LETTERS

Causes of Coral Reef Degradation

IN THEIR REPORT "GLOBAL TRAJECTORIES OF the long-term decline of coral reef ecosystems" (15 Aug., p. 955), J. M. Pandolfi *et al.* advocate a novel interpretation of the timing and causes of the worldwide decline of reef-building corals. Expanding on an earlier paper (*1*), they conclude that corals have been in decline for centuries and that overfishing was the leading cause. They consider the recent impacts of coral disease and bleaching to be ancillary effects and argue that reef ecosystems will not survive without immediate, draconian protection from fishing. Curiously, a Review by T. P. Hughes *et al.* in the same issue ("Climate change, human impacts, and the resilience of coral reefs," 15 Aug., p. 929) concludes that climate change and disease are the primary agents of increased coral mortality and that degraded reefs will survive, albeit with altered species composition. Pandolfi, Hughes, and J. B. C. Jackson are all authors on the two conflicting papers, rendering their message difficult to discern.

Pandolfi *et al.*'s evidence for the early decline of corals resides in a list of more than 400 references in an online supplement. That supplement would have been more useful had it described the method by which coral condition was ranked. Information on variances and sample sizes of the historical data should also have been included.

The proximal causes of the decline in coral cover on Caribbean reefs were recent disease outbreaks and hurricanes, whereas in the Pacific, coral mortality increased markedly because of recent, El Niño–Southern Oscillation–induced bleaching episodes [Hughes *et al.*; (*2*)]. Paleontological studies in Belize (*3*), the equatorial eastern Pacific (*4*), and Indonesia (*5*) suggest that corals grew actively and continuously for millennia until recent decades, although there is also evidence of localized reef degradation from terrigenous input dating to the time of European colonization (*6*).

None of this falsifies the hypothesis that the recent collapse of reef systems was driven by fishing pressure more than a century ago, but the burden of proof rests with Pandolfi *et al*. The hypothesis that overfishing caused corals to decline is argued by default, and no cogent mechanistic explanation is offered. Alternative explanations need to be falsified, including the release of nutrients and sediment as rising sea level flooded coastal areas centuries to millennia ago. If the authors are implying that overfishing removed herbivores earlier than suspected, releasing algae to overgrow corals at that time, they must reconcile their scenario with the observed transition from coral to algal dominance in the Caribbean beginning in the 1980s (*1*).

Protecting vertebrates from overexploitation is clearly a laudable goal. It is imperative, however, that policy-makers directly address the range of factors that are decimating coral populations and hindering their recovery. We are certain that Pandolfi *et al.* agree, and we encourage them to develop their argument more comprehensively.

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Letters to the Editor

Letters (~300 words) discuss material published in *Science* in the previous 6 months or issues of general interest. They can be submitted by e-mail (science_letters@aaas.org), the Web (www.letter2science.org), or regular mail (1200 New York Ave., NW, Washington, DC 20005, USA). Letters are not acknowledged upon receipt, nor are authors generally consulted before publication. Whether published in full or in part, letters are subject to editing for clarity and space.

Response

IN OUR REPORT, WE SHOWED THAT CORAL reef ecosystems were degraded long before more recent changes attributable to climate change or disease. Aronson *et al.* appear to discount historical data, preferring instead to assume that reefs were pristine until recently. Their comments focus on corals, which we show to have changed more recently than six other ecological guilds (categories of biota). Yet even for corals, there is clear evidence that recent changes represent an ongoing degradation that long predates modern ecological studies. While Aronson *et al.* attribute loss of coral cover in the Caribbean partially to hurricanes, it is the recent lack of recovery of contemporary Caribbean reefs following hurricanes that is the issue. We argued that when herbivores are lost from the system, disturbances from which corals are usually able to recover become more problematic, because faster-growing algae are able to exclude them. A previous contribution highlighted significant Caribbean coral decline well before the beginning of the 1980s [Fig. 2B in (*1*)], so decline is not simply a matter of the recent change in dominance from corals to

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algae. Although we agree that bleaching and disease are becoming more prevalent, the ability of reefs to absorb these impacts will clearly depend on the extent to which they are already degraded [our Report; (*1*)].

Aronson *et al.* identify coral bleaching and disease as "proximal" causes of coral decline, based on studies addressing coral cover during the past few decades. We show that these so-called proximal causes are not the ones that have acted over long time periods or that have caused the most intense degradation of reefs and associated ecosystems. Indeed, Aronson *et al.*'s assertions represent exactly the kind of conclusion that can arise without historical perspective.

Our study, which represents a significant departure from more traditional studies in experimental ecology (which use continuous, unidimensional data sets that are neatly described by a mean and variance) demonstrates that the ultimate causes of coral reef ecosystem decline are more subtle than recent proximal ones and reach further back in time than events observed in the past few decades. We agree with Aronson *et al.* that policy-makers need to consider all relevant factors in addressing coral reef degradation. However, history can be neither ignored nor changed, and shooting the messenger will solve nothing.

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Response

IN OUR REVIEW, WE SUMMARIZED CURRENT knowledge of the degraded status of coral reefs and of the human threats to reefs. We also identified new directions for research to inform the management of these vital natural resources. Aronson *et al.* misinterpret our Review by claiming that our conclusions (on the importance of overfishing on coral reefs) conflict with those of the accompanying Report by Pandolfi *et al*. In this Report, Pandolfi *et al.* emphasize the long trajectory of reef decline, while we focused more on contemporary threats and future solutions. Until recently, the direct and indirect effects of overfishing and pollution from agriculture and land development have been the major drivers of massive and accelerating decreases in abundance of coral reef species. We argued that human impacts and the increased fragmentation of coral reef habitat have undermined reef resilience, making them much more susceptible to current and future climate change. In particular, we presented clear, unambiguous evidence that overharvesting of herbivorous fishes can impair the resilience of coral reefs and inhibit their recovery from bleaching and other disturbances, leading to a phase shift to algal-dominated reefs. Consequently, our Review contained an entire section on the benefits of No-Take Areas (NTAs; where fishing is prohibited) and the need for effective management of fish stocks and of the ecosystem functions that fishes provide both inside and outside NTAs. We called for the establishment of 25% of reefs as NTAs. We do not consider our findings to be in conflict with those of Pandolfi *et al.* Rather, we placed the role of overfishing in the context of the range of current environmental pressures faced by reefs, and we called for a pluralistic approach to reef conservation that includes protection of fish stocks as a major component.

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Bio2010 Misinterpreted?

THE NATIONAL RESEARCH COUNCIL'S (NRC) study *Bio2010: Transforming Undergraduate Education for Future Research Biologists* (*1*) presents recommendations for educating undergraduate students planning careers in molecular biology, cell biology, and other fields central to biomedical research. This emphasis is the result of the charge given by the sponsors (NIH and Howard Hughes Medical Institute) to the authoring committee and is made abundantly clear in the text, even if it is not apparent in the title of the report.

I have been informed that some college and university educators and administrators have used this report to justify the diminution—or even the elimination—of ecology, population and evolutionary biology, and other nonbiomedical subject areas in undergraduate life sciences programs and curricula. This is certainly not an appropriate response to the committee's conclusions.

Consider the following quotation from the report's Preface: "Changes [to the life sciences curriculum] cannot be made solely to benefit future biomedical researchers. The impact on undergraduates studying other types of biology, as well as other sciences, cannot be ignored as reforms are considered" (pp. ix–x). Moreover, even though the report suggests course options for students interested in only one type of biology, these options explicitly incorporate coursework and electives from throughout the life sciences (e.g., evolutionary biology and ecology), as well as from other sciences.

Students are often exploring a variety of career options as they advance through a life sciences curriculum, and the report stresses the importance of exposing them to the breadth of biology:

> "The Committee… has concluded that the best preparation for the biomedical research of the future is a broadly based education in biology with a strong foundation in the physical sciences and mathematics. A well-educated biology major should understand the principles of population and evolutionary biology, ecology, cognitive neurobiology, and plant biology, irrespective of his or her future research area. The connections between biomedical research and other sciences will become more intimate and mutually reinforcing in the years ahead. Most compelling, the fundamental unity of biology speaks strongly against the desirability of compartmentalization too early in one's education" (p. 24).

Bio2010 also recommends that new ways be found to help life sciences faculty learn more about the emerging evidence and practices that can help improve undergraduate teaching and student learning, including an annual Summer Institute for biology faculty. A successful experiment focused on this last recommendation is described in the accompanying Policy Forum by Wood and Gentile (*2*), co-chairs of the organizing committee for the Institute.

"[S]ome college and university educators and administrators have used this report [*Bio2010*] to justify the diminution—or even the elimination—of ecology, population and evolutionary biology, and other nonbiomedical subject areas in undergraduate life sciences programs and curricula. This is certainly not an appropriate response to the committee's conclusions.

–ALBERTS

I urge those who might use *Bio2010* in restructuring undergraduate study in the life sciences to read this important report carefully.

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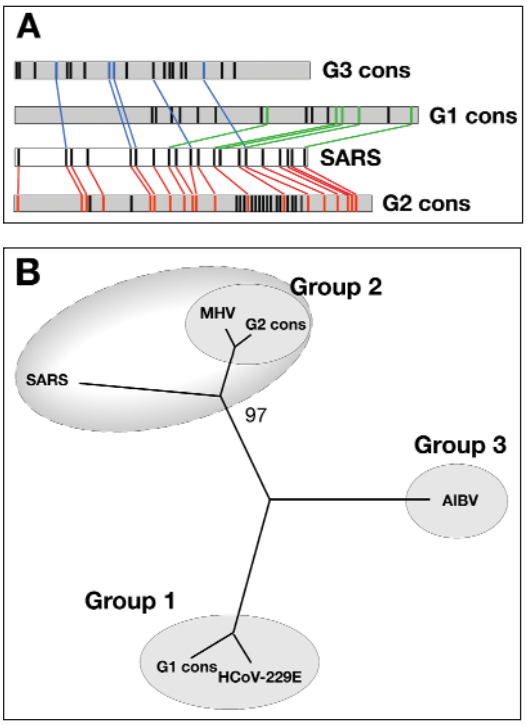
Phylogeny of the SARS Coronavirus

SEVERAL PAPERS HAVE BEEN PUBLISHED ON THE genomic sequence and phylogeny of the severe acute respiratory syndrome (SARS) coronavirus (*1–4*). We have determined the



(A) Schematic representation of cysteine positions in the NH_2-terminal domain of the SARS-CoV spike protein in comparison with the corresponding consensus sequences from group 1, 2, and 3 coronaviruses (G1 cons, G2 cons, and G3 cons). Colored lines connect conserved cysteines. (B) Neighbor-joining network inferred from the NH_2-terminal domain of the spike proteins. The value at node indicates number of 100 bootstrap trees presenting clusters distal of node. The same result was obtained when we performed a maximum parsimony analysis. The unrooted tree shows that the SARS-CoV virus clusters with group 2 coronaviruses. Similar trees were obtained using Nsp1, matrix, and nucleocapsid proteins. Branch lengths are proportionate to amino acid changes. MHV, murine hepatitis virus; AIBV, avian infectious bronchitis virus; HcoV-229E, human coronavirus 229E.

nucleotide sequence of the SARS coronavirus FRA isolate (accession number AY310120) and found the overall genome organization and predicted proteins to be in agreement with published studies. We also performed a phylogenetic analysis. Using conserved regions of the virus such as the polymerase gene and standard algorithms, we found that the new virus is not related to the known type 1, 2, and 3 coronaviruses and represents a novel, previously unknown type of coronavirus, in agreement with published studies.

However, when we examined the NH_2-terminal domain of the SARS spike protein, which in other coronaviruses is known to harbor the receptor-binding domain and to be responsible for the tissue tropism of the virus, we made the striking observation that 19 out of the 20 cysteines are spatially conserved with those of the group 2 consensus sequence. In marked contrast, only five of them are conserved in group 1 and group 3 sequences (see panel A of figure). Encouraged by this observation, we performed a phylogenetic analysis using the neighbor-joining algorithm on the amino acid sequences of the poorly conserved proteins such as spike, Nsp1, matrix, and nucleocapsid. In all cases, we found that SARS-CoV clustered more frequently with group 2 coronavirus (see panel B of figure), showing that a statistically significant relationship between SARS and group 2 coronavirus is found in different regions of the genome. Our findings suggest that group 2 coronaviruses and SARS are closely related and are likely to share a common ancestor. This observation makes unlikely an avian origin of the virus because avian coronaviruses are mostly type 3, and is consistent with the finding that a virus similar to SARS is found in mammalian species used as food in China (*5*). After submission of this Letter, Snijder *et al.* published a phylogenetic analysis based on the polymerase gene in which they reported that SARS-CoV derives from an early split of type 2 coronavirus (*6*). Our observation allows a rational, hypothesis-driven approach to study the origin of the virus and the animal species involved in transmission and to design measures to prevent and contain the infection.

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