space or by moving into blood and thence to other sensitive structures. With regard to the first alternative, injections of dye (10 μl, 5 percent bromophenol blue) showed intense staining limited to spinal segments not more than 1.0 to 1.5 cm distant in either direction. Moreover, the absence of an initial effect upon the forelimbs suggests that the more rostral spinal segments were not initially affected by the lumbar injection. With regard to the second alternative, the intravenous injection of 15 μg of morphine did not alter the response of the animal on any of the three measures employed (*8*). The assumption that the spinally administered morphine did not move into more rostral brain regions was further substantiated on the basis of studies in which ^{14}C-labeled morphine sulfate was administered through the spinal catheter and animals were sacrificed at intervals of up to 60 minutes after the injection. At even the longest intervals, neither forebrain nor brainstem radioactivity ever exceeded 0.15 percent of that recovered from the spinal cord. It thus seems certain that the behavioral effects observed resulted from a local pharmacological action of these narcotics on the lower spinal segments. Injections of the potent local anesthetic dibucaine (1 to 10 μg) not only blocked responding on the experimental tasks, but unlike equipotent doses of morphine, it resulted in pronounced motor incoordination. Moreover, such effects produced by dibucaine were not antagonized by naloxone.

Morphine can antagonize reflexes (*9*) and alter evoked spinal activity in animals with sectioned spinal cords (*10*), as well as depress the discharge (by local iontophoresis) of spinal units responding characteristically to strong peripheral stimuli (*11*). Moreover, narcotics given by arterial injections directed toward the spinal cord are more potent in blocking the monosynaptic reflex than when they are injected intravenously (*12*). Such findings, while showing a pharmacological action of narcotics at the cord level, do not indicate that the physiological effect observed after analgetics have been systemically administered plays any role in the animal's perception of or response to environmental stimuli. Our principal finding, therefore, is that narcotics exert a direct, pharmacologically specific effect on spinal function, which diminishes the response of the intact, behaving animal to otherwise aversive peripheral stimulation. Although the mode of action of spinally administered narcotics is not clear, the fact that they were effective in antagonizing responses to the hot-plate and pinch and elevated the operantly defined shock titration threshold indicates that the narcotic effect was not due simply to a local attenuation of reflex activity. Substantial stereospecific binding of a narcotic occurs within the spinal cord in the vicinity of the substantia gelatinosa (*13*). In light of this binding and of the proposed importance of the substantia gelatinosa to the transmission of noxious stimuli (*14*), it is reasonable, if speculative, to suggest that narcotics directly modulate the activity of the substantia gelatinosa. Two things remain to be determined: (i) the relative importance of this narcotic-sensitive spinal system compared to supraspinal systems, and (ii) the specific role played by the spinal cord in the mediation of the analgesia observed following the systemic administration of narcotics.

TONY L. YAKSH
THOMAS A. RUDY
School of Pharmacy, University of Wisconsin, Madison 53706

References and Notes

1. K. Tsou and C. S. Jang, *Sci. Sin.* **13**, 1099 (1964); A. Herz, K. Albus, J. Metys, P. Schubert, H. Teschemacher, *Neuropharmacology* **9**, 539 (1970); Y. F. Jacquet and A. Lajtha, *Science* **182**, 490 (1973); L. G. Sharpe, J. E. Garnett, T. J. Cicero, *Behav. Biol.* **11**, 303 (1974); A. Pert and T. L. Yaksh, *Brain Res.* **80**, 135 (1974); *Pharmacol. Biochem. Behav.* **3**, 133 (1975); T. L. Yaksh, J. C. Yeung, T. A. Rudy, *Brain Res.*, in press.
2. A length of polyethylene (PE-10) tubing was inserted through the hub of a disposable 20-gauge needle from which the needle segment had been removed. The tubing was then fastened with an epoxy cement so that 8.5 cm of tubing extended from the hub. During insertion, the catheter was slightly stiffened by inserting a length of 0.005-inch wire, which extended to within 1 cm of the catheter tip. By flexing the head downward and continually rotating the catheter during insertion, the incidence of damage to spinal tissue was limited to one or two rats in ten (T. L. Yaksh and T. A. Rudy, in preparation).
3. The solution consisted of an osmotically and ionically balanced mixture consisting of NaCl (7.46 g), KCl (0.19 g), $MgCl_2 \cdot 6\ H_2O$ (0.19 g), and $CaCl_2$ (0.14 g) made up in 1 liter of distilled water and filtered with 0.22-μm Millipore filter into pyrogen-free containers.
4. The tail flick, a spinally mediated reflex, was assessed by laying the tail across a slit through which light from a focused bulb projected. The time between the light's being switched on to the tail's being abruptly moved was the measured response. In the absence of a response, the light was turned off after 10 seconds to prevent tail damage.
5. The hot-plate response was defined as the interval between the time the animal was placed on a heated surface (55° ± 1°C) to the time it licked its hindpaw. The trial was terminated after 30 seconds if no response was observed.
6. The forelimbs and face were pinched lightly and systematically with a pair of forceps having a pressure area of 3 mm^2. In normal animals, this pinch resulted in squeaking, signs of agitation, and a vigorous effort to dislodge the forceps.
7. The animal was restrained in a plastic box with a lever at one end; its tail protruded from the other. Shock was applied to the tail in an ascending stepwise fashion from 0 to 4 ma by operant programming equipment. If the animal pressed the lever, the shock was reduced one step; if the lever was not pressed, the shock level was raised one step, and so on. In this manner, the animal was able to control or titrate the level of shock that it received [J. Yeung, T. L. Yaksh, T. A. Rudy, *Clin. Exp. Pharmacol. Physiol.* **2**, 261 (1975)]. With this paradigm, the shock titration threshold is the shock that will support an 80 to 90 percent level of escape behavior (T. L. Yaksh and T. A. Rudy, unpublished observation).
8. The four animals were manually restrained, and injections were made into the femoral vein. We entered the vein under visual guidance by exposing the vein through a 1-cm slit after first infiltrating the region with local anesthetic.
9. A. Wikler, *J. Pharmacol. Exp. Ther.* **80**, 176 (1944); W. Knoll, J. Hasse, G. Block, B. Muhlberg, *Int. J. Pharmacol.* **2**, 57 (1963); W. Grossman, I. Jurna, T. Nell, C. Theres, *Eur. J. Pharmacol.* **24**, 67 (1974).
10. W. Grossman and I. Jurna, *Eur. J. Pharmacol.* **29**, 171 (1974); N. Iwata and Y. Sakai, *Jpn. J. Pharmacol.* **21**, 413 (1971); L. Kitahata, Y. Kosaka, A. Taub, K. Bonikos, M. Hoffert, *Anesthesiology* **41**, 39 (1974).
11. O. Calvillo, J. L. Henry, R. S. Newman, *Can. J. Physiol. Pharmacol.* **52**, 1207 (1974).
12. J. G. Sinclair, *Eur. J. Pharmacol.* **21**, 111 (1973).
13. C. B. Pert, M. J. Kuhar, S. Snyder, *Life Sci.* **16**, 1849 (1975).
14. R. Melzack and P. D. Wall, *Science* **150**, 971 (1965).
15. This conversion was used to permit comparison between animals having slightly differing baselines, where the percent maximum effect is the difference between the postdrug and predrug latencies divided by the difference between the cutoff time and the predrug latency, times 100.
16. We thank M. Caine, P. Huang, M. Kuzmicki, R. Plant, and J. Yeung for their assistance in carrying out the experiments and in preparing the manuscript.

2 February 1976; revised 30 March 1976

Evolution on the Level of Communities

Abstract. *According to traditional models, natural selection is largely insensitive to an organism's effect on its community. Effects on the community at large cannot feed back differentially to the organisms that cause them, and, hence, cannot lead to the differential fitness of the organisms. However, if a spatial variation exists in community composition, organisms do differentially feel their own effects on the community, and this leads to a form of evolution on the community level. Without violating the principle of individual selection, the concept of an organism that exists for the "function" it performs in its community may be valid in some cases.*

The idea that biological communities are "super-organisms" has arisen many times in the history of science (*1*). In this analogy a species is likened to an organ whose function can only be understood in terms of its role in the maintenance of a larger whole.

At present there is little theoretical support for the super-organism concept. Current evolutionary theory explains the traits of species in terms of their advantage to individuals; community functions, if they exist, are viewed as coincidental.

Elsewhere I and others have presented a model of structured demes that leads to

a form of group selection (*2, 3*). Here I discuss the consequences of that process for community evolution.

Most models of community dynamics begin with a number (S) of species, at densities $N(1), N(2) \ldots N(S)$. The species are related to each other through a series of differential or difference equations, the change in density of each depending on the densities and interaction coefficients of the others.

The interaction coefficients form the familiar community matrix, which in its most general form includes all types of species interactions (*4*).

$$\begin{bmatrix} a_{11} & a_{12} & a_{13} & \cdot & a_{1S} \\ a_{21} & a_{22} & \cdot & \cdot & \cdot \\ a_{31} & a_{32} & \cdot & \cdot & \cdot \\ \cdot & \cdot & \cdot & \cdot & \cdot \\ a_{S1} & \cdot & \cdot & \cdot & a_{SS} \end{bmatrix} \quad (1)$$

Here $a_{i,j}$ refers to the direct per capita effect of species j on species i. Indirect effects (for example, j's effect on i through its effect on k) are obtained by reiterating the equations for more than one time interval. Any row of the matrix gives the (direct) effect of the community on the species, and the corresponding column gives the effect of the species on the community.

A species can have an effect on itself through its effect on the rest of the community. In fact, given sufficient reiterations, every effect of a species on its community will loop back to influence the species itself, either positively or negatively, by all possible pathways (a pathway being a chain of species connected by nonzero interaction coefficients). As an example, earthworms stimulate plant growth and thereby increase their own resource of dead plant parts, a positive indirect effect consisting of one link.

A species (or a genotype within a species) that cultivates its community so as to maximize its own fitness through indirect effects seems adaptive in the intuitive sense of the word, but it cannot be selected for according to traditional models (*5*). Consider two species (or genotypes within a species) whose interaction coefficients with the rest of the community are

$$\begin{bmatrix} a & b & c & d & e & f & g \\ a & b & c & d & e & f & g \\ h & m & & & & & \\ i & n & & & & & \\ j & o & & & & & \\ k & p & & & & & \\ l & q & & & & & \end{bmatrix} \quad (2)$$

In other words, the species are identical in terms of what they receive from the community (rows), but they differ in their effects on the community (columns). Even though the columns may have profound effects on population dynamics, they cannot alter the relative abundance of the two species. Mathematically the reason is obvious; identical rows in the matrix signify that the equations governing the two species are identical, and therefore a change in relative abundance is impossible. The biological interpretation is more interesting; a species (or genotype within a species) cannot affect itself differentially by its effect on the community at large. All indirect effects loop back to both species equally. In short, according to traditional models, natural selection is largely insensitive to an enormous class of variation—variation in the effect of a species on its community (*6*).

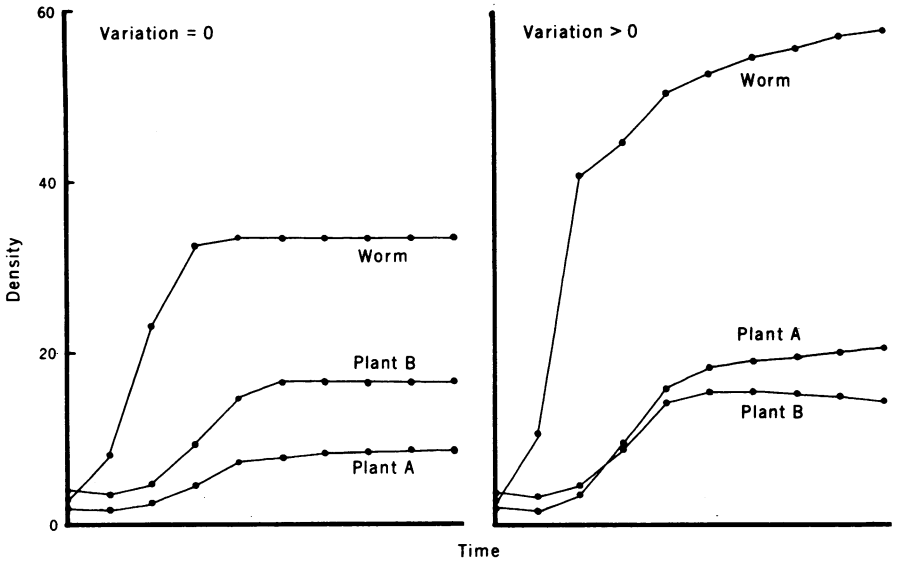

Fig. 1. Population dynamics of a three-species community with and without spatial variation. For this computer run $m_{A,B} = m_E = .01$, $L = 50$, $K_{A,B} = 100$, $K_E = 75$, and the variance in the density of each species was equal to its mean. However, the general patterns hold for any nonzero variance.

Traditional models carry an unstated assumption of spatial homogeneity; that is, they assume that the densities and relative proportions of the species are the same everywhere in the community. However, every community census that I am aware of shows intense spatial variation in these parameters. Variation occurs both over large geographical areas and on minute spatial scales that must be considered intrademic for the populations involved. The model presented here relies on intrademic spatial variation.

Given a nonzero variance in community composition, the average densities of the species become meaningless and must be replaced by weighted averages, or subjectively experienced densities, which differ for each species (*2, 7*).

$$\hat{N}(i,i) = N(i) + s^2(i)/N(i) \quad (3)$$

$$\hat{N}(i,j) = N(j) + \mathrm{cov}(i,j)/N(i) \quad (4)$$

where $\hat{N}(i,j)$ is the density of species j experienced by an average individual of species i; $N(i)$ is the average density of species i; and $\mathrm{cov}(i,j)$ and s^2_i are the covariances and variances of densities over space. If the species are distributed randomly with respect to each other then $\mathrm{cov}(i,j) = 0$ and variation in community composition merely causes an individual, on the average, to experience its own species at a greater density than actually exists in the community. As such it differentially experiences the effect of its own species on the community, and this "exposes" variations in indirect effects to natural selection.

The overall process may be modeled by a computer simulation as follows: (i) take any traditional model [that is, a system of difference equations with specified interaction coefficients and starting densities $N(1), N(2) \ldots N(S)$]; (ii) create a "population" of some number, T, of noninteracting communities, having mean densities of $N(1), N(2) \ldots N(S)$ and specified variances (this simulates spatial variation in species composition); (iii) allow each community to run for a specified number of reiterations (simulating species interactions within each community for a time interval); (iv) mix the system by adding species numbers from all communities and dividing by T to obtain new metacommunity densities, $N'(1), N'(2) \ldots N'(S)$. Then redistribute the organisms back into the communities with mean densities $N'(1), N'(2) \ldots N'(S)$, and the same specified variance as in (ii); (v) repeat (iii) and (iv) as long as desired. Steps (iii) and (iv) simulate a process of alternating dispersal and sedentary

stages fundamental to any model of intrademic spatial variation. The biological justification of the model has been discussed elsewhere (*2*).

As a hypothetical example, consider the earthworm, which improves plant growth through a variety of pathways (*8*). Because plants form the foundation of any biological community, the effects of the earthworm (both direct and indirect) on almost every member of its community are positive. However, the reverse is unlikely to be true; the effects of the community on the earthworm are variable. The success with which the earthworm operates depends to a large extent on the community that surrounds it. Plant litter may or may not be of a shape or texture that is easy to ingest. Secondary compounds leached into the soil may stimulate or inhibit.

Consider two plant species (A and B) that benefit equally from earthworm activity but differ in their effect on the earthworm (E). Let per capita fitnesses equal

$$\frac{N(\mathrm{A})_{t+1}}{N(\mathrm{A})_t} = \frac{N(\mathrm{B})_{t+1}}{N(\mathrm{B})_t} = 1 + m_{A,B}\left\{\left[\frac{N(\mathrm{E})_t}{L+N(\mathrm{E})_t}\right]K_{A,B} - N(\mathrm{A})_t - N(\mathrm{B})_t\right\} \quad (5)$$

$$\frac{N(\mathrm{E})_{t+1}}{N(\mathrm{E})_t} = 1 + m_E\left\{\left[\frac{N(\mathrm{A})_t}{N(\mathrm{A})_t + N(\mathrm{B})_t}\right]K_E - N(\mathrm{E})_t\right\} \quad (6)$$

These are modifications of the logistic equation in which the carrying capacity of each of the plants depends asymptotically on worm activity, while the carrying capacity of the worm depends on the relative proportions of A and B. The constants m and K represent the rate of increase and the maximum carrying capacity, respectively. The constant L governs the rate at which carrying capacities of the plants become asymptotic (*9*).

Simulation results for communities with and without variation are presented in Fig. 1. In traditional models (without variation) A and B retain their starting proportions. Natural selection cannot discriminate between them because, although they vary in their effects on the earthworm, these effects provide feedback to both plant species equally. However, given variation in community composition, each plant species differentially feels its own effect, A is selected for, and such selection eventually causes the extinction of B. The opposite would have occurred if the worm had a negative effect on the plants. In this case the worm and plant A would have been driven to extinction. If the equations are reconstructed such that an optimal density of worms exists as far as the plants are concerned, the proper ratio of A and B automatically results to produce it. Finally, if the worm varies in its effects on the plants, it will evolve to maximize plant fitness (and therefore its own, through indirect effects). The variance was equal to the mean densities in the simulation trials presented here, but the qualitative results occur whenever the variance in species composition is greater than zero.

In this example the earthworm's existence depends on the function it performs in its community. However, this does not violate the principle of individual selection, for per capita fitness is at all times the criterion of selection used in these models. Given sufficient control of the community over its component species, the concepts of individual and community adaptation become synonymous.

DAVID SLOAN WILSON
Department of Zoology,
University of Washington,
Seattle 98195

References and Notes

1. For a history, see M. T. Ghiselin, *The Economy of Nature and the Evolution of Sex* (Univ. of California Press, Berkeley, 1974).
2. D. S. Wilson, *Proc. Natl. Acad. Sci. U.S.A.* **72**, 143 (1975); *Am. Nat.*, in press.
3. E. L. Charnov and J. R. Krebs, *Am. Nat.* **109**, 107 (1975); W. D. Hamilton, in *Biosocial Anthropology*, R. Fox, Ed. [Halsted (Wiley), New York, 1975], pp. 133–155.
4. Matrices imply linear equations; they are used for illustrative purposes but are not necessary for the basic model.
5. Evolution involves a change in the relative proportions of competing "types;" for the purposes of this discussion, it does not matter whether these types are alleles, genotypes, or species. Thus, while evolutionary arguments are usually cast in terms of intraspecific genetic changes (competition between genotypes), interspecific changes in community composition can be the result of the same process (competition between species). The words "species" and "genotype" can be used interchangeably throughout this argument.
6. For a rigorous treatment of the concept, see R. Levins, in *Ecology and Evolution of Communities*, M. L. Cody and J. M. Diamond, Eds. (Harvard Univ. Press, Cambridge, Mass., 1975), pp. 16–50. Two exceptions to this rule are acceptable to traditional models: (i) symbiosis involves a one-to-one interaction between individuals, in which case indirect effects are automatically funneled back to the individual that caused them. In terms of the structured deme model, this corresponds to a trait-group size of one; (ii) if individual recognition is possible, indirect effects can be behaviorally redirected to the individuals that cause them.
7. M. Lloyd, *J. Anim. Ecol.* **36**, 1 (1967).
8. For a traditional view on earthworms, see G. C. Williams, *Adaptation* and *Natural Selection* (Princeton Univ. Press, Princeton, N.J., 1966).
9. In this model the plants depend completely on the earthworm and the earthworm depends completely upon plant *A* (K = 0 without the essential species). This is an unrealistic assumption. However, aside from being useful as an illustrative tool it has one other justification. Multispecies communities probably do have total or near total control over most of their members. The only way to simulate this dependency of a species on its community in a simple model is to increase the amount of control per species.
10. I thank A. B. Clark, T. W. Schoener, C. A. Istock, L. Van Valen, and M. E. Gilpin. Supported by NSF grant BMS 75-17663.

24 December 1975; revised 16 April 1976

Evaluation and Publication of Scanning Electron Micrographs

When applied appropriately and critically to biological materials, scanning electron microscopy (SEM) may reveal important, unanticipated morphological details and relationships. It is particularly well suited to the study of large specimens at high resolution and requires relatively short processing times. The rapid proliferation of published micrographs obtained by this relatively new technique has apparently not been accompanied by the establishment of widely accepted criteria for assessment of their scientific merit. It is our purpose to draw attention to some of the major interpretative pitfalls and to suggest practical guidelines for review and publication.

Initially, our attention was drawn to this problem by a difference of opinion concerning the normal configuration of the arterial endothelial surface. Smith *et al.* (*1*), describing the appearance of the arterial endothelial surface as revealed by SEM, identified luminal projections arising from the lining of canine pulmonary arteries as normal structures of endothelium. Wolinsky (*2*), in a subsequent technical comment, suggested that the projections might have been produced by retraction of the vessel wall before or during fixation and proposed that such structures might be absent if vessels were examined in the physiologic (that is, distended) state. Since that exchange, SEM descriptions of the luminal surface of undistended arteries have continued to appear, but the controversy regarding the appearance of the normal endothelial surface has remained unsettled. Quite recently, for example, Fujimoto *et al.* (*3*) considered endothelial microvilli in their own undistended vessels to be normally occurring structures. They cited the findings of Smith *et al.* in confirmation but ignored Wolinsky's challenge. In an attempt to resolve this problem we undertook a detailed study of the intimal surface appearance of arteries fixed in situ by perfusion at various controlled pressures. We found, as Wolinsky predicted (*2*), that most of the surface projections were absent when intraluminal pressures were maintained at physiologic levels during fixation. In addition, other endothelial projections and surface details, such as bridges and undulations,