Elimination of Two Reef-Building Hydrocorals Following the 1982–83 El Niño Warming Event

P. W. GLYNN AND W. H. DE WEERDT

One probable extinction and one range reduction of eastern Pacific reef-building hydrocoral (*Millepora*) species mark the first documented cases of species eliminations resulting from the worldwide 1980s coral reef bleaching events. Two of 12 Panamanian coral species were eliminated suddenly from their former ranges by prolonged high sea temperatures during the 1982–83 El Niño–Southern Oscillation event. Three conditions contributed to their demise: high sensitivity to sea warming, populations confined to a small geographic area, and bathymetric restriction to the euphotic zone (≤20 meters depth) where El Niño sea warming had its greatest effect.

EXTINCTION EVENTS ARE WELL DOCumented in the geologic record among marine and terrestrial biotas and in extant terrestrial ecosystems, especially in tropical latitudes during the present century (*1*). In contrast, no species extinctions have been noted in coral reef ecosystems despite widespread tropical marine habitat disturbances over the past few decades (*2–4*). We describe the disappearance of two zooxanthellate hydrocoral (*Millepora*) species that were present in reef coral communities on the Pacific coast of Panama before the extraordinarily strong 1982–83 El Niño–Southern Oscillation (ENSO) event. The hydrocorals were among the first reef-building species to lose their zooxanthellae during the ENSO sea warming event and subsequently experienced the highest mortalities of all affected coral reef organisms. The two hydrocoral species have not been seen alive in Panama since 1983, despite intensive searches over their formerly known ranges. This may represent the first documentation of an extinction (total loss of genetic diversity) and an extreme range reduction of coral reef species, perhaps a harbinger of future extinctions should habitat destruction and coral reef bleaching events continue.

Three species of hydrocorals (Phylum Cnidaria, Class Hydrozoa) were first discovered in the eastern Pacific, Gulf of Chiriqui, Panama (Fig. 1) in 1970 (*5, 6*). These hydrocorals were represented by two widely distributed Indo-Pacific species (*Millepora intricata* Milne Edwards and *Millepora platyphylla* Hemprich & Ehrenberg) and one still undescribed species (*Millepora* sp. nov.) (7). The nearest known populations of *M. intricata* occur in the Caroline Islands and *M. platyphylla* in the Marquesas Islands, 13,000 km and 6,600 km west of Panama, respectively (*8*). Subsequent studies revealed large populations of these hydrocorals throughout the Gulf of Chiriqui, but nowhere else in Panama. Before 1983, *Millepora* spp. represented 0.9% of the total cover of photosymbiotic corals in the lower forereef slope at the Uva Island reef (*20*). *Millepora* spp. have not been found in any of several coral reefs extensively studied in the Pacific Panamic Province (Mexico, El Salvador, Costa Rica, Colombia, mainland Ecuador, and the Galapagos Islands), and were probably restricted to the Gulf of Chiriqui (*9–19*).

The evidence linking coral bleaching with the high temperature anomalies of the severe 1982–83 El Niño event is cogent. All zooxanthellate reef coral species in the equatorial eastern Pacific experienced some degree of bleaching and mortality during the prolonged (5 to 6 months) warming (2° to 3°C above normal) period. Interregional comparisons in the eastern Pacific indicated that mortality rates were related to magnitude and deviation of high temperature anomalies (*23*). Although information on the thermal tolerance limits of *Millepora* spp. is lacking, their field responses to sea warming in Panama (*20*) and elsewhere (*4, 24*) were similar to those reported for scleractinian corals (*4, 25*).

The three *Millepora* species were severely bleached during January through March 1983 (Fig. 2), and by May 1983 no living colonies could be found in the Gulf of Chiriqui (*20*). Compared with photosymbiotic scleractinian corals, the hydrocorals appeared to be more sensitive (that is, all colonies were fully bleached) and most died shortly after the initial bleaching response. Numerous colonies of most scleractinian corals regained their zooxanthellae and were recovering by October 1983. By the end of April 1983, no live *Millepora* were observed to 20-m depth on the approximately 3-ha Uva Island coral reef or elsewhere in the Contreras Islands (four sites), at Coiba Island (three sites), or on mainland coral reefs around Bahia Honda (three sites). The only known surviving hydrocoral was a 2-cm branch of *M. intricata* found on a nearly 6-ha coral reef in the Secas Islands (four sites) in late October 1983 (*21*).

Intensive surveys in the Gulf of Chiriqui, from 1983 through 1990, have revealed only *M. intricata*. Several small colonies of *M. intricata* began to colonize basalt substrata at Uva Island in 1985, and by May 1987 recruits were seen on the Uva reef. Since no *Millepora* survivors were present on the reef (about 100 m from the earliest colonies that recruited on basalt), we assume that the reef recruits were sexually derived. Only seven colonies have grown to a large size (20- to 40-cm diameter) since 1987. Some of the large colonies are now reproducing by fragmentation. The parent colonies have realized rapid growth during 4 years (1987 to 1990) with a mean branch extension of 4.6 ± 0.7 mm per month (mean ± 95% confidence interval). If *M. platyphylla* or *Millepora* sp. nov. demonstrate equally rapid growth, then colonies recruiting from survivors of the 1983 mortality event should be clearly visible after 7.5 years.

Thus, we conclude that two species of *Millepora* have been eliminated from the Panamic Pacific Province. Recolonization of *M. platyphylla* could result from long distance transport of propagules across the

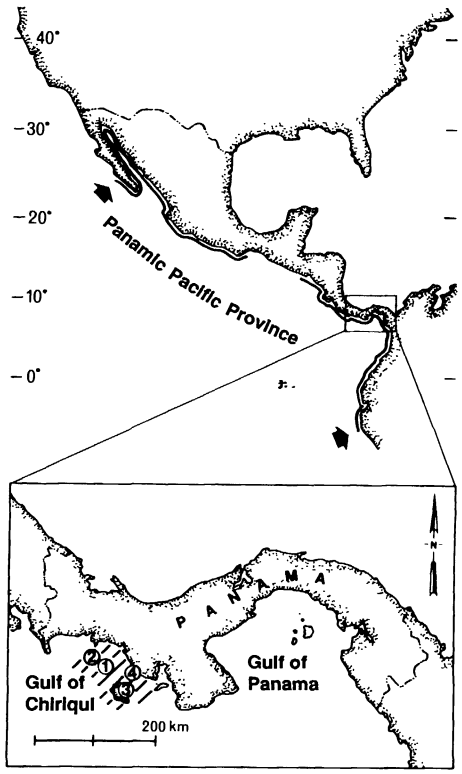

Fig. 1. The pre-1983 distributional range of *Millepora* spp. in the Gulf of Chiriqui, Panama, is denoted by diagonal lines. Circled numbers identify 1, the Contreras Islands; 2, Secas Islands; 3, Coiba Island; and 4, Bahia Honda.

P. W. Glynn, Division of Marine Biology and Fisheries, Rosenstiel School of Marine and Atmospheric Science, University of Miami, 4600 Rickenbacker Causeway, Miami, FL 33149.
W. H. de Weerdt, Department of Invertebrate Zoology, National Museum of Natural History, Smithsonian Institution, Washington, DC 20560.

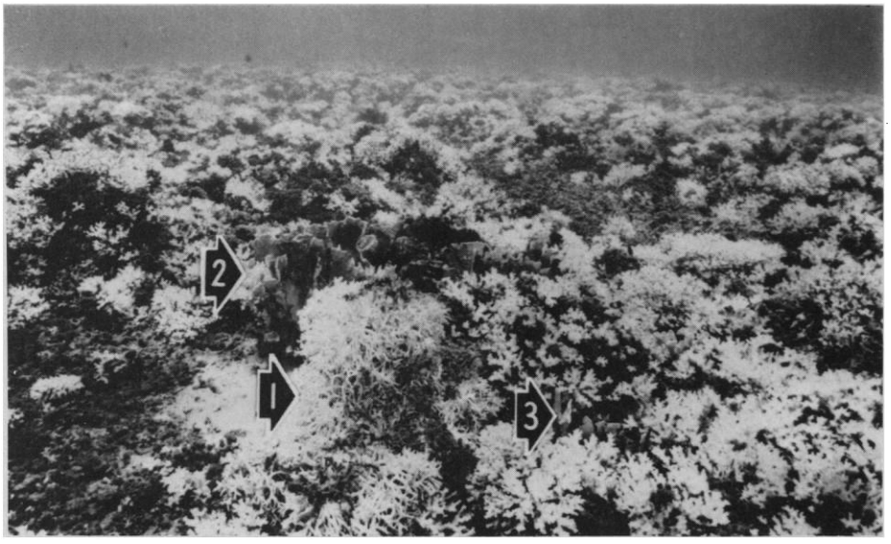

Fig. 2. Bleached scleractinian corals and hydrocorals, Coiba Island, 6 m depth, 27 April 1983. Numbered arrows identify 1, *Millepora intricata*; 2, *Millepora platyphylla*; and 3, *Millepora* sp. nov.

Pacific Ocean (*22*). Even if the planktonic larvae of *Millepora* are short-lived, *Millepora* is known to attach to pumice (*26*) and perhaps their initial arrival into the eastern Pacific occurred by long-distance rafting by way of the North Equatorial Counter Current.

An alternative hypothesis, that *Millepora* sp. nov. is present but unknown in the central-western Pacific, is less tenable than the extinction hypothesis for several reasons. The *Millepora* species of the Pacific Ocean are relatively well known and since *Millepora* sp. nov. was conspicuous and abundant in Panama, it would probably be noticed if it were present elsewhere. Also, *Millepora* sp. nov. was absent from six major museum collections of hydrocorals studied in Europe and North America. Additionally, there are far fewer species of reef-building hydrocorals than scleractinian corals (*27*). Only four new species of *Millepora* have been described since 1884. This description rate of one new *Millepora* species per 23.5 years indicates a low likelihood that new species remain to be discovered.

Unlike documenting the loss of conspicuous terrestrial species, the task of documenting marine extinctions is much more difficult (*28*). Only one other historical extinction of a marine invertebrate, a limpet inhabiting eelgrass, has been published (*29*). Because eastern Pacific *Millepora* spp. were restricted to the Gulf of Chiriqui, it was possible to conduct intensive searches over all habitats within their formerly (pre-1983) known range. Further, these hydrocorals are highly visible, sessile organisms that deposit massive calcareous skeletons. *Millepora* sp. nov. and *M. platyphylla* produced 30-cm and 80-cm-diameter colonies, respectively, prior to 1983.

The recent disappearance of *Acropora valida* (Dana) from the eastern Pacific reef coral fauna may represent another regional elimination related to the 1982–83 ENSO event (*30*). A small population of the wide-ranging Indo-Pacific *A. valida* was discovered at Gorgona Island, Colombia, in late 1983 and then vanished thereafter (*31*). Since the condition of this species was not observed during the El Niño sea warming period, the cause of its demise is not known. Other potentially stressful El Niño–related conditions that precede or follow sea warming events, such as dinoflagellate blooms and strong anti–El Niño upwelling, also cause coral mortalities (*25, 32*).

Several eastern Pacific scleractinian populations experienced local eliminations—that is, disappearances from particular reefs, islands or archipelagoes, following El Niño coral bleaching and mortality responses. For example, *Porites panamensis* Verrill was not present on most coral reefs in Panama after 1983, and *Pocillopora elegans* Dana, *Pocillopora damicornis* (Linnaeus), and *Tubastraea tagusensis* Wells in the Galapagos Islands disappeared from all sites where they were formerly abundant (*31, 34*). All of these species except *T. tagusensis* have reappeared, but in many instances they have not recolonized the coral reef habitats they occupied before the disturbance. It is possible that the non-zooxanthellate *T. tagusensis*, a Galapagos endemic, is also extinct. However, because this species has not been well studied its status is problematical.

The eastern Pacific reef coral fauna, consisting of several small, geographically isolated populations in a highly varying environment, would seem to be especially vulnerable to extinction (*32*). The extremely restricted distribution of *Millepora* spp., members of the peninsula-like Panamic Province, was confined further to offshore islands in the Gulf of Chiriqui, a relatively small (100 by 150 km) body of water. Eighteen to 65 El Niño events of magnitude comparable to the 1982–83 event are thought to have occurred during the Holocene (*35*). Therefore, such disturbances may in large part be responsible for the meager development of coral reefs and low coral species diversity in the tropical eastern Pacific region. How *Millepora* spp. managed to survive earlier warming episodes is not known, but may be related to the extensive geographic spread of the 1982–83 El Niño, which, unlike previous events, reached to 10°N latitude (*30*).

With the exception of Emiliani *et al.* (*36*), who invoked high temperatures to explain Late Cretaceous tropical marine extinctions, the cause of coral reef extinctions has focused on periods of global sea cooling (*37*). The recent patterns of coral bleaching and mortality associated with high temperature stress suggest that slight and sustained thermal increases should be explored further as a physical agent of tropical marine species extinctions. If present species eliminations associated with ENSO warming can be extended to possible future global warming, then reef-building corals with restricted ranges, or small populations at their distributional limits, could succumb to slight but sustained sea warming.

REFERENCES AND NOTES

1. P. W. Signor, *Annu. Rev. Ecol. Syst.* **21**, 509 (1990); E. O. Wilson, Ed., *Biodiversity* (National Academy Press, Washington, DC, 1988).
2. B. Salvat, Ed., *Human Impacts on Coral Reefs: Facts and Recommendations* (Antenne de Tahiti Museum E.P.H.E., Papetoai, Moorea, 1987).
3. United Nations Environment Programme/International Union for Conservation of Nature and Natural Resources, *Coral Reefs of the World* (IUCN Publications Services, Cambridge, England, 1988), vols. 1–3.
4. E. H. Williams, Jr., and L. Bunkley-Williams, *Atoll Res. Bull. 335* (1990), p. 1.
5. P. W. Glynn, *Bull. Biol. Soc. Wash.* **2**, 13 (1972).
6. _____, R. H. Stewart, J. E. McCosker, *Geol. Rundsch.* **61**, 483 (1972).
7. W. de Weerdt and P. W. Glynn, *Zool. Med. Leiden*, in press.
8. H. Boschma, *Zool. Verh. Leiden* **1**, 3 (1948).
9. J. W. Porter, *Bull. Biol. Soc. Wash.* **2**, 89 (1972).
10. P. W. Glynn and G. M. Wellington, *Corals and Coral Reefs of the Galapagos Islands* (Univ. of California Press, Berkeley, 1983).
11. J. W. Durham and J. L. Barnard, *Allan Hancock Pac. Exped. 16* (1952), p. 1.
12. D. F. Squires, *Bull. Am. Mus. Nat. Hist.* **118**, 367 (1959).
13. J. Cortes and M. M. Murillo, *Rev. Biol. Trop.* **33**, 197 (1985).
14. H. M. Guzman and J. Cortes, *Mar. Ecol.* **10**, 23 (1989).

15. H. von Prahl and M. Alberico, *Isla de Gorgona* (Talleres Graficos Banco Popular, Bogota, 1986).
16. C. Birkeland, D. L. Meyer, J. P. Stames, C. L. Buford, *Smithson. Contrib. Knowl. Zool.* **176**, 55 (1975).
17. H. G. Gierloff-Embden, *La Costa de El Salvador* (Direccion de Publicaciones, Ministerio de Educacion, San Salvador, 1976).
18. J. W. Durham, in *The Galapagos, Proceedings of the Symposia of the Galapagos International Scientific Project*, R. I Bowman, Ed. (Univ. of California Press, Berkeley, 1966).
19. G. J. Bakus, *Atoll Res. Bull. 179* (1975), p. 1.
20. P. W. Glynn, *Environ. Conserv.* **10**, 149 (1983); *ibid.* **11**, 133 (1984).
21. Sixteen sites in the Gulf of Chiriqui were searched for *Millepora* spp. from 1984 to 1990. Two coral reef sites were surveyed intensively during 10 (Secas reef, Secas Islands) and 14 (Uva reef, Contreras Islands) different research cruises. These were sampled by (i) 30 permanent chain transects, each 10 m long [J. W. Porter, *Ecology* **53**, 745 (1972)], with all corals counted in 300 transects, for a total search time of 44 diver hours; (ii) 12 permanent 1-m^2 and 20-m^2 quadrats with all corals mapped in a total of 620 m^2, 58 hours search time; and (iii) swimming surveys of forereef and reef base zones along each reef site, 40 hours. Forty hours of surveys at 14 other sites yields a total search effort for 1984 to 1990 of 204 hours.
22. R. H. Richmond, in *Global Ecological Consequences of the 1982–83 El Niño-Southern Oscillation*, P. W. Glynn, Ed. (Elsevier, Amsterdam, 1990).
23. P. W. Glynn, J. Cortes, H. M. Guzman, R. H. Richmond, *Proc. Sixth Intl. Coral Reef Symp.* **3**, 237 (1988).
24. B. E. Brown and Suharsono, *Coral Reefs* **8**, 163 (1990).
25. P. W. Glynn and L. D'Croz, *ibid.*, p. 181.
26. P. L. Jokiel, *Endeavour* **14**, 66 (1990).
27. J. B. Lewis, *Coral Reefs* **8**, 99 (1989).
28. G. C. Ray, in *Biodiversity*, E. O. Wilson, Ed. (National Academy Press, Washington, DC, 1988).
29. J. T. Carlton, G. J. Vermeij, D. R. Lindberg, D. A. Carlton, E. C. Dudley, *Biol. Bull.* **180**, 72 (1991).
30. P. W. Glynn, in *Global Ecological Consequences of the 1982–83 El Niño–Southern Oscillation*, P. W. Glynn, Ed. (Elsevier, Amsterdam, 1990).
31. H. von Prahl and A. Mejia, *Rev. Biol. Trop.* **33**, 39 (1985); H. von Prahl, personal communication.
32. H. M. Guzman, J. Cortes, P. W. Glynn, R. H. Richmond, *Mar. Ecol. Prog. Ser.* **60**, 299 (1990).
33. G. Robinson, in *El Niño in the Galapagos Islands: The 1982–83 Event*, G. Robinson and E. M. del Pino, Eds. (Fundacion Charles Darwin para Las Islas Galapagos, Quito, Ecuador, 1985).
34. R. H. MacArthur, *Geographical Ecology* (Harper & Row, New York, 1972); E. G. Leigh Jr., *J. Theor. Biol.* **90**, 213 (1981); J. M. Diamond, in *Extinctions*, M. H. Nitecki, Ed. (Univ of Chicago Press, Chicago, 1984).
35. M. W. Colgan, in *Global Ecological Consequences of the 1982–83 El Niño–Southern Oscillation*, P. W. Glynn, Ed. (Elsevier, Amsterdam, 1990)
36. C. Emiliani, E. B. Kraus, E. M. Shoemaker, *Earth Planet. Sci. Lett.* **55**, 317 (1981), E. G. Kauffman and C. C. Johnson, *Palaios* **3**, 194 (1988).
37. S. M. Stanley, in *Extinctions*, M. H. Nitecki, Ed. (Univ. of Chicago Press, Chicago, 1984); J. A. Fagerstrom, *The Evolution of Reef Communities* (Wiley, New York, 1987).
38. Facilities and logistical support were provided by the Smithsonian Tropical Research Institute and the Centro de Ciencias del Mar y Limnologia, University of Panama. This paper benefited from discussions with D. P. de Sylva, C. M Eakin, S. Snedaker, and H. R. Wanless The authors acknowledge the support of NSF, grants OCE-8415615 and OCE-8716726 (P W G.), and the Research Opportunity Fund, Smithsonian Institution (W.H.d.W.). Permission to work in Panamanian waters was granted by the Direccion de Recursos Marinos, Ministerio de Comercio e Industrias, Republic of Panama. Contribution from the University of Miami, Rosenstiel School of Marine and Atmospheric Science.

6 March 1991; accepted 21 May 1991

Identification of the Envelope V3 Loop as the Primary Determinant of Cell Tropism in HIV-1

STEPHEN S. HWANG, TERENCE J. BOYLE, H. KIM LYERLY, BRYAN R. CULLEN*

Cells of the monocyte-macrophage lineage are targets for human immunodeficiency virus–1 (HIV-1) infection in vivo. However, many laboratory strains of HIV-1 that efficiently infect transformed T cell lines replicate poorly in macrophages. A 20–amino acid sequence from the macrophage-tropic BaL isolate of HIV-1 was sufficient to confer macrophage tropism on HTLV-IIIB, a T cell line–tropic isolate. This small sequence element is in the V3 loop, the envelope domain that is the principal neutralizing determinant of HIV-1. Thus, the V3 loop not only serves as a target of the host immune response but is also pivotal in determining HIV-1 tissue tropism.

ALTHOUGH THE $CD4^+$ LYMPHOcyte is the major target for HIV-1 replication in the peripheral blood compartment, cells of the monocyte-macrophage lineage represent the predominant HIV-1–infected cell type in most tissues, including the central nervous system (*1–3*). An HIV-1 infection of macrophages, although less cytopathic than an infection of T cells, compromises macrophage function and may underlie many of the pathogenic effects of HIV-1 infection in humans (*1–3*).

S. S. Hwang, Department of Microbiology and Immunology, Duke University Medical Center, Durham, NC 27710.
T. J. Boyle and H. K. Lyerly, Department of Surgery, Duke University Medical Center, Durham, NC 27710.
B. R. Cullen, Howard Hughes Medical Institute and Department of Microbiology and Immunology, Duke University Medical Center, Durham, NC 27710.

*To whom correspondence should be addressed.

Despite the importance of macrophages as a primary target for HIV-1 infection in vivo, many laboratory isolates of HIV-1 are unable to replicate in these cells (*3–8*).

The HIV-1 isolates can be divided into two major subgroups on the basis of their cellular host range in vitro (*2–9*). Macrophage–T cell (MT)–tropic isolates efficiently infect both macrophages and $CD4^+$ peripheral blood lymphocytes (PBLs) but are unable to replicate efficiently in many transformed cell lines of either T cell or monocytic origin. A second class of viruses, termed T cell (T)–tropic, replicates efficiently in both PBLs and transformed T cell lines but poorly in macrophages (*2–9*). The tropism of HIV-1 is determined early in the viral replication cycle, between binding of the virus to the cell surface and initiation of viral reverse transcription (*6, 7*) and is independent of the viral long terminal repeat (*8*) but dependent on sequences in the viral gp120 envelope protein (*5, 6*).

The HTLV-IIIB isolate of HIV-1 is T-tropic, whereas the BaL strain is MT-tropic (*3, 8*). To determine which sequences were important for in vitro tropism, we cloned and sequenced the envelope gene and flanking viral sequences of BaL with DNA derived from BaL-infected macrophages (*10*). We then constructed a series of chimeric HIV-1 proviruses by substitution of BaL-derived sequences into an HTLV-IIIB proviral clone (Fig. 1). These chimeric viruses were then tested for tropism by analysis of their replication competence in PBLs (*11*), primary monocyte-derived macrophages (*12*), and the transformed $CD4^+$ T cell lines H9 and CEM (Table 1).

Both the parental HTLV-IIIB clone (pIIIB) and the provirus with the complete BaL *env* gene (pBaL) replicated equivalently in primary PBLs, as we determined by measuring secreted p24 Gag protein (Table 1) or supernatant reverse transcriptase activity (*13*). As predicted (*3, 8*), the pIIIB provirus also replicated efficiently in both the H9 and CEM cell lines but not in macrophages. In contrast, the pBaL provirus replicated efficiently in macrophages but not in H9 or CEM cells (Table 1). The various chimeric proviral constructs, like the parental pIIIB and pBaL clones, all displayed comparable replication competence in PBLs.

In addition, all chimeric clones were either fully T-tropic or fully MT-tropic; that is, no intermediate or dual tropism was detected. As previously reported (*5, 6*), tropism was determined entirely by sequences located within gp120. A 20–amino acid