N.D.) for ten locations at China Lake and for two locations at Boron are a direct result of interference due to VO_3^- and other anionic metal complexes. It also is significant that the data on As content from single-sweep polarography are in good agreement with the SDDC data only for a single well location, namely, location 29 of Table 1. In view of the projected importance of water pollution analysis, several conclusions have been derived from the work reported:

1) The underground waters of the Boron and China Lake basins are associated with heterogeneous rock formations, many of which contain large amounts of water-soluble minerals. Thus, the subsurface water more closely resembles dilute brines or even brackish seawater (*1*) than it does the usual surface waters for which the colorimetric SDDC method was developed. In the SDDC test procedure (*4*) the analyst is cautioned (paragraph 1.2) about interference with AsH_3 evolution caused by certain metals; all the metals listed in paragraph 1.2 were found in our mineralized water samples. Stratton and Whitehead (*9*) and Ballinger *et al.* (*10*) developed and evaluated the SDDC method for relatively pure river drinking water, and neither of these groups of authors found it necessary to use a second method of known accuracy and specificity for As to verify their results.

2) It is recommended that the SDDC method be used for the routine analysis of desert water only after verification of the As content by means of x-ray or single-sweep polarographic measurements. Angino *et al.* (*11*) cited the presence of the same interfering metals in detergents as those reported here but they evidently ignored the caution in paragraph 1.2 of the SDDC test procedure in their statement that a high degree of precision in the determination of As was attained. No mention was made of the degree of accuracy obtained.

3) Accurate and highly specific analytical methods should be employed by concerned agencies to correctly detect the presence of and to define the extent of water pollution. The accurate analysis of trace toxicants in water in the parts-per-billion range is difficult, at best, and research efforts in this area are vitally needed, as advocated by Smith (*12*).

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Paradox of Enrichment: Destabilization of Exploitation Ecosystems in Ecological Time

Abstract. *Six reasonable models of trophic exploitation in a two-species ecosystem whose exploiters compete only by depleting each other's resource supply are presented. In each case, increasing the supply of limiting nutrients or energy tends to destroy the steady state. Thus man must be very careful in attempting to enrich an ecosystem in order to increase its food yield. There is a real chance that such activity may result in decimation of the food species that are wanted in greater abundance.*

Schemes for increasing primary productivity by enriching an ecosystem's energy or nutrient flow are much in evidence today and are probably a reflection of the increasing demands of the world's population. Such schemes may end in catastrophe.

In 1963, Huffaker, Shea, and Herman (*1*) reported destabilization of a stable exploitation ecosystem which resulted in the extinction of both the exploiter (an acarophagous mite) and its victim (an herbivorous mite). They produced this result by trebling the herbivore's food density. By using a variety of realistic models, I predict that instability should often be the result of nutritional enrichment in two-species interactions.

Rosenzweig and MacArthur (*2*) showed that exploitation (or predator-prey) ecosystems do not necessarily exhibit any oscillations. Furthermore, even if there are oscillations, they do not last under ordinary circumstances. If the exploiter is quite proficient at reproducing in the presence of few of its victims, then the ecosystem does not persist. If, however, the victims are relatively proficient at escape or their exploiters have a relatively poor reproductive efficiency or digestive efficiency, then the system will persist in ecological time (*3*).

The dividing line between persistent and explosive systems is definable from a general graph of exploitation (*2*). The victim's density V is plotted against P, the exploiter's density. The collection of graph points at which $dV/dt = 0$ is called the victim's isocline. The collection of points at which $dP/dt = 0$ is called the exploiter's isocline. Any point of intersection between the two isoclines is an ecosystem equilibrium, but not all such equilibria will result in a steady state. The usual form of the prey isocline is a hump (*4*). If the equilibrium is at a point on the left side of the hump, the predator is too proficient and the system will ordinarily not persist. If equilibrium is at a point on the right-hand (downslope) side of the hump, the system will persist. Thus, the hump's peak is over a critical value of V, V^*. If the equilibrium value of V is larger than V^*, the system is safe. If not, it is in danger of extinction.

If the exploiters do not actually interfere with each other directly—if they never battle over the same individual victim or engage in cannibalism or territorial defense—then the P isocline is a simple vertical line ($V = J$). The position of this line is fully determined by the phenotypes of the exploiter and its victim. It does not change with nutrient flow or energy supply.

To discover the effect of enriching a system, one needs to find how V^* changes as enrichment proceeds. If enrichment increases V^*, then it is jeopardizing the system, because eventu-

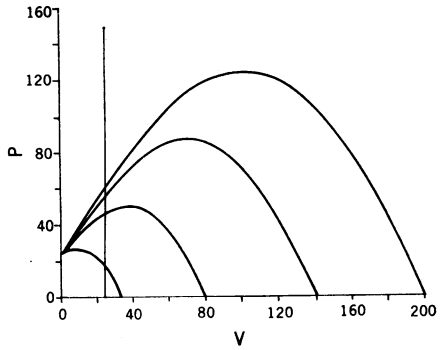

Fig. 1. Isoclines at four levels of productivity ($K = 34$, 80, 140, and 200) when model 4 is used. Symbol V is victim's density; P is exploiter's. The curved lines are V isoclines, which peak over higher values of V at higher K. The vertical line, $V = 25$, is the P isocline. From the slopes of the V isoclines at the points where they intersect the P isocline, one expects only the $K = 34$ system to have a steady state.

ally V^* will be made greater than J.

Briefly, the method is this. Set $dV/dt = 0$ and solve for P. This is the algebraic equation for the V isocline. Take $\partial P/\partial V$. The value of V that satisfies $\partial P/\partial V = 0$ is V^*. If K represents the standing crop of V where $P = 0$, then K must be directly proportional to the flow rates of limiting nutrients. Thus, enrichment implies K increase. The final step, then, is to obtain $\partial V^*/\partial K$. Since this is always positive, enrichment leads toward system instability.

Each analytical model used is the difference between inherent rate of increase of V and the number of V that die (or are not born) owing to the activities of P.

Assume that each V must receive a quota of nutrients at a rate Q in order just to replace itself. Nutrients flow to the lone individual V at a rate R. Thus, this individual reproduces at a net rate $r\ (R - Q)$. As V increases, however, each individual V is subjected to intraspecific competition. One may assume that an individual V's effective "feeding" rate diminishes with increasing V^a, where a is the victim's competition constant, $1 \geqslant a > 0$ (*5*). Thus the per capita reproductive rate is $r\ (RV^{-a} - Q)$ and the inherent rate is $rV\ (RV^{-a} - Q)$. From the parentheses one obtains $K = (R/Q)^{1/a}$. Hence, to increase K, R must be increased (a and Q are constant), and $\partial V^*/\partial K$ has the same sign as $\partial V^*/\partial R$ (*6*).

In addition to the above model, I have used the traditional Pearl-Verhulst logistic $rV\ (1 - V/K)$ and the Gompertz $rV\ (\ln K - \ln V)$.

Kill rate models. Lotka and Volterra's approach was to treat the two populations like molecules: kVP [see (*7*) and references therein]. This has been shown to be inadequate. Gause (*7*) obtained a reasonable fit to a kill rate curve by taking the square root of V. We can generalize this procedure by taking V to the gth power $0 < g \leqslant 1$. Thus, a kill rate model is kPV^g.

Another model is based on observations (*5*, *8*) that one lone exploiter will attack $k(1 - e^{-cV})$ victims in a fixed amount of time. Since the exploiters compete only by reducing each other's food supply, P exploiters will kill $kP(1 - e^{-cV})$.

Thus models for dV/dt include

$$dV/dt = rV(RV^{-a} - Q) - kP(1 - e^{-cV}) \quad (1a)$$

$$dV/dt = rV(1 - V/K) - kPV^g \quad (2a)$$

$$dV/dt = rV(RV^{-a} - Q) - kPV^g \quad (3a)$$

$$dV/dt = rV(1 - V/K) - kP(1 - e^{-cV}) \quad (4a)$$

$$dV/dt = rV(\ln K - \ln V) - kPV^g \quad (5a)$$

$$dV/dt = rV(\ln K - \ln V) - kP(1 - e^{-cV}) \quad (6a)$$

In view of the lack of convincing tests of any of the models as a general case for all systems, I have analyzed all six.

The first step in each analysis is omitted here: solution of each equation for P when $dV/dt = 0$. For example, Eq. 4a becomes

$$P = \frac{rV(1 - V/K)}{k(1 - e^{-cV})}$$

This set of equations is the set of V isoclines.

Next we obtain $\partial P/\partial V$ and determine the conditions under which this will be zero. These are the V^* equations:

$$R = \frac{Q(V^*)^a\ (e^{cV^*} - 1 - cV^*)}{(e^{cV^*} - 1)\ (1 - a) - cV} \quad (1b)$$

$$K = \frac{(2 - g)}{(1 - g)} V^* \quad (2b)$$

$$R = \frac{Q(V^*)^a\ (1 - g)}{(1 - a - g)} \quad (3b)$$

$$K = V^* \frac{(2e^{cV^*} - cV^* - 2)}{(e^{cV^*} - cV^* - 1)} \quad (4b)$$

$$\ln K = \ln V^* + 1/(1 - g) \quad (5b)$$

$$\ln K = \ln V^* + 1 + \frac{cV^*}{e^{cV^*} - 1 - cV^*} \quad (6b)$$

The final step requires a small explanaton. We need the sign of $\partial V^*/\partial K$ or $\partial V^*/\partial R$. Often the equation systems are easily solved for K or $\ln K$, but not V^*. However, $\partial V^*/\partial K$ is positive if and only if $\partial K/\partial V^*$ is. And $\partial K/\partial V^*$ is positive if and only if $\partial \ln K/\partial V^*$ is. Hence, we can readily proceed with these latter two partial derivatives. Three are positive for any set of values of the constants:

$$\frac{\partial K}{\partial V^*} = (2 - g)/(1 - g) \quad (2c)$$

$$\frac{\partial K}{\partial V^*} = \frac{2(e^{cV} - 1)(e^{cV} - 1 - cV - c^2V^2/2)}{(e^{cV} - 1 - cV)^2} \quad (4c)$$

$$\frac{\partial \ln K}{\partial V^*} = \frac{1}{V^*} \quad (5c)$$

Equation 4c is always positive because the MacLaurin series for e^{cV} is $1 + cV + c^2V^2/2 + c^3V^3/6 + \cdots$ (see Figs. 1 and 2).

The other three cases are not quite so readily handled. Equation 3b does not always have a positive solution for V^*. In fact V^* is negative if and only if $(1 - a - g)$ is also negative. The V isocline of Eq. 3a is humpless for such values of $(a + g)$. Values this great im-

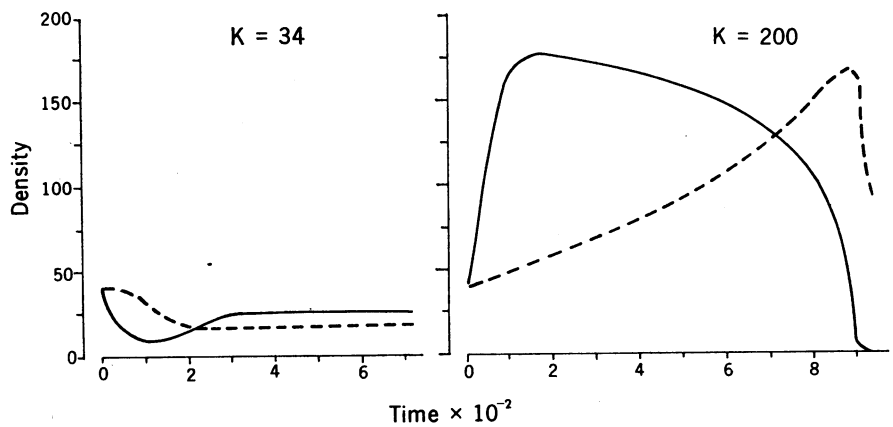

Fig. 2. Iteration of the model 4 exploitation at $K = 34$ and $K = 200$. Solid curve is V; dashed curved is P. Enrichment causes the simulated extinction of both species. The exploiter equation used was $dP/dt = AkP\ (e^{-cJ} - e^{-cV})$. Time units are in calculator cycles (*10*).

ply intense intraspecific producer competition and also a relatively low tendency for exploiters to become hungry or satiated and to modify their behavior accordingly. In such a system there is no tendency for extinction regardless of productivity.

However, if $(a + g)$ is less than 1, there is a positive V^* and

$$\frac{\partial R}{\partial V^*} = \frac{aQ(1-g)\ (V^*)^{a-1}}{(1-a-g)} \quad (3c)$$

Clearly Eq. 3c is always positive if V^* is biologically real. Hence, in model 3, if there is any threat of system extinction, it is increased by enrichment.

Models 1 and 6 are similar and most complex. It turns out that Eq. 6b is satisfied by two values of V. One is V^*. Another is a very small value of V that occurs over a trough in the V isocline. Thus, there is ambiguity in the following:

$$\frac{\partial \ln K}{\partial V^*} = \frac{(e^{cV^*} - 1)\ (e^{cV^*} - 1 - cV^* - c^2V^{*2})}{(e^{cV^*} - cV^* - 1)^2} \quad (6c)$$

This equation, set to zero, holds for both V^* and the V under the trough. The unstable equilibrium values of V are those between V (trough) and V^*. Model 6c is positive for V^* and negative for V (trough) (*9*). Hence, as enrichment proceeds, the range of unstable V is increasing at both ends. Therefore again, enrichment unambiguously tends to weaken the steady state. Model 1 has the same characteristics (*9*).

Until we are confident that the conclusions based on these systems do not apply to natural ecosystems, we must remain aware of the danger in setting enrichment as a human goal.

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11. I thank Drs. E. Leigh, III, S. Levin, D. McNaught, L. Segel, and M. Slatkin for valuable comments; and J. Riebesell, whose careful work was helpful in making mathematical errors scarce, if not entirely absent. Supported by the National Science Foundation and the Research Foundation of the State University of New York.

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Induction of Liver Acetaldehyde Dehydrogenase: Possible Role in Ethanol Tolerance after Exposure to Barbiturates

Abstract. *Mice were injected twice a day for 4 days with saline or phenobarbital or ethanol. Treatment with phenobarbital, but not ethanol, increased the amount of liver acetaldehyde dehydrogenase activity. More rapid removal of acetaldehyde, which is a toxic metabolic intermediate of ethanol, may contribute to the alcohol tolerance exhibited by persons who use barbiturates regularly.*

Acetaldehyde, a pharmacologically active metabolite of ethanol, appears to contribute to the actions of ingested ethanol (*1*). Acetaldehyde can induce nausea, vomiting, and sweating; it causes release of catecholamines and depression of oxidative phosphorylation in isolated brain tissue. Recently it was shown that acetaldehyde condenses with catecholamines (*2*) or potentiates a similar condensation by certain endogenous aldehydes (*3*) to form isoquinoline alkaloids that may possess biologic activity. Because the pharmacologic effects of acetaldehyde are generally subjectively unpleasant, it has even been suggested that "while ethanol actions may be the reason that people drink alcohol, the actions of acetaldehyde may be more related to why they stop" (*1*).

The purpose of our study was to see if phenobarbital, an inducer of many liver enzymes (*4*), would increase the levels of acetaldehyde dehydrogenase (AcDH), which is believed to be the enzyme primarily responsible for detoxication of acetaldehyde. We were interested in the tolerance to the effects of ingested alcoholic beverages exhibited by persons who use barbiturates regularly. If AcDH activity were elevated after exposure to barbiturates, the consequent more rapid removal of acetaldehyde could contribute to ethanol tolerance. Our results with mice support this hypothesis.

Male and female Paris R-III or C-57BL mice were injected intraperitoneally twice a day (at approximately 9:30 a.m. and 4:30 p.m.) for 4 days with either isotonic saline (buffered at *p*H 7.4 with 0.01*M* sodium phosphate) or with sodium phenobarbital (75 mg/kg) or with ethanol (2.4 g/kg, administered as a 30 percent by volume solution), each dissolved in the isotonic buffer. On day 5, the animals were killed by cervical dislocation, and the liver was homogenized (ten strokes in a Teflon and glass homogenizer) in 20 volumes of ice-cold water. The homogenate was kept at about 0°C for the duration of the experiment. Cell debris was removed by centrifugation at 700*g* for 5 minutes. Enzyme assays were completed within 2 hours after death.

Acetaldehyde dehydrogenase was assayed at room temperature by a modification of the method of Maxwell and Topper (*5*). Assay tubes contained 6 ml of buffer (0.1*M* glycine at *p*H 9.5, containing 4.0 m*M* mercaptoethanol and 2.0 m*M* ethylenediaminetetraacetic acid), 0.4 ml of nicotinamide adenine dinucleotide (NAD) solution (Calbiochem, 10 mg/ml), and 0.1 ml of acetaldehyde solution (Eastman, 1 percent by volume in water). A blank containing all of the above except acetaldehyde was used. The reaction was started by the addition of 0.2 ml of liver extract. Reduced NAD (NADH) concentration was measured by the optical density at 340 nm in a Gilford model 300 spectrophotometer after 3.5 and at 7.5 minutes; enzyme activity was given by the increase in optical density during the 4-minute interval, with the blank substracted.

In three separate experiments, increases in liver AcDH activity were observed after 4 days of treatment with phenobarbital, but AcDH activity was not increased after treatment with ethanol. Pooled results of the three experiments (Fig. 1) indicate that