Hudson's Bay Trading Company (*24*) as resulting from a pure Lotka-Volterra oscillation about a neutrally stable equilibrium point, which is to say, having an amplitude determined by some environmental shock over 100 years ago, is quite implausible: this system, with the maximum hare population being constant to within a factor of 2 over 100 years or nine cycles, is surely the outcome of some stable limit cycle. This outcome, as we have just seen, can easily arise from the nonlinear food-hare-predators web.

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References and Notes

1. These terms are used in the sense defined and discussed by C. S. Holling [*Annu. Rev. Entomol.* **6**, 163 (1961)].
2. Reviews of the evidence are given by E. J. Kormondy [*Concepts of Ecology* (Prentice-Hall, Englewood Cliffs, New Jersey, 1969)] or A. Macfadyen [*Animal Ecology* (Pitman, London, ed. 2, 1963), chaps. 12 and 16]; in particular, see A. J. Nicholson's results for the blowfly [*Aust. J. Zool.* **2**, 9 (1954)].
3. For example, see B. Goodwin, in *Towards a Theoretical Biology*, C. H. Waddington, Ed. (Edinburgh Univ. Press, Edinburgh, 1970), vol. 3, pp. 1–17.
4. F. M. Scudo, *Theor. Popul. Biol.* **2**, 1 (1971); A. Rescigno and I. W. Richardson, *Bull. Math. Biophys.* **27**, 85 (1967).
5. M. L. Rosenzweig, *Science* **171**, 385 (1971).
6. B. Gompertz, *Phil. Trans.* **115**, 513 (1825).
7. G. F. Gause and A. A. Witt, *Amer. Natur.* **69**, 596 (1935); F. E. Smith, *Ecology* **44**, 651 (1963).
8. K. E. F. Watt, *Can. Entomol.* **92**, 674 (1960).
9. V. S. Ivlev, *Experimental Ecology of the Feeding of Fishes* (Yale Univ. Press, New Haven, Conn., 1961).
10. G. S. Gause, *The Struggle for Existence* (Williams & Wilkins, Baltimore, 1934).
11. C. S. Holling, *Can. Entomol.* **91**, 293 (1959).
12. K. E. F. Watt, *ibid.*, p. 129.
13. N. S. Goel, S. C. Maitra, E. W. Montroll, *Rev. Mod. Phys.* **43**, 231 (1971).
14. T. I. Bulgakova, *Probl. Kibern.* **20**, 271 (1968), and references therein; see also (*5*).
15. M. L. Rosenzweig and R. H. MacArthur, *Amer. Natur.* **97**, 209 (1963); R. H. MacArthur and J. H. Connell, *The Biology of Populations* (Wiley, New York, 1966), chap. 6.
16. A. N. Kolmogorov, *Giorn. Inst. Ital. Attuari* **14**, 1 (1936). This theorem, which can be proved by the application of Poincaré-Bendixson methods to Eq. 1, is outlined by N. Minorsky [*Nonlinear Oscillations* (Van Nostrand, Princeton, N.J., 1962), chap. 2, sect. 9].
17. The theorem also holds in general when any of the conditions given by Eqs. 3 and 4 are equalities rather than inequalities. This can be seen if one uses the methods indicated in (*16*). There can be special exceptions here, but none of the prey-predator forms catalogued in the text constitutes such an exception, as the equalities are attained as sensible limiting cases of more general predator-prey terms which obey the inequalities given in Eqs. 3 and 4. In particular, in the example given in Eq. 2 the theorem is rigorously obeyed, although here $\partial h/\partial y = 0$ rather than a negative quantity (that is, there is no predator intraspecific interaction).
18. The criterion given in Eq. 3 usually requires that the per capita prey birth rate be a monotonic decreasing function of increasing x. This condition is violated if there is an "Allee effect" [W. C. Allee, *The Social Life of Animals* (Heinemann, London, 1939)] whereby the per capita birth rate falls off at small x. This result makes sense biologically: models incorporating the effect should permit the possibility of extinction. However, Kolmogorov's theorem still applies in a restricted part of the x-y plane, thus allowing the possibility of limit cycles wherein the minimum in the prey population remains large enough for its Allee effect not to operate.
19. This estimate is obtained from Eq. 2 on the assumption that the rates r and b are comparable, that $x^*/K << 1$, and that cx^*, fx^* are less than, or of the order of, unity (x^* and y^* are the populations satisfying $dx/dt = dy/dt = 0$). One may then obtain rough piecewise continuous approximations for the limit cycle trajectory $x\dagger(t)$, $y\dagger(t)$ in the four regions where $x\dagger(t)$ and $y\dagger(t)$ are small or large as compared with the (unstable) equilibrium point values x^*, y^*. I have tested the usefulness of this estimation procedure, which leads to Eq. 10, by comparing it with a series of exact computer solutions for the limit cycle. The approximation given in Eq. 10 gave good order-of-magnitude agreement in every case tested.
20. For example, see (*5*). On the other hand, R. H. MacArthur (personal communication) has empirically noticed limit cycle behavior in computer studies of such models.
21. M. S. Bartlett, *Stochastic Population Models in Ecology and Epidemiology* (Methuen, London, 1960).
22. L. C. Cole, *J. Wildlife Manage.* **18**, 107 (1954); L. B. Slobodkin, *Growth and Regulation of Animal Populations* (Holt, Rinehart & Winston, New York, 1961), chap. 14.
23. D. L. Lack, *The Natural Regulation of Animal Numbers* (Clarendon, Oxford, 1954), pp. 212–217.
24. D. A. MacLulich, *Univ. Toronto Stud. Biol. Ser. No. 43* (1937) [reproduced in Kormondy (*2*)].
25. I thank the people at Magdalen College, Oxford, and at the Institute for Advanced Study, Princeton, for their hospitality during my leave from the University of Sydney, Australia. I also thank M. L. Rosenzweig for some constructive comments. Research sponsored by NSF grant GP-16147 A 1.

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Enriched Predator-Prey Systems: Theoretical Stability

Rosenzweig has warned that enrichment may destroy predator-prey systems (*1*). Using the graphical predation theory he and MacArthur developed (*2*), Rosenzweig has predicted that enrichment may cause previously persistent ecological systems to explode. This will happen, he says, when enrichment shifts the peak of the prey zero isocline (the collection of points in the phase plane at which the prey population does not change in density) to the right of the predator zero isocline. McAllister *et al.* (*3*) have challenged this prediction on experimental grounds. In this technical comment I shall show that Rosenzweig's own models do not fully support his conclusions, that the theory on which his arguments are based was incomplete and insufficient, and that there is therefore no inconsistency here between theory and experiment.

Rosenzweig studied what he believed to be six reasonable deterministic models of predator-prey interaction (*1*). He concluded that all show danger of extinction with increasing enrichment. He particularly concentrated on the following model [model 4 in (*1*)]:

$$dV/dt = rV(1 - V/K) - kP(1 - e^{-cV}) \quad (1)$$

$$dP/dt = AkP(e^{-cJ} - e^{-cV}) \quad (2)$$

where V is the prey (victim) density, P is the predator density, and K is the carrying capacity of the prey. The predator zero isocline is at $V = J$. The other parameters in Eqs. 1 and 2 influence the shape of the prey zero isocline and the rate of the system's response.

Investigating Eqs. 1 and 2, Rosenzweig first shows that the prey zero isocline conforms to the expectations of graphical predation theory; that is, it is peaked. He next shows that enrichment, increased K, moves this peak to a larger value of V and that it may therefore move it to the right of the predator zero isocline. According to graphical predation theory, he then predicts that this may cause the populations to go to extinction. Rosenzweig numerically integrates Eqs. 1 and 2 and finds that, for a large value of K, this does indeed happen.

I have used a digital computer to study the behavior of Eqs. 1 and 2. My results are not in complete accord with Rosenzweig's predictions. I find that the populations do not go to extinction when the peak of the prey zero isocline lies to the right of the predator zero isocline.

The numerical values of the parameters in Eqs. 1 and 2 are relatively unimportant, so I have chosen the following numbers so as to make my system quantitatively similar to the system Rosenzweig depicts in figure 1 of (*1*): $r = 0.05$; $k = 0.025$; $c = 0.1$; $A = 1$; and $J = 20$. For a single predator zero isocline, I determined the dynamic response for five different prey zero isoclines (Fig. 1A). Figure 1B shows a system for which the enrichment is too low: the predator population goes to extinction. Figure 1, C and D, shows stable systems that approach a steady state at the point where the two zero isoclines intersect. These results are in accord with graphical predation theory.

Figure 1, E and F, shows systems that, according to graphical predation theory, may go to extinction. Obviously, they do not. Both reach stable limit cycles. In fact, no matter how great K may be, the populations modeled by

Eqs. 1 and 2 will never go to extinction (*4*). Other reasonable deterministic models of predation show analogous behavior; for example, Rosenzweig's model 6 (*1*), Gilpin's model (*5*), and apparently the model of McAllister *et al.* (*3*), although it is not clear whether the prey refugium was critically necessary.

The flaw in Rosenzweig's arguments lies not in the mathematics of (*1*), but in the development of graphical predation theory (*2*), which, as it stands, is insufficiently developed to permit predictions about global stability. Solutions to systems of autonomous differential equations, such as Eqs. 1 and 2, are trajectories in a phase plane. Such systems are globally stable if no trajectory that starts at positive P and V either crosses or asymptotically approaches one of the axes.

The phase plane of such systems consists of normal points, at which solution trajectories exist and are unique, and singular (equilibrium) points, which are defined by (*6*)

$$dP/dt = dV/dt = 0$$

Biologically, it is possible to sketch in several of these trajectories. There is one along the ordinate that corresponds to the dying off of the predator in the absence of prey. There are two others on the abscissa that in the absence of predators logistically approach the carrying capacity, K, from either side. Because solution trajectories must be unique at all normal points, no trajectory that starts at positive P and V can intersect the axes at any point that lies on these trajectories. Thus, if one of the populations is to go to zero, it must do so at one of the singular points that lie on the axes.

Systems such as those shown in Fig. 1, C–F, have three singular points: (0,0), (K,0), and the intersection of the two zero isoclines. It is obvious that the first two of these singular points are saddle points. Near the point (0,0), solutions first move downward parallel to the ordinate and then curve to move outward along the abscissa. Near (K,0), solutions first move parallel to the abscissa and approach $V = K$ from either side, whereupon they turn upward to increase P. Therefore, solution trajectories can nowhere intersect the axes. It is also clear that cycles very near the axes will spiral inward, for, if a trajectory starts from a value of V greater than K, it will fall below K after it completes its first cycle. This is shown in Fig. 1, C–E.

By studying the third singular point of the system, as Rosenzweig has done, it is possible to predict that this singular point will be unstable if the peak of the prey zero isocline is to the right of the predator zero isocline, which implies that solution trajectories will spiral outward from this point. Thus a complete graphical predation theory predicts that stable limit cycles must exist where the outwardly spiraling, small-amplitude cycles meet the inwardly spiraling, large-amplitude cycles.

The careful analysis of some reasonable deterministic models of predation suggests that ecological systems will persist after enrichment. This does not mean, however, that Rosenzweig's warning can or should be ignored. Systems such as that shown in Fig. 1F, although mathematically stable, could give rise to biologically unrealistic population densities, for example, less than one individual. Furthermore, if density-independent stochastic effects are added, such limit cycles would be susceptible to random extinction. Finally, Rosenzweig has noted (*7*) that if the idea of a

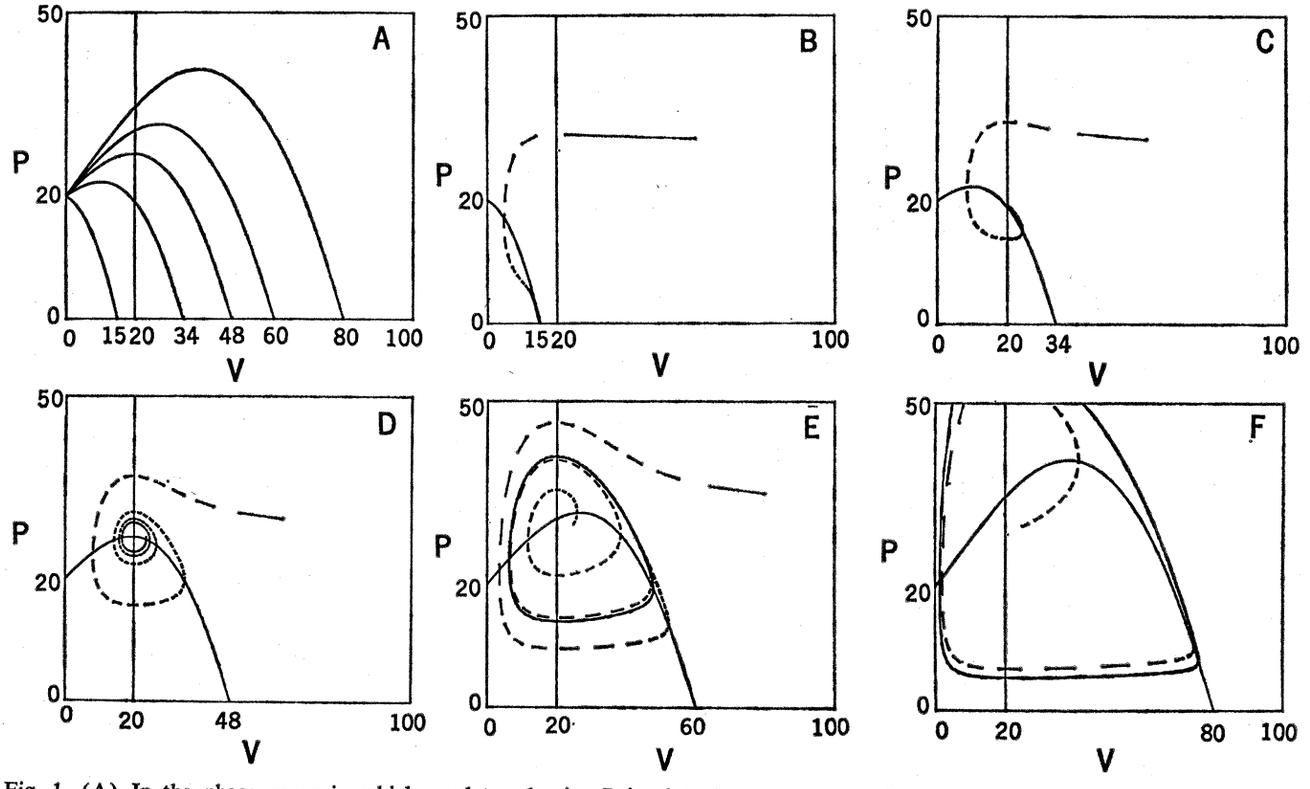

Fig. 1. (A) In the phase space in which predator density P is plotted against prey (victim) density V, the predator isocline is at $V = 20$ and five different values of K ($K = 15$, 34, 48, 60, and 80) give five different prey isoclines. In (B) through (F) the dynamic response for a particular starting configuration is shown for a single one of these values for K. Each dash and blank represents 20 iterations of Eqs. 1 and 2. The cycles are counterclockwise. (B) The predator population goes to extinction. (C) The two populations reach a steady state. (D) The two populations asymptotically approach a steady state. (E) The system reaches a stable limit cycle, both from within and without. (F) Again, the system reaches a stable limit cycle.

maximum predator life-span is included, this will perhaps make the model discussed above unrealistic during that part of the limit cycle where the victim density is very low.

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References and Notes

1. M. L. Rosenzweig, *Science* **171**, 385 (1971).
2. ——— and R. H. MacArthur, *Amer. Natur.* **97**, 209 (1963).
3. C. D. McAllister, R. J. LeBrasseur, T. R. Parsons, *Science* **175**, 562 (1972).
4. The calculations Rosenzweig carried out to obtain figure 2 in (*1*) are apparently in error. For $K = 200$, the prey population would be reduced to a value very close to zero during each cycle. The extinction of the prey (the extinction of the predators would soon follow) could very easily be the result of computer truncations.
5. M. E. Gilpin, in preparation.
6. T. V. Davies and E. M. James, *Nonlinear Differential Equations* (Addison-Wesley, Reading, Mass., 1966).
7. M. L. Rosenzweig, personal communication.
8. Research supported by PHS grant ES-00121-04.

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Most of what Gilpin says is quite right. Extinction in my simulation of model 4 *is* caused by a truncation; mathematically, model 4 does indeed reach a limit cycle. However, that truncation was designed into the system for the sake of biological reality; I maintain that it was proper to do that and proper to issue the warning based on it. The value of V at the low point of the cycle is just unrealistically small.

Riebesell (*1*) has undertaken simulations of a systematic series of two-species exploitative systems. He has included random environmental fluctuations. In general, he finds that the nontrivial equilibria of exploitative systems can be roughly sorted into four types. The first includes those in which the victim's equilibrium V (that is, $\hat{V}$) is so close to K that simulated uncertainty produces rapid predator extinction. The second includes those with slightly lower $\hat{V}$; they are steady states despite the randomness. The third type is a band of still smaller $\hat{V}$ values; these produce the limit cycles of Gilpin. The last type includes the smallest values of $\hat{V}$, values so low that their associated limit cycles are unrealistic; either or both species become extinct. Cases of this last type correspond to ones in which strong enrichment is simulated, because, as I showed in my report (*2*), an increase in K has the same effect on stability as a decrease in $\hat{V}$. Thus a totally realistic approach to the problem demands that one deal with the probability that enrichment can meaningfully increase extinction rates.

In addition to that, even a change from a steady state to an oscillation is worthy of the concern of resource managers. Oscillation would produce its own problems: among them are fluctuations in the food supply, fluctuations in the labor market, and the need for storage facilities to damp the effect of the oscillation on consumers.

In defense of MacArthur's and my original work (*3*), it should be stated that we noted the problem of global stability therein. The section devoted to it included several cases in which we deduced the existence of limit cycles. We also admitted that graphical theory was incomplete without a clear understanding of global stability.

I should add that Gilpin's proof of limit cycles is unconvincing. It depends on the assumption that, in order to reach extinction, a population must ride a vector which intersects an axis. That is not so. The vector might reach a confluence with the axis—merge with it without crossing it.

May (*4*) has developed a convincing argument for the generality of limit cycles. Therefore, Gilpin's conclusion is perfectly accurate.

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References and Notes

1. J. Riebesell, thesis, State University of New York at Albany (1971).
2. M. L. Rosenzweig, *Science* **171**, 385 (1971).
3. ——— and R. H. MacArthur, *Amer. Natur.* **97**, 209 (1963).
4. R. May, *Science* **177**, 900 (1972). By coincidence, both May and Gilpin sent me copies of their manuscripts within a period of several days during the winter of 1971–72. They clearly have arrived at the same conclusions independently of each other and of Riebesell.

19 June 1972

Meetings

Immunology and Genetics

Thirty West Coast geneticists met with specialists in various fields of immunology at La Jolla, California, 25–26 February, to explore research and clinical aspects of the interface of immunology and genetics. The workshop-symposium was supported by the National Genetics Foundation.

Genetic control of immune responsiveness to specific antigens and the possible relation of such control to histocompatibility systems of mice, guinea pigs, and man were considered first. The antibody response involves a complicated interaction between thymus-derived antigen-reactive lymphocytes (T cells) and bone marrow–derived, antibody-producing cell precursors (B cells). A T cell capable of recognizing the specific immunogen seems to stimulate proliferation of and specific antibody production by B cells. Immune response (Ir) genes and antigen recognition were discussed by H. O. McDevitt (Stanford) and B. Benacerraf (Harvard). For some Ir genes, both responder (R) and nonresponder (NR) strains of mice and guinea pigs give a primary 19*S* response, but only R strains give a 7*S* secondary response. T cells appear to respond to "carrier" determinants on a given antigen, while the structure of the antibody combining site (idiotype) is a function of the structural genes for the immunoglobulins and is expressed in B cells. Genetic loci controlling graft rejection and amount of antibody produced and affecting antibody specificity map in a region of mouse linkage group IX. Ir maps between H-2^K and H-2^D, the two well-separated cistrons of the major histocompatibility system in the mouse (homologous with the LA and Four cistrons in the human HL-A system). Ir seems to comprise a set of several or many linked genes with dif-