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# Changes in a coral population on reefs of the northern Florida Keys following a coral disease epizootic

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ABSTRACT: Populations of the coral *Dichocoenia stokesi* were quantitatively monitored on reefs of the middle and northern Florida Keys during and after a 1995 epizootic of the coral disease white plague type II. Three large-scale surveys were conducted in the falls of 1995, 1998, and 2002 at selected sites throughout the Florida Keys totaling 8478, 6280, and 8792 $m^2$ respectively. Between 1995 and 2002, the average number of *D. stokesi* colonies per 314 $m^2$ site decreased from 44.3 to 11.2, a decline of almost 75%. We found no evidence of coral recruitment in the 7 yr following the epizootic, and have found a continuing pattern of coral population decline. The colony size–frequency distribution pattern on these reefs changed over this time period as well, with the *D. stokesi* population exhibiting a trend to domination by large colonies, suggesting that the remaining population, while growing, is no longer reproducing. The shift in population to dominance by large colonies is typical of coral populations on degraded reefs. Here we report the results of a quantitative field study on the Florida reefs and compare these data with a similar study on reefs of Lee Stocking Island, Bahamas, that have not experienced a white plague type II epizootic.

KEY WORDS: Coral reefs · Coral disease · Reef degradation · White plague type II · *Dichocoenia stokesi* · Population structure



## INTRODUCTION

It is generally accepted that coral populations are exhibiting loss and degradation on a world-wide basis (Hughes et al. 2003, Pandolfi et al. 2003). Widespread occurrences of total coral colony mortality, partial mortality, population decline, and apparent decreases in coral recruitment have been reported on many reefs (Pandolfi et al. 2003). These problems are particularly prominent on reefs of the wider Caribbean, and it has been estimated that coral cover on Caribbean coral reefs has declined by 80% over the past 30 yr (Gardner et al. 2003).

Recently, the case has been made that the most important factors contributing to coral reef decline are phase shifts from coral-dominated to algal-dominated communities (Done 1992) and overfishing (Jackson et al. 2001). Coral diseases are often acknowledged as contributors to coral decline (see Pandolfi et al. 2003); however, for the most part, coral diseases are seen as small, short-term contributing factors. Yet, in 1 case a coral disease (white band disease) is believed to have instigated a phase shift in a coral population from framework Caribbean acroporids to small, non-framework *Agaricia* spp. (Aronson & Precht 1997). This occurrence suggests that coral diseases play an important, as opposed to a minor, role in reef degradation. It has, in fact, been pointed out that coral diseases that target different species can be considered to be a new form of coral reef degradation by affecting species richness and diversity (Done 1992).

In the past 2 decades, numerous reports have been published documenting the occurrence of coral disease outbreaks in both scleractinian (Bak & Criens 1982, Gladfelter 1982, Rützler et al. 1983, Edmunds 1991, Bruckner & Bruckner 1997, Bruckner et al. 1997,

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Richardson et al. 1998a,b, Nugues 2002, Croquer et al. 2003, Borger 2003) and gorgonian (Nagelkerken et al. 1997a,b) corals. Each of these studies concludes that coral disease is contributing to the observed degradation of coral reefs. Many of these reports include quantitative data describing disease incidence, prevalence, coral species affected, coral mortality rates, and coral tissue loss. Coral diseases are often frequently included in quantitative short- and long-term coral monitoring programs. Reports from such programs indicate that there has been an overall increase in the number of coral diseases, the number of cases of disease (incidence), and the number of coral species affected (Green & Bruckner 2000, Porter et al. 2001, Sutherland et al. 2004, Weil 2004). Together these studies countermand a common hypothesis that disease incidence has remained constant over time and that the reported increase in disease incidence is due to increased sampling intensity. Despite the above, little is known about the long-term effects of coral disease on coral community and population structure.

In 1995, a massive epizootic of the coral disease white plague type II (WPII) occurred on reefs of the middle and northern Florida Keys (Richardson et al. 1998a). Diseased colonies (Fig. 1) exhibited a sharp line between apparently healthy, pigmented coral tissue and freshly exposed coral skeleton (Richardson et al. 1998b). During the outbreak, 17 species of scleractinian corals were affected, the most susceptible of which was the elliptical star coral *Dichocoenia stokesi.*

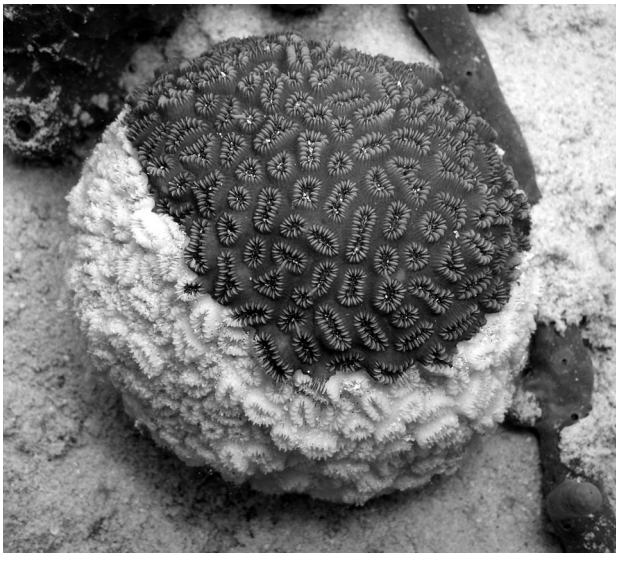

Fig. 1. *Dichocoenia stokesi. D. stokesi* colony infected with white plague type II. The disease is characterized by a sharp line between apparently healthy tissue (top) and freshly exposed coral skeleton. Tissue loss typically progresses upward from the base of colonies at rates of 2 cm $d^{-1}$

The primary pathogen of WPII was isolated during the 1995 outbreak (Richardson et al. 1998a), was determined to be a novel genus and species of the Alphaproteobacteria, and was given the name *Aurantimonas coralicida* (Denner et al. 2003). To date virulence factors, mode of disease transmission, and the mechanism responsible for coral tissue death are unknown.

The first case of WPII was observed in June of 1995. A quantitative study of the disease outbreak was initiated in August and conducted for the remainder of the period of disease activity, which lasted until mid-October (Richardson et al. 1998a). During this time period, assessments of the spatial distribution and dynamics of the disease were conducted to determine incidence and prevalence values, and mortality rate. It was found that during the last 10 wk of the approximately 19 wk long epizootic total colony mortality rates of up to 38% of the most susceptible species, *Dichocoenia stokesi*, occurred (Richardson et al. 1998a).

Since the 1990s, WPII epizootics have been reported throughout the wider Caribbean (Green & Bruckner 2000, Weil 2004). WPII is now considered to be one of the most serious of Caribbean scleractinian coral diseases. The potential recovery of the relatively slow-growing scleractinian populations susceptible to WPII after epizootics is not yet known, nor are the short-term and long-term effects of the disease on the population of the most susceptible species *Dichocoenia stokesi*.

To investigate the question of potential recovery versus non-recovery of *Dichocoenia stokesi* following the 1995 WPII epizootic, we conducted 2 large surveys in the falls of 1998 and 2002 on the same scale as was carried out for 1995. We report here the results of these surveys, and discuss our findings in terms of continued disease occurrence, coral population dynamics, and colony size–frequency distribution patterns within the population of *D. stokesi*. We also present, for comparative purposes, results of a parallel survey on coral reefs near Lee Stocking Island, Bahamas, conducted in 2002. No WPII epizootic has been observed or reported on these reefs.

## MATERIALS AND METHODS

Multiple surveys were conducted along 40 km of reef tract on reefs of the middle to northern Florida Keys during August and September of 1995, 1998, and 2002. Surveyed reefs (n = 11) were selected along the entire length of this part of the reef tract. Each survey was conducted using the radial transect method of Edmunds (1991), in which sites surveyed were 10 m in radius (314 $m^2$), and were examined in 2 m wide circular swaths centered around either a fixed mooring

buoy pin or a haphazardly selected location on the reef. Sites around mooring buoy pins were considered permanent sites, some of which were repeatedly surveyed in different years (see below). In 1995, surveys were conducted between August 7 and September 8, and consisted of 27 sites on 7 reefs (area = 8478 $m^2$), with the number of sites ranging from 1 to 9 per reef. In 1998, 20 sites on 6 reefs were surveyed August 3 through 6 (area = 6280 $m^2$; 2 to 5 sites per reef), and in 2002 a final survey was conducted between August 5 and 22 at 28 sites on 9 reefs (area = 8792 $m^2$; 2 to 4 sites per reef). Of the 11 reefs surveyed, 4 were surveyed in all 3 years, 3 in 2 of 3 years, and 4 in only 1 of the survey years. While efforts were made to resurvey the exact same sites, this was problematic because the numbering system of the mooring buoys was changed during this time. Additionally, some buoys were lost during storms and were redeployed at different locations on these reefs. In addition to the Florida Keys surveys, 39 sites on 9 reefs (area = 12 246 $m^2$; 3 to 12 sites per reef) were surveyed on reefs of Lee Stocking Island, Exuma Chain, Bahamas, from June 25 through July 27, 2002. The Lee Stocking Island site was included as a control site for comparison with the Florida site. Lee Stocking Island reefs are less impacted by proximity to human populations. Lee Stocking Island reefs exhibit similar coral communities to those on reefs of Florida, with both communities dominated by the framework building corals *Montastrea annularis*, *M. faveolata*, and *Siderastrea siderea* (Voss et al. 2004). Reefs of both Lee Stocking Island and Florida are predominately patch reefs with depths from <2 to 30 m. Since we had no baseline (pre-epizootic) data for the Florida reefs, we selected the Lee Stocking Island population for our control.

During each survey, each colony of *Dichocoenia stokesi* was counted and recorded as either healthy or diseased with WPII, and data were expressed both as incidence (number of diseased colonies per site) and prevalence (proportion, or percentage, of diseased colonies per site). Colonies of this species are characterized as hemispherical and discrete, and are easily recognizable as individuals. Colonies with old partial mortality, but no active disease, were recorded as healthy when they exhibited healthy tissue. In the 1998 and 2002 survey, colony size was also measured using underwater calipers or a ruler marked on a dive slate. In 1998, 2 dimensions (the widest diameter of healthy tissue area of a colony and the widest area at a 90° angle to the first measurement) were measured to the closest 1 cm. In 2002, in both Florida and Lee Stocking Island, only the widest diameter was recorded, and this diameter was measured only to the nearest 5 cm (5 cm size classes). When reporting these data as size–frequency distributions, data were not log-transformed because only 1 dimension was used (longest axis), which we deemed a valid size estimate since this coral species has the shape of an elliptical hemisphere. Since size classes in 2002 were recorded only to the nearest 5 cm, the 1998 data were divided into the 5 cm diameter size classes for comparison.

Colony size data were analyzed to determine both colony size–frequency distribution patterns and skewness, which refers to the pattern of asymmetry around the mean of the frequency distribution (Bak & Meesters 1998). Skewness ($g_1$) is either positive or negative, in that a positively skewed distribution has the majority of samples in the smaller size classes, and the tail is drawn out to the right. Similarly, a negatively skewed distribution has the majority of samples in the larger size classes, and the tail is drawn out to the left.

Variability within the *Dichocoenia stokesi* populations in terms of WPII (number of healthy and total colonies, disease incidence, and prevalence) as well as colony density (colonies per site) from each year in the Florida Keys were checked for normality (Kolmogorov-Smirnoff tests) and compared using a 1-way ANOVA and post hoc Bonferroni multiple comparison tests. Mean colony sizes were compared between (1) healthy and diseased colonies in 1998, and (2) healthy colonies in 1998 and 2002 using nonparametric Mann-Whitney *U*-tests. Size–frequency distributions were compared to normal distributions (single Kolmogorov-Smirnov test). All statistical analyses were conducted using SAS 9.0 (SAS Institute 2002).

## RESULTS

### Decline of *Dichocoenia stokesi*

As seen in Fig. 2 and Table 1, the population of *Dichocoenia stokesi* has not recovered in the years since the 1995 WPII epizootic and has, in fact, declined. Fig. 2 shows the mean number of *D. stokesi* colonies (total and apparently healthy) per site (each 314 $m^2$) as well as WPII disease incidence (number of diseased colonies per site) and prevalence (% diseased colonies within the population of *D. stokesi* per site). The mean number of colonies per site (n = 27, 20 and 28 sites for 1995, 1998, and 2002 respectively) decreased from 44.3 in 1995 to 11.2 in 2002 (Fig. 2, Table 1). The maximum number of colonies per site decreased from 95 to 43, and the minimum number of colonies per site decreased from 10 to 0 between 1995 and 1998, with an increase to 1 in 2002 (Table 1). WPII disease prevalence decreased dramatically between 1995 and 2002, from a mean of 16.7 to 0%. The zero value was specific for the *D. stokesi* population; during the 2002 survey, 11 colonies of 4 other coral species exhibited signs of

WPII indicating the continued presence of the disease on the reef.

ANOVA results indicated that the total number of *Dichocoenia stokesi* colonies (df = 2, $F = 21.8$, $p < 0.001$), healthy colonies (df = 2, $F = 16.4$, $p < 0.001$), WPII incidence (df = 2, $F = 28.4$, $p < 0.001$), and WPII prevalence (df = 2, $F = 37.4$, $p < 0.001$) were significantly different on Florida reefs between all 3 years surveyed. Post hoc Bonferroni multiple comparisons indicated that while 1995 differed in each of these measures when compared to both 1998 and 2002, 1998 and 2002 were not distinguishable (analysis not shown).

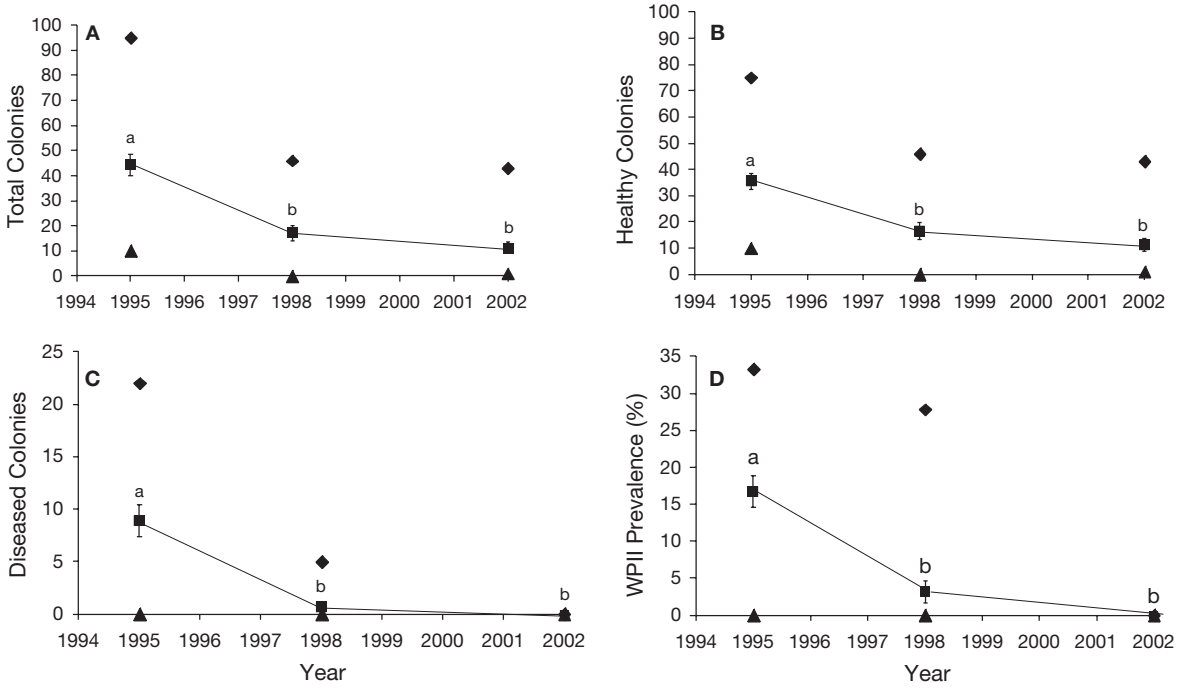


Fig. 2. *Dichocoenia stokesi.* Mean total (A), healthy (B), and diseased (WPII) colonies (C) for 1995, 1998, and 2002 per site surveyed. Prevalence (% diseased colonies) is shown in (D). ■: mean (±SE); ◆: maximum; ▲: minimum. In each graph, means with different lowercase letters indicate a significant difference with Bonferroni post hoc comparisons. Note different scales in (C) and (D) (to allow depiction of error bars)

Table 1. *Dichocoenia stokesi.* Summary of populations on Florida reefs during the 1995 WPII epizootic and in following years

| Distribution of *Dichocoenia stokesi* | Total colonies | No. of healthy colonies | No. of diseased colonies | Prevalence (% infected) |
|---|---|---|---|---|
| **1995** | | | | |
| All sites | 1196 | 956 | 240 | 20.1 |
| Minimum (per site) | 10 | 10 | 0 | 0 |
| Maximum (per site) | 95 | 75 | 22 | 33.3 |
| Mean per site (SE) | 44.3 (5.3) | 35.4 (4.0) | 8.9 (1.5) | 16.7 (2.1) |
| **1998** | | | | |
| All sites | 342 | 329 | 13 | 3.8 |
| Minimum (per site) | 0 | 0 | 0 | 0 |
| Maximum (per site) | 46 | 46 | 5 | 27.8 |
| Mean per site (SE) | 17.1 (3.1) | 16.5 (3.1) | 0.65 (0.24) | 3.2 (1.5) |
| **2002** | | | | |
| All sites | 314 | 314 | 0 | 0 |
| Minimum (per site) | 1 | 1 | 0 | 0 |
| Maximum (per site) | 43 | 43 | 0 | 0 |
| Mean per site (SE) | 11.2 (2.3) | 11.2 (2.3) | 0 (0) | 0 (0) |

Table 2. *Dichocoenia stokesi*. Mean size comparison of diseased versus healthy colonies (1998) and healthy colonies (1998 vs. 2002) on Florida reefs

| *Dichocoenia stokesi* colonies | n | Mean size (cm) | SD | SE | *U*-test | p-value |
|---|---|---|---|---|---|---|
| **1998** | | | | | | |
| Diseased | 13 | 10.8 | 5.336 | 1.48 | 1681 | 0.19 |
| Healthy | 329 | 9.51 | 4.498 | 0.248 | | |
| **1998/2002** | | | | | | |
| 1998 Healthy | 329 | 9.51 | 4.498 | 0.248 | 23064 | <0.001 |
| 2002 Healthy | 314 | 19.9 | 11.7 | 0.6605 | | |

### Colony size

In 1998, the mean sizes of diseased versus healthy *Dichocoenia stokesi* colonies (Table 2) were not significantly different (10.8 and 9.51 cm respectively, $p > 0.05$). When comparing the mean sizes of healthy colonies between 1998 and 2002, however, there was a statistically significant difference ($p < 0.001$), and the 2002 colonies were on average larger (9.51 cm in 1998 versus 19.9 cm in 2002, $p < 0.001$).

### Colony size–frequency distributions

Fig. 3 and Table 3 show the colony size–frequency distribution data for the 1998 and 2002 Florida surveys (Fig. 3A,B), and for the control population of *Dichocoenia stokesi* on reefs of Lee Stocking Island (Fig. 3C). Size data were not collected during the 1995 Florida survey. When comparing the size–frequency distributions of Florida's corals between 1998 and 2002 (3 and 7 yr after the epizootic respectively), it can be seen that there is a pronounced change in the size–frequency distribution pattern. The 1998 population (Fig. 3A) was dominated by small colonies that ranged from the 5 to 15 cm size classes. There were very few colonies in the 25 cm size class, and none in the larger classes. In 2002 (Fig. 3B), the average colony size of the population increased to 19.9 cm diameter (Table 3). In this year, there were many colonies larger than the maximum size class (25 cm) recorded in 1998, represented by colonies in every size class through 55 cm. Fig. 3A,B also reveals that there was an overall and pronounced decline in the relative and absolute numbers of small colonies. In 1998, there were 108 colonies in the 5 cm size class, which represents 32% of the population. This number declined to 51 in 2002, or 16% of the population. When summing the 2 smallest size classes, the percentage of colonies in these classes decreased from 78 to 32% between 1998 and 2002.

Fig. 3C and Table 3 present data collected in 2002 for the *Dichocoenia stokesi* population on reefs of Lee Stocking Island, Bahamas, which has no record of a WPII epizootic (although the disease was present in other species at a very low prevalence of 0.16% for all coral species during the survey period; Voss et al. 2004). The size–frequency distribution of *D. stokesi* at the Lee Stocking Island site shows a predominance of corals in the smallest 2 size classes, representing 61% of the coral population. The largest colonies were 50 cm in diameter, similar to the size of the largest colonies (55 cm class) in 2002 in Florida.

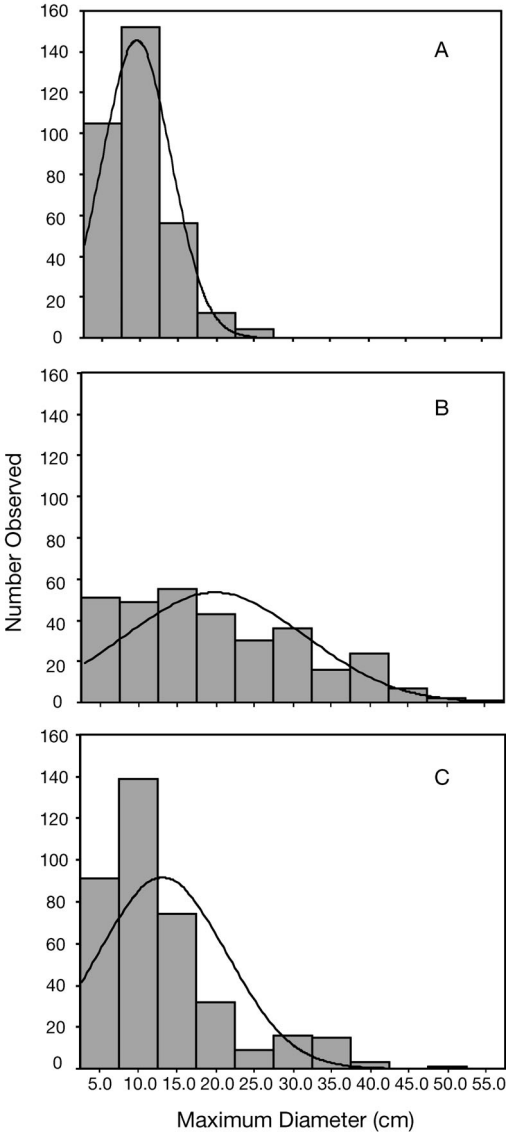


Fig. 3. *Dichocoenia stokesi*. Size–frequency distributions on reefs of the Florida Keys in 1998 (A) and 2002 (B), and on Lee Stocking Island in 2002 (C). Colonies were averaged to the nearest 5 cm for each size class. The curve represents the predicted normal distribution for each population

Table 3. *Dichocoenia stokesi*. Size-distribution parameters of populations on reefs of the Florida Keys and Lee Stocking Island (LSI). Mean size refers to the widest diameter of the colony. Data were tested for normality using the Kolmogorov-Smirnov test

| Site | Year | Mean size | SD | Skewness ($g_1$) | 95 per cent confidence | Probability of normal | n |
|---|---|---|---|---|---|---|---|
| Florida Keys | 1998 | 9.51 | 4.5 | 0.687 | 18 | <0.01 | 342 |
| Florida Keys | 2002 | 19.9 | 11.7 | 0.549 | 40 | <0.01 | 314 |
| LSI | 2002 | 13.1 | 8.3 | 1.480 | 30 | <0.01 | 380 |

### Skewness

Fig. 3 and Table 3 also present data related to the skewness of each population surveyed. The lines of each graph in Fig. 3 present the predicted normal distribution for each survey. All 3 distributions are skewed to the right (positive values, see Table 3), indicating a greater proportion of small size classes as compared to large size classes. In 2002, the Florida *Dichocoenia stokesi* population was less positively skewed than in 1998 ($g_1$ values of 0.549 and 0.687 respectively), had fewer corals in the small size classes, and more colonies in the large size classes. Thus, in addition to the loss of small *D. stokesi* colonies from 1998 to 2002, there was a shift to a more even distribution across small to large size classes. The Florida populations were much more negatively skewed in both years than the Lee Stocking Island population, which had a $g_1$ value of 1.48 (Table 3). All of the distributions differed from normal distributions.

In summary, in the 7 yr following the 1995 WPII epizootic, we found that within the population of *Dichocoenia stokesi* on Florida reefs, mean colony size had increased, the range of colony size had increased, and the size–frequency distribution pattern (skewness) had changed.

## DISCUSSION

Only a few studies have specifically addressed the long-term effects of scleractinian coral disease on coral reef ecosystems and coral populations (Richardson 1998). The effect of coral disease on coral reefs may potentially have both positive and negative aspects. A possible benefit of coral disease to the reef ecosystem is that disease-related loss of coral tissue may open up new substrate (exposed coral skeleton) for coral recruitment. However, this was shown not to be the case in 2 separate studies of recruitment onto new substrate exposed by black band disease (Edmunds 1991, 2000, Kuta & Richardson 1997). Coral diseases may also have long-term negative effects on reefs. The only coral disease examined in the context of long-term negative effects on coral populations is white band disease, which is believed to have induced a phase shift from reef-framework corals to small, encrusting species (Aronson & Precht 1997, 2001).

Coral community response to coral disease outbreaks, particularly at the population level, has not been studied to any great extent (Richardson 1998). Multi-year surveys of coral populations (species and numbers of individuals), and coral disease incidence and prevalence have revealed that an observed overall decline in coral populations, including on Florida's reefs (Porter et al. 2002), is often accompanied by a parallel increase in the numbers and incidence of coral disease. The exact role of coral disease in the observed decline, however, is not yet known.

In our study, we were interested in assessing the long-term effect of the 1995 WPII epizootic on the Florida Keys population of *Dichocoenia stokesi*. Prior to the epizootic, no quantitative data were collected by our group to document the population (numbers and size–frequency distribution) of *D. stokesi* on Florida's reefs; therefore, we have no baseline data with which to compare our findings (although some early data have been reported by 2 other groups, and are discussed below). As an alternative, we also surveyed *D. stokesi* populations on reefs of Lee Stocking Island to provide a control. The data presented in Fig. 2 clearly show that there is a steady decline in the population of *D. stokesi* occurring on middle to northern Florida Keys reefs. The mean number of colonies per $m^2$ has decreased by almost 75%. This finding is in agreement with the results of a multi-year monitoring survey documenting a severe decline in coral numbers on the Florida Reef Tract during the period 1996 to 2000 (Porter et al. 2002).

We found that, although declining in overall number, colonies in the existing *Dichocoenia stokesi* population on Florida reefs are growing as evidenced by the increase in corals in the larger size classes. Thus, while there were no colonies in size classes >25 cm in 1998, by 2002 coral colonies were present up through the 55 cm size class. Between 1998 and 2002, the proportion of corals in the 25 cm class and above increased from 0 to 27.4%. The presence of very large colonies in

the 2002 data set may have been affected by the fact that of 48 sites surveyed in 1998 and 2002, only 10 of these were repeat surveys of the same sites. Reports of growth rates for this species range from an average increase in diameter of up to 6.67 mm $yr^{-1}$ (Vaughn 1915) to linear (top of colony) growth of 1.36 to 7.58 mm $yr^{-1}$ (Morris 1993, E. Peters pers. comm.). The larger growth rate of 7.58 mm $yr^{-1}$ was calculated from an observed mean growth rate of ca. 3.5 cm over 24 wk (Morris 1993) and was conducted in the context of a study on the effect of dredging. There are no data specific to *D. stokesi* growth rates for our study sites during the period of our surveys.

Accompanying the increase in medium and large *Dichocoenia stokesi* colonies, we found a decline in small colonies. Between 1998 and 2002, the proportion of corals in the 5 and 10 cm size classes decreased from 78 to 32%. This can only be explained by either selective mortality of the smallest colonies, or by a decrease in recruitment. During the 1995 epizootic, it was observed by L. L. Richardson (but not quantified or recorded) that the small colonies of each susceptible coral species were most commonly diseased. If this pattern were consistent, the continued presence of active WPII in 1998 may be contributing to the loss of small colonies.

Dustan & Halas (1987) included size data on a per coral species basis during repeat transects on a northern Florida reef conducted during 1975 and 1982. They found that the mean *Dichocoenia stokesi* colony size (reported as the distance a transect line intercepted a colony, thus not necessarily the maximum width) increased from 4 to 6 cm over this 7 yr period. Size-frequency data were not reported, and there was only a small number of *D. stokesi* colonies observed along the transect lines (9 colonies in 1975 and 3 in 1982). In separate surveys in 1975, 102 colonies of *D. stokesi* were examined for the presence of white plague (type I), none of which exhibited white plague signs (Dustan 1977). There was no information reported as to the presence or absence of white plague on *D. stokesi* for the 1982 transect data (Dustan & Halas 1987). Thus, it is not possible to propose a role of this coral disease in the increase in average size class following the first documented outbreak of this disease.

Bak & Meesters (1999) also reported colony size-frequency distribution data for *Dichocoenia stokesi* populations in Florida. In this case, data were collected in 1994 (R. Bak pers. comm.), the year before the 1995 epizootic. Their data set utilized size categories of 10 cm (0 to 10, 10 to 20, etc.) and thus, do not conform to our size classes (nearest 5 cm); however, some comparisons can be made. Bak & Meesters 1999 (untransformed data) revealed that the highest proportion of colonies was in the 10 to 20 cm size class (>50% of the population) and the next highest proportion was in the smallest (0 to 10) cm size class (approximately 33% of the population). Approximately 15% of colonies were in the 20 to 30 cm size class, none in the 30 to 40 or 40 to 50 cm size classes, and a very small number (not provided but bar suggests <1%) in the 50 to 60 cm size class. When comparing these values with our data in Fig. 3A, it can be seen that these distributions are similar. Thus, while in 1994 >80% of colonies were 20 cm or smaller, in 1998, we found >90% of colonies to be in the size classes below 17.5 cm. In both studies (Bak & Meesters 1999 and the 1998 data set reported here), small *D. stokesi* colonies were much more common than what we found in 2002. Thus, the distribution pattern of *D. stokesi* on Florida reefs appears to have shifted to large colonies over a period of time several years after the 1995 epizootic (i.e. after 1998). We do not know the reason for this shift.

In another study, Meesters et al. (2001) included *Dichocoenia stokesi* size–frequency distribution data in association with a study conducted on reefs of Curaçao. In this study, they compared size–frequency distributions among populations of 13 coral species at 4 sites, 2 of which were considered to be healthy and 2 degraded. Degraded sites were directly offshore of a heavily industrial urban area, while healthy (control) reefs were 2 km upstream and away from coastal influence. They noted that the degraded reefs exhibited lower coral species diversity, lower living coral, and increased turbidity and eutrophication, but there was no mention of the presence (or absence) of disease (Meesters et al. 2001). These investigators found that only 2 (including *D. stokesi*) of the 13 species studied did not exhibit population differences (changes in size–frequency distribution patterns) between the 4 sites.

Bak & Meesters (1999) and Meesters et al. (2001) have proposed that relative skewness can be used as a measure of the health of coral populations. Specifically, they proposed that healthy populations are slightly negatively skewed (dominated by small colonies), and less healthy populations are increasing more negatively skewed (more large colonies). In the Curaçao study, they found that 48 of 52 (13 species at each of the 4 sites) size–frequency distribution patterns were negatively skewed, and that *Dichocoenia stokesi* populations were negatively skewed at all 4 sites. Only 2 of the 13 coral species exhibited populations that were less negatively skewed in the degraded area. They suggested that the pattern of relatively negative skewness in the degraded sites was due to fewer colonies in the smaller size classes, the result of lower coral recruitment (Meesters et al. 2001).

In our study, *Dichocoenia stokesi* populations had $g_1$ (skewness) values of 0.687 and 0.549 for Florida reefs

in 1998 and 2002, and 1.480 for Lee Stocking Island in 2002. This is in general agreement with the findings of Meesters et al. (2001) of lower $g_1$ values (more negatively skewed) for coral populations at degraded versus control sites; however, exact values cannot be compared because their data were log-transformed and ours were not.

It is not known why the Florida population is exhibiting an overall pattern in which the relative numbers of small colonies of *Dichocoenia stokesi* are decreasing. A possible, and untested, explanation for the observed population decline and the relative lack of small *D. stokesi* colonies in Florida is that the WPII pathogen is still present on Florida reefs and is detrimentally affecting corals by sub-acute (no disease signs) infection that selectively results in mortality of small colonies. During the period of the study, disease prevalence within the *D. stokesi* population decreased from an average of 20.1% in 1995 to 0% in 2002; therefore, the continued loss of individual colonies cannot be attributed to death caused by active disease. However, as noted previously, in 2002 we documented active white plague on 11 colonies of 4 other coral species during the course of the *D. stokesi* survey; thus, the pathogen is still present on the reef. It is not known whether the pathogen is still associated with apparently healthy *D. stokesi* colonies, whether the colonies that survived the 1995 epizootic were resistant, or whether these colonies developed immunity upon exposure to the pathogen. These questions are currently under study. Also of importance, it is not known whether a sublethal infection by *Aurantimonas coralicida* would directly and negatively affect *D. stokesi* reproduction. In 1996, we detected 2 *D. stokesi* recruits on a reef that was repeatedly surveyed in the 1995 study to document WPII induced mortality, suggesting that the population would rapidly recover from the severe perturbation of the disease. However, the recruits did not survive to the following year, and the number of *D. stokesi* colonies on this reef has decreased dramatically through August of 2002. Recruitment failure may also be due to unknown factors unrelated to disease.

The decline in *Dichocoenia stokesi* may be not be a result of the 1995 epizootic, and may in fact be related to general environmental degradation. Florida reefs are believed to be some of the most severely stressed of coral reefs (Porter et al. 1999). Relationships between general reef degradation and environmental perturbations (nutrification, global warming, etc.) are currently being studied by a number of investigators, but no definitive results have been obtained. Our preliminary data suggest that sedimentation (Voss et al. 2004) and nitrate levels (authors' unpubl. data) may impact coral disease prevalence. To date, limited ecological research has been performed in the field and the laboratory to understand the interactions between environmental factors and WPII incidence and prevalence. During the 1995 epizootic, there was a positive correlation between WPII incidence and both elevated temperature and water depth (>2 m) on Florida's reefs (Richardson et al. 1998a). Dustan found the same patterns in 1977; i.e. white plague-infected corals were most common between 8 and 18 m depth (Dustan 1977), and the rate of tissue destruction by white plague was positively correlated with increased water temperature (Dustan 1977). A similar depth pattern was found on reefs of Venezuela (Croquer et al. 2003) and Puerto Rico (E. Weil pers. comm.), but not on reefs of Dominica (Borger 2003). Laboratory experiments on the ecological physiology of *Aurantimonas coralicida* have revealed a temperature range and optima that are in agreement with the seasonal occurrence of the disease on Florida's reefs (Remily 2004).

At this point, the reasons for the change in Florida's *Dichocoenia stokesi* population in the 7 yr following the 1995 WPII epizootic are not known. It is possible that the WPII epizootic selectively killed susceptible colonies, and that resistant colonies survived and enabled an evolutionary shift to a naturally resistant population. This would explain the Caribbean-wide pattern in which WPII epizootics that occur in one region do not recur on the same reefs in subsequent years. We saw no evidence of active recruitment in the Florida Keys *D. stokesi* population, and believe that reproductive capacity may have been compromised in the years following the 1995 epizootic. To understand the underlying causes of coral decline, and the specific role of coral diseases in that decline, more studies targeting coral population structure and size distributions should be carried out on multi-year bases. Although time-consuming, the inclusion of size–frequency distributions for select coral species within monitoring programs might provide invaluable baseline data for ecological analyses aimed at understanding the reasons and relative importance of individual factors in coral reef decline. Ideally, such data sets would include measurement of colonies, especially in the smallest size classes, to the nearest 1 cm, which would allow data to be log-transformed and allow retrieval of much more information about coral population structure (see Vermeij & Bak 2002 and 2003 for rationale, methods, and a comparison of the information value of transformed versus non-transformed data sets). In the specific case of the role of coral diseases, a database that included pre-epizootic population structure information would be highly useful in assessing recovery (or non-recovery) from disease events. Finally, if at all possible, resurveying large numbers of permanent sites would provide specific data about fate, recovery, and growth of individual colonies.

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## LITERATURE CITED

Aronson R, Precht W (1997) Stasis, biological disturbance, and community structure of a Holocene coral reef. Paleobiology 23:326–346

Aronson R, Precht W (2001) White-band disease and the changing face of Caribbean coral reefs. Hydrobiologia 460:25–38

Bak RPM, Criens SR (1982) Survival after fragmentation of colonies of *Madracis mirabilis*, *Acropora palmata*, and *A. cervicornis* (Scleractinia) and the subsequent impact of a coral disease. Proc 4th Int Coral Reef Symp 2:221–227

Bak RPM, Meesters EH (1998) Coral population structure: the hidden information of colony size-frequency distributions. Mar Ecol Prog Ser 162:301–306

Bak RPM, Meesters EH (1999) Population structure as a response of coral communities to global change. Am Zool 39:56–65

Borger JL (2003) Three scleractinian coral diseases in Dominica, West Indies: distribution, infection patterns and contribution to coral tissue mortality. Rev Biol Trop 51 (Suppl 4):25–38

Bruckner A, Bruckner R (1997) The persistence of black band disease in Jamaica: impact on community structure. Proc 8th Int Coral Reef Symp 1:601–606

Bruckner AW, Bruckner RJ, Williams EH (1997) Spread of a black-band disease epizootic through the coral reef system in St. Ann's Bay, Jamaica. Bull Mar Sci 61:919–928

Croquer A, Pauls SM, Zubillaga AL (2003) White plague disease outbreak in a coral reef at Los Roques National Park, Venezuela. Rev Biol Trop 51(Suppl 4):39–45

Denner EBM, Smith G, Busse HJ, Schumann P, Narzt T, Polson S, Lubitz W, Richardson LL (2003) *Aurantimonas coralicida* gen. nov., sp. nov., the causative agent of white plague type II on Caribbean scleractinian corals. Int J Syst Evol Microbiol 53:1115–1122

Done TJ (1992) Phase shifts in coral reef communities and their ecological significance. Hydrobiologia 247:121–132

Dustan P (1977) Vitality of reef coral populations off Key Largo, Florida: recruitment and mortality. Environ Geol 2: 51–58

Dustan P, Halas JC (1987) Changes in the reef-coral community of Carysfort Reef, Key Largo, Florida: 1974–1982. Coral Reefs 6:91–106

Edmunds PJ (1991) Extent and effect of black band disease on Caribbean reefs. Coral Reefs 10:161–165

Edmunds PJ (2000) Recruitment of scleractinians onto the skeletons of corals killed by black band disease. Coral Reefs 19:69–74

Gardner TA, Cote IM, Gill JA, Grant A, Watkinson AG (2003) Long-term region-wide declines in Caribbean corals. Science 301:958–960

Gladfelter W (1982) Whiteband disease in *Acropora palmata*: implications for the structure and growth of shallow reefs. Bull Mar Sci 32:639–643

Green EP, Bruckner AW (2000) The significance of coral disease epizootiology for coral reef conservation. Biol Conserv 96:347–361

Hughes TP, Baird AH, Bellwood DR, Card M and 13 others (2003) Climate change, human impacts, and the resilience of coral reefs. Science 301:929–933

Jackson JBC, Kirby MX, Berger WH, Bjorndal KA and 15 others (2001) Historical overfishing and the recent collapse of coastal ecosystems. Science 293:629–637

Kuta KG, Richardson LL (1997) Black band disease and the fate of diseased coral colonies in the Florida Keys. Proc 8th Int Coral Reef Symp 1:575–578

Meesters E, Hilterman M, Kardinaal E, Keetman M, de Vries M, Bak R (2001) Colony size-frequency distributions of scleractinian coral populations: spatial and interspecific variation. Mar Ecol Prog Ser 209:43–54

Morris SM (1993) Effects of dredging-induced sedimentation and turbidity on two species of stony corals of Southeast Florida. Master's thesis, Nova Southeastern University Oceanographic Center, Dania

Nagelkerken I, Buchan K, Smith GW, Bonair K and 8 others (1997a) Widespread disease in Caribbean sea fans. I. Spreading and general characteristics. Proc 8th Int Coral Reef Symp 1:679–682

Nagelkerken I, Buchan K, Smith GW, Bonair K and 10 others (1997b) Widespread disease in Caribbean sea fans. II. Patterns of infection and tissue loss. Mar Ecol Prog Ser 160:255–263

Nugues MM (2002) Impact of a coral disease outbreak on coral communities in St. Lucia: what and how much has been lost? Mar Ecol Prog Ser 229:61–71

Pandolfi JM, Bradbury RH, Sala E, Hughes TP and 8 others (2003) Global trajectories of the long-term decline of coral reef ecosystems. Science 301:955–958

Porter JW, Lewis SK, Porter KG (1999) The effect of multiple stressors on the Florida Keys coral reef ecosystem: a landscape hypothesis and a physiological test. Limnol Oceanogr 44:941–949

Porter JW, Dustan P, Jaap WC, Patterson KL and 5 others (2001) Patterns of spread of coral disease in the Florida Keys. Hydrobiologia 460:1–24

Porter JW, Kosmynin V, Patterson KL, Porter KG and 13 others (2002) Detection of coral reef change by the Florida Keys coral reef monitoring project. In: Porter JW, Porter KG (eds) The everglades, Florida Bay, and coral reefs of the Florida Keys: an ecosystem sourcebook. CRC Press, Boca Raton, FL, p 749–770

Remily ER (2004) Ecological physiology of *Aurantimonas coralicida*: pathogen of the coral disease white plague type II. Master's thesis, Florida International University, Miami, FL

Richardson LL (1998) Coral diseases: what is really known? Trends Ecol Evol 13:438–443

Richardson L, Kuta K, Schnell S, Carlton R (1997) Ecology of the black band disease microbial consortium. Proc 8th Int Coral Reef Symp 1:597–600

Richardson LL, Goldberg WM, Kuta KG, Aronson RB and 5 others (1998a) Florida's mystery coral killer explained. Nature 392:557–558

Richardson LL, Goldberg WM, Carlton RG, Halas JC (1998b) Coral disease outbreak in the Florida Keys: plague type II. Rev Biol Trop 46(Suppl 5):187–198

Rützler K, Santavy D, Antonius A (1983) The black band disease of Atlantic reef corals. III. Distribution, ecology and development. PSZN I: Mar Ecol 4:329–358

Sutherland KP, Porter JW, Torres C (2004) Disease and immunity in Caribbean and Indo-Pacific zooxanthellate corals. Mar Ecol Prog Ser 266:273–302

Vaughn TW (1915) The geologic significance of the growth rate of the Floridian and Bahamas shoal-water corals. J Wash Acad Sci 5:591–600

Vermeij MJA, Bak RPM (2002) Inferring demographic processes from population size structure in corals. Proc 9th Int Coral Reef Symp 1:589–594

Vermeij MJA, Bak RPM (2003) Species-specific population structure of closely related coral morphospecies along a depth gradient (5–60 m) over a Caribbean reef slope. Bull Mar Sci 73:725–744

Voss JD, DK Mills, Richardson LL (2004) An integrative analysis of environmental factors, coral population and community structure, and microbial community composition associated with coral diseases near Lee Stocking Island, Bahamas. 10th Int Coral Reef Symp Abstracts, International Coral Reef Society, Okinawa, p 167

Weil E (2004) Coral reef diseases in the wider Caribbean. In: Rosenberg E, Loya Y (eds) Coral health and disease. Springer-Verlag, Berlin, p 35–68

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