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STUNTED GROWTH AND STEPWISE DIE-OFF IN ANIMAL COHORTS

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Abstract.—A model of an animal cohort foraging on logistically growing food is analyzed. The problem is captured in three differential equations, one for food density and two for the state of the animal cohort, keeping track of body weight and number of individuals, respectively. When the animals efficiently exploit their food to low densities, the model produces cycles. The cycles differ markedly from those produced by traditional predator-prey models. Consumer decline is associated with starvation mortality when individuals lose too much weight. This condition causes a stepwise decline in individual number, each step corresponding to one cycle. Because the survivors of each starvation period grow and because larger animals have lower weight-specific metabolic rates, the nature of the cycles changes over time. They acquire a slow-fast character because of the increasing difference between food and consumer speed and show distinct catastrophic features, as the fast phases are caused by jumping between an over- and underexploited state of the food population. The cycles may either continue toward extinction of the cohort or damp out in a stable state characterized by stunted individual growth. Data from fish communities are closely in line with the specific predictions from this generic model about patterns of die-off and stunted growth. The model behavior is robust. It does not depend on the type of functional response or the way in which mortality increases with individual loss. Furthermore, the same patterns are obtained from an elaborate, realistic individual-based model, which indicates that the results are not artifacts of simplifications like considering all individuals equal and assuming only one food source.

Ever since the work of Lotka and Volterra (Lotka 1925; Volterra 1926), predator-prey dynamics has been among the most popular topics in theoretical ecology (see, e.g., Rescigno and Richardson 1967; MacArthur 1972; May 1973; Tanner 1975). In almost all studies it is assumed that the state of a population can be characterized by some general quantity representing either number or biomass. Obviously, this is a rather crude simplification. The size structure of populations is often far from constant, and since small animals differ in many respects (metabolic rates, prey selection, predators, etc.) from large ones, this implies that the parameters of lumped models should actually vary considerably over time. Putting that into the model, however, requires awkward constructions with many underlying assumptions. An elegant way to overcome these problems is to construct models based on the characteristic properties of individual organisms. This

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can be done in several ways (Metz and Diekman 1986; Caswell 1989; DeAngelis and Rose 1992), but the conceptually simplest approach is to follow each individual separately (*i*-state configuration models sensu Metz and Diekman 1986). The advantages of the approach are numerous (Huston et al. 1988; Hogeweg and Hesper 1990). The parameters, such as speed of movement and amount eaten per day, are of the type typically measured by experimental biologists, and the models can often explain complex patterns on the population level from such readily available knowledge of individual biology. There are, however, also some disadvantages associated with individual-based modeling approach. Obviously, the computational costs of simulating large populations are generally very high, although there are some ways of reducing this problem (Rose et al. 1993; Scheffer et al. 1995). More importantly, the stochasticity inherent to most individual-by-individual (*i*-state configuration) models and the fact that simulation is the only way of revealing their properties make it difficult to arrive at generic results.

In this article we explore the importance of dynamics of body weight and individual metabolism of the consumer without entering the domain of real individual-based modeling with its associated problems. By focusing on the development of one cohort and assuming all individuals within the cohort to have the same size, we are able to describe the system by a simple three-differential equation model. It is shown that the dynamics of such developing cohorts should differ markedly from what one would expect on the basis of traditional predator-prey models.

The current analysis is limited to a system consisting of one consumer cohort and its food. The dynamics of a reproducing population with multiple cohorts competing for the same food will be treated in a separate article (M. Scheffer, unpublished results).

MODEL

The model describes a cohort of animals foraging on a single food population. It is analyzed using the program GRIND (De Boer 1983).

The dynamics of body weight, W (g), depend on consumption, C (g d^{-1}); respiration, R (g d^{-1}); and an efficiency for converting food to body weight, e (g g^{-1}):

$$dW/dt = e \cdot C - R. \tag{1}$$

Big animals have a lower metabolism per gram body weight than small ones. More precisely, respiration and consumption tend to be proportional to W^k with $k < 1$ (see, e.g., Straskraba and Gnauck 1985). Although k differs with the species, it is often close to 2/3. We formulate respiration relative to that of an animal of 1 g (r_{ref} [g d^{-1}]):

$$R = r_{ref} \cdot W^{2/3}. \tag{2}$$

The daily ration per gram body weight decreases with the size of the animal in the same way as respiration, but the actual uptake is also a function of food availability. Although most animals show a simple saturating functional response (Type II) in laboratory situations, a sigmoidal response (Type III) seems more

appropriate to mimic most field situations. Prey switching and prey refuges are well-known causes of sigmoidal responses (Begon et al. 1986), but even in the common situation in which an animal concentrates on patches with higher prey density, the resulting functional response becomes sigmoidal (May 1978). We therefore use a Type III functional response formulated as a Hill function of food, F(g m^{-3}), with a half-saturation constant, H (g m^{-3}):

$$C = c_{\text{ref}} \cdot W^{2/3} \cdot F^2/(F^2 + H^2) . \tag{3}$$

Since we follow the dynamics of only one developing cohort, reproduction is not considered and the dynamics of animal numbers, N(m^{-3}), are governed solely by mortality. In periods of food shortage, respiration can exceed consumption. The animals lose weight and are more likely to die. We define weight loss, L (g g^{-1}), relative to the maximum attained weight in the past, W_{max} (g):

$$L = (W_{\text{max}} - W)/W_{\text{max}} . \tag{4}$$

As observed for virtually all animals in nature, starvation mortality increases quite sharply to a certain maximum, m (d^{-1}), when a certain critical weight loss, L_{crit} (g g^{-1}), is exceeded. We formulate the increase of mortality as a Hill function of weight loss:

$$dN/dt = -m \cdot N \cdot L^p/(L^p + L_{\text{crit}}^p) . \tag{5}$$

The power (p) sets the sharpness of the sigmoidal increase of mortality around the critical weight loss. Although this is a crude way of summarizing the dynamics of energy budgets in animals, it captures the essentials for our current purposes.

Food dynamics are formulated in a simple logistic fashion with a carrying capacity, K (g m^{-3}); a maximum growth rate, g_{max} (d^{-1}); and losses due to consumption:

$$dF/dt = g_{\text{max}} \cdot F \cdot (K - F)/K - C \cdot N . \tag{6}$$

Although the model formulation is generic, we chose the default parameter setting to roughly represent young fish foraging on zooplankton ($g_{\text{max}} = 0.5$, $K = 50$, $m = 0.1$, $L_{\text{crit}} = 0.25$, $e = 0.4$, $c_{\text{ref}} = 0.25$, $H = 5$, $r_{\text{ref}} = 0.05$, $p = 5$), all biomasses being expressed as wet weights. Simulations start with an initial state of $N = 50$, $W = 0.1$, and $F = 50$ unless stated otherwise.

RESULTS AND DISCUSSION

Behavioral Repertoire

Simulation runs with the model have two possible final states: extinction of the consumer population or an equilibrium with both food and consumer present. Both states are typically preceded by a transient state of cycling characterized by stepwise die-off of the consumer population (figs. 1, 2). In the stable state, the growth of individuals is stunted. The animals are just able to maintain their body weight and neither starve nor grow.

A close-up on a smaller timescale (fig. 3) shows the biological mechanism

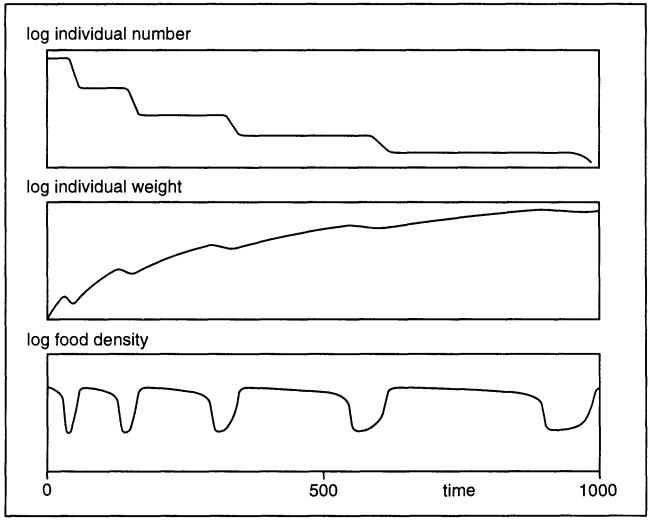

FIG. 1.—Simulation with the default parameter setting showing cycles of food depletion and mortality leading to extinction.

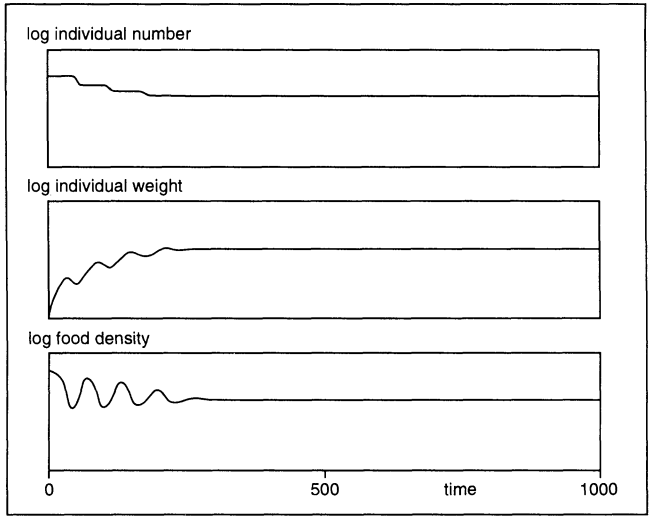

FIG. 2.—Simulation run with a higher half-saturation constant of the functional response (H = 10), causing the system to settle in a stable state with stunted individual growth.

behind the die-off cycles (fig. 4). The food level decreases because of increasing consumption by the growing individuals. When food is depleted below the minimum level required for consumption to balance respiration, the individual growth of consumers stops, and they start losing weight. When the critical weight loss is approached, starvation mortality starts taking its toll. When the grazing pressure on the food population is released sufficiently by the resulting consumer

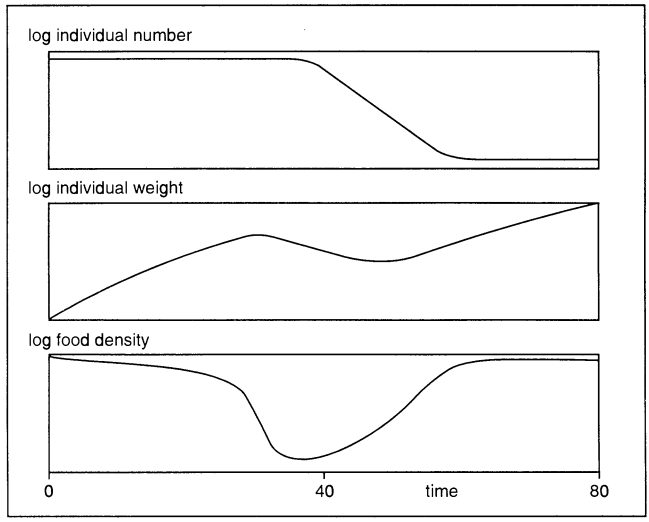

FIG. 3.–One-cycle period showing the timing of different phases

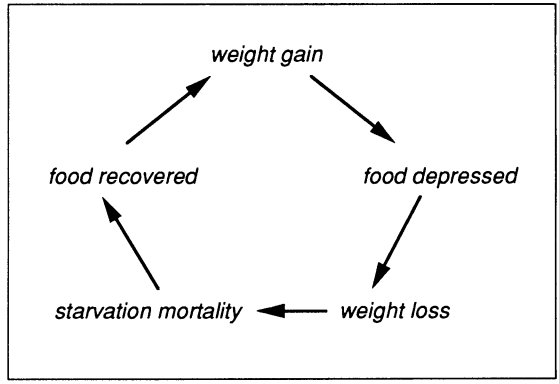

FIG. 4.—Mechanism behind stepwise die-off cycles

decrease, the food population starts to increase. This soon leads to high enough food levels for the consumer individuals to gain weight again. When they have gained enough weight, starvation mortality falls and a new cycle is set off.

Obviously, these cycles are not stable limit cycles, because their nature changes over time (fig. 1). In the course of the simulation the consumer cohort consists of increasingly fewer but larger individuals. Since weight-specific metabolism and consumption decreases with animal size, this implies that the "parameters of the predator" actually change over time.

Comparison to Traditional Predator-Prey Models

For the traditional two-differential equation predator-prey models, the construction of zero isoclines in the phase plane is often very informative. Unfortu-

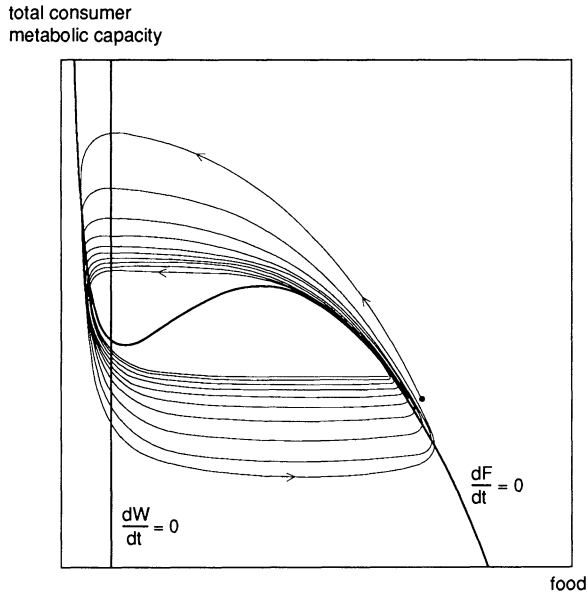

FIG. 5.—Phase plane of food density against the total metabolic capacity of the consumer cohort with the zero isocline of food and a trajectory of a 3,000-time-step simulation run ($H = 6$) showing how the system approaches a slow-fast cycle as individual size increases.

nately, there are some problems in applying that approach to the current model. The isocline for individual weight can easily be found. Since respiration and consumption change with weight in the model, in the same way there is a fixed food level at which net growth is zero. However, the derivative of the animal number is never positive in the current model without reproduction. The isocline of the third variable, food, should be constructed in a three-dimensional phase space since it depends on consumer weight as well as numbers. In order to be able to capture the behavior of the model in a two-dimensional phase plane with a fixed food isocline for comparison with traditional predator-prey models, we combine consumer numbers and individual weight in a new variable, the total metabolic capacity, M (g m^{-3} d^{-1}), of the cohort:

$$M = N \cdot c_{\text{ref}} \cdot W^{2/3}. \tag{7}$$

At first sight a trajectory of a long simulation run plotted in the new phase plane seems to settle at a flat type of limit cycle after a transient period of spiraling inward (fig. 5). However, the depicted behavior is somewhat different from that. Loosely phrased, we are actually looking at a limit cycle that shrinks with increasing individual weight of the consumer. The shown simulation is started on this cycle, but any other initial state quickly converges to it. The reason that this cycle becomes flatter as consumer animals grow bigger is that the weight gains and losses of the consumer become increasingly slower as larger animals have a lower consumption and respiration per unit of body weight, whereas the intrinsic

rates of the food population remain the same. This contrast in speed between the consumer and its food gives the system a distinct slow-fast character. Starting at the lower right point of one of the inner cycles, the system first moves up slowly along the food isocline. The consumer consumptive capacity increases because of individual growth, which depletes the food to an increasingly lower level. When the top of the food isocline is reached, a fast cycle phase brings the food into an overexploited state on the left side of the isocline for individual weight. A slow decrease in consumer biomass is now set off that causes the system to proceed downward along the food isocline. The consumer decrease results initially from individual weight loss but is speeded up by starvation mortality when the individuals have lost enough weight. After the consumer cohort's consumptive capacity has decreased to below the minimum in the food isocline, food can recover. The geometry of this slow-fast cycle can be recognized in the simulated time course of the food population (fig. 1). The time series also shows how the frequency of the cycle slows down in the course of the simulation due an increase in length of the slow phases determined by the speeds of growth and decline of the consumer cohort.

The shrinking of the cycle with increasing individual size is the clue to understanding why the stepwise die-off cycling can end up into a stunted growth state when consumer individuals grow bigger (fig. 2). The flattening out due to low growth rates of big animals simply implies less overshoot in consumer weight. This reduction in amplitude of weight oscillation facilitates an approach of the intersection of the isoclines depicted in figure 5 without exceeding the critical weight loss triggering starvation mortality and setting of a new cycle. The other requirement is, of course, that this intersection is not too far below the maximum of the food isocline. This depends largely on the half-saturation coefficient of the functional response. If the animal is inefficient in exploiting its food at low densities (high H value), the minimum in the food isocline does not arise at all, and stunted growth without die-off cycles is the typical behavior.

Obviously, the state of stunted growth cannot be a real equilibrium. There is always mortality for reasons other than starvation in nature, and all cohorts gradually go extinct. The thinning out of a cohort allows survivors to grow; hence, stunted growth should not be expected in situations where mortality for nonfood reasons is high.

THE CYCLES AS CATASTROPHIC TRANSITIONS

Another way to look at this slow-fast cycle is to plot consumption and food production together as a function of the food level (fig. 6), a visualization pioneered by Noy-Meir (1975). The consumption curve represents the functional response of the consumer multiplied by its total population level. The implicit assumption is that the consumer population is constant, or at least not responding instantaneously to the food level. Since larger consumer populations result in higher consumption curves, this curve can actually be thought of as moving up and down with changing consumer populations. The slow-fast transitions in our cycle can be understood from this. A low consumer population causes a low

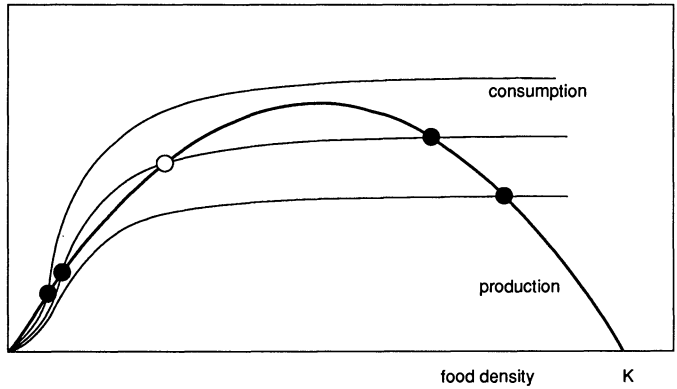

FIG. 6.—Food production and consumption as a function of food density. The height of the consumption curve is dependent on the total metabolic capacity of the consumer cohort. Net growth of food occurs only if production exceeds consumption. Intersections represent equilibria. The open circle is an unstable equilibrium.

consumption curve that has only one intersection point with the production curve, which represents an "underexploited" equilibrium with a high food level. Increase of the consumer population (due to individual growth in our case) moves this intersection toward lower food densities until the top of the production curve is reached and the intersection disappears, which causes a catastrophic transition to an overexploited state. Once the system is in this state, the consumer population needs to be reduced to a low level to allow recovery of the food because the over- and underexploited state exist as alternative equilibria over a range of consumer densities. Note that if the consumer and food speeds are sufficiently disjunct, the shape of a producer isocline (fig. 5) can actually be interpreted as a catastrophe fold sensu Thom (1975), with food as the fast variable (traditionally plotted on the Y-axis rather than the X-axis in catastrophe theory context).

The well-known three-dimensional cusp catastrophe representation is obtained by including a control variable as an extra dimension (fig. 7). The half-saturation constant (H) of the consumer functional response is an obvious control variable in this case. The fact that the catastrophe fold disappears for higher values of H can be understood geometrically from figure 6. If the half-saturation food density of the consumption curves moves too far to the right, it becomes impossible to get multiple intersections with the production curve corresponding to a "fold" in the isocline. Therefore, in order to get catastrophic transitions in the cycle, the half-saturation constant of the functional response must be small relative to the carrying capacity of the food. Put in biological terms, this means that the consumer must be relatively efficient in foraging at low food densities. Another condition, necessary to allow catastrophic collapse of food, is that the consumer population can grow enough to allow the consumption curve to rise above the peak of the production curve. This implies that the consumer must be able to still show net growth at food levels left of this peak, which, assuming logistic growth, is at half the carrying capacity. Note that this requirement coincides

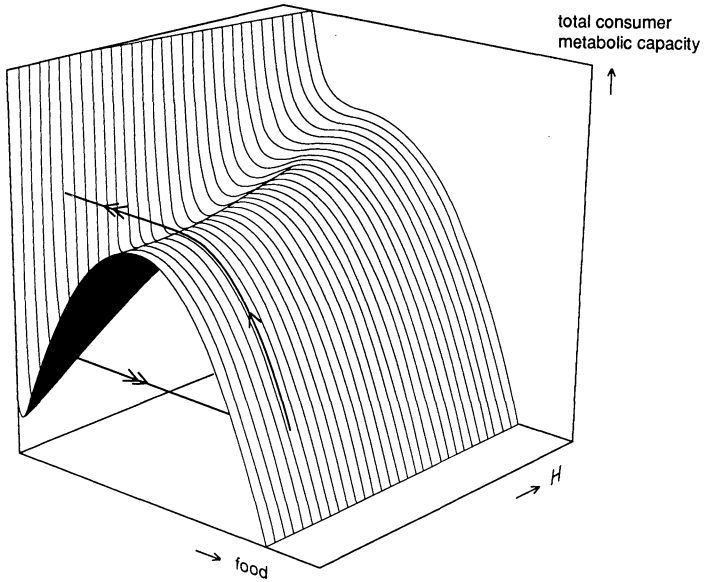

FIG. 7.—The food isocline plane shows a cusp catastrophe fold with the half-saturation constant (H) of the consumer functional response as control variable. The slow-fast cycles proceed along the surface during the slow phase and show a catastrophic transition during the fast phase.

nicely with the well-known general condition for limit cycles (Gilpin 1972) that the prey isocline needs to have a positive slope at the intersection with a vertical predator isocline, which boils down to the same requirement on predator efficiency.

COHORT DYNAMICS IN THE FIELD

The model analysis shows how phenomena like stepwise die-off and stunted growth equilibria can arise from the dynamic interaction between a cohort of consumer individuals and their food. Probably the best field studies of cohort dynamics are produced in fisheries research. Indeed, stunted individual growth as observed in the model simulations is a commonly described phenomenon among the older year classes in dense fish stocks (Bardach 1978). In freshwater populations "thinning" is the generally applied measurement to improve individual growth that provides larger fish, which are more valued by the fishermen. The model indicates two different explanations for the fact that especially older individuals tend to show stunted growth under food limitation, whereas starvation mortality is predominantly seen among young fish. In the first place, small fish starve more easily as they reach the critical weight loss sooner because of their higher weight-specific metabolism. An additional explanation, which is less easily obtained without the use of a model, is that large animals produce flatter slow-fast limit cycles. Put in biological terms: larger animals have lower rates of both

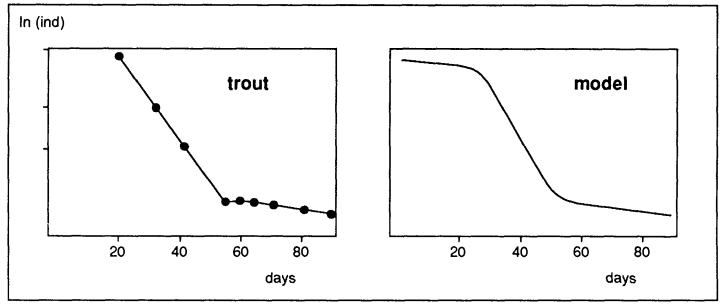

FIG. 8.—Discontinuous die-off in field data for brown trout (after Elliott 1989) closely resembles the pattern produced by the model over one die-off cycle.

growth and decline, which results in less overshoot in their response to food availability and hence a smaller probability to exceed the critical biomass loss that triggers mortality.

The dynamic patterns predicted by the model, such as the scenario of food depletion followed by stunted growth, individual weight loss, a distinct period of mortality and subsequent recovery of food and the surviving consumers, are much more difficult to trace in field data since detection requires frequent and accurate sampling of the cohort. Unfortunately, sampling young fish is notoriously laborious; consequently, accurate data are scarce, and the mechanisms regulating survival and growth in this life stage are relatively poorly understood (Shepherd and Cushing 1990). Nonetheless, it is well-known that cohorts of young-of-the-year fish often deplete their food to a low level and suffer high mortality losses (Braum 1978). The few intensively studied cases in which the dynamics have been documented in some detail provide especially interesting information. In Lake IJssel (The Netherlands), for instance, stunted growth and severe weight loss have been observed among young-of-the-year pike perch at the end of the summer, followed by a short period of massive mortality wiping out almost the whole cohort in a few weeks (E. H. R. R. Lammens, unpublished results). Probably the most detailed study of the dynamics of a young fish cohort, however, has been published by Elliott (1989, 1990*a*, 1990*b*). The author intensively monitored the dynamics of young-of-the-year cohorts of migratory trout, *Salmo trutta* L., over 8 yr. In each year class, there was a distinct discontinuity in survival. Invariably, an abrupt transition from a period of high mortality to a low-mortality phase is seen after a "critical survival time." This pattern closely resembles the model results over the first 100 d (fig. 8) produced from the default parameter setting (actually chosen to represent young fish) with a small constant background mortality. The fish that die during the high-mortality period are indeed shown to suffer from an energy deficit (Elliott 1990*a*), but the matter is complicated in the trout case by the fact that the animals lose a lot of energy in interference competition for territories.

Although the observed field patterns fit the model predictions well, there are some obvious caveats in the interpretation of this fit. In the first place, a modeled

mechanism is generally just one of many possible explanations for an observed phenomenon. Therefore, observation of a predicted pattern in the field cannot be interpreted as proof of the soundness of a model. The mechanism described by the model may or may not play a role in nature, and if it operates in real-world systems, a similar pattern may still be caused by a totally different mechanism in any specific field situation (Oreskes et al. 1994; Scheffer and Beets 1994).

A NOTE ON ROBUSTNESS AND GENERALITY

A general caveat of minimal models is that the behavior can be an artifact from the mathematical formulation rather than a result of real biological relationships (Roughgarden 1983). We therefore checked whether the observed patterns of stunted growth and stepwise die-off hinge on particular aspects of the model formulation.

We first focused on two parts of the formulation that may seem more or less peculiar, the sigmoidal functional response and the steep increase in mortality beyond a critical weight loss. It appears that after some tuning of the value of H, the model can produce the same qualitative behavior (figs. 1, 2) if the sigmoidal response is replaced by a simple saturating one (Type II) by eliminating the square from equation (3) (results not shown). Also, the steepness of increase in mortality around the starvation threshold hardly influences the model behavior. The value of p in equation (5) can even be decreased to one (a simple saturating increase of mortality with weight loss) without losing the stepwise die-off pattern.

Two obviously crude simplifications are the fact that there is only one food source and that all animals in the cohort are of the same size. It is therefore interesting that the predicted discontinuous survival curve has been produced independently from a much more elaborate model (DeAngelis et al. 1993). This detailed individual-by-individual model describes a cohort of young smallmouth bass foraging on an array of different prey species. In this case the largest individuals are the survivors because they can include larger prey species in their diet and are therefore less affected by food shortage during the die-off phase.

Resuming, the patterns produced by our generic model appear quite robust. They do not depend on the shape of the functional response or the way in which mortality increases with weight loss. Neither are they artifacts introduced by simplifications such as considering only one food source or assuming all animals in the cohort to be of the same size. Although, obviously, neither of the tested model variants is a faithful description of any particular real-world situation, the fact that they all produce the same pattern is a good sign. To borrow a phrase from Levins (1966, p. 423): "Our truth is the intersection of independent lies."

The fact that the behavior is robust against changes in the model formulation suggests that it should be robust against biological details too. Therefore, we do not expect the phenomenon of stepwise die-off to be restricted to the discussed fish-plankton case. The basic ingredients are food depletion, variance in fitness, and the fact that an animal is more likely to die when it has lost more weight. The latter aspect is not addressed in traditional predator-prey models. In our opinion the general point can be made that this omission has led to a far too

sterile view of predator-prey cycles. Whenever a predator population decreases markedly because of food shortage, a period of starvation may be expected to wipe out part of the population. Obviously, the starving animals will not be a random subgroup but rather the smallest or otherwise most vulnerable individuals. This implies that after each die-off phase of a cycle the characteristics of the population will be changed, and hence the "parameters" of the model should actually be adapted. The current analysis is limited in that it follows only one cohort and its food. Long-term dynamics of discretely reproducing populations will also be influenced by interaction between cohorts. In another article (M. Scheffer, unpublished results), we will show how this easily results in several types of population cycles and chaotic dynamics.

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