

Disease prevalence and population density over time in three common Caribbean coral reef sponge species

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Reports of disease in sponges are increasing, but the paucity of data on disease prevalence over time makes it uncertain how much this trend reflects increased attention to sponges rather than increased sponge disease. Population and community influences on disease dynamics, and the consequences of disease at these levels, are also little known. Five censuses, over 14 y, of a small plot on a shallow coral reef at San Blas, Panama, provide data for the three most abundant species on population dynamics (number of individuals and total volume) and disease prevalence (number of individuals with active lesions). Although data for the three species, combined in broad categories (i.e. high vs low), support a general conclusion that disease prevalence was greater from 1994–1998 than from 1984–1988, the data do not demonstrate a steady increase over time, and disease prevalence for two of the species decreased in each of the final two censuses from a high in 1994. Fluctuations in population density (total volume) and disease prevalence were nearly synchronous within individual species, but asynchronous among the three species, suggesting that population density, measured as total sponge volume per unit area, may influence disease dynamics in these sponges.

INTRODUCTION

Diseases of coral reef organisms are being reported with an increasing frequency and sense of urgency, as data accumulate on the speed with which abundant and functionally important species can be diminished by disease. As the chief generators of reef framework building material, scleractinian corals have received the great majority of the attention (e.g. Weil et al., 2006 for a recent review), but it is clear that diseases are playing definitive roles in population dynamics of other cnidarians and also sponges (e.g. Smith et al., 1996; Harvell et al., 1999; Acosta, 2001; Weil, 2004; Webster, 2007).

Although dramatic episodes of disease in sponges have been reported, especially in commercially valuable species (Smith, 1941; Pansini & Pronzato, 1990; Pronzato et al., 1999) or in conspicuous large-bodied species (e.g. Nagelkerken et al., 2000; Cervino et al., 2006), the aetiological agents have been elusive in all but a few cases (Rützler, 1988; Webster et al., 2002; Cervino et al., 2006). Our minimal understanding of the epidemiology, and the population or community consequences, of disease in sponges reflects the difficulty of studying sponge disease in the field. Diseased sponges, or portions of sponges, disappear rapidly once the tissue is killed and the skeletal fibres exposed, erasing the history—even history as recent as a few weeks previously—of disease in a population (Wulff, 2006b). At any particular moment, currently active lesions constitute the only evidence of disease, and estimates of actual losses to disease over time can only be acquired by long periods in the field or by repeated censuses (e.g. Stevely, 1996; Wulff, 2006a). Interpretation of disease observations in the context of community and population

dynamics is further complicated by the possibilities of recovery from disease and asexual propagation by breakage at lesions.

Sponges benefit coral reefs and associated marine ecosystems by efficiently filtering small organic particles from the water column, increasing survival of live corals by binding them to the reef frame, facilitating regeneration of damaged reefs, providing food for spongivores, protecting mangrove roots and corals from boring organisms, sheltering animals such as juvenile spiny lobsters, and harbouring nitrifying and photosynthesizing microbial symbionts (reviews by Diaz & Rützler, 2001; Wulff, 2001). Bright colours and interesting shapes of tropical sponges attract human divers, inspiring responsible stewardship. The possible immediate seriousness of loss of sponges, and all they contribute to healthy marine ecosystems, is suggested by two studies in which sponge communities were censused over time: (a) during only a few months, as many as 90% of the sponges died or were damaged in Florida Bay sites influenced by cyanobacterial blooms in 1991–1993 (Butler et al., 1995; Stevely, 1996; Stevely & Sweat, 2001); and (b) over 1/3 of the original sponge biomass and half of the original 39 sponge species were lost over the course of 14 y (1984–1998) of monitoring a shallow coral reef site in San Blas, Panama (Wulff, 2006a).

A recent review of disease in sponges (Webster, 2007) highlights both the apparently increasing influence of sponge disease on marine ecosystem dynamics and the great gaps in our knowledge about disease in sponges. Although reports of such events are definitely increasing, the paucity of baseline data makes it difficult to know if these reflect greater attention to sponges, or if disease is actually on the rise

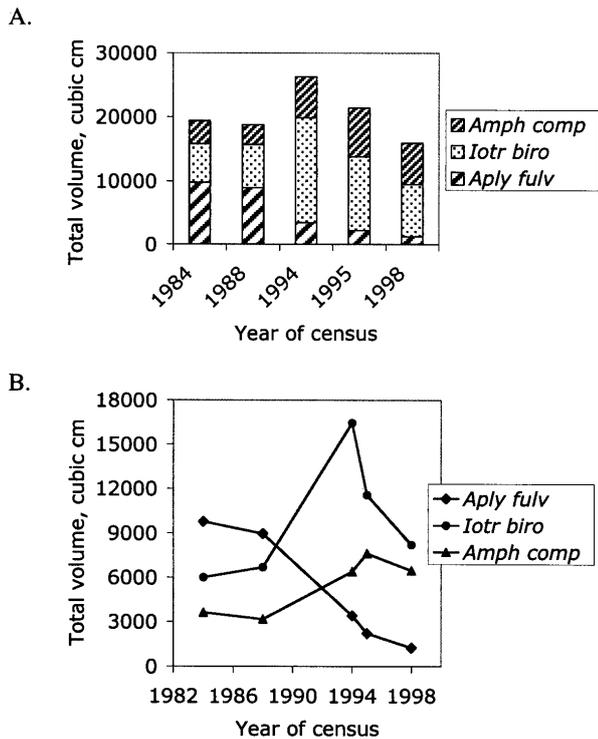


Figure 1. Relative abundance, measured by total sponge volume, of *Iotrochota birotulata*, *Amphimedon compressa* and *Aplysina fulva*, in 16 m² of a shallow reef at San Blas, Panama, at five census dates between 1984 and 1998, plotted: (A) to highlight lower variation in total combined volume relative to variation in volume of individual species; and (B) to highlight the lack of synchrony in population fluctuations of these three sponge species.

(Webster, 2007). Also unknown is if high sponge population densities can increase vulnerability to disease, perhaps by facilitating transmission. Dramatic losses to disease from high density sponge farms (e.g. Smith, 1941) suggest that density–disease relationships warrant evaluation.

What caused declines in sponge volume and diversity on a shallow coral reef in San Blas, Panama is not known for certain because monitoring was at long intervals, but disease appeared to be among the most plausible agents (Wulff, 2006a). Indications of disease were recorded at each census. Three of the species, all of erect branching growth forms, were sufficiently abundant that combined disease prevalence and population dynamics data may provide insight on the questions: (1) did disease prevalence increase over the 14 y between 1984 and 1998?; and (2) is it possible that population density influences disease prevalence in these common coral reef sponges?

MATERIALS AND METHODS

A coral reef community, in which sponges were the most abundant sessile organisms, was censused five times over 14 y (details of census methods and results in Wulff, 2001, 2006a). Only 16 m² were censused completely, but diversity and abundance were high, so that at the first census this small area held 1395 individual sponges representing 39 species (Wulff, 2001, 2006a). Sponge abundance was evaluated

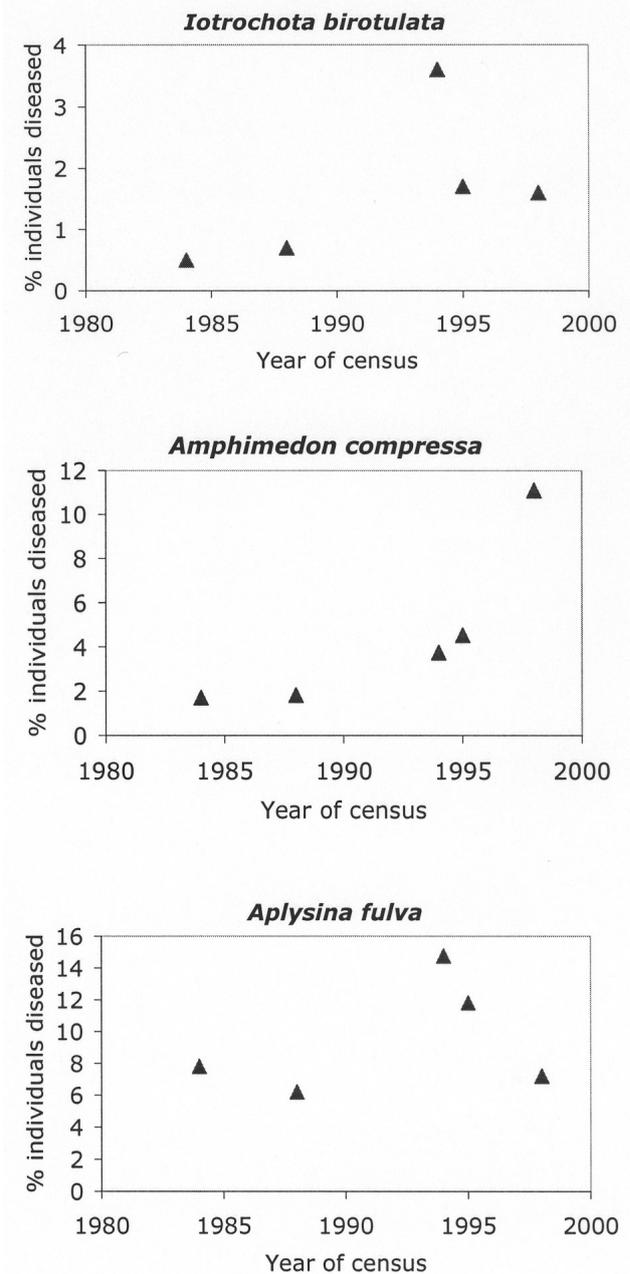


Figure 2. Disease prevalence (% of individuals diseased) over time, at five census dates between 1984 and 1998, in a 16 m² censused plot at San Blas, Panama.

by number of individuals, area covered, and volume; and during each census, all indications of damage or disease were recorded. Disease had a characteristic appearance and pattern of progression for each species (described in Wulff, 2006b), and diseases appeared to be species-specific (Wulff, 1997). Disease prevalence was recorded as the number of physiologically independent individual sponges exhibiting a clearly active lesion (or, in some cases, lesions) characteristic of disease in that species.

RESULTS

Initial population densities of the three most abundant species, *Iotrochota birotulata* (Higgin), *Amphimedon compressa* (Pallas) and *Aplysina fulva* (Pallas), in the 16 m² were 307,

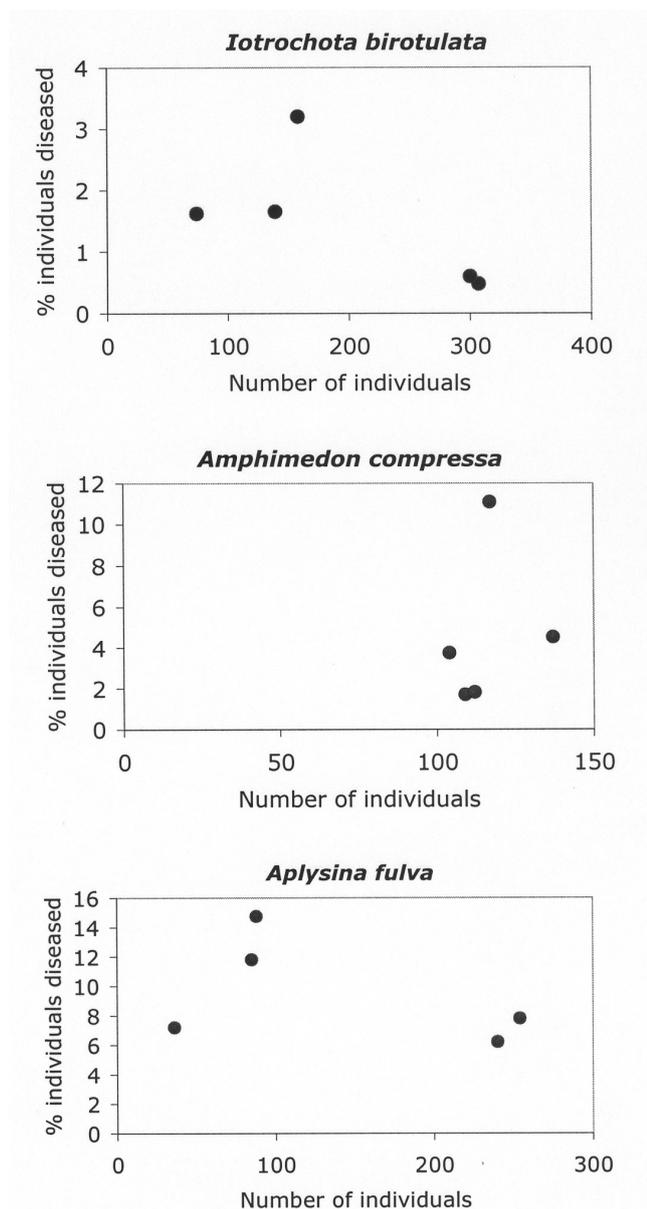


Figure 3. Disease prevalence (% of individuals diseased) and number of individuals in 16 m² at five census dates between 1984 and 1998, at San Blas, Panama.

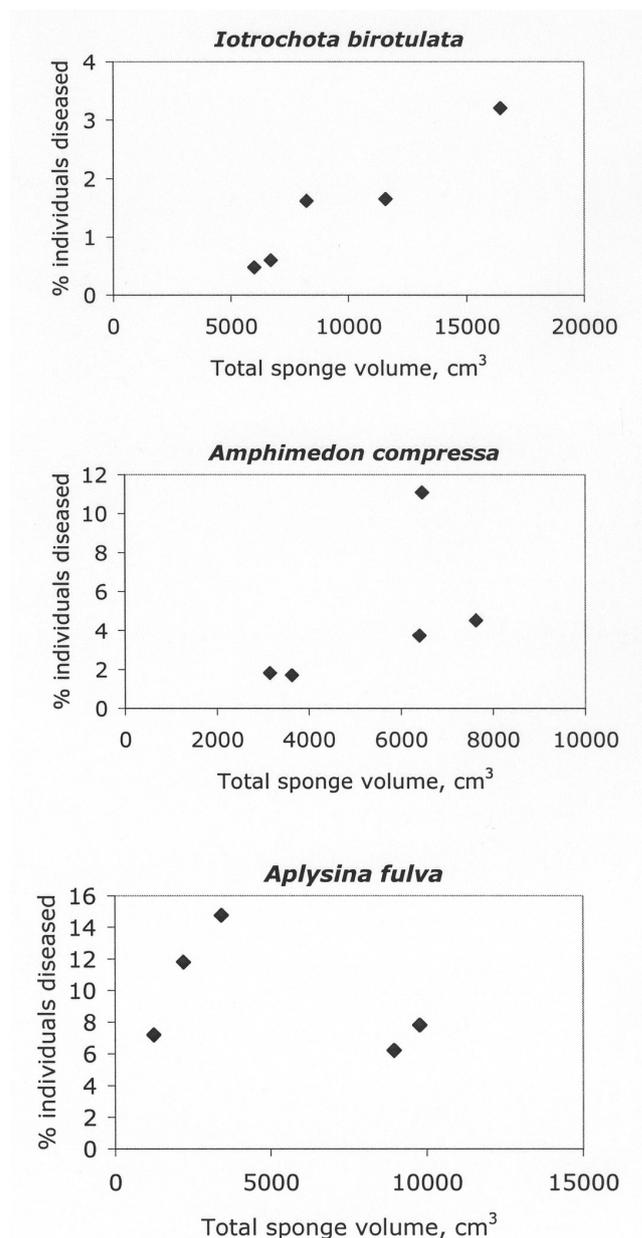


Figure 4. Disease prevalence (% of individuals diseased) and total sponge volume in 16 m² at five census dates between 1984 and 1998, at San Blas, Panama.

109, and 254 individuals, and 6001.3, 3626.3, and 9767.3 cm³ total volume, respectively. Although the combined total volume of these three species varied relatively little, especially during the first four censuses, the proportion contributed by each species changed over time (Figure 1A). Standard errors of the mean volumes for the five censuses for *I. birotulata*, *Amphimedon compressa* and *Aplysina fulva* are 0.18, 0.14, and 0.31, respectively; whereas the standard error of the mean for their combined volume is only 0.08. Presenting these data in an x:y scatter plot highlights the lack of synchrony in population fluctuations of the three species (Figure 1B).

At every census (i.e. in February 1984, August 1988, June 1994, September 1995 and March 1998), disease was observed in individuals of *I. birotulata*, *Amphimedon compressa* and *Aplysina fulva*. Disease was also observed in individuals

of eight other species, but they were not sufficiently abundant within the plot to document disease dynamics meaningfully.

For each of the three sponge species, disease prevalence was analysed with respect to time (Figure 2), number of individuals (Figure 3) and total sponge volume (Figure 4). By Kendall's rank correlation coefficient, the only associations that were significantly ($P=0.05$) different from random were disease prevalence and total volume for *I. birotulata*, and disease prevalence and year of census for *Amphimedon compressa*; but with only five census dates, a single mismatch in the joint ranking of the variables renders an association not significant by this non-parametric test. Disease prevalence was especially high in 1994 for both *I. birotulata* and *Aplysina*

fulva. For *A. fulva* there appear to be two different relationships of volume, and also time, with disease prevalence.

Although the specific relationships of disease prevalence with time, number of individuals, and volume were different for each species (and there is no reason to expect them to be the same), data from the three species could be combined in broad categories to test for possible general patterns. For the data in Figures 2, 3 and 4, the G-test was used to test for association of relatively high disease prevalence (i.e. the highest 3 data points for each species) vs low disease prevalence (the lowest 2 data points for each species) and the highest 3 vs lowest 2 data points for each of (a) time, (b) number of individuals, and (c) total volume. The data give no reason to reject a null hypothesis that the association between disease prevalence and number of individuals is random for these three species. However, the association between high disease prevalence and later monitoring years was significant ($P < 0.025$), as was the association between high disease prevalence and high sponge volume ($P < 0.025$).

DISCUSSION

Disease prevalence over time

Although the association between disease prevalence and time is statistically significant when both variables are divided into two categories, the data do not indicate that disease steadily increased between 1984 and 1998. Prevalence was highest in *Amphimedon compressa* in 1998, the final year in which a census could be made on this reef, but in both *Iotrochota birotulata* and *Aphysina fulva* prevalence was highest in 1994, and then decreased in 1995, and again in 1998. Fluctuations in disease prevalence over time were not otherwise synchronous in these three sponge species, suggesting that they were not driven by a particular external environmental variable.

These data raise the question of what the appropriate time scale is for considering if disease is, or is not, increasing in sponges. In any attempt to demonstrate significant change over time, a long-term trend can be obscured by short-term fluctuations. Perhaps the significant difference in disease prevalence between the 1980s and the 1990s is truly indicative of an overall increase, and the asynchronous fluctuations during 1994–1998 reflect short-term responses of individual species to population level or abiotic parameters. Unfortunately these data cannot be placed in a longer time-scale context, because there are no data on sponge disease in San Blas before 1984, and, after the 14 y of censusing, this reef was no longer available to researchers. The simple question ‘Is sponge disease increasing?’ cannot be answered until we have made repeated community-wide sponge censuses, accompanied by systematic recordings of disease, at many more sites.

Disease prevalence and density in fragmenting sponges

For asexually fragmenting organisms, such as these branching sponges, population density can be evaluated in several ways, including: (a) number of physiologically independent individuals; (b) total volume; and (c) number of genotypes. These density measures differ in the insights they can provide, and also in their methodological difficulties.

Density evaluated as numbers of individuals

For the three branching sponge species, association between number of individuals and disease was not significantly different from random, and no patterns emerge from plots for individual species. This is not surprising, as the multiple ways in which disease can influence, and be influenced by, the number of individuals in these asexually propagating sponges are likely to obscure patterns. Life histories of all three species are dominated by a cycle of growth, fragmentation, partial mortality, fusion and regeneration (Wulff, 1991). It is possible that transmission of infectious pathogens might be slowed if a given volume of sponge is divided into many physiologically independent individuals, because each individual would have to be independently colonized. On the other hand, disease itself can contribute to the fluidity with which biomass is distributed among physiologically independent ramets. In these branching sponges, the skeleton deteriorates shortly after a disease lesion develops (a mean of 5.5 d in *I. birotulata*, 13.3 d in *Amphimedon compressa*, Wulff, 2006b), so mid-branch lesions result in asexual propagation for sponges that halt disease progression and recover.

In a 15-month study of disease in the branching species *Aphysina cauliformis* in the Bahamas (Olson et al., 2006), the lowest density of sponge individuals was measured (by random transects rather than repeated censuses) in the same monitoring period as the lowest disease prevalence, but these variables were not otherwise associated. The authors suggested that, although number of individuals may have been decreased by disease, two major hurricanes probably played a larger role. The hurricanes may have also decreased disease prevalence, if mortality of diseased sponges was higher due to increased breakage at lesions (Olson et al., 2006).

Thus the net effect of number of individuals and disease prevalence on each other depends on a combination of rate of colonization of individuals by an infectious pathogen, rate of disease progression through each individual, and the relative frequency of cases in which disease progresses until it kills an entire individual vs cases in which new individuals are created as branches break at lesions.

Density evaluated as total volume

Evaluating density of readily fragmented organisms by volume instead of number of individuals avoids interpretation problems caused by the possibility that individuals are created by disease. Another advantage for sponges is that most of their functional roles are played out in proportion to their volume rather than number of individuals (e.g. Wulff, 2001). In this study, total volume and disease prevalence were associated significantly when the data were combined across species and analysed by broad categories. Individual plots of these variables for each of the species show generally positive associations, with the exceptions of a single data point spike in disease prevalence for *Amphimedon compressa* at intermediate volume, and apparently two different positive associations for *Aphysina fulva*. An additional suggestion that disease dynamics may be influenced by population or community level dynamics, instead of (or in addition to) abiotic factors, is provided by the lack of synchrony in fluctuations of both volume and disease prevalence among

these three species. To conclusively determine if density (volume) influences disease would require data from multiple populations differing in density at each census date.

Density evaluated as number of genotypes

Density of genotypes was not evaluated within the census plot, but in an adjacent plot of 10×20 m, clonal diversity was estimated using a combination of somatic tissue grafting, morphological characters and colour (Wulff, 1986). Density of genotypes was much lower than density of individuals. The number of genotypes represented by 90 physiologically independent individuals of *Amphimedon compressa* and 90 of *Aplysina fulva* were, respectively, 12 and 13; and in the 60 largest individuals of *I. birotulata* 24 genotypes were represented, confirming the importance of asexual propagation in these populations. Influence of genotypic diversity on vulnerability to disease is not known for sponges, but may be worth explicit study, given the potential importance of asexual propagation for sponge mariculture. The dramatic commercial sponge epidemic in the 1930s involved underwater farms, in what was then called British Honduras (now Belize), in which at least 700,000 individuals, mostly of two species of *Hippospongia*, were 'planted' (Smith, 1941). Not only were densities up to 30 times normal, but the sponges were propagated by cuttings, creating lower than natural genotypic diversity.

Effects on disease of low genotypic diversity and high density may be hopelessly confounded for organisms in which asexual propagation serves to efficiently achieve high density. Asexual propagation was also important for maintenance of the spectacularly dense stands of the Caribbean corals, *Acropora cervicornis* and *A. palmata*, that until recently were the most striking features of shallow Caribbean fore-reefs (e.g. Highsmith et al., 1980; Tunnicliffe, 1981; Highsmith, 1982; Aronson & Precht, 2001). It is not known if low genotypic diversity has been a factor in rapid losses of these species to disease. Near extermination by disease of the sexually reproducing sea urchin *Diadema antillarum* (e.g. Lessios, 1988) indicates that low genotypic diversity is not a prerequisite for disease to be devastating, but the importance of genetic variation may vary among pathogens and hosts.

Density and infectious disease in the sea

Although greater transmission efficiency of infectious pathogens in denser populations is a central tenet of terrestrial animal epidemiology (e.g. Lloyd-Smith et al., 2005 for a review), disease prevalence in marine organisms has been explicitly related to density in only a few studies. The disease white plague, which afflicts many scleractinian coral species, has been correlated with coral colony density (Richardson et al., 1998); and prevalence of disease in the zoanthid *Palythoa caribaeorum* in Brazil was density-dependent with respect to area covered, but not with respect to number of colonies (Acosta, 2001). Prevalence of disease caused by the terrestrial fungus *Aspergillus sydowii* in the Caribbean sea fan, *Gorgonia ventalina*, was density-dependent with respect to number of colonies near Akumal, Mexico (Mullen et al., 2006). The Caribbean reef corals, *Acropora cervicornis* and *A. palmata*, and the long-spined black sea urchin, *Diadema antillarum*, that have been recently decimated by disease,

lived at particularly high population densities prior to disease (e.g. Gladfelter, 1982; Lessios et al., 1984; Lessios, 1988; Aronson & Precht, 2001; Bruckner et al., 2002), but density differences among sites have not been demonstrated to exert influence on disease prevalence.

The difficulty of disentangling density from other influences in among-site comparisons may account for some of the rareness of demonstrations of density-dependence of disease in the sea. Higher disease prevalence has often been documented at sites that are nearer to shore or to anthropogenic pollutant sources (e.g. for corals: Green & Bruckner, 2000; Sutherland et al., 2004; and for sponges: Cervino et al., 2006; Webster, 2007), although this is not always the case (e.g. Page & Willis, 2006). Density would also not be expected to influence disease that is caused by normal symbionts running amuck, rather than by infectious pathogens (e.g. Rützler, 1988, for a sponge example; Lesser et al., 2007 for a general review).

A possible community consequence of density-dependent disease

The leap from disease prevalence data to population and community consequences of disease is not made gracefully without additional data on rates of incidence, rates of disease progression within individuals, and the frequency of recovery vs mortality. For *I. birotulata*, *Amphimedon compressa* and *Aplysina fulva*, the means of linear lesion expansion rates recorded in the field are, respectively, 0.42, 0.64, and 1.3 cm per day, and halting of lesion expansion, followed by recovery has been observed (Wulff, 2006b). However, the additional details of disease dynamics that are required for predicting community consequences are unknown even for these relatively well-studied species. It is tempting to speculate that a positive association between disease prevalence and density could help to maintain a balance over time in the relative abundance of these three species, as each in turn goes through a cycle of increased density, increased disease, lower density, lower disease. Balance in their relative abundances could be advantageous, as genet survival of a variety of hazards is increased when a sponge of one of these species adheres to a neighbouring heterospecific sponge (Wulff, 1997), and similar population densities may increase the probability that a neighbour represents a different species.

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