REPORT

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A simple model of growth form-dependent recovery from disease in coral reef sponges, and implications for monitoring

Abstract For clonal organisms that can suffer high levels of partial mortality and still recover, the conditions that influence infection and development of disease (e.g., abiotic stressors, population density) may be very different from the conditions that influence recovery. Recovery from infectious disease may increase if an individual can mount a defense before infection spreads throughout its body. If pathogens spread within an organism from an initial infection point, growth form—in conjunction with size—can influence the amount of time before all the tissue is diseased, and recovery precluded. A simple model of pathogen progression within individual sponges predicts that species with massive growth forms will be most susceptible to being overwhelmed by pathogen infection, and branching species will be most likely to recover. These predictions may help to explain the seemingly contradictory observations that branching species had the greatest prevalence of disease, and massive species the greatest rate of loss, in a monitored coral reef community. Disease may be observed disproportionately frequently in the organisms that are most likely to recover, resulting in underestimation, by standard monitoring procedures, of the effect of disease on losses from the community.

Keywords Growth form · Infectious pathogens · Coral reefs · Recovery from disease · Sponges

Introduction

In a paper that drew attention to the fundamental importance of growth form to the lives of clonal sessile animals, Jackson (1979) used simple but compelling

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models to point out how growth form can influence success in competition with neighbors, and in avoiding or recovering from damage caused by abiotic disturbances or predators. At that time pathogens did not appear to be important in the lives of clonal marine animals. But disease has become an increasingly prevalent agent of mortality and community change on coral reefs (e.g., Harvell et al. 1999; Green and Bruckner 2000; Aronson and Precht 2001; Sutherland et al. 2004), and infectious pathogens have often been implicated. In many cases the cause of disease has not yet been identified, much less details of the mechanisms by which the host is able to halt its progression (e.g., Sutherland et al. 2004 for colonial zooxanthellate cnidarians). While infection and development of biotic disease may depend largely on habitat characteristics, taxon-specific vulnerability, and population density; recovery may depend on very different factors, such as body size and, as suggested in this contribution, growth form.

Coral diseases have received a great deal of attention (e.g., Green and Bruckner 2000; Santavy et al. 2001; Sutherland et al. 2004), but mortality due to disease has also been reported in sea fans (e.g., Harvell et al. 1999), and reports of diseased sponges are increasingly frequent (e.g., Rützler 1988, 2004; Butler et al. 1995; Goreau et al. 1998; Pronzato et al. 1999; Perez et al. 2000; Williams and Bunkey-Williams 2000; Wulff 2006; and a recent website devoted to sponge disease in the Caribbean at http://www.reefbase.org/spongedisease). Historically, diseases have had tremendous effects on sponge populations (e.g., Smith 1941). Disease is more difficult to observe and quantify in sponges than in corals. Whatever the overall growth form of a coral, live coral tissue is a thin sheet over the surface of a rock-like skeleton, so disease progression is readily observed. Calcareous skeletons of corals provide a record of their lives long after they have died, so mortality can be quantified months or even years afterwards. Sponges, by contrast, are 3-dimensional masses of living cells pervaded homogeneously by canals, choanocyte chambers, and skeletal framework. Disease can progress invisibly, deep

inside. Denuded skeletal fibers disintegrate, and so dead sponges quickly disappear, rendering quantification of disease incidence and mortality impossible without continuous monitoring.

Loss of sponges to disease is cause for concern because of important functional roles played by coral reef sponges, such as filtering bacteria from the water column, binding live corals to the reef frame, and facilitating reef regeneration (reviews in Diaz and Rützler 2001; Wulff 2001). Sponges are the most abundant sessile animals by biomass in many Caribbean reef areas (e.g., Diaz and Rützler 2001), but taxonomic and quantification challenges have discouraged inclusion of sponges in monitoring schemes. Compounding the problem, monitoring at time intervals short enough to observe diseased sponges before they disintegrate and vanish (i.e., 1–10 weeks) is impractical at most sites. The need for other strategies to learn how disease could affect sponges, including how to focus field data collection, motivated exploration of models of disease progression. Sponges are useful starting points for exploring influence of growth form on disease recovery in sessile animals in general, because sponges are structurally simple and homogeneous and yet display a great variety of growth forms.

Disproportionately frequent reports of disease in unusually large sponges, e.g., "loggerhead" (*Spheciospongia vesparium*) and "barrel" (*Xestospongia muta*) sponges (e.g., Butler et al 1995; Goreau et al. 1998; Williams and Bunkley-Williams 2000), may reflect how conspicuous they are due to their size. But disease may also be more likely to be observed in large sponges because it takes longer for a pathogen to progress throughout their tissue. For example, a bathtub-sized *X. muta* in Curaçao was observed by many divers as it took a couple of months to entirely succumb to disease (I. Nagelkerken, photo and observations in Reef Encounter 28:14–15). In the medium-sized vase-shaped species, *Mycale laxissima*, degeneration due to a pathogen progressing upwards from the base resulted in death within 2 months (Reiswig 1973). By contrast, smaller sponges of the commercially important genera *Spongia* and *Hippospongia* were dead within a week after initially internal lesions became evident at their surfaces (Smith 1941). Encrusting species, with very small volumes, can fully disintegrate within a few days of the first signs of disease (Hummel et al. 1988, personal observation).

Larger bodied sponges may also be more likely to recover from disease because they have more time to respond to a pathogen and halt its spread before it has infected the entire sponge. Among sponges that recovered from disease in Florida Bay were individuals of *S. vesparium*, typically the largest sponges in the system (Butler et al. 1995); and, on a Panama reef, disease in an *Ircinia strobilina* over 1 m in diameter and in several large *X. muta* (1–1.5 m in diameter) halted when 5–20% of the initial living mass was still alive. Subsequently the sponges regenerated (personal observation).

Here I suggest that recovery of sponges can also be influenced by growth form. A simple model of pathogen progression through sponge individuals relates the time required for infection to progress throughout a sponge, and also the rate of increase of the area of the interface between diseased and healthy tissue, to sponge growth form. The model predicts surprisingly large differences, suggesting that growth form may strongly influence recovery if success is increased by (1) more time between an initial lesion and spread of infection throughout, and (2) less area of diseased-healthy interface over which to block pathogen advance.

Materials and methods

Disease progression in the field

Diseased sponges were monitored in the field, so that parameters in the models could be made as realistic as possible with respect to how disease progresses within individual sponges and how sponges react to disease. In massive species, disease is often invisible until it reaches the surface; but in branching sponges, the progression can be followed from day to day along each branch. Disease progression within individuals of three common erect branching species, *Iotrochota birotulata* (Higgin), *Amphimedon compressa* Duchassaing & Michelotti, and *Aplysina fulva* (Pallas), was monitored on a shallow Caribbean coral reef in San Blas, Panama (site description in Wulff 2001) during May 1998 and June 1999. These species, which represent different orders of Demospongiae (respectively Poecilosclerida, Haplosclerida, and Verongida), differ in the proportions of their skeletal fibers that consist of siliceous spicules (means of respectively 83, 40, and 0%, Wulff 1995). Sizes (by volume) of individuals in monitored populations ranged from 1 cm^3 to several 100 cm^3; and the 3-dimensional space occupied by the branches of physiologically confluent individuals ranged from < 1 cm up to 2 m in largest dimension (Wulff 1991).

Disease in *I. birotulata* (green surface and dark purple interior) was first evident as dulling of the surface, followed by loss of tissue from the purple-stained skeleton, which soon turned white. In *A. compressa* (crimson red throughout), disease first appeared as glossing of the surface, and the subsequent advance of a narrow band of brown was followed by exposure of the completely bared skeleton. In *Aplysina fulva* (ochre yellow), surfaces also became glossy, and progression of a narrow band of brown was followed by exposure of the golden skeletal fibers. Species-specific pathogens are the suspected etiologic agents, as disease could be induced in undiseased individuals by contact with a diseased portion of a conspecific, but the diseases could not be induced in heterospecific sponges; and naturally adherent pairs of sponges of the same species were sometimes both diseased, whereas those of different species were never both diseased (Wulff 1997, unpublished data).

Daily measurements of all dimensions of diseased areas were made to the nearest 1 mm, and records were kept of barrier development, sloughing, and breakage of diseased and dead portions. A total of 71 lesions were followed for from 2 to 4 days each. In most cases there was only one lesion on a sponge individual, but 12 individuals had more than one lesion.

Results

Disease progression in the field

For *I. birotulata* the mean rate of linear advance of diseased patches that could be measured on successive days was 0.42 cm day^{-1} (SE = 0.12; maximum was 1.2 cm day^{-1}; $n = 29$ lesions). For *A. compressa* the mean rate of advance was 0.64 cm day^{-1} (SE = 0.09; maximum was 1.1 cm day^{-1}; $n = 36$ lesions), and in *A. fulva*, the mean rate of advance was 1.3 cm day^{-1} (SE = 0.31; maximum was 2.2 cm day^{-1}; $n = 6$ lesions). Typical examples are illustrated in Fig. 1. Progression halted during monitoring in five of the diseased patches in *I. birotulata*. Within a few days after tissue had died,

Fig. 1 Representative examples of disease progression in three species of branching sponges (two different individuals for each species), drawn in the field **a** on the first day of monitoring and **b** 2 days later: *A.c. Amphimedon compressa*, *I.b. Iotrochota birotulata*, and *A.f. Aplysina fulva*. All examples are drawn to the same scale: branches of *A. fulva* are 1 cm in diameter

the *I. birotulata* skeletons began to crumble. No physical barriers were observed between diseased and healthy portions, and skeletal fibers appeared to deteriorate as a function of the number of days since they were exposed by loss of living cells. In *A. compressa* progression halted in eight diseased patches. In each case a barrier, possibly of spongin, developed between diseased and healthy tissue, and dead portions were sloughed as if the skeletal fibers were severed by the sponge.

The elapsed time between disease initiation and skeletal disintegration determines how frequently sponges must be monitored to accurately assess disease incidence. This window of time during which disease can be observed in a particular sponge individual can be estimated from the maximum lengths of dead segments between healthy branch portions (i.e., longer dead segments are not found because the skeleton has crumbled or sloughed), which varied little within each species. Dividing the length of a dead segment by twice the rate of disease progression estimates the number of days since the initial lesion in the center of the dead segment. For *I. birotulata*, the mean length of the five longest dead segments was 4.6 cm (SE = 0.09), and for *A. compressa*, the mean was 16.2 cm (SE = 0.71). Similar calculations for *A. fulva* were not possible because too few monitored individuals had lesions that were flanked by living tissue on each end. Based on the mean rate of disease advance for each species, mean time between the initial lesion and skeletal deterioration to the point of breakage was 5.5 days for *I. birotulata* and 13.3 days for *A. compressa*. These measurements were made during a period of calm weather, when branch breakage was solely the result of deterioration of the spongin binding the spicules together. The longer time period between evidence of disease and skeletal deterioration in *A. compressa* coincides with a significantly higher spongin to spicule ratio in the skeletal fibers of *A. compressa* relative to *I. birotulata* (Wulff 1995).
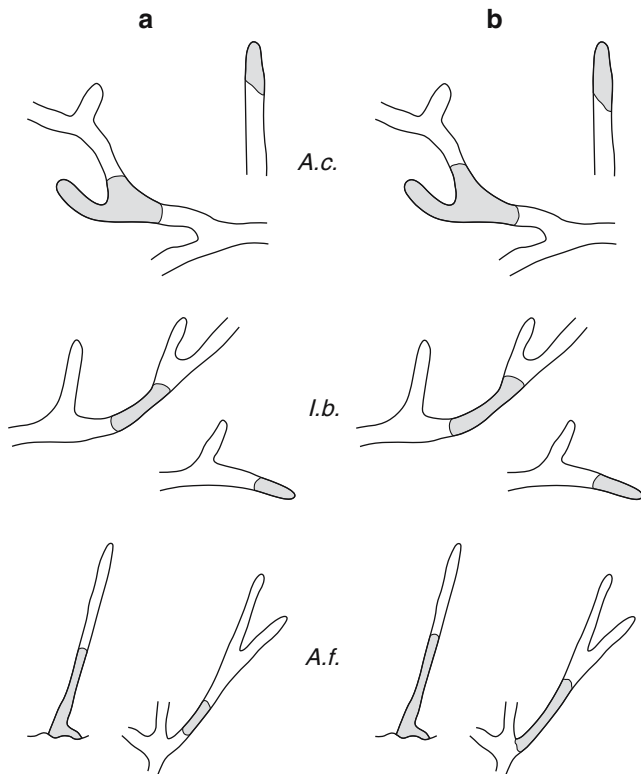
Model of disease progression

A simple graphical model of progression of infection within individual sponges focused on two basic growth forms: massive and branching [corresponding to Jackson's (1979) mounds and trees]. The assumption underlying the model is that degeneration spreads from an initial lesion in all possible directions.

Expansion of diseased portions

If diseased areas expand in all directions from the initial lesion, the volume of diseased tissue at a given time can be described as a simple geometric solid. Thus, if lesions expand at a rate of 1 linear unit per day, the total volume of diseased tissue on day D for a massive sponge with the initial lesion on the surface is a hemisphere, $2/3 \pi D^3$; and if the infection begins inside the sponge, it

is a sphere, $4/3 \, \pi D^3$. For a branching sponge, diseased portions expand as a cylinder with constant radius, r, so that the volume diseased on day D is $D\pi r^2$ if progression along the branch is in one direction, and twice that if progression is in both directions. To graph changes in diseased volume over the first 10 days using realistic numbers, a radius for the branching sponge was chosen to be 1 cm, in the normal range for common Caribbean species. The rate of linear advance was chosen to be 0.5 cm per day, based on the data from field observations of branching species.

Volume of diseased tissue is predicted to expand rapidly for massive sponges relative to branching sponges (Fig. 2). After only 5 days, the volume of diseased tissue for a massive sponge in this example is 4.2 times that of diseased volume for a branching sponge. After 10 days, the volume of diseased tissue for a massive sponge is 16.7 times that for a branching sponge. These comparisons are for the simplest case, in which disease has not progressed into additional branches. In large sponges with side branches, the rate of expansion of the diseased portion will increase incrementally as disease spreads to each new branch, and decrease whenever a branch becomes entirely diseased, halting further expansion in that direction. The amount and timing of increases and decreases in expansion rate will depend on the branching pattern and the location of the initial lesion relative to the branching pattern.

Interface between diseased and healthy tissue

Recovery depends on halting the progress of disease by deployment of a defense system or construction of a barrier at the interface between diseased and healthy tissue. The rate of increase of the area of this interface is growth-form dependent. For massive species, the area of the interface is the surface of the hemisphere or sphere centered on the initial lesion, $2\pi r^2$ or $4r^2$, depending on if the initial lesion is at the surface or inside the sponge, with r increasing at the rate of progression. For branching species, r (branch radius) is a constant, and the interface area is the cross-sectional area of a branch, πr^2, or twice that if the infection is traveling in both directions.

Just 5 days into the infection, the area of interface between diseased and healthy tissue in a massive sponge is 12.5 times that of a branching sponge, and after 10 days, the area of interface in a massive sponge is 50 times that of the branching sponge. In the branching sponge this area remains the same as disease progresses (Fig. 3), always the cross-sectional area of the branches, or twice that if the initial lesion is mid-branch. In large sponges with side branches, the area of interface will increase and decrease in increments of branch cross-sectional areas, as disease progresses into additional branches and progresses through them to their tips.
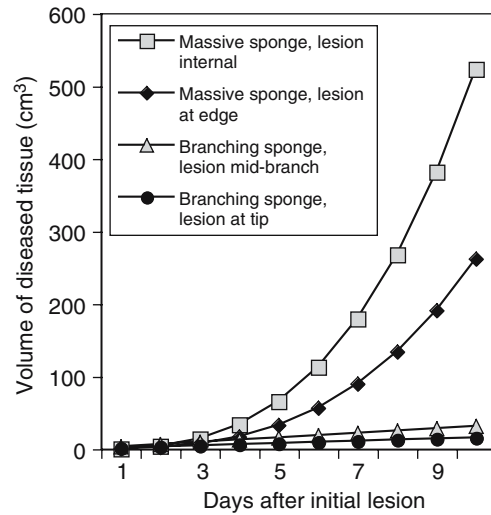

Fig. 2 Rates of increase of the volume of infected tissue for massive and branching sponges. Lesions are assumed to expand in all directions at the rate of 0.5 linear unit per day. Branch radius for the branching sponge was chosen to be 1 cm, and there are no side branches. The maximum volumes of diseased tissue for each day are for lesions beginning in the center of the sponge, and the minimum volumes are for lesions beginning at the edge of the sponge

Disease progression through the same tissue volume in different shapes

To distinguish effects of size and growth form, the results of the model can be illustrated in terms of the number of days to total mortality for sponges of different growth forms, but identical volume. To make comparisons using realistic numbers, the volume chosen was 500 cm^3, the size of a medium bath sponge. For a
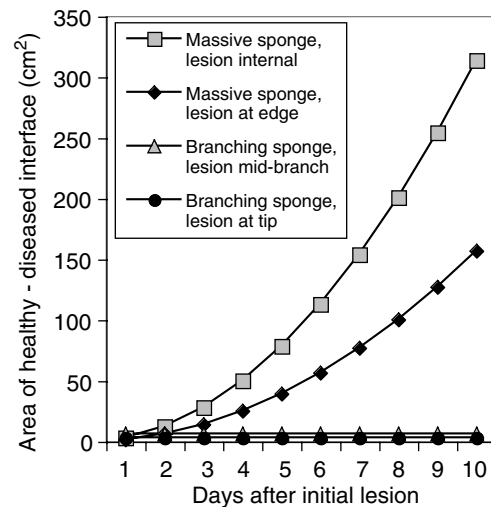

Fig. 3 Rates of increase of the area of the interface between healthy and diseased tissue. Lesions are assumed to expand in all directions at the rate of 0.5 cm per day. The maximum area for each day is for lesions that began inside a sponge, and the minimum area for each day is for lesions that began on the edge of the sponge

volume of 500 cm^3, a massive sponge shaped as a hemisphere has a radius of 6.2 cm, and a branching sponge of radius 1 cm has a total branch length of 159.1 cm. Linear progression of disease through the tissue was set at 0.5 cm day^{-1}, based on the rates measured in the field.

The model predicts substantial differences in the amount of time it would take for a pathogen to spread throughout sponges of different growth forms, but the same volume. Depending on whether the initial lesion is at the edge or center, the time to spread throughout the individual massive sponge is 12–25 days. The time to spread throughout an individual branching sponge depends on where the initial lesion is relative to the branching pattern. In the example pictured, it would take 72–132 days (depending on location of the initial lesion) for disease to spread throughout the sponge from a single initial lesion; six times as long as for the massive sponge (Fig. 4). For the massive sponge to have as many days in which to marshal defenses as the branching sponge, it would have to have a diameter of 74 cm (i.e., exceptionally large), corresponding to a volume 221 times that of the branching sponge.

Discussion

Studies of disease in clonal organisms must consider the possibility of recovery and regeneration after substantial partial mortality. This model teases out one aspect of recovery, the effect of growth form, which is independent of the particular host or pathogen. Growth form may influence recovery by determining the number of dimensions in which infection can radiate simultaneously. For sponges, the model predicts that the volume of infected tissue and the area of the interface between infected and uninfected tissue can increase much more rapidly for massive species than for branching species, perhaps outstripping the ability of a sponge to deploy deterrents.
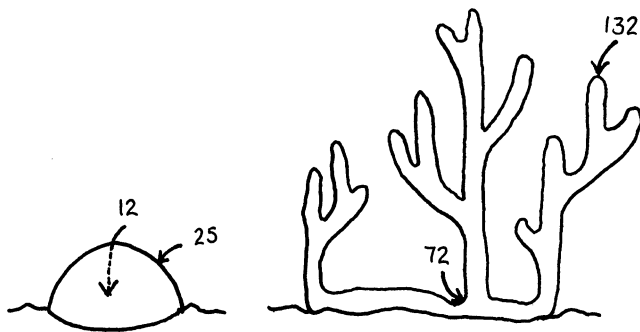

Fig. 4 The number of days for pathogen infections to spread throughout sponges of the same volume but of different growth forms. The tissue volume chosen for both sponges was 500 cm^3. The branching sponge has a radius 1 cm and total branch length of 159.1 cm. The massive sponge has a radius of 6.2 cm. The rate of progression of the disease through the tissue was set at 0.5 cm per day. Maximum and minimum number of days depend on whether the initial lesion is in the center or edge of the sponge individual

Assumptions underlying, and applications of, the model

The primary assumption underlying this simple model is the equal rate of progression of disease in all possible directions within a sponge. This assumption was based on observations of three branching species reported on here, of progressive degeneration from the base upwards in a vase-shaped species (Reiswig 1973), and of steadily enlarging lesions in an encrusting species (Hummel et al. 1988), a finger-shaped species (Shield and Witman 1993), a flabellate species (J. Cervino, personal communication), and an open basket-shaped sponge (Williams and Bunkley-Williams 2000; I. Nagelkerken, photo and observations in Reef Encounter 28:14–15). By cutting open massive commercial sponges in the midst of an episode of heavy mortality due to disease in 1938–1939, Smith (1941) was able to determine that infections first took hold deep inside the sponges, and then spread to reach the surface.

The rate of progression of disease through a sponge is likely to differ among sponge species and pathogens, but differences among taxa would have to be large to overwhelm the predicted differences between branching and massive sponges of 4 fold in volume of diseased tissue after only 5 days, and 17 fold in 10 days. The three branching species in which disease progression was followed are each in a different order of Demospongiae, but differed in mean rates of progression by a factor of only 2.6 (i.e., only a 2.6 fold difference in diseased volume after 10 days). Modifications of the model can readily accommodate variations, such as multiple points of initial infection within the same sponge, variable rates of disease progression in different directions, different branching patterns, or slowed progression rates just before halting. The model is based on simple linear dynamics, in the absence of information to the contrary, but more complex dynamics would give the same general result, i.e., that disease progression and enlargement of the area of interface are much more rapid within a massive sponge than within a branching sponge.

Halting disease progression may involve construction of a barrier between diseased and healthy portions in at least some sponges. The few survivors of the commercial sponge epidemic walled off the infected portion with "a skin or callous" (Smith 1941). *Geodia papyracea* also walls off diseased zones with a spongin barrier and sloughs decaying tissue (Rützler 1988). Other species, e.g., *Amphimedon compressa*, *Spongia* cf. *obscura*, and *Agelas* spp. erect barriers, possibly of spongin, between diseased and healthy tissue and then sever connections with the necrotic portion, allowing it to slough off (personal observation). In the Mediterranean sponge *Petrosia ficiformis*, recovery is facilitated by bundles of spicules that separate damaged from healthy portions (Cerrano et al. 2001). The variety of ways in which sponges inhibit further spread of disease raises questions relating to the possibility of taxonomic patterns in efficiency of halting disease within an individual. For example, species in keratose orders have been particularly prominent in

reports of sponges lost to disease in the Mediterranean, tropical Pacific, and Caribbean (e.g., Smith 1941; Pansini and Pronzato 1990; Vacelet et al. 1994; Pronzato et al. 1999; Butler et al. 1995; Cerrano et al. 2000; J. Cervino, personal communication; Perez et al. 2000; Wulff 2006). It has been suggested that this reflects their greater vulnerability to disease in warmer water (Vicente 1989), but it is also possible that skeletal or other traits of species in these orders make them less efficient at recovering from disease.

Ultimately, recovery depends on a sponge marshalling an effective defense before it is overwhelmed by expansion of the interface between diseased and healthy tissue, and before all the tissue is diseased. Results of regeneration experiments (review in Henry and Hart 2005) show that recovery requires resources. Boury-Esnault and Doumenc (1978) demonstrated that cells with glycogen reserves migrate to, and concentrate at, wounded sites, indicating integrated response at the level of the sponge individual. Thwarting disease progression may consume additional resources in cases in which barriers are erected at the interface area. Time may be critical, as small (1 cm by 1 cm) experimental lesions in three species of large-bodied Caribbean coral reef sponges required 5.2–27.6 days to complete initial scar formation (Hoppe 1988). With growth forms differing in the expansion of the interface area of 12.5 fold in just the first 5 days after infection (and 50 fold after 10 days), even a small effect could be magnified to a physiologically meaningful point.

Observations of commercially valuable sponges that were completely necrotic within a week of first signs of disease (Smith 1941) are compatible with the estimate from the model of 12 days until demise for a 6.2 cm radius massive sponge with an internal initial lesion. Also compatible with model predictions are observations of disease halting in branching sponges and of disproportionately frequent recovery of very large individuals of massive species; but data on mortality or recovery of diseased sponges are scarce. The example in Fig. 4, comparing disease progression in massive versus branching sponges of the same total volume, illustrates how many fewer days a massive sponge may have in which to mount an adequate defense before all tissue is infected. A massive sponge individual may have to be very large in order to have any living portions remaining from which to regenerate, after defenses are organized.

Adapting this model to predict growth-form dependent recovery in other groups of clonal animals would have to take into account greater internal differentiation and integration (including directional transport systems) that might influence spread of pathogens within individuals. Taxa may differ in the degree to which they are able to deploy physical barriers or cellular level deterrents to disease progression (e.g., Sutherland et al. 2004 for cnidarian examples). Size-dependence of disease resistance and recovery has been demonstrated in gorgonians (e.g., Dube et al. 2002; Cerrano et al. 2005). In the Mediterranean, Cerrano et al. (2005) demonstrated

less damage and better regeneration in smaller sea fans during a mass mortality. Intricate branching patterns of sea fans and other gorgonians add another level of complexity. For scleractinian corals overall growth form influences ability to capture light, compete, resist breakage, and shed sediment. But with respect to progression of, and responses to, disease, it may be useful to consider living portions of all corals to be essentially encrusting (i.e., 2-dimensional), with the interface between diseased and healthy portions best described as a perimeter instead of an area.

The real world—monitoring community dynamics

The possibility that sponge growth form can influence mortality due to disease is addressed by patterns of species loss from a censused plot. Community dynamics of sponges were followed for 14 years with five censuses of a small plot (initially 1,395 sponge individuals in 39 species, in 16 m^2) on the same San Blas, Panama reef where disease progression was followed (other aspects of this study are presented inWulff 2001, 2006). Of the original 39 species, 16 were in the massive or branching growth form categories considered in the model. Eight of these species (i.e., 50%) had vanished from the plot by the end of the 14 years. Losses were disproportionately great for massive species (80% lost). No branching species were lost (Fig. 5a).

What eliminated species from this plot is not known for certain, but predators and abiotic physical disturbance were not likely responsible, in general, as many of the lost species are especially resistant to these mortality sources (e.g., Wulff 1994, 1995). Disease is the most likely agent. Disease was noted in 119 individuals, representing 11 of the 39 species, at some point during the censusing. Although this may not seem like a high incidence, mean rates of disease advance of 0.42, 0.64, and 1.3 cm per day through common branching sponges demonstrate how quickly live tissue can be lost. Crumbling of skeletons within 5.5–13.3 days after the initial lesion emphasize the ephemeral nature of evidence that a sponge had existed. If diseased individuals disappear within 10 weeks (published observations range from 1 to 9 weeks in Smith 1941; Reiswig 1973; I. Nagelkerken, photo and observations in Reef Encounter 28:14–15), and observations of disease were confined to censusing times (i.e., five 3-week periods over 14 years), the great majority of the actual disease incidence could not have been observed. The possibility of losses to disease that are essentially invisible, unless monitoring intervals are impractically short (i.e., shorter than the time between infection and deterioration of a sponge individual), prompts development of models and other ways to gain insight on population and community effects of disease in coral reef sponges.

If disease was an important source of mortality in the plot, the model may help to resolve an apparent paradox in the observations: disease prevalence, as recorded
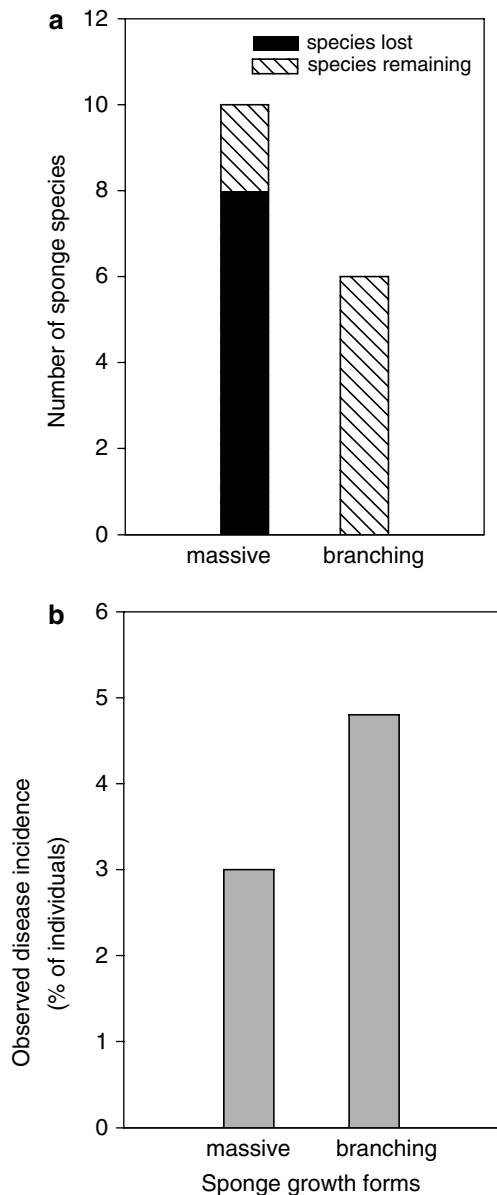
Fig. 5 a The number of sponge species of massive versus branching growth forms that vanished from 16 m² on a shallow reef (San Blas, Panama) that were completely censused five times in 14 years. **b** The percent of individual sponges of massive versus branching growth forms that were observed to be diseased during the five censuses

throughout a branching sponge of 500 cm³, but only 12–25 days in a massive sponge of the same volume, Fig. 4).

For other sponge growth forms, very small body size (often less than a few cubic centimeter for encrusting species, e.g., Wulff 2001) may be more important than growth form per se in determining how many days an individual has in which to mount a defense before being overwhelmed by disease. Encrusting sponge species are rarely reported to be diseased, but this could reflect the small amount of time between development of the initial lesion and complete disintegration (personal observation of a couple of days in tropical sponges, and 3–5 days in a temperate encrusting sponge, reported by Hummel et al. 1988). In sponges of larger volumes, growth form may influence whether the result of pathogen infection is death or recovery. The amount of time a sponge has in which to mount a defense may determine both (1) how likely disease will be observed in that individual, and also (2) how likely a particular individual is to recover. Growth form—in conjunction with body size—could therefore directly influence perceptions of the importance of disease in ways that must be considered in interpretation of monitoring data.

The importance of disease in determining community dynamics of coral reef sponges cannot be known unless monitoring is frequent enough to catch disease in progress before diseased sponges disappear completely; and in most cases such short intervals between surveys are impractical. Disease prevalence data, collected in the course of monitoring at intervals longer than a few months, is likely to be misleading if used alone to indicate importance of disease in sponges. However species-specific, or growth form-specific, conversion factors based on measured rates of advance of pathogens through individual sponges, or recovery rates of sponges that have been diseased, can be applied to observed disease prevalence to estimate actual influence of disease on population and community dynamics. Results from this model caution that massive sponge species may succumb especially rapidly, and may therefore require special attention in monitoring programs, perhaps in the form of rapid surveys for disease at short time intervals.

Focus in field monitoring of sessile coral reef invertebrates has been largely on disease prevalence, which can be readily measured as part of a standard monitoring protocol. Net influence of disease on community dynamics, however, reflects the balance between initiation and progression rates vs. recovery rate in each of the species. If growth form and body size affect this balance, observed disease incidence at any particular moment may be disproportionately high in the very organisms that are most likely to recover.

during the five censuses, was highest in branching sponges (Fig. 5b), but these same species increased their relative representation in the plot during the 14 years; while eight of the ten massive species were lost, and fewer were observed to be diseased (Fig. 5b). The model predicts this pattern: individual massive sponges are more likely to succumb to disease, whereas individual branching sponges succumb more slowly and are more likely to survive. This provides more opportunities for observation of disease in progress in branching than in massive species (e.g., 72–132 days for disease to progress

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