**Abstract** Despite increased efforts in coral-reef monitoring and coral-disease research, reports on rapid recovery of diseased corals are rare. Here, I report fast recovery of *Porites cylindrica* colonies from live tissue loss, a reported and yet unnamed disease, at Shiraho, Ishigaki Island, Japan. Loss of live tissue and exposure of skeleton in *P. cylindrica* were first observed in August and September 2007. At the end of January 2008, recovery from the tissue loss was quantified. Mean proportion (± SD) of affected *P. cylindrica* branches decreased from 0.52±0.16 in September 2007 to 0.08±0.06 in January 2008. The majority of branches in the September sample had multiple, especially small, lesions; whereas in January, branches with multiple lesions were absent and most branches had no lesions. These results indicate that, by the end of January 2008, *P. cylindrica* colonies in the study area had almost fully recovered from the summer tissue loss by regrowth of live tissue over the denuded skeletons. Except in cases of coral bleaching, the rapid recovery from a disease observed in this study has not previously been reported. The observed quick recovery suggests that some coral-disease outbreaks may have been overlooked in the past and, without proper monitoring, will be overlooked in the future as well. Effects of overlooked diseases may accumulate and can have long-lasting impacts on reef communities, suggesting a need for frequent monitoring.

**Keywords** recovery, coral disease, *Porites cylindrica*, tissue loss, high temperature

**Introduction**

There is an increasing consensus that the number of incidents of coral diseases has multiplied rapidly during the last few decades (Sutherland et al. 2004; Ward and Lafferty 2004), and to date more than thirty disease names have been described (Lesser et al. 2007). The proposed primary driving force of disease increase is high sea water temperature (Harvell et al. 2002; Lesser et al. 2007). High water temperature stresses hosts and makes them more susceptible to infection while increasing pathogen virulence and growth (Rosenberg and Ben-Haim 2002; Lesser et al. 2007). In fact, many coral-disease outbreaks are associated with higher-than-normal sea water temperatures, and they often accompany or follow coral bleaching (Harvell et al. 2001; Jones et al. 2004; Lesser et al. 2007).

Shiraho Reef is a well-developed fringing reef located along the southeast coast of Ishigaki Island, Japan (Hasegawa and Yamano 2004). It is about 850 m in width and 1.5 km in length (Harii and Kayanne 2003). During low
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Along the inshore edge of the *Heliopora coerulea* aggregation, the *Porites* wall was interrupted, in some places, by *H. coerulea* or by non-live substrata, but individual colonies were indistinguishable.

Recovery of *P. cylindrica* from the disease, i.e., regrowth of live tissue over the bare skeletal areas caused by the disease, was accessed in two ways. First, the proportion of affected *P. cylindrica* branches was estimated by linear point intercept method (Nadon and Stirling 2006) on September 6, 2007, and again on January 26, 2008 (see below for the definition of affected branches). Six 10-m transects, each marked at 20-cm-interval sampling points, were randomly placed along a 150-m-long portion of the wall. In each transect, I counted the number of times a *P. cylindrica* branch (including a dead branch that was entirely denuded of live tissue, see below) was found under a sampling point and the number of times an affected *P. cylindrica* branch was found under a sampling point. By dividing the latter by the former, I obtained the proportion of affected *P. cylindrica* branches.

In September sampling, only *P. cylindrica* branches having the characteristic symptoms of the disease were counted as ‘affected.’ Dead branches, entirely denuded of live tissue, were not scored as ‘affected’ because causes of their death were unknown. The characteristic symptoms of the disease became unrecognizable by the end of November 2007 and remained so in January 2008 (pers. obs.). Denuded skeletal patches were present on many branches, but they were surrounded by a growing-lip of whitish, half-transparent tissue. Therefore, in January, 2008, *P. cylindrica* branches with any tissue loss, including dead branches with no live tissue, were classified as ‘affected.’ This necessary treatment meant that dead branches were regarded as *Porites* branches rather than non-live substrata. Therefore, in order to have the same measure in the denominator of the proportion calculation for the two sampling periods, the number of dead branches was included in the count of the number of *P. cylindrica* branches. In short, proportion calculated for each transect in September was proportion of live, disease-affected branches in all *P. cylindrica* branches, dead or alive; whereas in January, it was proportion of branches with any tissue loss in all *P. cylindrica* branches, dead or alive. Proportion data were arcsine-transformed. Because cor-

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**Materials and methods**

The study was conducted at the southern end of Shiraho Reef, where the disease was originally observed. At this site, *Porites cylindrica* colonies formed an almost-continuous, vertical coral wall (about 1.5–2.0 m high) and the reef crest is exposed in the air and separates the reef-flat water from the outer ocean (Harii and Kayanne 2003). Loss of live tissue and exposure of skeleton were originally observed in *Porites cylindrica* Dana 1846 colonies around the largest *Heliopora coerulea* De Blainville 1830 aggregation on the Shiraho back reef during the 2007-summer bleaching event (Asoh 2008). The disease is characterized by circular or irregularly shaped patches of bare skeleton surrounded by a narrow, opaque, white line of tissue (Fig. 1a, b). Tissue margins differ from the advancing edge of growing tissue, which is whitish and half-transparent, and are peeling off of the underlying skeleton around large skeletal patches. None of these symptoms were found in adjacent *H. coerulea* colonies.

Here I report recovery of *P. cylindrica* colonies from the 2007-summer tissue loss, which was quantified in January 2008.

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*Fig. 1* *Porites cylindrica* branches in September 2007 and in January 2008. **a**: representative branches in September, **b**: close-up view of some branches in September, **c**: representative branches in January, and **d**: branches with growing lips in January.
relation between *Porites* c*ylindrica* cover (i.e., number of times a live or dead *P. cylindrica* branch was found under a sampling point / total number of sampling points) and proportion of affected branches was suspected, transformed data were analyzed by ANCOVA (analysis of covariance) with *P. cylindrica* cover as covariate.

Secondly, in order to estimate changes in the number and size of affected areas (hereafter referred to as ‘lesions’) with time, pictures of quasi-randomly chosen branches were taken on September 7, 2007, and again on January 26, 2008. Fifty sampling points were drawn randomly from 150 points, representing 1-m-interval points along a line placed in the 150-m long *Porites* wall, and a branch just below a sampling point was photographed. When there was no *Porites* branch under a given sampling point, the sampling point was skipped. In all, 34 branches were photographed in September, 2007, and 39 branches in January, 2008. Each photographed branch was classified into one of the following categories: with no lesion, with a single small lesion, with only small but multiple lesions, with small and large lesions, and only with large lesions. Small lesions were lesions having a width less than the branch diameter estimated at the midpoint along the branch axis, and large lesions were ones having a width reaching the branch diameter. Branches with large lesions were not divided into two sub-categories by the number of lesions because doing so produced cells with expected counts of less than one in a Chi-square analysis. Definition of ‘affected’ follows that defined in the previous paragraph.

**Results and discussion**

Mean proportion (±SD) of affected *P. cylindrica* branches was 0.52±0.16 on September 6, 2007, and was 0.08±0.06 on January 26, 2008. There was a significant difference in the proportion of affected branches between the two months, after adjusting for *Porites* cover (*F*₁,ₙ = 40.45, *P* < 0.0001; Fig. 2). Affected branches in January sampling included those with tissue loss from any cause, and hence the proportion of affected branches in this sample was overestimated. In contrast, affected branches in September sampling included only those with the characteristic symptoms. Denuded branches were not included even though tissue loss in some of these branches might have been caused by the same agent that caused the tissue loss in question. Despite the probable underestimation of affected branches in September, the amount of decrease in January was quite large. The very low proportion of affected branches at the end of January indicates that, by this time, *P. cylindrica* colonies in the study area had almost fully recovered from the summer tissue loss by re-growth of live tissue over the denuded skeletons.

Recovery from the summer tissue loss was also evident in the number and size of lesions on quasi-randomly chosen branches (Table 1). The distribution of lesions of different categories differed significantly between the two samples (χ²=65.9, df=5, *P* < 0.0001). The majority of branches in the September sample had multiple, especially small, lesions (Fig. 1a, b). Branches with these lesions were absent in January, and most branches had no lesions (Fig. 1c, d). It takes about 1.5 months for exposed skeletal areas of broken *P. cylindrica* branches of about 1-cm diameter to be covered with live tissue (Yoshikwa, Asoh, and Maekawa unpublished data). Because recovery from the summer tissue loss is likely to have started by the end of November, there was enough time for the skeletal areas denuded in summer to be covered by growing tissue by the end of January.

In theory, a low proportion of affected branches similar to that observed in January could be obtained in the
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following situation: branches affected during summer died off, were buried in sand or silt, and were scored as non-live substrata, and only unaffected branches survived and were examined. This, however, does not appear to be the case. *Porites cylindrica* constituted a 1.5–2 m high vertical wall at the study site, and hence the probability of dead branches being buried in sand or silts should be low. Thus, if there had been no recovery, dead branches should have remained and would have been scored as dead branches. That being the case, the proportion of affected branches in the January sample should have been close to the September value.

The initiation and cessation of the tissue loss in *P. cylindrica* are likely to be related to changes in sea-water temperature. The tissue loss was observed in summer, when other coral species in the reef bleached due to high sea-water temperature (Fig. 3; Maekawa and Suzuki 2007; Nadaoka et al. 2007), and the progression of tissue loss had halted in fall and winter. Many coral-disease outbreaks are correlated with high summer temperature and disease cessations with low winter temperature (reviewed in Sutherland et al. 2004; Lesser et al. 2007). Correlation between disease outbreaks and high water temperature is thought to result from increased host susceptibility, increased pathogen virulence, or increased vector transmission (Rosenberg and Ben-Haim 2002); however, the causal link between temperature and a disease outbreak is not fully understood and much less is known about the mechanisms by which disease progression is halted. Detailed experimental and microbiological studies are needed to determine how initiation, progression, and cessation of the observed tissue loss are causally linked to changes in sea-water temperature.

Except in cases of coral bleaching, the fast recovery from a disease observed in this study has not previously been reported. The lack of reports of quick recovery may partly be due to a reporting bias toward severe disease outbreaks, such as ones resulting in an apparent ecological phase-shift of communities. Whether regeneration of live tissue over denuded skeletons should occur depends on a balance between energy required and energy available for tissue regeneration, as well as on various environmental factors such as temperature, sedimentation, and settlement of colonizing organisms (Kramarsky-Winter and Loya 2000; Hall 2001; Croquer et al. 2002). The amount of required energy, in turn, is influenced by lesion size, and the amount of available energy is influenced by colony size and physiological states of remaining live tissue (Oren et al. 1997, 2001; Kramarsky-Winter and Loya 2000; Lirman 2000; Fine et al. 2002). If reports of disease

### Table 1

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Fig. 3 Daily average water temperature before and during the study (solid line). Each tick mark on the horizontal axis indicates the first day of the month. Solid triangles indicate days when quantitative data were taken (September 6–7, 2007 and January 26, 2008). An open triangle indicates the day when the disappearance of disease-typical symptoms was confirmed by casual observation (November 26, 2007). During a period between two open diamonds (August 10–September 21, 2007), the author casually checked the reef daily and confirmed the presence of disease-typical symptoms. Daily average water temperature from May 1, 2008 to January 31, 2009 is also given for comparison (dotted line).
outbreaks are strongly biased toward severe ones, in which effects are so catastrophic that there is little live tissue remaining for tissue regeneration, the number of reports of quick recovery would be few. Given the temperature-dependence of many coral diseases, it is likely that corals undergo cycles of disease infection and recovery. Quick recovery from a disease might be more widespread than has been reported.

The observed quick recovery of *P. cylindrica* from tissue loss suggests the possibility that some coral-disease outbreaks may have been overlooked in the past and, without proper monitoring, will be overlooked in the future as well. Even when there is quick recovery, disease infections certainly carry costs of reduced growth and reproduction (Meesters et al. 1994; Kramarsky-Winter and Loya 2000; Oren et al. 2001). Effects of overlooked diseases may accumulate and can have long-lasting impacts on reef communities, suggesting a need for frequent monitoring.

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