# Climate change induces demographic resistance to disease in novel coral assemblages

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**Climate change is reshaping biological communities and has already generated novel ecosystems. The functioning of novel ecosystems could depart markedly from that of existing systems and therefore obscure the impacts of climate change. We illustrate this possibility for coral reefs, which are at the forefront of climatic stress. Disease has been a principal cause of reef degradation and is expected to worsen with increased future thermal stress. However, using a field-tested epizoological model, we show that high population turnover within novel ecosystems enhances coral resistance to epizootics. Thus, disease could become a less important driver of change in the future. We emphasize the need to move away from projections based on historic trends toward predictions that account for novel behavior of ecosystems under climate change.**

Climate change is altering ecosystems (1, 2) and causing unprecedented degradation in sensitive systems such as coral reefs. For millennia, Caribbean coral reefs were built by large, long-lived corals whose life history strategy tolerated disturbance and only rarely allowed colonization of new space (3). However, proliferation of coral epizootics over the last few decades has led to a massive decline of the reef-building corals (4). The increased disease incidence has been linked to rising sea temperatures that may simultaneously stress the coral host and enhance virulence of the pathogens (5, 6). The strong link between infectious disease outbreaks and rising sea temperature has inevitably led to projections of increased epizootics in the future (7). Indeed, the overwhelming current trend in ecosystems science emphasizes additive or synergistic deleterious effects of climate change (2, 8).

However, climate change has resulted in the emergence of novel coral assemblages, whose ecological properties are in marked contrast to those seen in previous millennia (9). Whereas the Caribbean coral assemblages were once dominated by large, long-lived species (e.g., *Acropora cervicornis* and *Montastraea annularis*), they now increasingly comprise small-bodied, fast-growing species that brood their larvae and recruit frequently (e.g., *Porites astreoides* and *Agaricia agaricites*) (10) (Fig. 1). By investigating changes in coral demography, likely borne of climate change, we discover mechanisms that counter the current projections of climate impacts on coral epizootics. We find that allowing for a more dynamic population turnover in an epizoological model of coral disease not only gives a superior fit to empirical data, but also suggests that emerging coral assemblages could be far less prone to epizootics. In challenging current understanding of the importance of disease in coral reefs of the future, our analysis highlights the necessity of considering novel functionality of the novel ecosystems resulting from climate change and other anthropogenic effects.

## Results and Discussion

Even our highly generalized expression for infection (*Methods*) offers insight into patterns of disease among coral reefs. Observations of epizootics constrained by threshold levels of coral cover (6) represent a classic epidemiological phenomenon. For an outbreak of disease with transmission rate $\beta$ to occur, the following condition must be met (11):

$$S^* > \frac{\mu}{\beta}. \quad [1]$$

The general observation within the Caribbean that coral species with high population turnover rates are naturally more resistant to epizootics is explained by the direct proportionality between the coral coverage necessary for an outbreak ($S^*$) and the coral population mortality rate ($\mu$). In this way, higher infection transmission rates ($\beta$) necessitate lower densities of susceptible corals for an epizootic to occur. Conversely, populations with a high turnover (thus, high $\mu$) require higher densities of susceptible corals to counter the fact that individual colonies tend not to persist long enough to become infected and to pass on the infection.

The reduced disease susceptibility of postepizootic coral populations is normally attributed to adaptive immunological resistance in surviving individuals (12, 13). An additional explanation for this phenomenon arises when underlying ecological processes are considered. For undisturbed populations of large reef builders, there is negative skewing in the colony size structure (14), meaning the population is dominated by large coral colonies with inherently low turnover rates. Whereas such populations are primed for epizootics (because of a lowered critical threshold coral density, Eq. **1**), the opposite is true for positively skewed coral populations that have recently experienced severe perturbations.

We fit our simple model (Eqs. **3a** and **3b**) to the best time series of coral epizootics available: an outbreak of White Plague type II in a population of *Dichocoenia stokesi* in the Florida Keys (Fig. 2). The model offers a parsimonious explanation for the observed reduction in secondary disease outbreaks. Coral mortality from the first disease outbreak reduces the average size of a colony in the postoutbreak population. Because smaller colonies have a higher rate of mortality, the average turnover of the postoutbreak population is higher than that before the outbreak. This increase in population turnover makes it more difficult for a second epizootic to occur. For an outbreak to occur, a colony must survive long enough to become infected and then infect, on average, more than one additional colony. This outcome becomes decreasingly likely for a population consisting of small, ephemeral colonies. In short, whereas coral might exhibit adaptive immunological responses (13), we show that higher demographic rates naturally reduce the ability of the disease to spread within a population consisting of diminutive colonies under high flux. A previous model (15), which had greater complexity but lacked the life history dynamism included here, was unable to resolve the secondary epizootic and achieved a poorer fit to the data (Fig. 2). Inclusion of dynamism in demographic rates not only generates a better model fit but also does so with fewer parameters and without explicit space.

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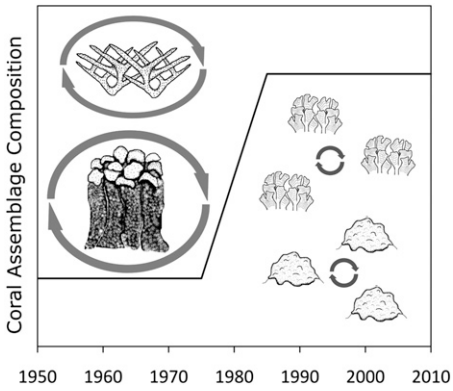


**Fig. 1.** The recent switch in the dominant coral species within Caribbean reefs. Species with low demographic rates, *Acropora cervicornis* (*Upper Left*) and *Montastraea annularis* (*Lower Left*), have been replaced by species with high demographic rates, *Agaricia* spp. (*Upper Right*) and *Porites asteroides* (*Lower Right*).

We conclude that climate change has antagonistic influences on epizoology. First, it has a positive effect on coral susceptibility to infection at an individual level because of rising stress upon hosts and perhaps elevated pathogen abundance or virulence (7). Set against this effect are changes at the assemblage and population level that reduce the risk of epizootics through increased average population turnover. These effects are not confined to novel ecosystems because the elevated mortality rate of corals anticipated under more frequent coral bleaching (8) will also elevate population turnover, attenuating the risk of epizootics. Overall, the net outcome of these conflicting mechanisms depends on the relative magnitudes of the antagonists (Fig. 3), which are currently unclear and will doubtless vary over time and space.

The results of our model also provide a unique perspective to explain the contrasting patterns of coral epizootics in different oceans. In the Caribbean, outbreaks have primarily affected coral species with slow natural turnover rates (16). However, in the Indo-Pacific, epizootics are most common in acroporids that actually exhibit relatively rapid population dynamics (17). On the basis of the conflicting mechanisms illustrated in Fig. 3, we hypothesize that disease transmission rates are higher in the Indo-Pacific to counter the demographic resistance of rapid turnover coral species.

Biotic and abiotic causes of ecosystem novelty have been described (18, 19) but a formal framework is needed. To place our

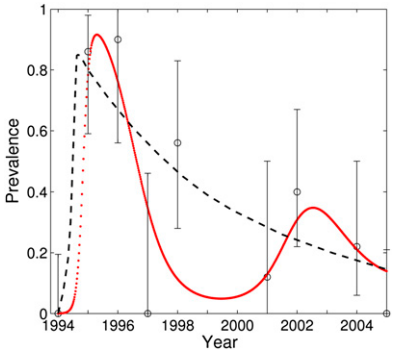


**Fig. 2.** White Plague type II prevalence data (circle markers with SE bars) from *Dichocoenia stokes* coral in the Florida Keys. Parameter values are $r$ = 0.25, $\mu_1$ = 0.2125, $\mu_2$ = 0.05, $\beta$ = 1, and $\sigma$ = 0.5 [sum of squared residuals 0.36, our model (red line) vs. 0.48, previous model (black dashed line)] (15).

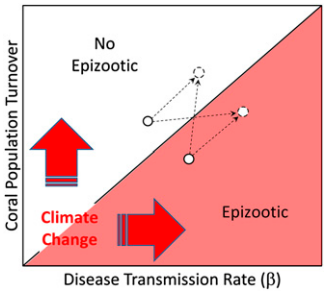


**Fig. 3.** The effects of climate change on the coral population turnover and the transmission rate of infectious disease act antagonistically on the probability of epizootics. The epizootic threshold occurs at R0 = 1, where R0 is calculated as $\beta/\mu_1$. Different scenarios of the net effect are illustrated and depend on the relative influence of competing mechanisms.

results in a generic context, we categorized the mechanisms that can potentially give rise to novel ecosystems (Table 1). Our coral reef case study highlights a variety of possible routes to ecosystem novelty. The Caribbean Sea constitutes a novel environment in that it is warmer now than in the recent past (20) and species have responded asymmetrically to change, thereby nullifying the likelihood that such assemblages are merely an early successional state of the original ecosystem (i.e., type 2.2 with a novel ecosystem, Table 1). Alternatively, if the pathogen is a novel biotic agent (21), then a novel ecosystem could arise through both abiotic and biotic drivers (type 3). Our classification also allows us to distinguish an unfamiliar local environment, which is not unprecedented for the biome, from an environment that is completely novel. Distinguishing such changes is important because ecosystem novelty is more likely to occur under a novel environment (19). Our results imply that projecting the future of a novel ecosystem from trends in the recent past may have misleading results. The net outcome of biotic and abiotic change may not be easily generalizable; however, armed with the view of a dynamic and adapting system, predictive efforts can at least be expected to improve.

## Methods

In the simplest case, corals ($C$) are recruited into a susceptible class ($S$) and become infected ($I$) with disease transmission rate $\beta$. Using the classic mass action approach (22), the rates of change in the susceptible and infected classes are

$$\frac{dS}{dt} = rC - \beta SI - \mu_1 \quad [2a]$$

$$\frac{dI}{dt} = \beta SI - (\mu_1 + \mu_2)I, \quad [2b]$$

where $r$ represents new coral recruitment, and $\mu_1$ and $\mu_2$ are the respective natural mortality rate and the disease-induced mortality rate of coral. Following disturbance-associated mortality (e.g., epizootic induced), coral coverage is reduced and colony size structure becomes more positively skewed (23). Recruitment rates increase as more free space is made available (24) and average colony mortality rates will be increased with a greater proportion of smaller colonies (25). This life history dynamism can easily be incorporated into our framework:

$$\frac{dS}{dt} = (S + \sigma I)\, r\left(1 - \frac{C}{K}\right) - S\mu_1\left(1 - \frac{C}{K}\right) - \beta SI \quad [3a]$$

$$\frac{dI}{dt} = \beta SI - I\left(\mu_1\left(1 - \frac{C}{K}\right) + \mu_2\right). \quad [3b]$$

**Table 1. Typology of ecosystem responses to anthropogenic changes in biotic and abiotic environments**

| Type | Primary driver | Subtype | Examples of mechanism | Ecosystem outcome |
|---|---|---|---|---|
| Type 1 | Biotic | 1.1: Invasive species | Direct anthropogenic disturbance | Novel ecosystem |
| | | 1.2: Species loss | Direct anthropogenic disturbance | Novel ecosystem |
| Type 2 | Abiotic | 2.1: Locally unfamiliar environment | Species resist change | No change |
| | | | Symmetrical response of species to change | Ecosystem migration |
| | | | Asymmetrical response of species to change | Novel ecosystem |
| | | 2.2: Novel environment | Species resist change | No change (unlikely) |
| | | | Either symmetrical or asymmetrical response to change | Novel ecosystem |
| Type 3 | Biotic and abiotic | 3.1: Biotic driver does not have abiotic cause | Direct anthropogenic disturbance | Novel ecosystem |
| | | 3.2: Abiotic change causes biotic driver | Species range shift | Novel ecosystem |

Here, $K$ is the carrying capacity: the level of coral coverage for which new recruitment and natural mortality are effectively zero. $0 \leq \sigma \leq 1$ allows for reduced recruitment associated with infected colonies.

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1. Parmesan C, Yohe G (2003) A globally coherent fingerprint of climate change impacts across natural systems. *Nature* 421:37–42.
2. Halpern BS, et al. (2008) A global map of human impact on marine ecosystems. *Science* 319:948–952.
3. Edmunds PJ, Elahi R (2007) The demographics of a 15-year decline in cover of the Caribbean reef coral *Montastraea annularis*. *Ecol Monogr* 77:3–18.
4. Gardner TA, Côté IM, Gill JA, Grant A, Watkinson AR (2003) Long-term region-wide declines in Caribbean corals. *Science* 301:958–960.
5. Rogers C (2008) Coral bleaching and disease should not be underestimated as causes of Caribbean coral reef decline. *Proc R Soc Lond Ser B Biol Sci* 276:197–198.
6. Bruno JF, et al. (2007) Thermal stress and coral cover as drivers of coral disease outbreaks. *PLoS Biol* 5:e124.
7. Cróquer A, Weil E (2009) Changes in Caribbean coral disease prevalence after the 2005 bleaching event. *Dis Aquat Organ* 87:33–43.
8. Hoegh-Guldberg O, et al. (2007) Coral reefs under rapid climate change and ocean acidification. *Science* 318:1737–1742.
9. Pandolfi JM, Jackson JBC (2006) Ecological persistence interrupted in Caribbean coral reefs. *Ecol Lett* 9:818–826.
10. Green DH, Edmunds PJ, Carpenter RC (2008) Increasing relative abundance of *Porites astreoides* on Caribbean reefs mediated by an overall decline in coral cover. *Mar Ecol Prog Ser* 359:1–10.
11. Anderson RM, May RM (1991) *Infectious Diseases of Humans: Dynamics and Control* (Oxford Univ Press, New York).
12. Mydlarz LD, McGinty ES, Harvell CD (2010) What are the physiological and immunological responses of coral to climate warming and disease? *J Exp Biol* 213: 934–945.
13. Reed KC, Muller EM, van Woesik R (2010) Coral immunology and resistance to disease. *Dis Aquat Organ* 90:85–92.
14. Colgan MW (1987) Coral reef recovery on Guam (Micronesia) after catastrophic predation by Acanthaster planci. *Ecology* 68:1592–1605.
15. Sokolow SH, Foley P, Foley JE, Hastings A, Richardson LL (2009) Editor's choice: Disease dynamics in marine metapopulations: Modelling infectious diseases on coral reefs. *J Appl Ecol* 46:621–631.
16. Cróquer A, Weil E (2009) Spatial variability in distribution and prevalence of Caribbean scleractinian coral and octocoral diseases. II. Genera-level analysis. *Dis Aquat Organ* 83:209–222.
17. Sutherland KP, Porter JW, Torres C (2004) Disease and immunity in Caribbean and Indo-Pacific zooxanthellate corals. *Mar Ecol Prog Ser* 266:273–302.
18. Hobbs RJ, et al. (2006) Novel ecosystems: Theoretical and management aspects of the new ecological world order. *Glob Ecol Biogeogr* 15:1–7.
19. Hobbs RJ, Higgs E, Harris JA (2009) Novel ecosystems: Implications for conservation and restoration. *Trends Ecol Evol* 24:599–605.
20. Strong AE, et al. (2008) Implications for our coral reefs in a changing climate over the next few decades—hints from the past 22 years. *Proceedings of the 11th International Coral Reef Symposium*, pp 1130–1134. Available at www.reefbase.org.
21. Harvell CD, et al. (1999) Emerging marine diseases—climate links and anthropogenic factors. *Science* 285:1505–1510.
22. Kermack WO, McKendrick AG (1927) A contribution to the mathematical theory of epidemics. *Proc R Soc Lond A* 115:700–721.
23. Hughes TP, Jason ET (2000) Recruitment failure, life histories, and long-term decline of Caribbean corals. *Ecology* 81:2250–2263.
24. Edmunds P (2000) Recruitment of scleractinians onto the skeletons of corals killed by black band disease. *Coral Reefs* 19:69–74.
25. Hughes TP, Jackson JBC (1985) Population dynamics and life histories of foliaceous corals. *Ecol Monogr* 55:141–166.