Fish larval behavior, as glimpsed through the handful of studies completed to date, appears diverse and fascinating (reference 3 of Bellwood *et al.*). What Bellwood *et al.* do not mention is that their own research has revealed such swimming and orientation abilities only in late-stage larvae. For a large proportion of the dispersal phase, fish larvae may have much less control over transport. Undoubtedly, there will be some species whose behavior will lead to high levels of local retention. How else can we account for species with very restricted ranges in regions where reefs are highly interconnected by currents (*1*)? But this is only one end of a spectrum of behavior, and there will be many other species whose dispersal will be more passive. Such behaviors will result in a spectrum of transport distances ranging from short to long.

Furthermore, while both letters emphasize fish behavior, the interests of managers extend to all of the other taxa that compose reef ecosystems. As Bellwood *et al.* point out, many invertebrate larvae seem to have little capacity to modify their transport by currents. The transport envelopes I describe cannot capture the detail of individual species' behaviors but, then, managers require broad guidance rather than reductionist complexity.

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## Rapid-Wasting Disease: Pathogen or Predator?

Researchers have raised concern about the sudden emergence of coral diseases and the contagious nature of many of these diseases (*1*). A new "disease" was recently identified on star coral (*Montastraea annularis* species complex) and brain coral (*Colpophyllia natans*) from reefs of Bonaire, Netherlands Antilles. This affliction has been named "rapid-wasting disease" (RWD) because of the rate of tissue destruction (advancing 7.5 centimeters in 24 hours), associated with the removal of the top layers of calcium carbonate (Random Samples, 27 June 1997, p. 1979). While the extent of tissue loss ob-

## Framework for Federal Science Policy

**REQUEST FOR COMMENTS**

The AAAS Board of Directors invites you to help develop a framework for our nation's science policy. The Board is preparing a paper on goals and directions for American science as input to the National Science Policy Study being conducted by the Science Committee of the U.S. House of Representatives. Members can read and comment on a draft of the Board's paper on the AAAS website at http://www.aaas.org/spp/fedsci. Feedback will be most helpful if received by April 8, 1998.

served may be reason for concern, we do not believe that this affliction is the result of a disease, but rather is caused by parrotfish bites.

In June 1997, we surveyed reefs of Bonaire and Curaçao to determine the incidence of disease among massive reef-building corals. In Curaçao, star corals (*Montastraea* spp.) exhibiting the described signs of "RWD" were present (less than 0.5%) in shallow water (less than 10 meters). In Bonaire (at depths of from 3 to 20 meters), these signs were present on a higher proportion of corals (0.5 to 5%); damage was concentrated on (but not confined to) reefs along the northwestern half of the island.

Several factors lead us to conclude that these signs are caused by something other than a disease. "Rapid-wasting disease" has been characterized by removal of tissue and the top layers of the skeleton; coral diseases typically damage coral tissue alone. Elevated portions of the colonies are affected; live, apparently healthy tissue remains within the depressions between lobes. Coral tissue at the margin of the tissue-denuded, abraded skeleton appears fully pigmented and does not exhibit signs of disease or necrosis. Calcium carbonate skeletal material may be found on or near the coral with "RWD." The greatest number of damaged colonies occurs in areas with a high abundance of large, territorial stoplight parrotfish (*Sparisoma viride*).

We monitored colonies of M. *annularis* in Bonaire with recent tissue and skeletal destruction for up to 70 minutes each (at depths of from 3.5 to 15 meters). Approximately 70% of the colonies with fresh lesions identified during our dives ($n$ = 14) were attacked by S. *viride* at least once, and usually repeatedly. During these periods, one to three terminal–phase male and one or more initial–phase adult S. *viride* were observed removing live coral tissue and skeleton from a single coral head, returning at 2- to 15-minute intervals. In two cases, fish removed the majority of the tissue from one lobe, then began grazing on an adjacent, previously undamaged lobe. S. *viride* were also observed biting C. *natans*; fish removed tissue and skeletal material in a band, moving methodically across the coral.

Stoplight parrotfish use an excavating feeding mode that leaves deep grazing scars (2). In a previous study from Bonaire (3), terminal-phase males took 9.3% of their bites from live coral (primarily M. *annularis*), creating conspicuous white spots on the coral. Damage by S. *viride* is the most serious cause of chronic coral tis-

sue loss to M. *annularis* in the U.S. Virgin Islands (*4*). In our study, a maximum of 17 bites were taken from the same coral in a 70-minute period. If territorial fish repeat this behavior throughout the day, it would result in substantial damage (*5*), which might be correlated with high rates of tissue destruction characteristic of "RWD."

*Sparisoma viride* have been known to create large lesions on M. *annularis* for nearly 20 years (*6*). The abundance of macroalgae has increased on many Caribbean reefs since the loss of *Diadema antillarum* in the mid 1980s. A greater biomass of algae may support a larger population of herbivorous fish such as S. *viride*, resulting in a higher density of territories, more conspecific interactions, and consequently more territorial markings on live coral. Parrotfish white spot biting may be more noticeable on coral reefs in Bonaire, which have had protected fish populations for almost two decades. We have made similar observations on coral reefs of La Parguera, Puerto Rico.

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**Letters to the Editor**

Letters may be submitted by e-mail (at science_letters@aaas.org), fax (202-789-4669), or regular mail (*Science*, 1200 New York Avenue, NW, Washington, DC 20005, USA). Letters are not routinely acknowledged. Full addresses, signatures, and daytime phone numbers should be included. Letters should be brief (300 words or less) and may be edited for reasons of clarity or space. They may appear in print and/or on the World Wide Web. Letter writers are not consulted before publication.