Financing Nuclear Liability

IN LIGHT OF THE 2011 FUKUSHIMA DISASTER, recent discussion has focused on finding the best nuclear storage options (1) and maximizing the oversight power of global institutions (2). However, even with the best risk-informed planning and guidelines, accidents at nuclear power plants (NPPs) could still occur (3). The 1990 report from a U.S. presidential commission estimates that the catastrophic nuclear accident probability in the United States (about 100 nuclear reactors) in the remaining lifetime of 40 years per plant is one accident in 250 thousand years (4). There are currently 438 NPP units worldwide (predicted to increase to 500) (5); extrapolating the U.S. figure with some uncertainty considerations to obtain the worldwide average time to an accident yields an estimate of one accident in 5 to 50 thousand years for remaining lifetimes. Given the possibility of another accident, in addition to strengthening safety measures, we should develop dependable liability coverage that can be tapped in an emergency.

In 1957, the United States enacted the Price-Anderson nuclear liability regime for managing the risk of nuclear accidents. The legislation aimed to establish a mechanism for compensating the public for losses and to encourage the private development of nuclear power. With 104 operating reactors, the United States has a total of $11.975 billion in coverage (as of 2011) (6). The U.S. Nuclear Regulatory Commission (US NRC) has determined that the Price-Anderson Act is inadequate in light of the increased amount of coverage required in the wake of the Fukushima disaster. The US NRC has noted that the nuclear insurance market is unable to provide the required financial protection and that “the United States, if an accident occurs, needs to be able to pay both the physical and social costs of an accident” (6).

The US NRC has started the process of establishing the possibility of another accident, in addition to strengthening safety measures, we should develop dependable liability coverage that can be tapped in an emergency.

IN THEIR REVIEW “PROJECTING CORAL REEF FUTURES UNDER GLOBAL WARMING AND OCEAN ACIDIFICATION” (22 July, p. 418), J. M. Pandolfi et al. argue that the threat of climate change for corals has been exaggerated by pointing to variability in coral heat tolerance, adaptation potential, and the recent fossil record. In doing so, they fail to consider the full range of model assumptions and the precision of the fossil record.

The Future of Coral Reefs

IN THEIR REVIEW “PROJECTING CORAL REEF FUTURES UNDER GLOBAL WARMING AND OCEAN ACIDIFICATION” (22 July, p. 418), J. M. Pandolfi et al. argue that the threat of climate change for corals has been exaggerated by pointing to variability in coral heat tolerance, adaptation potential, and the recent fossil record. In doing so, they fail to consider the full range of model assumptions and the precision of the fossil record.

Under the section “Projecting coral reef futures,” Pandolfi et al. predict that adaptation of the dinoflagellate symbionts that provide the host with energy is likely to help coral reefs maintain their structure in the near future. The argument is based on an untested model of symbiont population dynam-
This model incorporates tradeoffs between symbionts’ thermal tolerance and symbiont population growth, but it ignores tradeoffs that involve essential properties of coral reefs (such as documented reductions in calcification of corals harboring thermally tolerant symbionts). For symbiont adaptation to ensure the future of coral reefs, they would have to simultaneously evolve the following characteristics: (i) thermal tolerance to the synergistic effects of increased atmospheric pCO₂ on sea water; (ii) capability to maintain metabolic exchange in energy and nutrients between themselves and their coral hosts; and (iii) compatibility across a broad range of available coral hosts and environments. Each of these adaptations is unlikely on its own, and there is little evidence that any have occurred to date (2), making the probability of the simultaneous evolution of all these traits in the near future highly improbable. Even if these adaptations were to evolve simultaneously, coral reef structure would only be sustained if the intrinsic capability of corals to maintain high rates of calcification, above the rates of erosion, were preserved.

Despite acknowledging the absence of analogous periods that match the current rate of change in ocean acidity and temperature, Pandolfi et al. drew on the fossil record for insights into past coral reef response to climate change. However, the precision of these records over ecologically relevant time scales (decadal to centennial) is inherently problematic because of the low resolution of radiocarbon dating and the uncertainty associated with the order in which reefs developed and the dating results (3, 4). In the example presented by Pandolfi et al., proxy records of temperature from sediment cores were sampled on average every 133 ± 7 years, whereas radiocarbon ages were sampled every 1321 ± 329 years; accurate comparison of rates of change that differ over decadal to centennial scales is consequently highly problematic. Conversely, the proxy temperature records from the same core record (5) indicate remarkably stable conditions during the past 10,000 years (25.9° to 27.7°C). Consequently, most paleoecological perspectives are limited in their usefulness for interpreting the rapid trends and impacts that are occurring today.

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References

Response
HOEGH-GULDBERG ET AL. ASSERT THAT EVOLUTIONARY RESPONSES TO CLIMATE CHANGE IN CORALS ARE HIGHLY IMPOSSIBLE IN THE NEAR FUTURE—CITING TRADEOFFS BETWEEN TOLERANCE TO WARMING AND ACIDIFICATION, AND TWO FACTORS RELATED TO THE INTEGRITY OF THE CORAL-SYMBIONT PARTNERSHIP. IN SUPPORT, THEY CITE AN EARLIER PAPER MAKING SIMILAR CLAIMS (1), BUT THIS REFERENCE PRESENTS NO empirical OR THEORETICAL EVIDENCE FOR THIS THESIS.

In fact, the hypothesis that adaptation cannot occur over decadal time scales has been shown repeatedly to be incorrect: Numerous and complex physiological, metabolic, and morphological changes can occur rapidly and repeatedly among independently evolving lineages (2–5). Specifically, tradeoffs are ubiquitous in nature, and, although they influence the rate and direction of evolution (as emphasized in our Review), they do not prevent any evolution from happening. Moreover, contrary to Hoegh-Guldberg et al.’s assertions that characteristics of endosymbiosis will impede adaptation in corals, studies of other organisms have found that endosymbions and hosts, if anything, evolve more rapidly than their free-living counterparts (6, 7).

In our projections section, we discuss Baskett et al. (8) because it is the only study that attempts to rigorously determine the potential effects of thermal adaptation on coral cover. Moreover, Baskett et al. (8) explicitly include feedbacks of symbiont thermal tolerance on coral colony growth. More broadly, the fact that the model in (8) has important limitations does not justify eschewing attempts to model evolutionary dynamics entirely.

Our Review presented fossil evidence for varying sensitivity of coral reefs to climate change over multiple temporal scales. Hoegh-Guldberg et al. argue that the geological record lacks resolution to detect such changes, such as during the well-known and accepted periods of Abrupt Climate Change (ACC) during the early to mid-Holocene (9). However, the decadal- to centennial-scale chronology and paleoclimate from the Cariaco Basin study (10) shows substantial fidelity to high-resolution Greenland ice-core records and adheres to all of the best practices recommended in the relevant paper cited by Hoegh-Guldberg et al. (11). The Cariaco Basin sediments record continuous annual episodes of sedimentation that generally lack the bioturbation and time-averaging typical of reef deposits, so the concerns with using radiometric dating to study the fine-scale chronology of reef accretion cited by Hoegh-Guldberg et al. (12) are irrelevant.

Our Review is emphatic that “coral reefs are indeed threatened by climate change” (Pandolfi et al.). However, we believe that the best way for coral reef scientists to inform policy responses to this threat is to do our best to comprehensively and rigorously account for all the processes likely to deter-
mine reefs’ responses to climate change—physiological, ecological, biogeographical, and evolutionary.

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References

CORRECTIONS AND CLARIFICATIONS

News & Analysis: “8.7 million: A new estimate for all the complex species on Earth” by D. Strain (26 August, p. 1083). The article noted that Nigel Stork of Griffith University in Queensland, Australia, and colleagues published a paper estimating that there are some 3.7 million arthropod species on Earth. This figure was based on a calculation error and was subsequently revised in a corrigendum. Their median estimate, depending on the model, was revised to 6.1 million or 7.8 million species.

Technical Comments: Response to Comments on “Drought-induced reduction in global terrestrial net primary production from 2000 through 2009” by M. Zhao and S. W. Running (26 August, p. 1093; www.sciencemag.org/cgi/content/full/333/6046/1093-e). The sentence “the reduction of NPP in the 2005 drought of the Amazon is 5.56% of the mean NPP from 2000 to 2004” was incorrect. The reduction was 9.28%, not 5.56%.

Research Articles: “Local and long-range reciprocal regulation of cAMP and cGMP in axon/dendrite formation” by M. Shelly et al. (29 January 2010, p. 547). The paper included a misleading description of the method of FRET imaging. The last sentence on p. 547 (beginning eight lines from the end) should read, “Bath application of the membrane-permeant cAMP analog Sp-8-Br-cAMPS (20 µM) or the AC activator forskolin (20 µM) resulted in a global increase of cAMP and PKA signals in ICUE- and AKAR-expressing cells, respectively, as measured by the increase in the ratio of yellow fluorescent protein (YFP) to cyan fluorescent protein (CFP) fluorescence at the neurite (Fig. 2, A, B, D, E, and G) for AKAR, and the ratio of CFP to YFP fluorescence for ICUE.” In addition, there was a mistake in the name of a FRET probe used in Fig. 2B and Fig. 4. The label on the left in Fig. 2B should be “AKAR (PKA),” not “ICUE (cAMP).” In the Fig. 2 legend, the first sentence describing panels B and C should read, “FRET signals observed at the neurite tip of 16-hours neurons expressing AKAR or cGES-DES.” In the Fig. 4 legend, the first sentence describing panel A should begin, “YFP fluorescence and FRET signals for PKA-activity in an AKAR-expressing hippocampal neuron at 16 hours. . . .”

Reports: “Label-free, single-molecule detection with optical microcavities” by A. M. Armani et al. (10 August 2007, p. 783). The authors reported the use of optical microresonators immersed in aqueous solutions and functionalized with antibodies to detect small concentrations of the analytes recognized by the antibodies. The Report presented discontinuities in the resonant response, which the authors took to represent the responses from binding individual analyte molecules. The amplitude of these discontinuities was too large to be caused by the direct effect of the analyte binding; to explain their large size, the authors proposed a thermo-optic effect, in which local heating of the resonator surface from light-analyte interaction amplified the effects of analyte binding. However, as noted by Arnold et al. [Optics Express 18, 281 (2010)], the thermo-optic effect cannot account for the size of the discontinuities. The origin of the large wavelength discontinuities is being investigated by several independent efforts.