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LETTERS

Hydrocoral Species Not Extinct

Prolonged seawater warming that accompanied the 1982 to 1983 El Niño–Southern Oscillation (ENSO) disrupted marine ecosystems in the eastern Pacific Ocean, bleaching (through the loss of endosymbiotic zooxanthellae) and killing reef-building corals (*1*). Two hydrocoral (*Millepora*) species, *M. platyphylla* Hemprich and Ehrenberg and *M. boschmai* de Weerdt and Glynn, were not found alive in their former range in the Gulf of Chiriqui, Panama, for almost 9 years. *M. intricata* Milne Edwards, a third Indo-Pacific hydrocoral species in the eastern Pacific also virtually disappeared at that time. A few small colonies reappeared in 1985 on the Uva Island reef in the Gulf of Chiriqui, and by 1987 populations had expanded to several scattered patches of up to 5 square meters.

A thorough search from 1984 through 1990 over the former ranges of *M. platyphylla* and *M. boschmai* in the Gulf of Chiriqui revealed only dead colonies. As *M. platyphylla* lives throughout the Indo-Pacific Province, its disappearance in the eastern Pacific was regarded as a local extirpation (*2*) and the disappearance of *M. boschmai*, a newly named eastern Pacific endemic species (*3*), as an extinction event.

The discovery of five live colonies of *M. boschmai* at Uva Island, Gulf of Chiriqui, Panama, in 1992 shows that this species has survived. The colonies exhibited normal pigmentation and appeared healthy in February and June, although this was not the case for all reef-building corals over this period. Partial bleaching occurred in five scleractinian coral species in the Gulf of Chiriqui when ambient reef temperatures were anomalously high, a result of the strong 1992 El Niño (*4*). Zooxanthellae densities and chlorophyll *a* concentrations indicated that bleaching was a result of zooxanthellae loss rather than reduced chlorophyll concentration per zooxanthella. Mean sea surface temperature (SST) was 30.0°C (n = 20) on Uva Reef from 16 to 22 February, which is about 1.0° to 1.5°C above normal. Satellite-derived data documented that SST values of 29° to 30°C dominated the Gulf of Chiriqui from February through April 1992 (*5*).

We assume that the skeletal elongation rate of the platy *M. boschmai* is one-half that of the dendritic *M. intricata*, which is 5.5 centimeters (cm) per year (*2*), and estimate that the smallest colony of *M. boschmai* (12 cm high) is about 4 years old and the largest colony (21 cm high) about 7.5 years old. Recently discovered colonies that were recruited to Uva Island after the 1983 ENSO dieoff could have been derived from remnant colonies that dispersed from local cryptic refugia (*1*, *6*). It is also possible that propagules originated from yet-undocumented populations in the central or western Pacific.

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Lyme Disease: Asking the Right Questions

I would like to elaborate on some of the issues discussed in Marcia Barinaga's article about the controversy at the Fifth International Conference on Lyme Borreliosis (News & Comment, 5 June, p. 1384). The academic Lyme disease researchers would have us believe that there is a methodological conflict between their own studies and their clinician opponents' "anecdotal" findings. Nothing could be further from the truth. Although criticisms of the offending abstracts submitted before the conference were not entirely without merit, the presumption that the existing body of academic Lyme disease literature represents some sort of scientific ideal is ludicrous.

The central flaw in the current Lyme disease orthodoxy is the persistent myth of "post-Lyme syndrome." This condition was suggested by researchers to account for the troublesome fact that many patients do not

fully recover after supposedly curative antibiotic therapy, but continue to suffer from chronic headaches, cognitive deficits, debilitating fatigue, and paresthesias. These persistent symptoms are explained away by the fibromyalgic syndrome (*1*), which provides a convenient sense of closure to researchers but leaves patients in the throes of devastating chronic illness.

There is ample evidence to retire this model in favor of one involving chronic infection. Some researchers have successfully cultured *Borrelia burgdorferi* from the skin or cerebrospinal fluid of patients after antibiotic regimens generally accepted as curative by academic researchers (*2*), while other clinicians have recovered the bacteria from patients who have undergone even long-term high-dosage antibiotic therapy (*3*). In addition, researchers have demonstrated that *B. burgdorferi* can penetrate and persist within human endothelial cells (*4*) and fibroblasts (*5*). Yet most academic researchers continue to deny the prevalence of chronic infection in Lyme disease.

There are also flaws with the academicians' diagnostic protocols. Lyme disease presents physicians with a diagnostic dilemma because its symptoms are so diverse and the commonly available serological tests used in diagnosis are known to be unreliable. Thus, while it may seem reasonable for researchers to insist that clinicians confine their studies on long-term therapy to patients who are demonstrably seropositive, it is scientifically—and morally—indefensible to advocate a rigid adherence to this overly restrictive diagnostic procedure in a clinical setting to determine treatment. Arbitrarily withholding antibiotic therapy from all seronegative patients guarantees that an unacceptably high percentage of them will go on to develop incurable late-stage Lyme disease. Such a policy also can lead to the underreporting of the real incidence of Lyme disease. The artificially low figures are in turn used by researchers to reinforce their claim that Lyme disease is actually quite rare. Thus the cycle of denial is complete. The facts, of course, strongly suggest that Lyme disease is seriously underdiagnosed. New testing techniques presented at the international conference (6) indicate that Lyme disease will be found to be significantly more common than previously recognized.

The rejection of the offending abstracts at the conference had much more to do with their conceptual challenge to current paradigms in Lyme disease research than with their alleged scientific deficiencies. Good science is as much about asking the right questions as it is the sensible pursuit of answers, and many Lyme disease patients do not feel that the mainstream Lyme disease researchers are asking the right questions.

The existing theories need to be reevaluated in light of the emerging evidence on chronic infection in late Lyme disease.

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Enantiomerically Pure Drugs

In response to an article by Ivan Amato about asymmetric synthesis ("Looking glass chemistry," Research News, 15 May, p. 964), John F. Beary III and C. Robert Eaton, the senior vice president and the manager of the research and development programs of the Pharmaceutical Manufacturers Association, urge discretion in the development of a drug regimen in which all new chiral entrants are marketed only as the pure active enantiomer, in cases where the bioactivity resides mainly or solely in that enantiomer (Letters, 10 July, p. 145). They cite the potential additional cost and delay incurred in the synthesis of pure enantiomers on a clinical scale but do not mention impending developments in chemical synthesis (the thrust of Amato's article) which will reduce the time required for such a regimen to well within the time frame of 12 years cited by Beary and Eaton as that needed for the launch of a drug de novo.

To illustrate their points they cite the case of ibuprofen, the Boots nonsteroidal anti-inflammatory agent currently marketed as a racemate. Only the S enantiomer is active, but as they remark, the R enantiomer is converted into S in a unidirectional manner in vivo. This fact is used to support their case that the therapist might as well administer the racemic mixture as the pure active S enantiomer. Unfortunately, a