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# Bacterial Predator-Prey Interaction at Low Prey Density

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A bacterial predator-prey interaction was studied using *Bdellovibrio* and bioluminescent prey bacteria. The attacking bdellovibrio causes decay of bioluminescence, which is correlated with bdellovibrio penetration into the prey. The behavior of the prey and predator populations over time was found to be well described by a Lotka-Volterra model. By using this model, the probability of bdellovibrio penetration after encountering a prey cell was found to be approximately 3.0%. The prey density required to give the bdellovibrios a 50% chance of survival was calculated to be at least $3.0 \times 10^6$ cells per ml, and the density required for population equilibria was calculated to be about $7 \times 10^5$ prey bacteria per ml. These values, not generally characteristic of natural habitats, suggest that the existence of *Bdellovibrio* in nature is limited to special ecological niches.

The simplest model for a prey-predator system is described by the classical Lotka-Volterra differential equations:

$$\frac{dN}{dt} = aN - \alpha NP \quad (1a)$$

$$\frac{dP}{dt} = -bP + \beta NP \quad (1b)$$

The prey population, $N(t)$, increases exponentially, limited by predation ($\alpha NP$); the predator population, $P(t)$, dies at a rate of $bP$ and at the same time grows at a rate which depends on the prey abundance ($\beta NP$). Thus, $a$ is the net growth coefficient of the prey, $b$ is the net extinction (death or inactivation) coefficient of the predator, $\alpha$ is the predation coefficient, and $\beta$ is the predator efficiency coefficient ($a$, $b$, $\alpha$, and $\beta$ are all non-negative quantities). These two equations are expressed in sequential oscillations: the abundance of predators rises when food is plentiful, but when they become sufficiently numerous the density of the prey falls, since growth of the prey cannot keep up with the pace of destruction. However, as the food supply becomes sparse, it limits further growth of the predator, and the predator population declines. This in turn provides an opportunity for the prey to proliferate once again.

Sequential oscillations predicted by this model have been observed in laboratory experiments (1, 10); however, they are not readily discernible in nature (1). Various known and unknown differences exist between laboratory and natural conditions. One of these concerns population density levels. Although the laboratory experimenter favors high-density cultures, spatially averaged natural densities are usually several orders of magnitude lower. The effect of prey density on the functional response of the predator cannot be simply predicted: a predator can be relatively less effective at high prey densities, or, according to other models, it may become increasingly efficient as prey numbers increase, until saturation eventually sets in (2, 5). A second factor is population distribution. Control of laboratory cultures is easier to achieve under homogeneous conditions, but a great deal of theoretical and experimental work indicates that non-homogeneity may play a crucial role in promoting the coexistence of predator and prey populations in natural ecosystems (3, 4, 6, 8, 9, 12, 15, 16, 22).

Our purpose was to study a prey-predator interaction under laboratory conditions at low prey densities, comparable to those which have been suggested as characteristic in natural environments (14). The system was composed of two marine bacteria: the predatory bacterium *Bdellovibrio* and its prey, a luminous *Photobacterium*. A bdellovibrio attacking a prey cell penetrates into its periplasmic space within approximately 10 min. At about the same time the light-emitting mechanism of the prey is damaged, and the intensity of the light of the suspension declines. This effect can be measured sensitively and accurately with suspensions of low cell densities that cannot be studied by other means. Moreover, the process need not be interrupted to determine the effect of the predator on the prey, since the intensity of the light can be directly monitored in the mixture.

In this paper we report the results of experiments intended to determine the predation coefficient $\alpha$. Based on this determination, we were able to estimate equilibrium populations using the Lotka-Volterra model. Our results confirm earlier speculations by Hespell et al. (7) that in order to maintain *Bdellovibrio* in the natural environment, the density of the prey, if dispersed homogeneously, should be higher than it usually is. We note that such predictions about predator-prey coexistence in nature can be made most reliably using a mathematical model, since it is otherwise difficult to combine prey and predator growth and death rates with the interaction factors measured experimentally or calculated from the laboratory data. In follow-up studies we intend to introduce heterogeneity approximating natural marine habitats into our experimental system in an effort to resolve the *Bdellovibrio* survival question.

## MATERIALS AND METHODS

**Bacterial strains and growth condition.** *Bdellovibrio* BM4 was isolated off the Mediterranean coast of Israel. Its properties as well as its growth conditions have been described (11). The prey bacterium used in the experiments, *Photobacterium leiognathi* E28, was isolated from the Red Sea (the Gulf of Elat) and was chosen because of its strong bioluminescence. It was grown on medium MPY (11) in a shaking water bath at 26°C for 24 h.

Both prey and predator were washed by centrifugation (5 min, 12,000 × *g*) and resuspended in buffer containing 0.01 M tris(hydroxymethyl)aminomethane, pH 7.4, and the following salts (in grams per liter): NaCl, 29.72; $MgSO_4 \cdot 7H_2O$, 6.16; $MgCl_2 \cdot 6H_2O$, 5.0; $CaCl_2 \cdot 2H_2O$, 1.47; KCl, 0.75 (Tris-salts buffer).

**Experimental procedure.** The cell concentration was adjusted turbidimetrically with a Klett-Summerson photometer. For each experiment, 0.9-ml samples of the prey suspension were distributed into glass scintillation vials. The experiment was started with the addition of 0.1 ml of bdellovibrio suspension or 0.1 ml of buffer for a control. The vials were incubated at room temperature (22 to 26°C) and shaken by hand once every 2 min before light reading.

Light intensity was measured by a photometer similar to that described by Mitchell and Hastings (13) (for prey suspensions of $10^5$ to $10^8$ cells per ml) or by a Packard scintillation counter (for prey suspensions of $10^3$ to $10^5$ cells per ml).

**Presentation of results.** Since control suspensions very often showed a decline in light intensity during the experiment, all results are presented in the form of the amount of light in the experimental vial as a percentage of the control vial. The time required to reduce the light to 50% of the initial ($t_{1/2}$) was taken from a log-log plot of the light intensity against time of the interaction. It was found that for slow interactions this plot produced straighter lines than a semilog plot and so could be used for extrapolation.

## RESULTS

Figure 1 shows the effect of *Bdellovibrio* attack on the bioluminescence of a high-density ($10^8$ cells per ml) *Photobacterium* suspension. At this density one can observe an initial increase in light, followed by an exponential decay 12 to 15 min after mixing prey and predator. The initial increase is only observed in dense suspensions and will not be dealt with in this report. On the other hand, the pattern of light decay is the same throughout the range of prey suspensions from $10^8$ to $10^3$ cells per ml.

The onset of light decay coincides with the beginning of measurable penetration of bdellovibrios inside the prey, suggesting that light extinction is caused by bdellovibrio penetration. To prove this point, bdellovibrios and prey bacteria ($10^8$ cells per ml) at a ratio of 5:1, 10:1, and 15:1 were mixed together in the presence of chloramphenicol (100 μg/ml). This antibiotic has been found to inhibit penetration of marine bdellovibrios without affecting their attachment, similarly to its effect on other, nonmarine bdellovibrios (20). These experiments showed that in the presence of chloramphenicol the initial transient increase in light intensity occurred as in the absence of the antibiotic, but no significant decay was measured for at least 30 min.

The rate of light decay is a function of the initial predator/prey ratio (input ratio [IR]): it is low at low IRs and increases with increased IR. The relationship between the bioluminescence decay and the IR for a suspension of $10^8$ prey bacteria per ml is shown in Fig. 2. A semi-

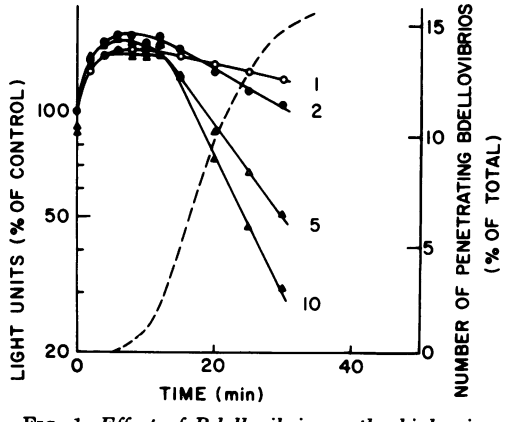


FIG. 1. *Effect of Bdellovibrio on the bioluminescence of prey bacteria. The density of the prey suspensions was $10^8$ cells per ml, and the IR was as noted on the figure. The broken line describes the kinetics of penetration of the bdellovibrios, based on data of A. Marbach (Ph.D. thesis, The Hebrew University, Jerusalem, 1977).*

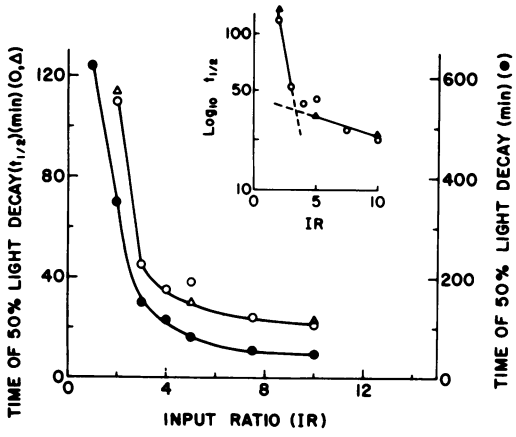


FIG. 2. *Effect of IR on the rate of bioluminescence decay of a prey suspension of $10^8$ cells per ml. The results of three separate experiments are described. Insert, Semilogarithmic plot of the results of two of the experiments.*

logarithmic plot of the same results (inset, Fig. 2) yields a broken line with two markedly different slopes. The point at which the slope changes was taken as the "saturating" IR. Results of experiments with prey densities ranging from $10^3$ to $10^8$ cells per ml show that the IR at which the rate of decay is "saturated" is quite constant for any given density: for a high-density prey population the value ranges from 3.0 to 3.5, and it becomes higher as the density of the prey decreases (Table 1).

The rate of light decay is also a function of cell density. Figures 3 and 4 show that with the same IR, $t_{1/2}$ varies by several orders of magnitude, depending on prey density. The decay rate in a population of a given density may vary somewhat from one experiment to another, depending on the activity of the bdellovibrio suspension used (always less than one order of magnitude, Fig. 2), but the variations are small compared to the range of rates measured for the full range of prey densities.

## DISCUSSION

The results described above reflect the fact that the probability of a bdellovibrio meeting its prey becomes smaller as prey density decreases. Does the interaction depend on chance collision only, or are there any special search mechanisms (such as chemotaxis) which would compensate to a degree for the sparseness of the prey population?

If the probability of a bdellovibrio meeting its prey is dependent on chance collision only, i.e., on the product of prey ($N$) and predator ($P$) densities, then the rate of prey extinction should be given by equation 1a, where $\alpha$ = (rate of encounter) × (probability of penetration of each encounter). Our data allow us to estimate $\alpha$ as follows: at high IR we may assume that the number of predators does not change significantly during the interaction. Recalling that prey (being suspended in buffer) do not reproduce ($\alpha = 0$), equation 1a becomes

$$\frac{dN}{dt} = -\alpha NP(0) \qquad (2)$$

Let $x(t)$ be the light emitted by the prey at time $t$, expressed as a percentage of that at time 0. We assume that the light emitted is propor-

TABLE 1. *Required IRs at different prey densities*

| Prey density (cells per ml) | IR required to obtain saturated decay rate[a] | Critical IR predicted from equation A3[b] |
|---|---|---|
| $10^8$ | 3.0–3.5 | 0.02 |
| $10^7$ | 7.5–8.0 | 0.2 |
| $10^6$ | 26–27 | 2 |
| $10^5$ | 80–90 | 20 |
| $10^4$ | 250 | 200 |
| $10^3$ | 2,000–2,500 | 2,000 |

[a] The experimental values were taken from a semilogarithmic plot of Fig. 2 (for a density of $10^8$ cells per ml) and of corresponding plots for lower prey densities.
[b] See Appendix 1.

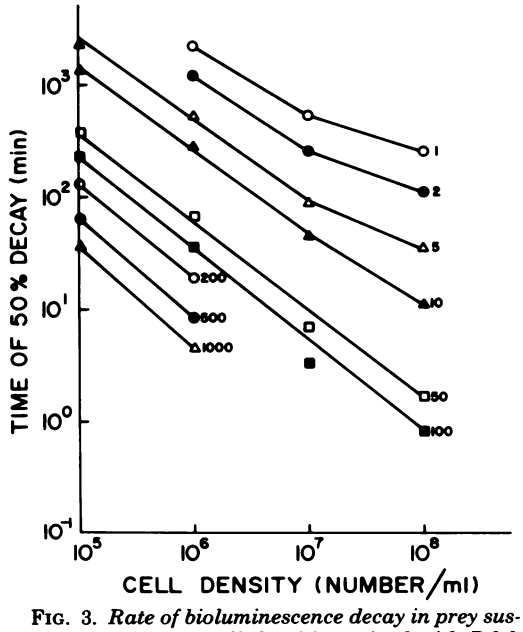


FIG. 3. *Rate of bioluminescence decay in prey suspensions of various cell densities, mixed with Bdellovibrio at IRs of 1 to 1,000 as noted on the figure. All experiments were done with the same bdellovibrio suspension.*

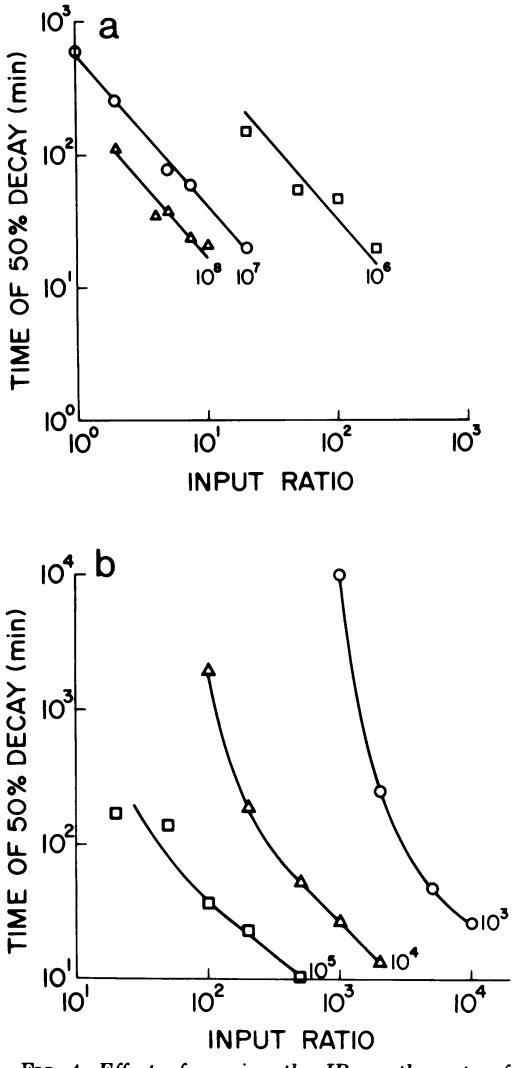


FIG. 4. *Effect of varying the IR on the rate of bioluminescence decay of prey suspensions of different cell densities. The cell density is noted on the figure. All experiments were done with the same bdellovibrio suspension.*

tional to the number of prey, i.e., that $x(t) = [N(t)/N(0)] \times 100$. Substituting for $N(t)$ in equation 2 leaves its form unchanged, and since $x(0) = 100$, its solution is:

$$x(t) = 100e^{-\alpha P(0)t}$$

or

$$\ln x(t) = \ln 100 - \alpha P(0)t$$

At time 0

$$P(0) = \mathrm{IR}\ N(0)$$

and by substitution we get

$$\ln x(t) = \ln 100 - \alpha \mathrm{IR}\ N(0)t$$

If $t_{1/2}$ is the time of 50% light decay, then

$$x(t_{1/2}) = 50$$

and by substitution we get

$$t_{1/2} = \frac{\ln 2}{\alpha \mathrm{IR}\ N(0)}$$

or taking logarithm

$$\ln t_{1/2} = \ln\ln 2 - \ln \alpha - \ln \mathrm{IR} - \ln N(0) \qquad (3)$$

Figure 3 shows the relationship between time to 50% decay and prey density for different IRs. Figure 4 shows the relationship between the time to 50% decay and the IR for different cell densities. It can be seen that at high prey densities and high IR [$N(0) \times \mathrm{IR} \geq 10^7$ per ml], the prediction of equation 3—that $\ln(t_{1/2})$ should decrease with unit slope with increasing ln IR and ln $N(0)$—is borne out. From the intercepts of the experimental lines with the vertical axis in Fig. 4a, we can estimate $\alpha = 3.0 \times 10^{10}$ per min.

It can also be seen that at low prey densities and/or at low IR, deviation from this prediction is apparent. In Appendix 1 we show that this deviation can be largely explained by taking the predator extinction rate coefficient, $b$, into account. Indeed, our experiments are consistent with the estimate $b = 1.0 \times 10^{-3}$ per min. This corresponds to a *Bdellovibrio* half-life of about 12 h and agrees with the length of time available for a bdellovibrio to find a potential prey estimated on the basis of viability measurements during starvation (7).

Hespell et al. (7) estimate the rate of encounter between predator and prey based on chance collision alone to be approximately $1.0 \times 10^{-8}$ per min. Our estimate of $\alpha$ thus yields a probability of predator penetration into prey of approximately 3.0% ($3.0 \times 10^{-10}/1.0 \times 10^{-8}$). A simulation, described in Appendix 3, confirmed the general correctness of the above estimates.

We note that although chemotaxis is not ruled out by our results, they suggest that it is unlikely to play a role in our experiments. This is because any increase in the rate of encounter due to chemotaxis would require a concomitant decrease in the probability of penetration (to be compatible with the obtained estimate of $\alpha$). However, this probability already seems rather low. Recent experimental evidence also confirms that bdellovibrios, although capable of a weak chemotaxic response to certain chemical compounds, do not show chemotaxis to potential

prey bacteria (17). Thus, we feel justified in accepting the null hypothesis that encounter is a purely chance process.

These results suggest that, under conditions such as those used in this study, interaction between *Bdellovibrio* and its prey depends on chance collision only. They also show that to obtain an inactivation of a significant fraction of a low-density prey population, either a high IR or a long time is necessary, neither of which is easy to find in natural aquatic environments.

*Bdellovibrio* counts from seawater yielded numbers in the range of 0.1 to 50 per ml (18, 21). The number of potential prey bacteria was not determined in those experiments but can be estimated to be in the range of $10^3$ to $10^5$ per ml (14). Thus, natural IRs may be much lower than 1. Based on chance encounter and a 10-h half-life, Hespell et al. (7) calculated that *Bdellovibrio* could not be maintained in a population of less than $1.5 \times 10^5$ prey organisms per ml.

Appendix 2 uses our estimates of $\alpha$ (predation coefficient) and $b$ (*Bdellovibrio* extinction rate coefficient) to show that a prey density of at least $3.0 \times 10^6$ per ml is required to give the *Bdellovibrio* a 50% chance of survival. Thus our calculation, which is more rigorous than that of Hespell et al. (7), requires yet an order of magnitude higher prey density for *Bdellovibrio* existence. Moreover, using the Lotka-Volterra equations (equation 1), we calculate that for population equilibria $7 \times 10^5$ prey bacteria per ml are required. These results are in obvious discordance with the above-quoted estimates for aquatic environment. A longer half-life for the bdellovibrios, such as might be achieved by cyst formation (19), would not appreciably change the above estimate: if the *Bdellovibrio* half-life were as high as 100 h, this would only reduce the required prey density to about $10^5$ per ml.

Still, bdellovibrios do exist in the marine environments, and their characteristics (11, 18) rule out the possibility that they are of nonmarine origin. Our results suggest that bdellovibrios could exist in seawater only if local concentrations of high-density prey bacteria exist, even though the average prey density is low. Indeed, it is known that most of the bacteria in the aquatic environments are attached to the surfaces of suspended matter, live as epiphytes on planktonic algae, or are concentrated on the surfaces of aquatic plants and animals (3, 15). High local concentration of heterotrophic bacteria has also been found in the thin upper layer of the sediment (4), as well as in thin layers near the air-water interface (16) or in the thermocline between the epilimnion and hypolimnion of lakes (14). Such areas could provide the required niche for the development of the bdellovibrios from which they could be dispersed in the large volume of water. Pollution of the waters either with bacteria (sewage) or with nutrients which cause multiplication of the microbial flora already present in the sea could lead to further enrichment of the bdellovibrios. According to our results, local concentrations of prey bacteria would be quickly annihilated by a few predator bacteria. However, current work in theoretical ecology (Nisbet and Gurney, personal communication; 22) shows that although the Lotka-Volterra model would not be applicable, such a situation may be globally stable under appropriate population exchange conditions. For example, transportation of predator "infections" from concentration to concentration might be affected by means of turbulence and mixing currents or by fish. Future experiments should aim to subject these hypotheses to experimental test.

## APPENDIX 1

If in equation 1b we do not neglect the natural predator extinction rate (but continue to neglect the reduction of the predator population due to penetration into prey), we obtain the equation

$$\frac{dP}{dt} = -bP$$

This is readily solvable for $P(t)$, which can then be substituted into equation 1a to yield

$$\frac{dN}{dt} = -\alpha N P(0) e^{-bt}$$

This equation has the solution

$$\ln N(t)/N(0) = \frac{\alpha P(0)}{b}(e^{-bt} - 1) \qquad \text{(A1)}$$

and setting $N(t_{1/2}) = N(0)/2$ and IR $N(0) = P(0)$ yields

$$t_{1/2} = \frac{1}{b} \ln \left[ \frac{1}{1 - \dfrac{b \ln 2}{\alpha \mathrm{IR} \cdot N(0)}} \right] \qquad \text{(A2)}$$

We note that it is the product IR·$N(0)$ rather than the individual factors IR and $N(0)$ which determines the value of $t_{1/2}$. Figure 5 displays a typical curve of $t_{1/2}$ versus IR·$N(0)$. Note that $t_{1/2}$ becomes infinite when IR·$N(0)$ = ($b\ln 2/\alpha$), and decreases as IR·$N(0)$ increases. This reflects the fact that for small enough predator densities, the death rate of predators becomes an important factor in the interaction. In the extreme, densities are so low that predators die out before half of the prey population is penetrated.

For fixed initial prey density $N(0)$, let $\mathrm{IR}_{\mathrm{critical}}$ be the value of IR at which $t_{1/2}$ becomes infinite (the critical value of IR). Then

$$\mathrm{IR}_{\mathrm{critical}} = \frac{b \ln 2}{N(0)\alpha} \qquad \text{(A3)}$$

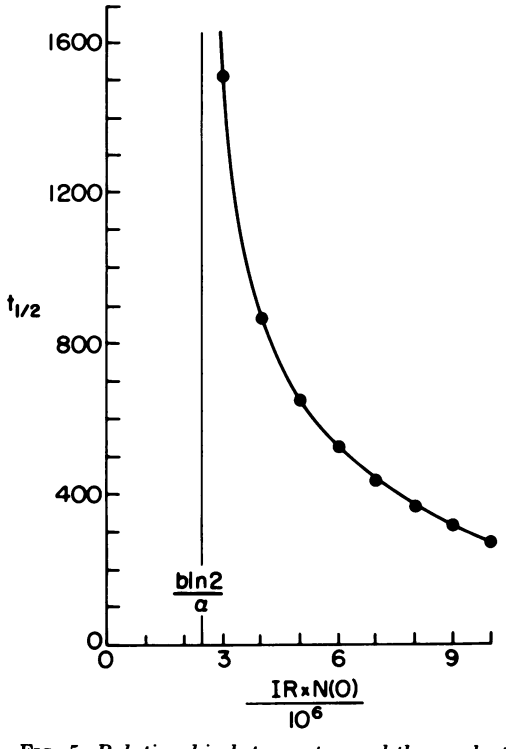


FIG. 5. *Relationship between $t_{1/2}$ and the product $N(0) \cdot IR$ as determined by equation A2.*

In Table 1 we compute values of $IR_{critical}$ from equation A3, using $b = 1.0 \times 10^{-3}$ per min (a half-life of ca. 12 h). These are juxtaposed with values of IR, obtained experimentally, at which there is a marked increase in the measured value of $t_{1/2}$ (see Results and Discussion). We note that $IR_{critical}$ is always less than the saturating value of IR, as would be expected if our hypothesis is correct. At low prey densities where predator death rate is significant, the two values are quite close.

## APPENDIX 2

It can be shown that the probability that a *Bdellovibrio* survives until penetrating a prey is

$$p = \frac{1}{1 + bT}$$

where $p$ is the probability of survival, $b$ is the death rate coefficient, and $T$ is the mean time to encounter and penetration; i.e., $T = 1/\alpha N$. (This assumes that the death and penetration processes are independently exponentially distributed random variables.) With $b = 1.0 \times 10^{-3}$ per min and $\alpha = 3.0 \times 10^{-10}$ per min, we find that a prey density $N$ of at least $3.0 \times 10^6$ per ml is required to give the predator a 50% chance of survival.

The equilibrium densities predicted by the Lotka-Volterra equations (equations 1a and 1b) are calculated as follows. Setting the left-hand sides to zero, we obtain

$$P_{eq} = \frac{a}{\alpha}$$

and

$$N_{eq} = \frac{b}{\beta}$$

where $P_{eq}$ and $N_{eq}$ are the predator and prey equilibrium densities, respectively.

We shall assume that predator penetration into prey results in five new predators on the average (consistent with laboratory findings) so that $\beta = 5\alpha$. Estimating the generation time of bacteria in marine environments is more problematic. Thus, we shall use values of 20 min, 10 h, and 100 h (with corresponding $a = 0.05$, $1.2 \times 10^{-3}$, and $1.2 \times 10^{-4}$ per min, respectively) ranging from conditions holding in the laboratory to maximum estimates in nature. We obtain the results given in Table 2. We see that an order of magnitude lower prey density is required for system equilibrium than for 50% survival of predator. Also, the predator equilibrium, even under the slowest growth conditions of the prey, greatly exceeds all estimates for aquatic environments (50 per ml).

## APPENDIX 3

In the simulation model, prey are initially located at all intersections of a planar grid. Each predator performs a random walk over the grid until it succeeds in penetrating a prey or its lifetime expires.

At each contact with a prey, there is a probability of penetration. If penetration occurs, the prey and predator are removed from their respective populations, but the light is not extinguished until 12 min (the experimentally determined value) later. The predator random walk is carried out as follows: after remaining on a prey a short time, the predator chooses in equally likely fashion a next site from its closest neighbors. After a time sampled from an exponential distribution with mean = (rate of encounter)$^{-1}$, the predator is placed at the selected site and the cycle repeats.

To calibrate the simulation with an experiment with given IR, IR predators are initially placed at each site. In the simulation model, the initial prey density is not represented directly in the grid spacing but is represented equivalently in the rate of encounter parameter, which is set to $10^{-8} \times N(0)$ per min$^{-1}$ (the pure chance collision estimate discussed in the text). Each predator's lifetime is sampled initially from an exponential distribution with mean $b^{-1}$, where $b$ is death rate estimated in Appendix 1. Using a 10 by 10 grid and probability of penetration as estimated in the text, we obtained excellent agreement between time re-

TABLE 2. *Predator and prey equilibrium densities calculated for different generation times of the prey*

| Generation time | Density (cells per ml) | |
|---|---|---|
| | $P_{eq}$ | $N_{eq}$ |
| 20 min | $1.7 \times 10^8$ | $7.0 \times 10^5$ |
| 10 h | $4.0 \times 10^6$ | $7.0 \times 10^5$ |
| 100 h | $4.0 \times 10^5$ | $7.0 \times 10^5$ |

quired to 50% light decay in the simulation and that derived from the data for almost all settings of IR and $N(0)$. Deviations were apparent, however, for very low IR and $N(0)$.

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