vulnerable *E. coli* resumed their population decline in three of the four replicates. These observations lead to two hypotheses concerning evolutionary change in our system. Our first hypothesis is that invasion of the chemostats by invulnerable mutants leads to changes in the resource level that, in turn, lead to changes in competitive dominance among the prey types. The invasion by the invulnerable mutants may have resulted in an overshoot of the invulnerable population's equilibrium density and a severe (but temporary) decrease in the glucose concentration in the chemostats. This severe decrease would confer a transient advantage to the more vulnerable *E. coli* because it is a superior competitor for glucose. This reversal of the competitive advantage would temporarily halt the decline of the more vulnerable *E. coli* population, as we observed. However, as the invulnerable population approached a stable equilibrium density the glucose concentration in the chemostat would rise, restoring the competitive advantage of the less vulnerable *E. coli* and resulting in the continued decline of the more vulnerable *E. coli*, as we observed in three of the four replicates of the high productivity treatment.

Why did we not observe the continued decline of the more vulnerable *E. coli* in all four replicates? This question leads to our second hypothesis: there are significant differences in the magnitude of the trade-off between invulnerability and competitive ability among the invulnerable mutants in the replicate chemostats. The magnitude of this trade-off would determine the equilibrium glucose concentration in the chemostats and, thus, would determine whether the less vulnerable or more vulnerable *E. coli* would be competitively superior after the invulnerable mutants reached their equilibrium. Invasion by a mutant with a relatively high cost of invulnerability would depress the resource level in the chemostat to a lesser degree, resulting in a competitive advantage for the less vulnerable *E. coli* relative to the more vulnerable strain, the persistence of the less vulnerable strain, and the decline of the more vulnerable strain. This is what we observed in three of the four replicates. Conversely, invasion by a mutant with a relatively low cost of invulnerability would depress the resource level in the chemostat to a greater degree, resulting in a competitive advantage for the vulnerable *E. coli* relative to the less vulnerable strain and persistence of the vulnerable strain. This is consistent with our observations of one of the four replicates.

In summary, we have demonstrated, using a chemostat community of phage and bacteria, that the relative im-

portance of predation and competition changes with changing productivity. We observed that the more vulnerable prey population declined at higher productivity but not at lower productivity, as predicted by theory. Furthermore, we observed that the less vulnerable prey population declined at lower productivity but not at higher productivity, as predicted by theory. The rate of decline in some replicates was slower than predicted, and this led us to propose some possible hypotheses for this outcome. We also observed evolutionary change in our model community, and these observations led to additional hypotheses concerning the evolutionary ecology of shared predation. One of the advantages of working with microbial model communities is that such hypotheses are testable. It is possible, for example, to determine the effect of wall growth on population dynamics (Chao and Ramsdell 1985; Schrag and Mittler 1996), to measure the magnitude of the trade-off between invulnerability and competitive ability (Lenski and Levin 1985; Lenski 1988), and to track resource levels during invasion (e.g., by using a sensitive technique such as high-performance liquid chromatography), and in future work we will do so. By understanding the role such complexities play in a relatively simple laboratory community, we can better understand the roles they may also play in more complex communities outside of the laboratory.

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