BOOKS: ECOLOGY

Streamlined Complexity

Ilkka Hanski

The community of birds, mammals, and lizards on the Pacific island of Guam has been fundamentally transformed over the past 50 years, in the wake of introductions of alien species—most notably a brown tree-snake (*1*). There is no doubt that the changes in the species composition, especially the extinction of 22 native terrestrial vertebrates, have been caused by complex interactions among the introduced and native species. But could ecologists have predicted the changes that actually occurred? Or consider the renowned population cycles of boreal lemmings and voles. In northern Fennoscandia the impressive regularity of the 4- to 5-year cycle, documented for the previous 40 years, has been failing since the mid 1980s. Do ecologists know why? Is the current pattern the result of climate change, an effect of the introduced American mink, or mere complexity—the sort of behavior that we should expect from complex dynamics (*2*)?

Untangling Ecological Complexity
The Macroscopic Perspective
by Brian A. Maurer
University of Chicago Press, Chicago, 1999. 261 pp. $50, £39.95. ISBN 0-226-51132-4. Paper, $18, £14.50. ISBN 0-226-51133-2.

These were the kinds of questions about ecological communities that I had in mind when picking up *Untangling Ecological Complexity*. Community ecology is a vast subject, so vast that ecologists have typically managed to consider only one perspective at a time. Maurer presents an enlightening précis of past dichotomies: communities as tightly knit complexes versus communities as haphazard assemblages of individual species; communities modeled by sets of differential equations versus communities described by techniques of clustering and ordination; communities teased apart by detailed experimental studies versus communities viewed from a far distance as statistical entities.

This third dichotomy has been dubbed "microecology" versus "macroecology," and it is the focus of *Untangling Ecological Complexity*. Maurer himself helped to establish macroecology a decade ago (*3*). In this book, he ranges from commentaries on experimental and modeling studies of local communities (the micro-scale) to exposés of large-scale ecological patterns and their explanations. The macroecological patterns he discusses include relationships among body size, geographic range size, and population density; links between species diversity and area and between distribution and abundance; and a bell-shaped decline of population density toward the periphery of the geographical range. (Curiously, Maurer does not offer a single example of this decline; the one map of spatial distribution of abundance that he illustrates implies something quite different.)

Maurer is tempted to dismiss much of the experimental research on community ecology as a mistaken focus on noise. In his view, although local populations and their interactions can be enumerated, continuing to study them will lead to descriptions of unique situations rather than to useful generalizations. He argues that the scale of experiments is too small and that it cuts off populations and communities from their proper spatial context. Maurer's gaze is on continents.

What does the case of Guam suggest? Could the community dynamics in Guam have been predicted by intensive studies of the constituent populations and by current microecological theory? I cannot see why not. Theory predicts that a haphazard assembly of species, such as the mix of the introduced and native species in Guam, is likely to produce an unstable, complex community (*4*). Ecological theory armed with differential equations would have predicted drastic abundance changes, cascading effects, and species extinctions; a paradise lost. We do not see such dramatic changes happening everywhere around the world. Is this because in most places ecological communities are not, after all, haphazard assemblages? And if so, at what spatial scale are they structured? (This is a macroecological question not asked by Maurer.) The demise of Guam's fauna was apparently brought about by the lack of coevolution between a versatile introduced predator and native, but naïve, prey; the abundance of more resistant introduced prey, which maintained high snake abundances (up to 100 snakes per hectare); and habitat loss, which forced native prey to use the disturbed habitats favored by introduced predators. These are useful and testable microecological generalizations.

Maurer's emphasis is on continents and on bird communities. From that perspective, his impatience with the community ecology of differential equations and small-scale experiments is intelligible. Yes, bird "communities" in small plots of prairie belong to something much more extensive. But what about plant communities in the prairie? What about insect communities? Much of our understanding of these, and many other, communities—though admittedly enriched in recent years by the increasing attention paid to spatial coupling among neighboring communities—has emerged from detailed studies of local interactions. What worries me about glossing over the local "noise" is that while neat patterns may emerge, the cost may be reduced hopes of answering the kinds of questions that community ecologists are generally asked.

To be fair, Maurer is not suggesting that macroecology should replace all of community ecology. And he has important points to make about the limitations of experiments on communities, which he illustrates with informative examples. For instance, failure to detect a treatment effect beyond a short period of time may be due to unexpected large-scale effects influenc-

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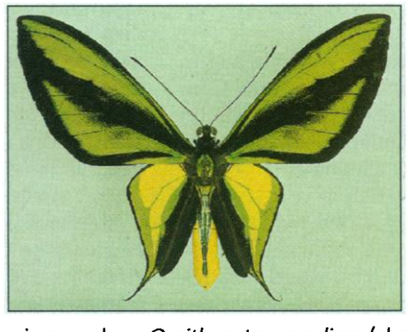
BROWSINGS



The Butterflies of Papua New Guinea. Their Systematics and Biology. *Michael Parsons.* Academic Press, San Diego, 1999. 916 pp. $275, £185. ISBN 0-12-545555-0.

Epitomized by the giant birdwings, such as *Ornithopetra paradisea* (above, male, 125-mm wingspan), New Guinea's rich butterfly fauna (960 species) has long attracted evolutionary ecologists and amateur naturalists. Building on his more than 15 years of museum and field study, Parsons describes all the major forms and morphs from Papua New Guinea and summarizes their ecology, evolution, and conservation.

CREDIT: MICHAEL PARSONS/*BUTTERFLIES OF PAPUA NEW GUINEA*

ing both the treatment and the control. Communities may not retain a stationary state for the duration of long experiments—which, however, is also a problem for observational studies.

The key conceptual constructs in *Untangling Ecological Complexity* draw on A. J. Lotka's comparison of populations with an ideal gas in a container, where the actual space occupied is settled by the tension between the internal pressure to expand and the constraining external pressures. This research program becomes ambitious, because its aim is to predict the sizes of geographic ranges from species' demographic characteristics. Though I find it difficult to reconcile species' range dynamics with thermodynamic principles (given the limited mobility of individuals in most species), Maurer's perspective leads to analyses of continental species-area and distribution-abundance relationships that have merit. He shows how sampling alone cannot explain the patterns in the distribution of species. More successful is a model of structured geographic ranges—that is, species having dissimilar range sizes and

EXHIBITIONS: DISEASES

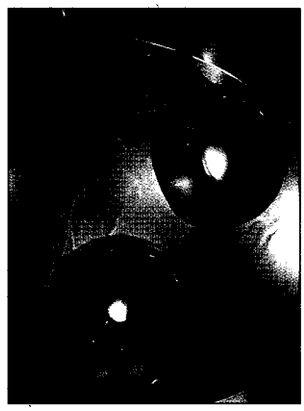
Magical Microbe Mystery Tour

Epidemic!
The World of Infectious Disease
Rob DeSalle, curator

At the American Museum of Natural History, New York City, through 6 September 1999.

It is 1993. In a dusty cabin inhabited by the scurrying shadows of deer mice, a flickering television screen carries news bulletins about the inexplicable deaths of several young Navajo in the remote southwestern United States from a respiratory illness. Fast forward six months, and the bulletins tell us how this medical mystery was finally solved and the culprits (deer mice carrying hantavirus) identified. So begins *Epidemic! The World of Infectious Disease*, the latest exhibition at the American Museum of Natural History. Three years in the planning, this sophisticated show is a masterpiece of creativity. The visitor journeys through different sections where dioramas, interactive touch-screen computer displays, videos, and text explain the biology of microbes, how they infect us, and how a local outbreak of an infectious disease can quickly escalate into a pandemic.

The highlight of the exhibition is the "Microbe Gallery" graced by exquisite three-dimensional models of microbes that took 25 modelers 5 months to make. (Top right, a green *Shigella* bacterium nears completion, with a finished pinkish-blue HIV particle in the background). Iridescent green globes of flu virus hover overhead, their surface convolutions mimicking the topography of the glycoprotein markers that distinguish different strains of the virus. Red blood cells infected by malaria parasites prepare to release their cache of merozoites that will invade new erythrocytes (center). The sinister pork tapeworm, *Taenia,* leers at visitors over a plexiglass wall, and *Giardia* parasites lurk like enemy space shuttles ready to invade their human hosts.



Floating spheres of HIV and flu virus lead visitors on to a section showing the different ways that microbes infect the human body. White spandex shrouds evoke mucosal membranes in the mouth—an entry site for flu virus. In a huge cross-sectional model of a T cell, the steps of HIV infection—invasion, reverse transcription, DNA integration, protein production, virus assembly, and budding—are illuminated in sequence. Nearby, visitors can choose to be a microbe or the host immune system in a computer game where the protagonists battle to the death.

The part of the exhibit devoted to the study of microbes is noteworthy for its landscape of contrasts. A handheld 17th-century microscope designed by van Leeuwenhoek sits near a model of an electron microscope. A bank of interactive computer displays allow visitors to carry out virtual diagnostic tests—an ELISA assay, a Western Blot, or a PCR reaction—for different microbes. A model of a Biosafety Level 4 laboratory with a scientist in a sealed life-support suit stands close to microscopes through which visitors can view slide preparations of deadly microbes.

The segment of the exhibition devoted to epidemics displays a number of imaginative dioramas. An air-conditioning pipe rumbles overhead signifying the 1976 outbreak of Legionnaire's disease in a Philadelphia hotel, which resulted in 30 deaths and was finally traced to *Legionella* contamination of the hotel's cooling system. There is a model of the Broad Street pump that John Snow identified as the source of contaminated water, the cause of a cholera outbreak in London in 1854. Ropes tethering a merchant ship in a Venetian port provide the perfect escape route for stowaway rats carrying bubonic plague, which decimated the population of Venice in 1347.

The exhibition's organizers do not shy away from presenting one of the greatest plagues of this century: AIDS. In a simple diorama, a series of disembodied arms pass around a blood-stained needle and syringe, making clear the ease with which HIV can be transmitted through sharing contaminated needles. In "Talking about AIDS," the closing part of the show, familiar settings—a subway platform, a park bench, a newspaper kiosk—project audio information about the latest AIDS statistics, bringing home the presence of AIDS in everyday life.

Accompanying *Epidemic* are a series of eight lectures on the biology of infectious diseases and their impact on public health, an AIDS symposium, and a series of documentaries and films about microbes. Those unable to make the trip to New York City may still enjoy a virtual version of the exhibition at http://www.amnh.org/exhibitions/epidemic/, and the exhibit is set to tour the United States after its run in New York.

Epidemic is a fitting sequel to *The Exhibition Illustrating the World's Work Against Tuberculosis* mounted by the Museum in 1908, which drew nearly a million visitors during its limited 6-week run. The goal of that exhibit was to educate the public about tuberculosis, an incurable disease rampant among the poor immigrants of New York City's overcrowded tenements. Ninety years later, the Museum continues its commitment to education and public health with *Epidemic*, which should reach an audience of many millions more than its predecessor.

—ORLA SMITH

positions, which determine the composition of local communities. Perhaps an even more fundamental element in Maurer's model is its assumption about the positive relationship between range size and average local density.

Macroecology emerged to challenge an approach to community ecology obsessed with experiments and conducted at a small spatial scale in simplified communities. Whatever you call the macroscopic perspective Maurer advocates, we need to document large-scale ecological patterns. We need to retain the broader ecological contexts of local communities and to understand how regional processes influence them. Disentangling ecological complexity is a vast task in which many approaches are required. Whether the one that Maurer provides will help us meet our unresolved challenges remains to be seen.

References

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PERSPECTIVES: NEUROBIOLOGY

Dissecting Dendrite Dynamics

Stephen J. Smith

The functioning of the brain depends on the interconnections of billions of neurons through trillions of synapses. But what developmental processes could possibly guide the correct formation of such vast numbers of synaptic connections? This question is at the heart of understanding brain development and the storage and processing of information throughout life. Although the surface of this problem has barely been scratched, some of the scratches have become a little deeper with new observations by Maletic-Savatic and colleagues reported on page 1923 of this issue (*1*). A confluence of new optical imaging methods enabled these investigators to take a much closer look at the dynamics of neuronal structure in developing brain tissue. They conclude that patterns of electrical activity may shape the morphology of developing neurons by promoting new dendritic extensions called filopodia, which may in turn initiate the formation of new synapses (synaptogenesis). The authors go on to show that activation of the *N*-methyl-D-aspartate (NMDA) receptor by electrical activity may be the event that triggers filopodial extension and synapse formation.

Enhanced online at www.sciencemag.org/cgi/content/full/283/5409/1860

Two decades of experiments have demonstrated that the electrical activity of neurons can shape patterns of synaptic interconnections during early development (*2*). For instance, nerve impulses in the pathways that carry sensory information influence the functional maps of the brain areas that receive these impulses. But it is not just the total number of impulses that count; temporal or spatial patterning of impulse activity may critically influence the shaping of such brain maps. The formation of memory in the mature brain also may involve activity-dependent morphological changes in neurons similar to those seen in early development (*3*). Although there are numerous well-documented examples of electrical activity driving neuronal morphogenesis, there have been few clues to indicate how this comes about.

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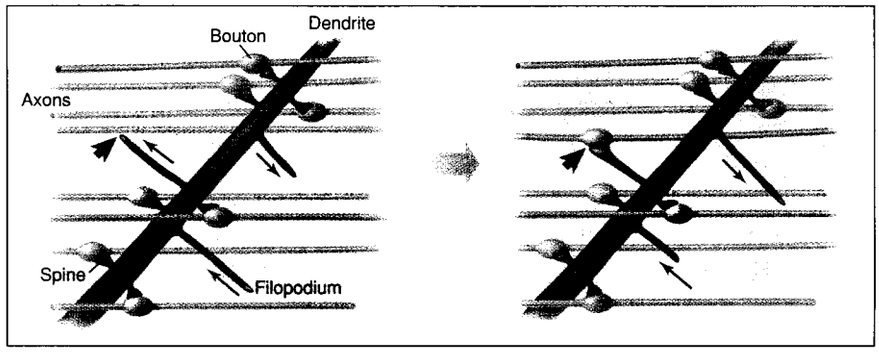
One molecular lead implicates the NMDA receptor, which binds the neurotransmitter glutamate at excitatory synapses in the central nervous system (*4*). Compared to other types of glutamate receptor found at excitatory synapses, the NMDA receptor plays a minor role in generating postsynaptic electrical responses. Rather, upon activation by glutamate, the NMDA receptor promotes a local influx of calcium ions (*5*). The effects of NMDA receptor activation are thus much more localized than those of other glutamate receptors whose electrical signals are conducted over much greater distances. Another property peculiar to the NMDA receptor is that it must be activated both by glutamate and by membrane depolarization to permit local influxes of calcium ions. This "associative" property enables NMDA receptors in the postsynaptic membrane to potentially discriminate between temporal and spatial patterns of impulses arriving at a given neuron. Evidence that NMDA receptors affect the neuronal morphogenesis of early development primarily comes from experiments with highly selective antagonists of these receptors such as APV [D,L(−)-2-amino-5-phosphonovaleric acid]. But, there have been no clues to indicate which steps in neuronal morphogenesis are affected by NMDA receptor activation and calcium influx.



Dendrite dynamics. The spines on the dendrites of neurons are relatively stable structures. In contrast, the slender extensions of dendrites called filopodia are dynamic, exhibiting both extension and retraction (blue arrows). The red arrowhead indicates the site where a protruding filopodium contacts a neighboring axon, possibly initiating the formation of a synaptic junction. After contact, the filopodium becomes a dendritic spine. The process of filopodial extension and synapse formation is triggered by electrical activity and the activation of NMDA receptors. [Adapted from (*1, 6, 7, 13*)]

Provocative ideas about how electrical activity and neurotransmitter release might affect the morphology of neurons come from dynamic optical microscopy. For example, in living, dissociated neurons in culture the growing tips (growth cones) and filopodia of both axons and dendrites exhibit motility (in the form of membrane protrusions) that can initiate cell-cell contact and synaptogenesis (*6–8*). This form of motility may be modulated by neurotransmitters and