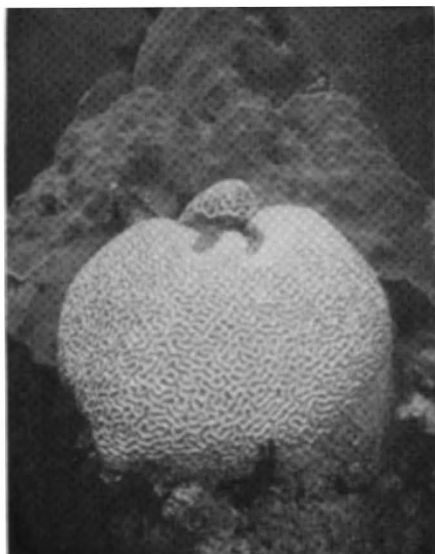


## Coral reef bleaching alert

SIR—Goreau, in a recent Scientific Correspondence<sup>1</sup>, drew attention to the coral reef bleaching of photosymbiotic hosts in the Caribbean in 1987–88. That bleaching event was world-wide, not just in the Caribbean, as we reported in a paper<sup>2</sup>



A totally bleached brain coral, *Colpophyllia natans*, among unbleached mushroom-shaped star coral, *Montastrea annularis*.

published after Goreau's letter. Bleaching of organisms in coral reefs caused bleaching complexes to be formed throughout the world in 1979–80, 1982–83 and 1986–88. Each complex was made up of two or three bleaching events, with a preceding event occurring in 1979, 1982 and 1986, and major bleaching events occurring in 1980, 1983 and 1987 (ref. 2). Preceding events occur in the warm-water season toward the beginning of a short-term warming event, and major bleaching events in the normal warmwater season at the height of a short-term warming event. We suggested that preceding events could be used to predict major events.

As a result of Goreau's letter, we distributed our third bleaching summary and have so far received 40 reports of bleaching in mid-to-late 1989 from many areas of the Caribbean. This bleaching could represent a preceding event and we believe that a world-wide, major event may occur in 1990. A new El Niño Southern Oscillation (ENSO) may be beginning<sup>3</sup>. ENSOs have coincided with the past two (and most extensive) world-wide bleaching events, if this association continues, a major world-wide bleaching event could accompany the new ENSO. The timing between past major events also suggests the next may occur this year.

We believe that the world-wide coral reef bleaching cycle is caused by increased seawater temperatures produced by the combination of increased global temperatures of the 1980s, temporary warm-

ing events such as ENSOs, and seasonal warming. The progressive deterioration of inshore regions, including coral reefs, may have contributed to the intensity of the events by reducing the resilience or resistance of photosymbiotic hosts to the bleaching process.

Major marine ecological disturbances, such as bleaching, usually appear without warning and are over before adequate investigation can be performed. We hope this prediction will allow the scientific community to prepare for the event. Study of the occurrences, causes and interrelationships among events is an important challenge<sup>4</sup>. Predicting a world-wide marine ecological disturbance is, as far as we know, an entirely new concept.

We support Goreau's efforts to estab-

lish a Caribbean-wide research effort on bleaching through the Association of Marine Laboratories of the Caribbean. Members of the Caribbean Aquatic Animal Health Project (telephone 809 899 2048, ext. 211) are attempting to follow coral bleaching and other ecological disturbances, and would appreciate receiving information concerning these events.

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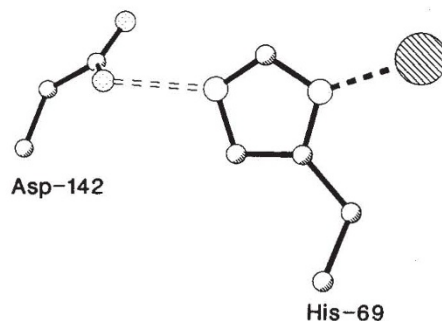
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- Goreau, T. J. *Nature* **343**, 417 (1990).
- Williams, E. H. Jr & Bunkley-Williams, L. *Atoll Res. Bull.* **335**, 1–71 (1990).
- Monastersky, R. *Sci. News* **137**, 135 (1990).
- Williams, E. H. Jr & Bunkley-Williams, L. *J. Aqu. Anim. Hlth* (in the press).

## Another catalytic triad?

SIR—David Blow has recently commented in News and Views<sup>1</sup> on the convergent importance of the catalytic triad as observed in recently determined lipase structures. Blow, with Janet Thornton, searched the Brookhaven Protein Data Bank<sup>2</sup> for Asp<sup>-</sup> -- His --- Ser triads that could be related to those of the serine proteases<sup>1</sup>. They identified two candidates (taka-amylase and immunoglobulin Kol), but each of these suffers from poor hydrogen-bond stereochemistry.

Our recent analysis of the Brookhaven



database<sup>3</sup> complements that of Blow and Thornton. We reported that Asp<sup>-</sup> -- His -- Ser triads with good geometry for hydrogen bonding are present in only serine proteases. We noted that the Asp(Glu)<sup>-</sup> -- His hydrogen bond occurs, on average, about one per unique structure in the databank, even though some of these couples display poor geometry for hydrogen bonding. We also reported that the implicated histidine can also hydrogen-bond to a backbone carbonyl oxygen or a water molecule.

The Asp(Glu)<sup>-</sup> -- His couple is a component of another triad found in the zinc enzyme family. These are base-histidine couples as zinc ligands in carboxypeptidase A, thermolysin and carbonic anhydrase as noted by Liljas and Rossmann<sup>4</sup>. We find

that the triad Asp(Glu)<sup>-</sup> -- His --> Zn<sup>2+</sup> is present in the three-dimensional structure of at least 36 zinc enzymes and their homologues<sup>5</sup>. Indirect carboxylate-zinc interactions (across bridging histidines) may contribute to biological metal-ion recognition and affinity, and such interactions could be important factors in metalloprotein folding.

We speculated<sup>3</sup> that the Asp<sup>-</sup>--His couple of the serine protease active site may be an evolutionary/structural motif related to that of the zinc protease active site. The Asp 142/His 69 couple, for example, ligands the zinc ion of carboxypeptidase A (ref. 5, see figure), and the Asp 170/His 142 couple ligands the zinc ion of thermolysin<sup>6</sup>. These zinc-binding motifs are intriguing in view of the fact that the Asp<sup>-</sup>--His couple of the serine protease active site selectively binds transition metals, for example, as contained in platinum complexes<sup>7</sup> and even the Zn<sup>2+</sup> ion itself<sup>8</sup>. This versatile couple is certain to turn up in additional biological circumstances.

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- Blow, D. *Nature* **343**, 694–695 (1990).
- Bernstein, F. C., Koetzle, T. F., Williams, G. J. B., Meyer, E. F., Jr., Brice, M. D., Rodgers, J. R., Kennard, O., Shimanouchi, T. & Tasumi, M. *J. Mol. Biol.* **112**, 535–542 (1977).
- Christianson, D. W. & Alexander, R. S. *J. Am. Chem. Soc.* **111**, 6412–6419 (1989).
- Liljas, A. & Rossmann, M. G. A. *Rev. Biochem.* **3**, 475–507 (1974).
- Rees, D. C., Lewis, M. & Lipscomb, W. N. *J. molec. Biol.* **168**, 367–387 (1983).
- Holmes, M. A. & Matthews, B. W. *J. molec. Biol.* **160**, 623–639 (1982).
- Brothers, H. M. & Kostic, N. M. *Biochemistry* **28**, 1944 (1989).
- Fujinaga, M. & James, M. N. G. *J. molec. Biol.* **195**, 373–396 (1987).