LETTER

Susceptibility of coral-disease models

The scarcity of empirical data on marine diseases highlights the need for epidemiological models that explain patterns and processes. Yakob and Mumby (1) used a generic susceptible-infected model to describe the prevalence of white plague type II disease on a coral population (*Dichocoenia stokesii*). They compared their model with the metapopulation model of Sokolow et al. (2). Yakob and Mumby (1) stated that rapid population turnover explained the observed oscillations in disease prevalence. Previous marine-disease models have largely ignored life-history traits. Indeed, systems subjected to high return periods of stress can be dominated by weedy species with rapid turnover rates. Although the Yakob and Mumby (1) model incorporated life-history traits and fits the data better than the model of Sokolow et al. (2), we find four conceptual inconsistencies within their model.

First, Yakob and Mumby (1) indicated that recruitment increased with the availability of settlement space. The loss of coral colonies usually has the opposite effect, reducing recruitment (3). Similarly, *D. stokesii* most likely suffered recruitment reduction after the initial disease outbreak, because no *D. stokesii* recruits were observed over a 7-y period following the initial outbreak (4). Therefore, it is unlikely that high recruitment could drive rapid population turnover, as suggested by Yakob and Mumby (1), and, in turn, explain the observed dynamics of white plague type II prevalence. Moreover, the model was highly sensitive to recruitment rate changes. Increasing recruitment by ~8% offset the second peak in disease prevalence, whereas decreasing recruitment by the same amount removed the second peak entirely (Fig. 1*A*).

Second, the authors parameterized natural mortality (21%) more than four times higher than disease-induced mortality (5%). White plague type II is one of the most aggressive coral diseases, potentially killing tissue at several centimeters per day. Such virulence can cause colony mortality within days of initial infection. Therefore, disease-induced mortality should be modeled substantially higher than natural mortality, but doing so radically changes the outcome of the model (Fig. 1*B*). Clearly, the model presented is not robust to variations in the input parameters.

Third, the model of Yakob and Mumby (1) parameterized the transmission rate at 100%. The authors argued that higher transmission rates among Pacific Ocean acroporids can cause differences in disease dynamics between the oceans; however, transmission rates higher than 100% are not possible.

Fourth, although the ultimate goal of modeling is to generate scale-invariant equations, applying population models [e.g., the model of Yakob and Mumby (1)] to metapopulation data [e.g., those presented by Sokolow et al. (2)] may misalign critical spatial, temporal, and environmental processes. Instead of suggesting that coral populations can evade disease through rapid turnover, it may be more fruitful to incorporate potential adaptive-like immune systems (5) into coral-disease models. It may be just as appropriate to conceptualize some coral diseases as noncontagion systems that express disease prevalence in relation to environmental thresholds, especially in a rapidly warming ocean.

***Adan G. Jordán-Garza*[1], *Erinn M. Muller*[1], *Scott G. Burman*[1], *and Robert van Woesik*[1,2]**

Department of Biological Sciences, Florida Institute of Technology, Melbourne, FL 32901-6988

1. Yakob L, Mumby PJ (2011) Climate change induces demographic resistance to disease in novel coral assemblages. *Proc Natl Acad Sci USA* 108:1967–1969.
2. Sokolow SH, Foley P, Foley JE, Hastings A, Richardson LL (2009) Disease dynamics in marine metapopulations: Modeling infectious diseases on coral reefs. *J Appl Ecol* 46: 621–631.
3. Hughes TP, et al. (2000) Supply-side ecology works both ways: The link between benthic adults, fecundity, and larval recruits. *Ecology* 81:2241–2249.
4. Richardson LL, Voss JD (2005) Changes in a coral population on reefs of the northern Florida Keys following a coral disease epizootic. *Mar Ecol Prog Ser* 297: 147–156.
5. Reed KC, Muller EM, van Woesik R (2010) Coral immunology and resistance to disease. *Dis Aquat Organ* 90:85–92.

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[1]A.G.J.-G, E.M.M., S.G.B., and R.v.W contributed equally to this work.

[2]To whom correspondence should be addressed. E-mail: rvw@fit.edu.

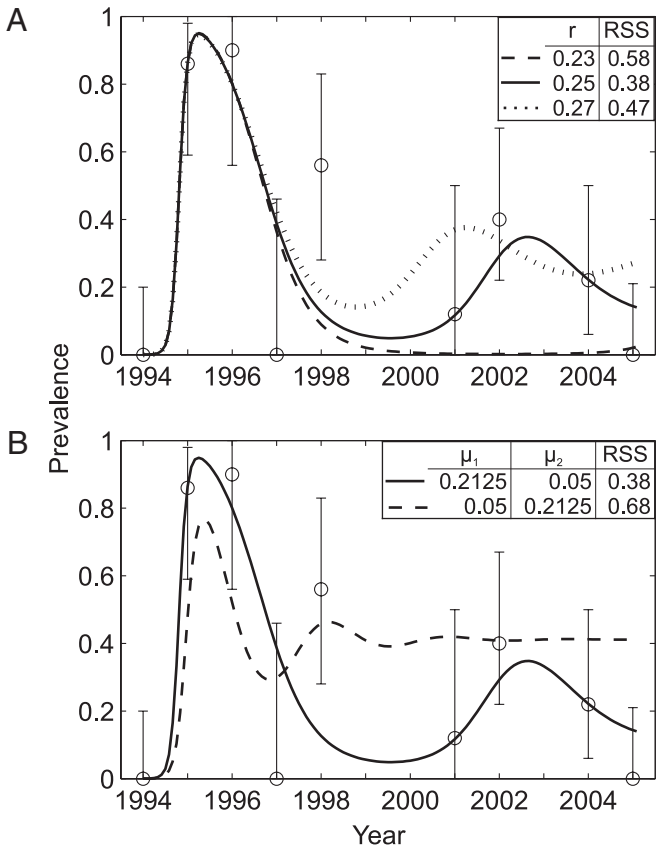

Fig. 1. A series of examples displaying the sensitivity of the model of Yakob and Mumby (1) to changes in recruitment (*A*) and mortality (*B*). Solutions for the original Yakob and Mumby model (solid black lines in *A* and *B*) are reproduced with the original parameterizations of recruitment (r = 0.25), natural and disease-induced mortality (μ_1 = 0.2125 and μ_2 = 0.05, respectively), transmission rate (β = 1), and reduced recruitment rates associated with infected colonies (σ = 0.5). Within a metapopulation context, recruitment represents the colonization of uncolonized sites; μ_1 quantifies the probability of site extinction under natural conditions, and μ_2 quantifies the probability of site extinction under disease conditions. The original white plague type II data (4) are also presented (circles with 95% confidence intervals). The broad overlapping 95% confidence intervals of the data, especially between 2001 and 2004, and the lack of information in 1999 and 2000 suggest that the second peak in disease prevalence could be an artifact generated by the model. Disease prevalence was the total number of sites with the disease divided by the total number of surveyed sites each year (2). The disease data were fitted to each model, and the residual sum of squares (RSS) was reported. (*A*) Results are shown by slightly altering (±0.02 or ±8% relative change) the recruitment value (r = 0.23 is represented by the hatched line, r = 0.27 is represented by the dotted line); all other parameters are the same as in the original model of Yakob and Mumby (1). (*B*) Model values are displayed when natural and disease-induced mortality are switched, where μ_1 = 0.05 and μ_2 = 0.2125; all other parameters are the same as in the original model. Reducing recruitment, a likely scenario when coral densities decrease, eliminated the oscillating pattern. Increasing recruitment increased the oscillation frequency. Changing the mortality parameters produced a model with a higher oscillating frequency and lower amplitude, which continued to decrease over time, reaching equilibrium when the disease became endemic. In all cases, changing the parameters resulted in a worse-fitting model (i.e., higher RSS).