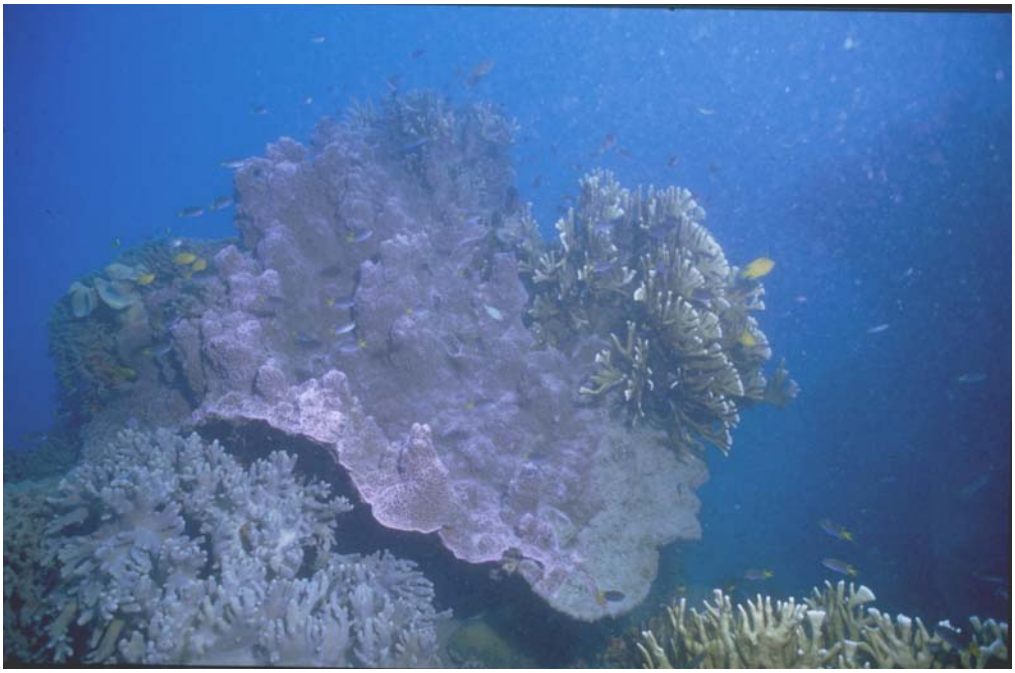
APPENDIX 1

Photographic stimuli used in the perceptual survey. The photographs were presented in a separate album from the questionnaire sheet

1

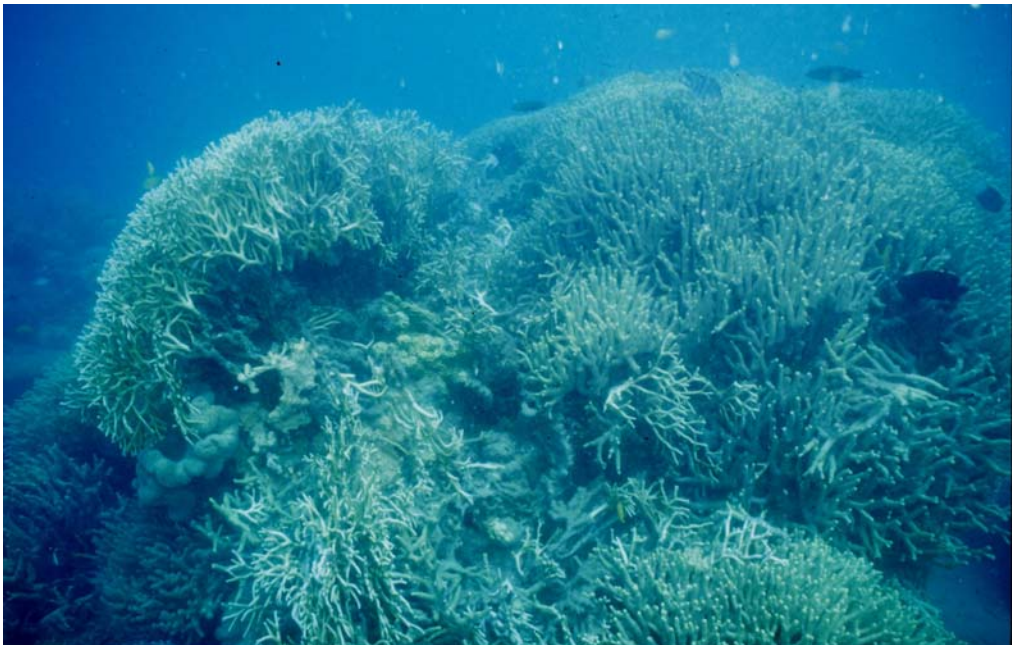
2

3



4

5



6

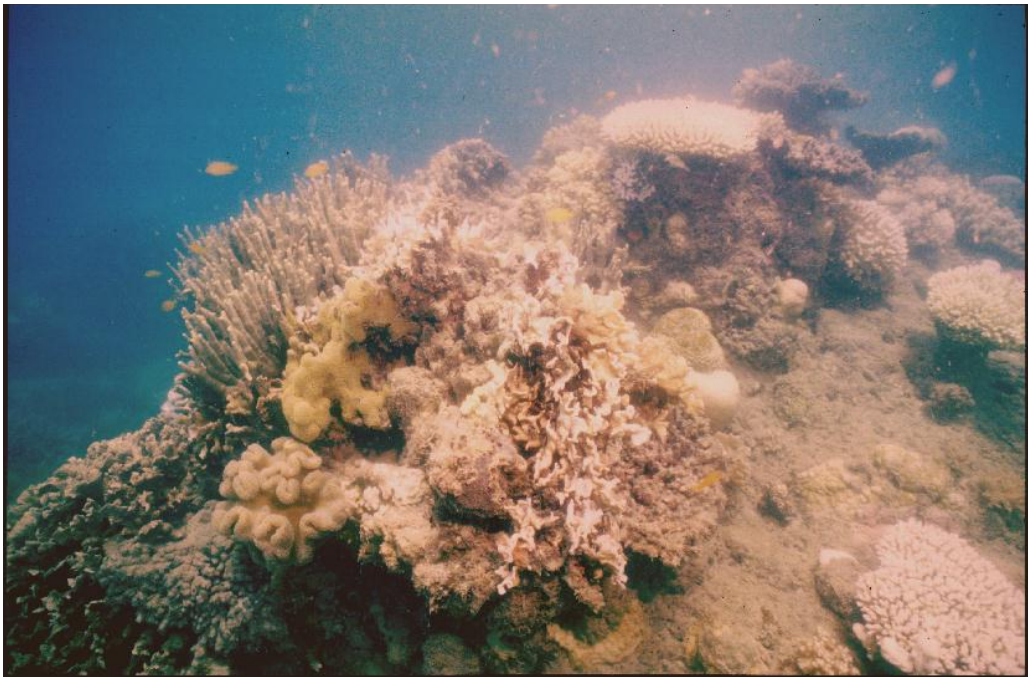
7

8

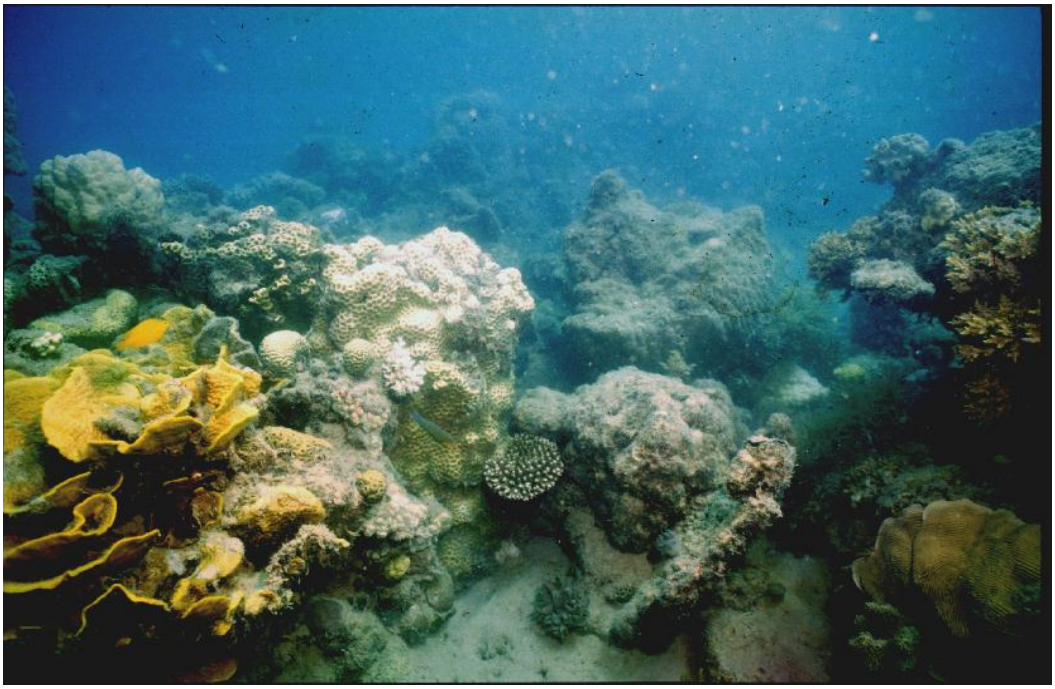
9

10

11

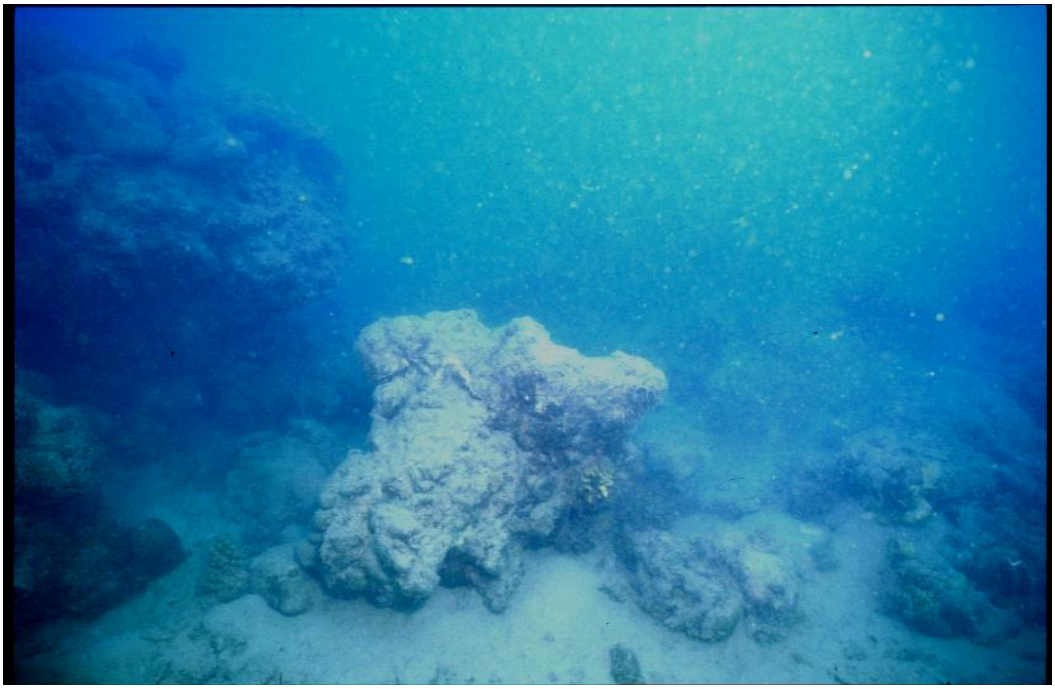


12



13

14

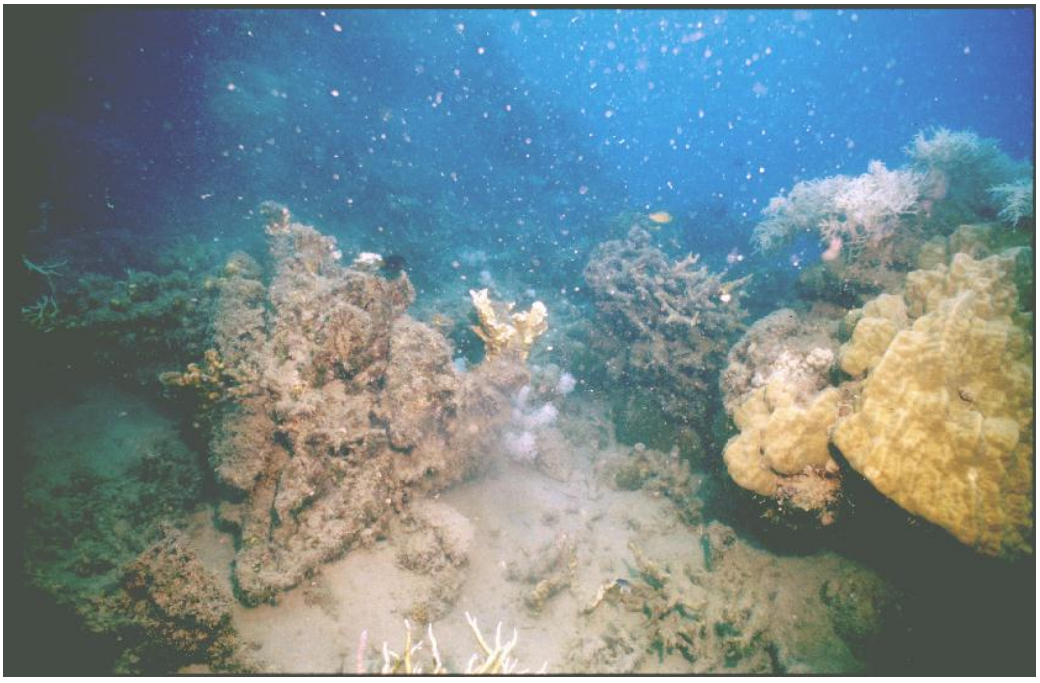


15

16

17

18



19

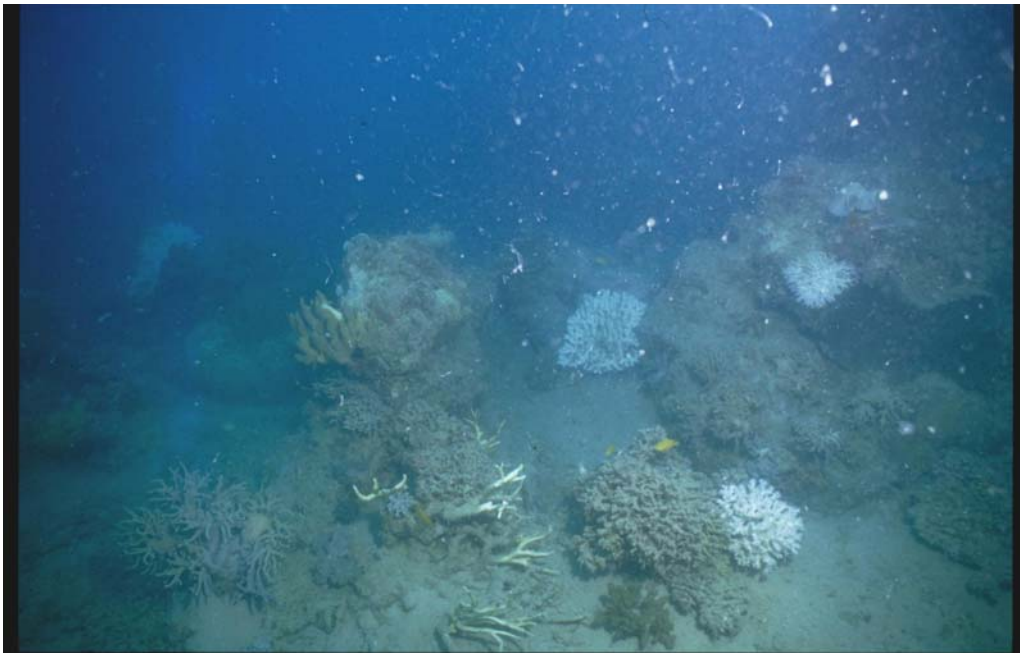
20

21

22

23

24



25

26

Appendix 2.

Proceedings 9th International Coral Reef Symposium, Bali, Indonesia 23-27 October 2000, Vol. 2.

Abundance of black-band disease on corals from one location on the Great Barrier Reef: a comparison with abundance in the Caribbean region.

E. A. Dinsdale[1]

Abstract Black-band disease (BBD) occurs on the Great Barrier Reef, Australia, with at least 21 species in five families being affected. Surveys of reef crest corals at Lizard Island found 2.8% of 4569 coral colonies were affected with BBD. Acroporidae, in particular *Acropora hyacinthus, Acropora intermedia* and *Acropora millepora,* were susceptible to BBD, compared with the Caribbean where acroporid corals are resistant to BBD. Pocilloporidae, including *Pocillopora damicornis, Pocillopora verrucosa* and *Stylophora pistillata,* were identified as important host species. The abundance of BBD varied between sites, ranging from 1.3% to 4.9% of colonies affected. The percentage of BBD-affected colonies varied between coral families. The percent of colonies affected with BBD were: 6.0% for the Pocilloporidae, 3.6% for the Acroporidae, 3.0% for the Faviidae and 2.1% for the Poritidae. The level of BBD-affected colonies on the GBR was within the range of affected colonies reported in the Caribbean.

Keywords Black-band disease, Great Barrier Reef, Coral disease.

Introduction

Black-band disease (BBD) affects scleractinian and gorgonian corals from the Caribbean and Red Sea (Antonius 1981a, 1988a; Garret and Ducklow 1975; Feingold 1988; Peters 1993). Over half of the 34 species of scleractinian corals that have been reported as susceptible to BBD occur in the Caribbean region (Rützler et al. 1983; Antonius 1988b; Edmunds 1991; Green and Bruckner 2000). BBD in the Caribbean region has caused partial mortality of individual colonies and led to reduced coral cover on some reefs (Santavy and Peters 1997; Kuta and Richardson 1996; Bruckner and Bruckner 1997; Bruckner et al. 1997). In contrast, there are few reports of BBD affecting corals in the Pacific region. Antonius (1985) found two susceptible coral species, *Goniastrea pectinata* and *Platygyra lamellina,* in the Philippines. BBD has been observed but not quantified on reefs of the Great Barrier Reef (GBR) and Fiji (Miller 1996; Littler and Littler 1996). As noted by Green and Bruckner (2000), there is a "relative scarcity" of records of coral diseases in the Pacific region compared with the Caribbean. The lack of reports of BBD from the GBR could be related to a lack of studies specifically focused on BBD or to a genuine lack of BBD on these reefs. In this paper, I quantify for the first time the prevalence of BBD at one location within the GBR.

Researchers have quantified BBD by describing the prevalence of the disease within coral communities and identifying susceptible coral species. Prevalence "refers to the number of cases of disease that exist, in a defined population, at some point in time" (Christie et al. 1997). The prevalence of BBD has been found to vary spatially and temporally. Surveys have recorded a range of 1% to 10% BBD-affected colonies within a population (Green and Bruckner 2000). Edmunds (1991) found that BBD prevalence in individual coral species at 7 locations in the Virgin Islands varied from 0% to 5.5%, with a mean of 0.25%. Dustan (1993) surveyed 19 reefs in the Florida Keys and found 6.2% of all colonies affected with BBD. The prevalence of BBD fluctuated from 0% to 3.2% on three different reefs in the Key Largo region (Kuta and Richardson 1996). The prevalence of BBD varies seasonally, with higher levels of BBD-affected colonies occurring during summer (Edmunds 1991).

Coral taxa appear to have different susceptibility to BBD (Peters 1993). In the Caribbean region, coral species from the family Faviidae are most often affected with BBD (Santavy and Peters 1997). Faviidae are important framework corals and have the highest diversity in Caribbean reefs (Walton-Smith 1971). *Diploria strigosa* and *Montastraea annularis* are most often affected with BBD, with lower levels of infections in closely related species, *Montastraea cavernosa, Diploria labyrinthiformis* and *Diploria clivosa* (Antonius 1981b; Rützler et al. 1983). Non-faviid corals are also susceptible to BBD (Green and Bruckner 2000). *Siderastrea siderea* was thought to be resistant to BBD (Antonius 1981a). However, infections on this species were recorded in 1992 on reefs of Jamaica and this species is now frequently recorded with BBD (Bruckner et al. 1997). Natural infections of BBD have not been recorded on corals in the families Acroporidae and Pocilloporidae in the Caribbean region. Experimental inoculations conducted on acroporids from the Caribbean suggest they are able to resist the BBD pathogens (Antonius 1981b, 1985; Rützler et al. 1983). However, in aquarium studies in the Red Sea, BBD was transferred

[1] EA. Dinsdale: School of Tropical Environment Studies and Geography, James Cook University, Townsville, Q. 4811, Australia. Email: elizabeth.dinsdale@jcu.edu.au

successfully to Acroporidae and Pocilloporidae after colonies were injured, suggesting that these species may be susceptible to the disease when stressed or injured (Antonius 1988b). If some species are susceptible to disease while others are not, there is a possibility for susceptible species to be lost from the community and be replaced by non-susceptible species. The different levels of affected colonies within certain taxa could indicate that these species are more susceptible to BBD.

Four major coral diseases; black-band disease, white-band disease type II, white plague type II and *Aspergillosis*, have been described (Santavy and Peters 1997; Richardson 1998; Green and Bruckner 2000). The effects of diseases on coral communities have ranged from partial mortality of a few individuals to community-level changes (Edmunds 1991; Aronson and Precht 1997). White-band disease has caused profound effects on coral communities in the Caribbean region. *Acropora palmata* stands in the US Virgin Islands have been decimated by a combination of white-band disease (Gladfelter 1982) and hurricane damage (Bythell et al. 1993). *D. strigosa* and *Porites* species were the major corals recruiting after these events (Bythell et al. 1993), suggesting a community shift mediated by disease and disturbance. Aronson and Precht (1997) described a disease-induced replacement of the dominant shallow water coral *Acropora cervicornis* by the less dominant *Agaricia* species in Channel Cay, Belize.

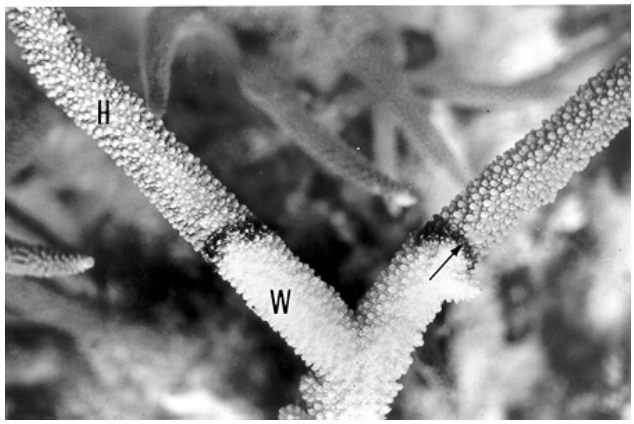
The aims of my study were to identify species of corals from the Great Barrier Reef that are susceptible to BBD and to determine the prevalence of BBD-affected colonies. I investigated whether the characteristics of BBD on the GBR are similar to those in the Caribbean region. The important characteristic of different levels of BBD-affected colonies between individual taxa was investigated by quantifying the prevalence of BBD in major coral families. Different susceptibility of coral species to BBD was investigated by comparing the number of individuals within a species to show signs of BBD when experimentally placed in direct contact with necrotic tissue.

Methods

Corals on four reefs around Lizard Island (14°40'S, 145°28'E), Australia were surveyed for the presence of BBD during summer (January – February), 1994. Replicate belt transects (10 x1m) were surveyed on the reef crest of the four sites in 1 to 3m water depth. Seven to ten transects were laid haphazardly parallel to the shoreline, at South Island, Lizard Head, Osprey Island and Patch Reef. Within each transect, benthos in the categories Scleractinia, Alcyonacea, Gorgonia and Hydrozoa were counted. Coral colonies displaying signs of BBD were recorded and identified to species, when possible.

For disease studies, the number of colonies in an area is usually recorded (Edmunds 1991; Kuta and Richardson 1996). A colony was defined as any autonomous coral skeleton with living tissue. BBD was identified in the field using these gross morphological characteristics: a black band of necrotic tissue abutting relative healthy tissue on one side and bare white skeleton on the other (Fig. 1). I quantified the number of BBD-affected colonies and the numbers of non-affected colonies in each transect.

Fig 1. *Acropora intermedia* from Lizard Island affected with black-band disease, illustrating the typical gross morphological features of the disease. Apparently healthy tissue (H) is abutted by a black band of necrotic tissue (arrow), with white (W) skeleton denuded of tissue posterior to the necrotic band.



To analyze the distribution of BBD between the four sites at Lizard Island, the percentages of BBD-affected colonies were compared using a one-way analysis of variance (anova). A post hoc Tukey's test was used to analyze if sites within habitats displayed similar prevalence of BBD (Underwood 1997). Due to the lack of normality in the data, an arc-sin square root transformation was performed prior to the analysis and homogeneity of variance was tested using the Levene statistic. Alpha level was set at $P < 0.05$.

To analyze whether BBD-affected colonies were spread evenly between taxa, a Pearson chi-square test (Zar 1999) was conducted. Data were pooled for all sites, since there were no obvious groupings of sites from the Tukey's test. To meet the assumptions of a chi-square test, it is necessary to minimize the number of cells with frequencies of less than five. Therefore, coral species were grouped into five categories; Acroporidae, Faviidae, Pocilloporidae, Poritidae and other Cnidaria (composed of the remaining scleractinian families, alcyonaceans, gorgonians and hydrozoans). A subdivided chi-square analysis with Yates' continuity correction was performed to identify which coral family was causing the variation in levels of BBD prevalence (Zar 1999).

To examine whether the different levels of affected colonies within a species were due to differences in susceptibility of that species, I compared the rate of infection of BBD on five individuals in each of eight coral species. It would be predicted that if a species was highly susceptible, all individuals would become affected with BBD. BBD was transferred by attaching a piece of BBD-affected *A. hyacinthus* to the experimental colony in the field. The necrotic area was placed in direct contact with a healthy colony, and fixed using plastic coated wire. Although care was taken not to injure the experimental corals, two control groups were established. In the first control group, five individuals from each species had a piece of healthy *A.*

hyacinthus attached using plastic coated wire, to control for the effects of contact between corals. The second control group was a tagging control, where five individuals in each species were tagged and handled. The experimental group and two control groups were collected after 48 h and presence or absence of BBD was noted. The time period was chosen due to the rapid movement recorded for BBD (Rützler et al. 1983). Coral species used during the trial were, *A. hyacinthus,* and *A. cuneata* in the family Acroporidae; *P. damicornis,* and *Seriatopora hystrix* in the family Pocilloporidae; *Hydnophora ridiga,* and *Platygyra daedalea* in the family Faviidae; *Symphyllia recta* in the family Mussidae; and *Porites lichen* in the family Poritidae. Colonies used in this experiment were located 1 to 3m deep within a $30m^2$ area on a reef in the Lizard Island lagoon. The rates of transmission and levels of BBD-affected colonies recorded during the survey were compared to identify if differences in susceptibility between species were apparent.

Results

Seventeen species of corals in five families (Pocilloporidae, Acroporidae, Faviidae, Poritidae and Mussidae) were affected by BBD in transects surveyed at Lizard Island (Table 1). Corals in the family Acroporidae and Pocilloporidae had 69 and 33 colonies affected with BBD. The species affected with BBD varied between sites. *A. hyacinthus, A. millepora* and *P. damicornis* were affected with BBD at all sites. *A. intermedia* was affected at Osprey Island and Patch Reef and *Goniastrea retiformis, P. verrucosa* and *Acropora gemmifera* were affected with BBD at South Island and Lizard Head. Four additional species, *Goniopora stokesi, Acropora polystoma, Acropora divaricata* and *Montipora tuberculosa*, were recorded with BBD off-transect.

Based on a sample size of 4569 corals, 2.8% of all colonies surveyed at Lizard Island were affected with BBD. The percentage BBD-affected colonies was variable between sites and ranged from 4.9% at South Island to 1.3% at Patch Reef (one-way anova $MS=2.52*10^{-2}$, $df = 3$, $F = 4.6$, $P<0.001$). However, a Tukey's test did not distinguish any patterns in BBD abundance between the sites.

The prevalence of BBD was not spread evenly between the five coral families affected with BBD (chi-square test $X^2 = 54.266$, $df = 4$, $P < 0.001$). Pocilloporidae had the highest level of affected colonies with 6.0% affected (Table 2). The subdivided chi-square analysis showed that Pocilloporidae colonies were affected at a higher rate than corals in the families Acroporidae, Poritidae and Faviidae ($X^2 = 6.546$, $df = 1$, $P < 0.05$). The percentage of BBD-affected colonies was spread evenly between the Acroporidae, Poritidae and Faviidae ($X^2 = 2.125$, $df = 2$, $P = 0.346$). This suggests that pocilloporid corals may be more susceptible to BBD than corals in other families. Apart from one BBD-affected colony in the family Mussidae on Lizard Head, other Cnidaria, such as alcyonaceans, gorgonians and hydrozoans, were not identified with BBD during this survey.

Table 1. Seventeen species were identified with black-band disease (BBD) on Lizard Island, suggesting multiple host species are a feature of BBD. The numbers of BBD-affected colonies compared with the numbers of unaffected colonies (in parentheses) are given for each site. The total number BBD-affected and unaffected colonies for each family are listed. Only BBD-affected colonies that were identified to species are listed. (NQ, species not quantified)

Family	Species	South Island	Lizard Head	Osprey Island	Patch Reef	Total
Pocilloporidae		13(105)	16(203)	3(111)	1(79)	33(549)
	Pocillopora damicornis	3 (82)	4 (111)	2 (77)	1 (41)	10 (311)
	Pocillopora verrucosa	5 (48)	6 (53)	0 (0)	0(1)	11 (102)
	Stylophora pistillata	5 (20)	6 (33)	1 (17)	0(9)	12 (79)
Acroporidae		18(459)	20(766)	20(421)	11(259)	69(1905)
	Acropora hyacinthus	4 (114)	5 (121)	5 (47)	3 (27)	18 (339)
	Acropora intermedia	0 (11)	0 (3)	4 (121)	6 (45)	10 (180)
	Acropora gemmifera	5 (58)	3 (154)	0 (7)	0 (5)	8 (224)
	Acropora millepora	1 (29)	1 (17)	2 (32)	2 (16)	4 (94)
	Acropora microclados	3 (3)	0 (5)	0 (3)	0 (4)	3 (15)
	Acropora monticulosa	0 (16)	2 (118)	0 (0)	0 (0)	2 (134)
	Acropora florida	0 (1)	1 (4)	1 (23)	0 (2)	2 (35)
	Acropora microphthalma	0 (0)	0 (0)	2 (12)	0 (7)	2 (19)
	Acropora robusta	0 (0)	0 (5)	2 (12)	0 (0)	2 (17)
	Acropora humilis	0 (10)	1 (17)	0 (3)	0 (13)	1 (43)
	Acropora palifera	0 (0)	0 (0)	1 (2)	0 (18)	1 (20)
	Acropora sarmentosa	1 (7)	0 (0)	0 (10)	0 (4)	1 (21)
Faviidae		9(79)	6(115)	0(167)	0(90)	15(496)
	Goniastrea retiformis	4 (12)	3 (23)	0 (18)	0 (54)	7 (97)
	Favia matthaii	1 (NQ)	NQ	NQ	NQ	1 (NQ)
Poritidae		3(38)	4(155)	0(72)	0(71)	7(336)
Mussidae		0(2)	1(7)	0(18)	0(20)	1(47)

The percentage of BBD-affected colonies in each family varied with site (Table 2). High levels of affected colonies were found in the Pocilloporidae, Faviidae and Poritidae at South Island with lower levels at Lizard Head. The percentage of affected Pocilloporidae colonies was lower at Osprey Island and Patch Reef, and no affected colonies were found for the Faviidae or Poritidae. Similar percentages of BBD-affected colonies in the family Acroporidae were found at all sites (Table 2).

Table 2. Percentage of black-band disease affected colonies in each family at each site.

Family	South Island	Lizard Head	Osprey Island	Patch Reef	Total
Pocilloporidae	8.3	7.9	2.7	1.3	6.0
Acroporidae	3.9	2.6	4.5	4.2	3.6
Faviidae	11.4	6.3	0	0	3.0
Poritidae	7.9	2.6	0	0	2.0
Other Cnidaria	0	0.4	0	0	0.1
Total	4.9	3.3	1.9	1.3	2.8

BBD transferred to all nine species in the transmission experiment after a 48-hr period. Of the five colonies tested for each species, *A. hyacinthus* had four individuals affected with BBD, while *P. damicornis*, *S. hystrix* and *H. ridiga* each had three individuals that were affected. In the other five species, BBD only transferred to two individuals in each species. None of the individuals in the two control groups showed any signs of BBD. Only four control colonies showed the presence of white tissue areas and small patches of denuded skeleton, suggesting handling stresses were low.

Discussion

I have recorded 129 coral colonies from five scleractinian families affected with black-band disease at one study location on the Great Barrier Reef, out of a total of 4569 colonies surveyed. This would suggest that BBD affects coral communities on the GBR, as well as in the Caribbean region. Corals from the family Acroporidae in the Caribbean region appear resistant to BBD (Rützler et al. 1983; Antonius 1988a). In contrast, Acroporidae from Lizard Island are susceptible to BBD, with 14 species affected. The family Pocilloporidae has eleven species that occur on the GBR; of the five species present on transects three were susceptible to BBD. The high percentage of Pocilloporidae colonies affected with BBD (6.0%) suggests this family may be an important host for BBD on the GBR.

A total of 2.8% of coral colonies were recorded with BBD during the survey at four sites around Lizard Island. The percent occurrence of BBD identified was more than the 0.25% of corals affected in US Virgin Island reefs (Edmunds 1991). However, other sites such as Floridian reefs (Dustan 1993) and Jamaican reefs (Bruckner and Bruckner 1997) had 6.2% and 6.1% of BBD affected colonies respectively, higher than recorded for Lizard Island. Feingold (1988) reported that 13.8% of octocorals were affected over a two-year period on the Northern Florida Keys, which is substantially higher than the prevalence of BBD on Lizard Island. My study suggests BBD is evident within coral populations on Lizard Island and the percent of affected colonies is within the range of BBD reported in the Caribbean region.

BBD often occurs seasonally, with the highest prevalence during the summer months (Edmunds 1991). The Lizard Island survey was conducted during the summer and therefore, may represent a maximum prevalence of BBD. Further surveys would be required to determine if seasonal variation also occurs on the GBR.

BBD is characterized by affecting multiple species, and there is variation in the level of affected colonies within a species between reefs (Peters 1993; Bruckner et al. 1997; Green and Bruckner 2000). These trends of BBD were also evident during my study on Lizard Island. Pocilloporidae had high levels of affected colonies compared with Acroporidae, Faviidae and Poritidae. The level of affected colonies within families also varied between sites. Bruckner et al. (1997) found similar variation in levels of affected colonies, with *M. annularis* (morphotype I and II), *D. clivosa* and *D. strigosa* most frequently affected in sheltered habitats. Conversely, *M. cavernosa* and *S. siderea* were most frequently affected with BBD in fore-reef habitats.

BBD transferred to all eight species in the transmission experiment and was found on a large number of species in a range of families during this and other surveys, suggesting that many species are susceptible to BBD. The levels of BBD-affected colonies recorded during a survey may depend on how BBD is transferred, not on differences in susceptibility of the corals. Research directed at testing susceptibility of species in different environments is required to understand the variation in levels of BBD-affected colonies between species.

Substantial reduction in living coral tissue occurs with BBD (Bruckner and Bruckner 1997), and <5% to 30% of colonies affected with BBD will eventually die (Green and Bruckner 2000). Mortality can be estimated using the prevalence of BBD recorded during the surveys and the mortality range from the literature. Therefore, if 2.8% of colonies were affected with BBD around Lizard Island, it is estimated <0.14% to 0.84% of coral colonies could die due to the disease. Compared with catastrophic events like *Acanthaster planci* outbreaks (Moran 1986), hurricane damage (Woodley et al. 1981) or white-band disease (Gladfelter 1982), where 60% to 90% of coral colonies die, mortality due to BBD is probably low at Lizard Island. However, the time between catastrophic events may be greater than for BBD, suggesting BBD may be a chronic mortality agent.

In conclusion, the survey of Lizard Island corals demonstrated that BBD affects coral communities on the GBR. BBD has multiple host species, with the level of affected colonies within a species varying between sites, similar to BBD in the Caribbean region. Corals in the family Acroporidae and Pocilloporidae are important host species on the GBR, in contrast to Faviid corals being important host species in the Caribbean region. The abundance of BBD at four sites surrounding Lizard Island was within the range of BBD recorded in the Caribbean region.

Acknowledgments I would like to thank T.P. Hughes and L. Owens for their support and supervision. Thanks to V. Hall, L. Smith and C. Syms for assistance during fieldwork. This work was supported by grants from Australian Research Council to T.P. Hughes. Thanks to A. Baird, J. Wolstenholme and the Coral Ecology Discussion Group at James Cook University for their comments on the manuscript. This is contribution no. 185 of the Coral Group at JCU.

References

Antonius A (1981a) Coral Reef Pathology: a review. Proc 4th Int Coral Reef Symp 2: 1-6

Antonius A (1981b) The "band" diseases in coral reefs. Proc 4th Int Coral Reef Symp 2: 7-14

Antonius A (1985) Coral disease in the Indo-Pacific: a first record. PSZNI Mar Ecol 6: 197-218

Antonius A (1988a) Distribution and dynamics of coral diseases in the eastern Red Sea. Proc 6th Int Coral Reef Symp 2: 293-298

Antonius A (1988b) Black-band disease behavior on Red Sea reef corals. Proc 6th Int Coral Reef Symp 3: 145-150

Aronson RB, Precht WF (1997) Stasis, biological disturbance, community structure of a Holocene coral reef. Paleobiology 23: 326-346

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

Bythell JC, Gladfelter EH, Bythell M (1993) Chronic and catastrophic natural mortality of three common Caribbean reef corals. Coral Reefs 12: 143-152

Christie D, Gordon I, Heller R (1997) Epidemiology. An introductory text for medical and other health science students. UNSW Press, Sydney

Dustan P (1993) Developing methods for assessing coral reef vitality: a tale of two scales. In: Ginsburg RN, Compiler, 1994, Proceedings of the Colloquium on Global Aspects of Coral Reefs: Health, Hazards and History, Rosenstiel School of Marine and Atmospheric Science, University of Miami, 1993, pp. 38-44

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

Feingold JS (1988) Ecological studies of a cyanobacterial infection on the Caribbean sea plume *Pseudopterogorgia acerosa* (Coelenterata: Octocorallia). Proc 6th Int Coral Reef Symp 3: 157-162

Garret P, Ducklow H (1975) Coral diseases in Bermuda. Nature 253: 349-350

Gladfelter WB (1982) White-band disease in *Acropora palmata*, implications for 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

Kuta KG, Richardson LL (1996) Abundance and distribution of black-band disease on coral reefs in the northern Florida Keys. Coral Reefs 15: 219-223

Littler MM, Littler DS (1996) Black-band disease in the South Pacific. Coral Reefs 15: 20

Miller I (1996) Black-band disease on the Great Barrier Reef. Coral Reefs. 15: 58

Moran PJ (1986) The *Acanthaster* phenomenon. Oceanogr Mar Biol Ann Rev 24: 379-480

Peters EC (1993) Disease of other Invertebrate Phlya: Porifera, Cnidaria, Ctenophora, Annelida, Echinodermata. In: Couch JA, Fournie JW (Eds.) Pathobiology of marine and estuarine organisms. CRC Press, Boca Raton, FL pp 393-449.

Richardson LL (1998) Coral diseases: what is really known? TREE 13: 438-443

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

Santavy DL, Peters EC (1997) Microbial pests: coral disease in the Western Atlantic. Proc 8th Int Coral Reef Symp 1: 607-612

Underwood AJ (1997) Experiments in ecology. Their logical design and interpretation using analysis of variance. Cambridge University Press, UK

Walton-Smith FG (1971) Atlantic reef corals. University of Miami Press, Florida

Woodley JD, Chornesky EA, Clifford PA, Jackson JBC, Kaufman LS, Knowlton N, Lang JC, Pearson MP, Porter JW, Rooney MC, Rylaarsdam KW, Tunnicliffe VJ, Wahle CM, Wulff JL, Curtis ASG, Dallmeyer MD, Jupp BP, Koehl MAR, Neigel J, Sides EM (1981) Hurricane Allen's impact on Jamaican coral reefs. Sci 214: 749-755

Zar JH 1999 Biostatistical analysis. 4th Edition. Prentice Hall Inc. New Jersey

Ecology, 83(2), 2002, pp. 436–451

DETECTING REGIONAL VARIATION USING META-ANALYSIS AND LARGE-SCALE SAMPLING: LATITUDINAL PATTERNS IN RECRUITMENT

T. P. HUGHES,[1,6] A. H. BAIRD,[2] E. A. DINSDALE,[2] V. J. HARRIOTT,[3] N. A. MOLTSCHANIWSKYJ,[2,4] M. S. PRATCHETT,[2] J. E. TANNER,[2,5] AND B. L. WILLIS[2]

[1]*Centre for Coral Reef Biodiversity, Department of Marine Biology, James Cook University, Townsville, Queensland 4811, Australia*
[2]*Department of Marine Biology, James Cook University, Townsville, Queensland 4811, Australia*
[3]*CRC Reef Research Center, James Cook University, Townsville, Queensland 4811, Australia*
[4]*School of Aquaculture, University of Tasmania, Launceston, P.O. Box 1214, Tasmania 7250, Australia*
[5]*SARDI Aquatic Sciences, P.O. Box 120 Henley Beach, South Australia 5022 Australia*

Abstract. Regional-scale variation of recruitment by marine organisms may reflect geographic patterns in adult stock sizes or fecundities, large-scale hydrodynamic features that influence the transport of larvae (e.g., currents, upwelling), and patterns of early mortality. In turn, recruitment may play a vital role in determining patterns of adult abundance and community structure, from local to biogeographic scales. We examined spatial variation in recruitment by corals at a regional scale, along 3300 km of the tropical and subtropical coast of eastern Australia (10°–31° S). We used two complementary approaches: (1) a meta-analysis of 21 different studies undertaken over a 16-yr period, each of which was generally conducted at a single reef, and (2) a large-scale sampling effort in which recruitment was measured in two years on 33 reefs arrayed along the length of the Great Barrier Reef (GBR). Our goal is to compare the emergent large-scale picture derived from many small-scale studies with patterns revealed by shorter-term regional sampling.

The two approaches show very similar large-scale patterns. Recruitment by spawning corals (mainly acroporids) was highest in the central GBR and declined steadily with increasing latitude by up to more than 20-fold. A smaller decline occurred on the northern GBR between Australian and Papua New Guinea. Recruitment by brooding corals (mostly pocilloporids) was greatest in the northern GBR and also declined to the south. The latitudinal decline in brooders was three- to fivefold, i.e., not as great as for spawners. Consequently, the proportion of brooded recruits increased to the south, and they generally exceeded spawners on the southern GBR and on isolated subtropical reefs at higher latitudes. Our meta-analysis shows that fully half of the variation in the ratio of spawners to brooders is attributable to one of 11 variables that we extracted from the published studies: the month when the recruitment panels were deployed. This result suggests that the intensity and timing of spawning have a crucial impact on large-scale patterns of recruitment. Elsewhere, we tested this hypothesis in the field, and confirmed that regional variation in recruitment by spawning acroporid corals was driven by spatial and temporal variation in the extent of mass spawning. Together, large-scale sampling and meta-analyses provide a powerful, combined approach for investigating large-scale patterns and the mechanisms underlying them.

Key words: coral reefs; Great Barrier Reef; larvae; meta-analysis; population dynamics; recruitment; spatial scale.

INTRODUCTION

The ability to compare and synthesize across studies is crucial for revealing general patterns and for scaling up from small-scale investigations to unveil regional or global phenomena. Meta-analysis (defined as the quantitative analysis of data that originated from several independent studies) provides major advantages over more traditional narrative syntheses and reviews (e.g., Hedges and Olkin 1985, Gurevitch and Hedges 1993). Following the lead from the social sciences (e.g., Glass et al. 1981) and medicine (e.g., Sachs et al. 1987), applications of meta-analysis to ecological data are becoming increasingly common (see recent overviews by Arnqvist and Wooster 1990, Osenberg et al. 1999). Regardless of whether the primary studies under investigation are descriptive or experimental, the underlying approach and objectives are the same: to quantify emergent patterns by applying statistical procedures, and to test for effects of ecological factors or methodology by analyzing subgroups of the overall data.

A growing awareness of scale dependency, advances in technology (e.g., satellite imagery, supercomputers)

Manuscript received 10 January 2000; revised 2 February 2001; accepted 18 February 2001; final version received 9 April 2001.

[6] E-mail: terry.hughes@jcu.edu.au

and increasing concern for environmental issues (e.g., habitat fragmentation, global warming) are encouraging ecologists to focus more on larger scale phenomena (e.g., Dayton and Tegner 1984, Edwards et al. 1994, Rosenzweig 1995). However, most ecological experiments and measurements are undertaken at relatively small scales of space and time, using relatively small sampling units (Schneider 1994). Ecologists often implicitly extrapolate their results across scales, typically inferring large-scale patterns from smaller scale investigations that are logistically easier to conduct. Unfortunately, these extrapolations are invalid because numerous processes (e.g., dispersal, evolution) prevail at larger scales which cannot be explored locally in space or time (see, e.g., commentaries by Wiens 1989, Rosenzweig 1995, Thrush et al. 1997). Schneider et al. (1997) suggested several solutions: undertaking larger-scale experiments (e.g., Carpenter 1990), combining or replacing small-scale experiments with large-scale surveys (e.g., Eberhardt and Thomas 1991), and iterative cycling between observation, small-scale experiments on components of a larger system, and refinement of theories on how the larger system works (e.g., Rastetter et al. 1992, Wiens et al. 1993). As yet, there is no clear consensus on how best to relate patterns and processes across multiple scales.

Meta-analysis is likely to be a useful tool for detecting large-scale patterns that extend beyond the resolution or capability of conventional experimental and descriptive studies. Thus, the limited scale of focus of most ecological investigations does not preclude the detection of large-scale phenomena if results can be integrated across many studies. A classic example is the long-term dynamics of the birds of Great Britain, revealed by censuses conducted since the 1930s by thousands of members of the British Trust for Ornithology (O'Connor 1985, Taylor 1987). Similarly, biogeographers can ascertain even global-scale spatial patterns based on many localized censuses or surveys, each conducted at one or a few locations, often for different purposes and by many different individuals (e.g., Stehli and Wells 1969). In some cases, it may be feasible to examine large-scale processes or patterns in a single intensive study (e.g., Hughes et al. 1999, 2000). Whether the results emerging from a meta-analysis and a large-scale study would actually be similar is an interesting question. We are unaware of any such comparison in the ecological literature.

In this paper, we set out to compare a meta-analysis of small-scale investigations with a single large-scale study. Comparing published studies would have been easy if they had all been done and reported in the same way. However, for reasons of logistics or personal choice, each one varied, often in ways that almost certainly affected the results. Our task therefore is to identify extraneous sources of variation in the data (e.g., due to methodology), account for them with statistical models, and explore the remaining variance that is attributable to the variables of interest. The large-scale pattern we investigated is the density and taxonomic composition of coral recruits along a 3300 km tropical–subtropical latitudinal gradient. Recruitment has a major influence on the size and composition of adult populations at all spatial scales (e.g., Gaines and Roughgarden 1985, Hughes 1990, Karlson and Levitan 1990, Caley et al. 1996, Connell et al. 1997, Hughes and Tanner 2000). Biogeographic patterns in the composition of coral assemblages (e.g., Stehli and Wells 1969) and their latitudinal limits (e.g., Crossland 1988) are likely to be strongly influenced by patterns of dispersal and recruitment. We focussed on the Great Barrier Reef and on isolated reefs to its south, where there have been 21 published reports on early recruitment by corals (on 18 separate reefs). In addition to these, we conducted a large-scale investigation of coral recruitment on 33 reefs from 10° S to 23° S, along the length of the Great Barrier Reef (Hughes et al. 1999, 2000). For convenience, we refer here to these two data sets as the small- and large-scale studies, respectively.

Harriott and coworkers were the first to compare several small-scale studies to examine latitudinal patterns in the density and composition of coral recruits along the east coast of Australia (Harriott and Simpson 1996). Based on data from seven studies conducted between 16° S and 31° S (see Table 2 in Harriott and Simpson 1996), she concluded that ''there is an apparent decline in the rate of recruitment of broadcasting (spawning) coral species with increasing latitude, with brooding corals being the dominant recruits at high-latitude sites'' (quote from Banks and Harriott 1996). Similarly, Dunstan and Johnson (1998) stated that ''the emerging picture (from the literature) is a transition from dominance of recruitment on settlement plates by (spawning) acroporids in central and northern regions of the GBR to dominance by (brooding) pocilloporids at the southern extremities of the GBR and on subtropical reefs to the south.'' However, neither of these conclusions was based on a formal meta-analysis of the literature. These patterns, if they occur, raise important issues concerning the mechanisms involved, and their ecological, biogeographical, and evolutionary consequences. Similar large-scale gradients in recruitment of benthic organisms occur on coastlines elsewhere. For example, changes in the abundance and population structure of echinoids along the west coast of North America (Ebert and Russell 1988), and of barnacles, mussels, and starfish on the east and west of New Zealand (Menge et al. 1999) are due in part to patterns of upwelling and the delivery of larvae. Recent modeling studies by Connolly and Roughgarden (1998, 1999) indicate the potential effects of regional-scale variation in recruitment on latitudinal patterns of adult abundances and community structure.

Study System

The Great Barrier Reef (GBR) is a continuous chain of nearly 3000 discrete reefs that stretches in a south-

easterly direction from 10° S to 23° S, along the coast of Queensland, Australia. Most of the reefs are 35–150 km offshore, depending in part on the width of the continental shelf. Isolated reefs and coral assemblages also occur south of the GBR, as far as Lord Howe Island (31° S), the southernmost coral reef in the world. The species richness of reef-building corals falls by ~25% between the middle and southern end of the GBR (to 245 species; Veron 1993). Eighty-seven of these extend 1100 km further south to Lord Howe Island (Veron and Done 1979, Harriott et al. 1995; T. P. Hughes, *unpublished data*). Patterns of water flow on the GBR are complex, largely because of the many gyres and eddies created by nearly 3000 reefs, and the effects of tides and variable winds. The main large-scale current flows westward towards Australia from the Coral Sea at 14°–18° S before bifurcating into a long-shore flow to the north and south (the Coral Sea Coastal Current and the East Australian Current, respectively; see Wolanski 1994).

Corals can be classified into two reproductive groups, brooders and spawners. Brooders release sperm, but not their eggs, which are fertilized internally to form relatively large planulae. After their release, brooded planulae have a short precompetency period (when they are not yet capable of settling) ranging from minutes to 2 d, depending on the species (Harrison and Wallace 1990). However, planulae may remain competent for weeks if they are deprived of a suitable settlement surface (under laboratory conditions, see, e.g., Richmond 1987). The release of planulae in brooders usually follows a lunar cycle, for up to 12 mo/yr depending on species and location (see review by Tanner 1996). The most abundant brooders on the GBR are species of Pocilloporidae (*Pocillopora, Stylophora, Seriatopora*), members of the *Acropora* subgenus *Isopora,* and some species of Poritidae (Harrison and Wallace 1990).

In contrast to brooders, broadcast spawners release both eggs and sperm, and fertilization is external. Most species of spawners on the GBR release their gametes in a multispecies spawning event which occurs over a period of a few days, in one or two months during the early austral summer (see Harrison et al. 1984, Babcock et al. 1986). Consequently, recruitment by most spawners is much more seasonal than brooders, with a major peak of settlement following closely after spawning (e.g., Wallace and Bull 1981, Wallace 1985*a*, Dunstan and Johnson 1998). The precompetency period of spawners is typically 3–7 d, more than twice as long as brooders. However, like brooders, broadcast-spawned larvae can remain viable for weeks (e.g., Wilson and Harrison 1998). Over 85% of coral species on the GBR are spawners. Levels of gene-flow in corals along the GBR range from modest to low (particularly for brooders; Ayre and Hughes 2000), with minimal genetic exchange occurring between the GBR and Lord Howe Island (D. J. Ayre and T. P. Hughes, *unpublished data*).

In this paper we conducted a meta-analysis of the existing literature to (1) quantify large-scale (latitudinal) patterns in recruitment by corals along the east coast of Australia, (2) measure regional changes in the proportion of recruit taxa, specifically brooders vs. spawners, and (3) compare patterns that emerge using the meta-analysis of small-scale investigations to those revealed by a single large-scale study. Our analyses points to the valuable role of meta-analysis in synthesizing results from many studies, but also highlights some limitations compared to large-scale investigations that are explicitly designed to examine regional phenomena.

Methods

The small- and large-scale studies share a basic characteristic: they all involved the deployment of replicate artificial substrata (recruitment panels), which were subsequently retrieved and censused. To avoid bias in our selection of cases for the meta-analysis, we included any publications from the study region (east Australia) that examined recruitment by corals onto artificial panels attached to hard substratum. There are 21 such primary studies, published from 1985 to 1999. The universal metric reported in these studies (and in the large-scale study) is the density of coral recruits per panel. In most cases, recruits were also classified into taxonomic categories, which allows us to examine spatial variation in both their total abundance and composition. We first plotted regressions of recruitment vs. latitude, to compare the two data sets. Then we conducted a detailed meta-analysis of the small-scale studies to further explore sources of variation in recruitment (e.g., due to methodological differences).

Meta-analysis of small-scale studies

Our task was to account for variation among previously published studies due to differences in methodology and latitude, using multiple regression models. We examined four dependent variables separately in the meta-analysis: total recruits per panel (all coral taxa combined), number of spawners, number of brooders, and the proportion of spawners to brooders. Analyses were done on both the mean number of recruits per panel, and the standardized number per 286 cm^2 (the surface area of all panels in the large-scale study). The results were very similar, so we report here only on the latter. We recorded the following 11 independent variables for each small-scale study: the size and composition of panels; the method of deployment; the month, year, and duration of deployment; whether the deployment period included the month when mass spawning occurred; depth and habitat; distance offshore; and latitude. Many of these variables are correlated (see *Results*). We chose the following variables

because they are very likely to have affected the amount of recruitment:

1) Panel size: Large panels should have a greater number of recruits, but they may have a lower overall density (due to ''edge effects'' which occur when new recruits are clustered close to the edge, presumably in response to gradients of light and water flow).

2) Panel composition: The chemical composition of recruitment panels and their rugosity or texture may affect patterns of settlement and early mortality (e.g., Harriott and Fisk 1987). The small-scale studies used six types of panels that were made of ceramic, fired clay, cement, PVC, and flat slices of dead corals.

3) Method of deployment: The published studies used three methodologies for the deployment of settlement surfaces (individual deployment of panels; panels bolted to racks side by side; panels attached to racks in vertical pairs, forming a ''sandwich'').

4) Duration: In any recruitment study, the longer panels are submerged the greater the opportunity for receiving multiple cohorts of larvae. However, losses of recruits due to post-settlement mortality will also be greater the longer panels are exposed. We recorded the duration of each study in weeks.

5) Month, year, and the timing of spawning: Temporal patterns of recruitment often reflect variation in the availability of competent larvae, e.g., in response to seasonal breeding cycles, or changes in hydrodynamic conditions (e.g., Babcock 1988, Milicich 1994, Hughes et al. 2000). We recorded the year and the month of initiation (when the panels were deployed) for each study. Whether or not the deployment included the month when mass spawning occurred (for that year and location) was recorded as a discrete variable, hereafter called ''spawning.''

6) Latitude, distance from shore, habitat, depth: The latitude and distance from shore were recorded for each of the 21 small-scale studies. We categorized the habitat of each study into three types (lagoon, reef crest, reef slope). Finally, we recorded depth as a continuous variable in meters.

We used generalized additive regression models (GAM, see Hastie and Tibishirani 1990) to determine the best combination of these 11 factors that explained variation in total recruitment and recruitment by spawners and brooders separately. We first examined the correlations among the independent variables and the proportion of the variation in recruitment that was explained by each one on its own. Subsequently, we used a forward-backwards stepwise method to select the best subset and best sequence of predictors among the independent variables. At each step, we added to the model the next variable with the highest F value and lowest P value (provided $P < 0.05$). After the addition of each new variable, all existing variables in the model were rechecked to ensure that they still contributed appreciably to the fit, and variables with $P < 0.05$ were deleted. This process continued iteratively until no other variable remained which added significantly to the model. We constructed alternative models if at any stage it was unclear which variable to add or delete from the model (e.g., because of similar F and P values), and the completed models were compared using an analysis of deviance (F test). To examine variation in the proportion of spawners and brooders, we followed a similar procedure, except a binomial distribution (rather than a normal one) was used, and alternative models were compared using the χ^2 distribution. Five of the independent variables were continuous (size of panel, duration of deployment, depth, latitude, and distance from the shore), while four others were categorical (composition of panels, method of deployment, whether or not the deployment overlapped with the annual mass spawning, and habitat). The remaining two independent variables of the 11 we examined, the month and year of deployment, were entered as both continuous and categorical variables, and the one with the best fit was retained. Continuous variables were entered into the model as linear variables with one degree of freedom, or as nonlinear variables using spline functions with four degrees of freedom (if the GAM indicated that the nonlinear component was significant with $P < 0.05$). One degree of freedom fits a straight line whereas n degrees joins all points. Four produces ''modest'' smoothing (Hastie and Tibshirani 1990).

Independence of data

The definition of an independent result is important, especially for selecting multiple observations arising from a single primary study. Data collected by the same person, on the same reef, and repeatedly over time, are unlikely to be statistically independent. However, identifying which data are spatially or temporally independent from the published literature is usually impossible, or at best very subjective and a potential source of bias (e.g., Downing et al. 1999, Englund et al. 1999). Operationally, meta-analysis of published results precludes rigorous testing for spatial and temporal autocorrelations, since the original raw data are usually unavailable. Because our aim is to explore the published data as much as possible, we used multiple results from each publication wherever we could, i.e., whenever different sets of panels were deployed as part of a single study at different sites, depths, or times. This approach to meta-analysis is not unusual as a descriptive tool, e.g., Goldberg et al. (1999) examined the relationship between competition and productivity in plants using a database of 296 cases from only 14 primary studies. Similarly, our meta-analysis is based on up to 253 cases from the 21 published studies.

The large-scale study

The large-scale study examined variation in recruitment by corals at multiple scales, from meters to the length of the Great Barrier Reef (from 10° S to 23° S;

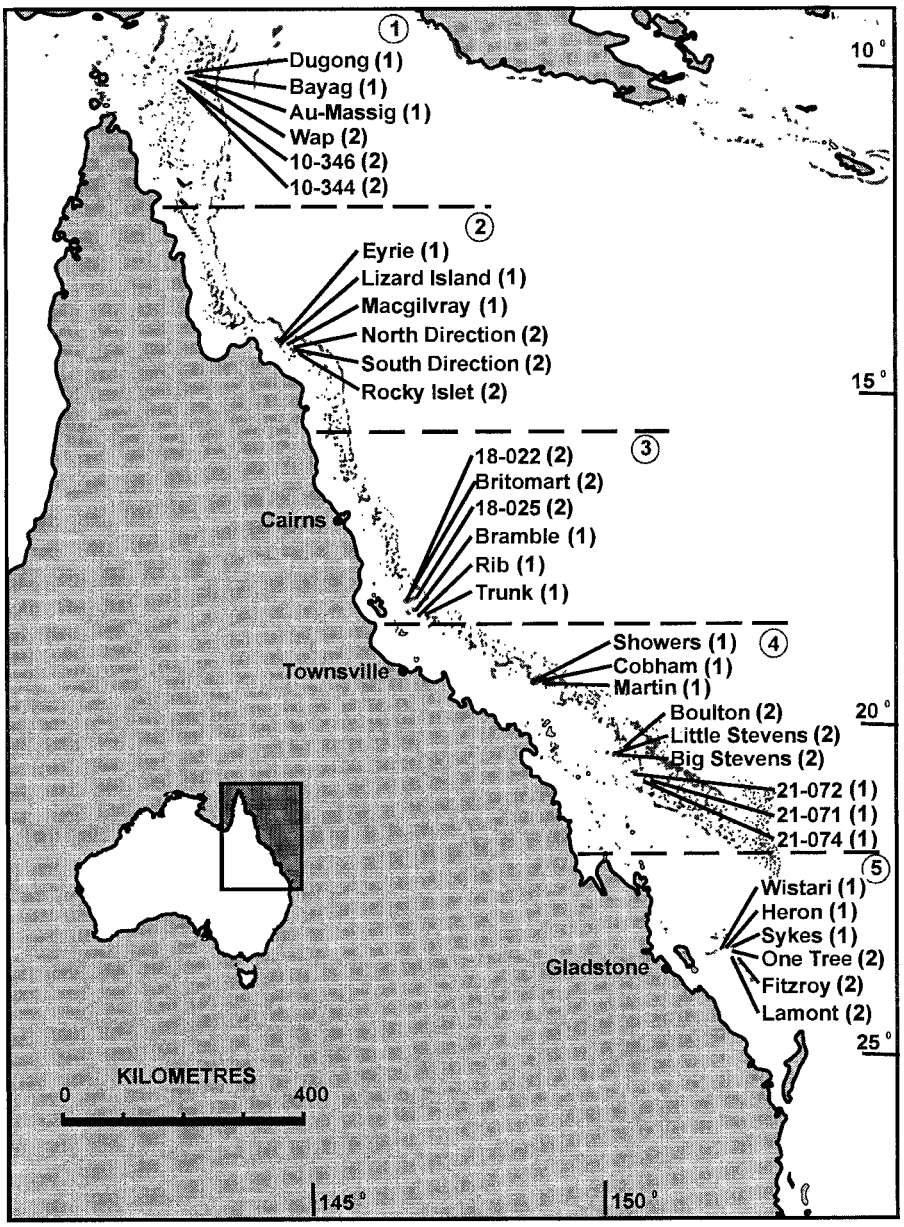


FIG. 1. Map of the Great Barrier Reef (GBR), indicating the location of reefs where recruitment of corals has been measured by the large-scale study. Note the hierarchical design, with 18 reefs in five sectors sampled during 1995-1996 (1), and a further 15 reefs during 1996–1997 (2). Sectors of the GBR are each 250–500 km apart, numbered 1–5 from north to south. The Coral Sea extends eastward, offshore from the GBR.

Fig. 1). We used a hierarchical sampling design which allowed us to partition variation (using nested ANOVAs) among four spatial scales: i.e., among five adjacent sectors from north to south along the GBR, among three to six neighboring reefs nested within each sector, among four replicate sites on each reef, and among 10 recruitment panels deployed at each site (see Hughes et al. [1999, 2000] for these analyses). Deployments were done in two consecutive years, 1995/6 and 1996/7 (year one and two, respectively). In year one, we targeted 18 reefs. In year two, panels were placed on 15 additional reefs. Thus, 40 panels were placed on each of the 33 reefs. Note that the panels were deployed on different reefs in separate years since our objective was to measure the effects of spatial scale rather than the predictability of recruitment at any particular site or reef (which would take many years to establish). To facilitate comparison with the small-scale studies, we report here on reef-scale patterns using the mean amount of recruitment (averaged for the 40 panels) on each reef plotted as a data point against latitude.

In contrast to the small-scale studies, the method-

TABLE 1. Attributes of the 21 small-scale studies used in the meta-analysis (see Fig. 1 for the locations) and of the large-scale study.

No.	Study	No. years	Month initiated	Deployment duration (d)	Latitude (° S)	No. reefs	Depth (m)	Panel material	Panel size (cm^2)
1	Baird and Hughes (1997)	1	Jan	56	14.41	1	1	clay	484
2	Harriott (1985)	1	Nov	56	14.41	1	1, 9	coral	100
3	Maida et al (1995b)	1	Oct	266	14.41, 18.40	2	4, 5	ceramic	675
4	Fisk and Harriott (1990)	2	Oct, Mar	182	15.50–16.45	3	4	ceramic	900
5	Harriott and Fisk (1987)	1	Oct	140	16.39	1	4	various	150–800
6	Harriott and Fisk (1988)	2	Apr	182	16.32–16.45	3	4	coral	400
7	Fisk and Harriott (1994)	1	Nov	147	16.41	1	3	ceramic	900
8	Sammarco and Carleton (1981)	1	Nov	119	18.26	1	10	coral	600
9	Sammarco (1991)	1	Jan, Jul	182, 365	18.16–18.49	3	3, 15	coral	150–158
10	Sammarco and Andrews (1988)	1	Oct to Mar	210	18.38	1	18	coral	600
11	Maida et al (1995a)	1	Oct	266	18.40	1	4	pvc	150
12	Wallace & Bull (1981)	1	Jul, Oct	112, 224	18.55	1	0–12	coral	200
13	Wallace (1985a)	3	Feb, Jun, Oct	119	18.55	1	0–15	coral	200
14	Wallace (1985*b*)	1	Oct	119	18.55	1	0–15	coral	200
15	Babcock (1988)	1	Dec, Sept	45	19.00	1	8	ceramic	576
16	Mundy (2000)	1	Jan	112	23.26	1	9	clay	572/286
17	Bothwell (1981)	1	Mar, Jul, Nov	122	23.27	1	2	cement	79
18	Dunstan and Johnson (1998)	4	Sept	135, 365	23.27	1	1	ceramic	400
19	Banks and Harriott (1996)	2	Jul, Nov	56–175	26.38	3	10, 19	ceramic	900
20	Harriott & Banks (1995)	3	Oct, Mar	182	30.18	1	8, 6	ceramic	900
21	Harriott (1992)	2	Nov, Mar	56–238	31.33	1	6, 13	ceramic	900
	Large-scale study	2	Nov, Dec	55–57	10.28–23.38	33	1	clay	286

ology of the large-scale study was standardized as much as possible so that nearly all of the 11 independent variables described above were controlled for (the major exception being latitude). In each year, the panels were deployed synchronously at all sites on all reefs, 10 d (±1) before the predicted annual mass spawning of corals, and retrieved 8 wk later. This uniform duration allowed enough time for large numbers of corals to settle, and for recruits to grow to a sufficient size (usually 1–2 mm) to allow limited taxonomic resolution (generally at the family level). The habitat and depth was the same on all reefs: shallow reef crests, ~1 m below datum. The panels were identical, unglazed clay tiles (11 × 11 × 1 cm) attached individually to the substratum by a bolt that held them 2–3 cm above the reef surface. A total of 1135 panels (87%) were relocated using GPS at the 132 sites on the 33 reefs. The retrieved panels were bleached and recruits on all surfaces were counted using a dissecting microscope. Juvenile corals were identified to family (or genus where possible), and classified as spawners or brooders.

RESULTS

Comparison of data sets

The scope of the large- and small-scale studies was quite similar. The former is based on a total of 58 471 recruits on 1135 panels that were deployed on 33 reefs. The 21 published studies have a combined sample size of 47 682 recruits on 538 panels from 18 different reefs (Table 1). In the large-scale data set, 83% of the recruits were spawners and 17% were brooders. In the small-scale studies, 33 370 recruits (70% of the total) were classified into different taxonomic groupings. Of these, 61% were spawners and 39% were brooders. The higher proportion of brooders in the small-scale studies reflects differences in methodology compared to the large-scale data set, and the greater southerly range of the individual studies.

As expected, there were huge differences among the small-scale studies in the 11 independent variables that we examined. For example, the censused surface area of panels was generally constant within studies, but

varied 11-fold among them (from 79 to 900 cm^2). Similarly, the duration of each study varied eightfold, from 6.5 to 52 wk. In comparison, the large-scale study used relatively small panels (121 cm^2) and had a short, uniform duration of 8 wk. The small-scale studies were conducted in 14 of the 16 separate years between 1979 and 1994 (inclusive), with initial deployments in ten different months (none were in May or August, during the Austral winter). Ten of the 21 primary studies had deployment periods that did not include the peak summer mass spawning of corals. In contrast, the large-scale study sampled only 2 yr, and the deployment was highly synchronized to precede the predicted mass spawning by 9–11 d. The depth range of the small-scale studies ranged from zero (intertidal) to 19 m, compared to a uniform 1 m depth for the entire large-scale data set. The small-scale studies were located at muddy inshore sites, on mid- and outer-shelf reefs, and on oceanic islands up to 580 km offshore. In contrast, the large-scale study was restricted to midshelf reefs, roughly halfway between the Australian mainland and the edge of the continental shelf (see Fig. 1).

The spatial array of study locations differed substantially between the large-scale and small-scale studies. Reefs in the large-scale data set were distributed in five to six sectors from north to south, more or less uniformly along the length of the GBR (Fig. 1). Not surprisingly, the regional spread of reefs comprising the small-scale data set was more haphazard (Fig. 2) since the individual studies were never designed to be a single sampling exercise. No reefs were sampled in the top 30% of the GBR to the north of Lizard Island (14°40′ S), while over two-thirds of the studies were conducted very close (<100 km) to Cairns or Townsville in the central portion of the GBR (roughly 17° and 19° S, respectively). Only three small-scale studies were undertaken on the southern 40% of the GBR to the south of Townsville, all of them on a single reef, Heron Island (23° S). Three additional studies were conducted south of the GBR (see Table 1), extending the small-scale data set from 14° to 31° S, compared to 10°–23° S for the large-scale study. The geographic extent of the overlap between the two data sets is ~1000 km, or 9° of latitude.

Latitudinal patterns of recruitment

The large- and small-scale studies both reveal a steady 20-fold decline in total recruitment (all taxa combined) from approximately 14° S to the geographic limit of coral reefs, 2100 km to the south (Fig. 3a; adjusted $r^2 = 0.288$, $P < 0.01$, and 0.246, $P < 0.001$, respectively). Recruitment by all taxa and by spawners exhibits a similar large-scale pattern, due to the numerical dominance of spawners (Fig. 3a, b). The large-scale study shows a greater effect of latitude, which accounted for 29% of the variance in spawners compared to 20% in the small-scale data set. The trend, however, is not a simple north–south gradient. According to the large-scale study, recruitment peaked in the central portion of the Great Barrier Reef in both years, and declined to the north as well as the south (Fig. 3a, b). The smaller-scale studies did not sample the northernmost portion of the Great Barrier Reef, but they confirm the southerly decline and establish that the trend extends beyond the highest latitudes of the large-scale study to the southern limits of coral reefs in the Pacific Ocean.

Recruitment by brooders also shows a north–south decline in both data sets (Fig. 3c), although the trend was not significant for the small-scale studies, with latitude explaining only 2% of the variation. In contrast, latitude accounted for a third of the variation in brooders in the large-scale study (adjusted $r^2 = 0.344$, $P < 0.001$). Brooders did not decline as quickly to the south as spawners in either data set (compare Fig. 3b and c). Consequently, the proportion of spawners declined at higher latitudes (Fig. 4). In the middle two-thirds section of the GBR (~12°–20° S; Fig. 1), spawners predominated in the large-scale study, making up close to 90% of recruits. The southern and northern ends of the GBR both show a decline in numbers of spawners, with a corresponding rise in the proportion of brooders. Consequently, the proportion of spawners was highly correlated with latitude in the large-scale study (adjusted $r^2 = 0.330$, $P < 0.001$, Fig. 4). The small-scale studies showed a much more variable and generally lower proportion of spawners than the large-scale data set, but also exhibited a significant (but much weaker) trend for a greater proportion of brooders at higher latitudes (adjusted $r^2 = 0.048$, $P < 0.05$, Fig. 4).

In summary, both data sets reveal significant regional-scale variation in the amount and composition of recruits. Less variation in recruitment was explained by latitude in the small-scale data set, i.e., ''unexplained'' variation within latitudes was greater, particularly for brooders where the latitudinal signal was not statistically significant. Next we use meta-analysis to explore how methodological differences among the small-scale studies contributed to this large residual variation.

Meta-analysis of small-scale studies

Here we use as many cases as possible from the published papers, i.e., multiple deployments of panels at different sites and times were treated as replicates. We found highly significant, but generally weak, correlations among all of the continuous independent variables that we examined (Table 2). Of particular interest, latitude was confounded with the year that the study began ($r = 0.50$, $P < 0.001$), the size of panels ($r = 0.37$, $P < 0.001$), distance from shore ($r = 0.37$, $P < 0.001$), depth ($r = 0.30$, $P < 0.001$), the month of deployment ($r = 0.21$, $P < 0.001$), and the duration of deployment ($r = -0.19$, $P = 0.003$). Specifically, compared to northern studies, those done in the south were

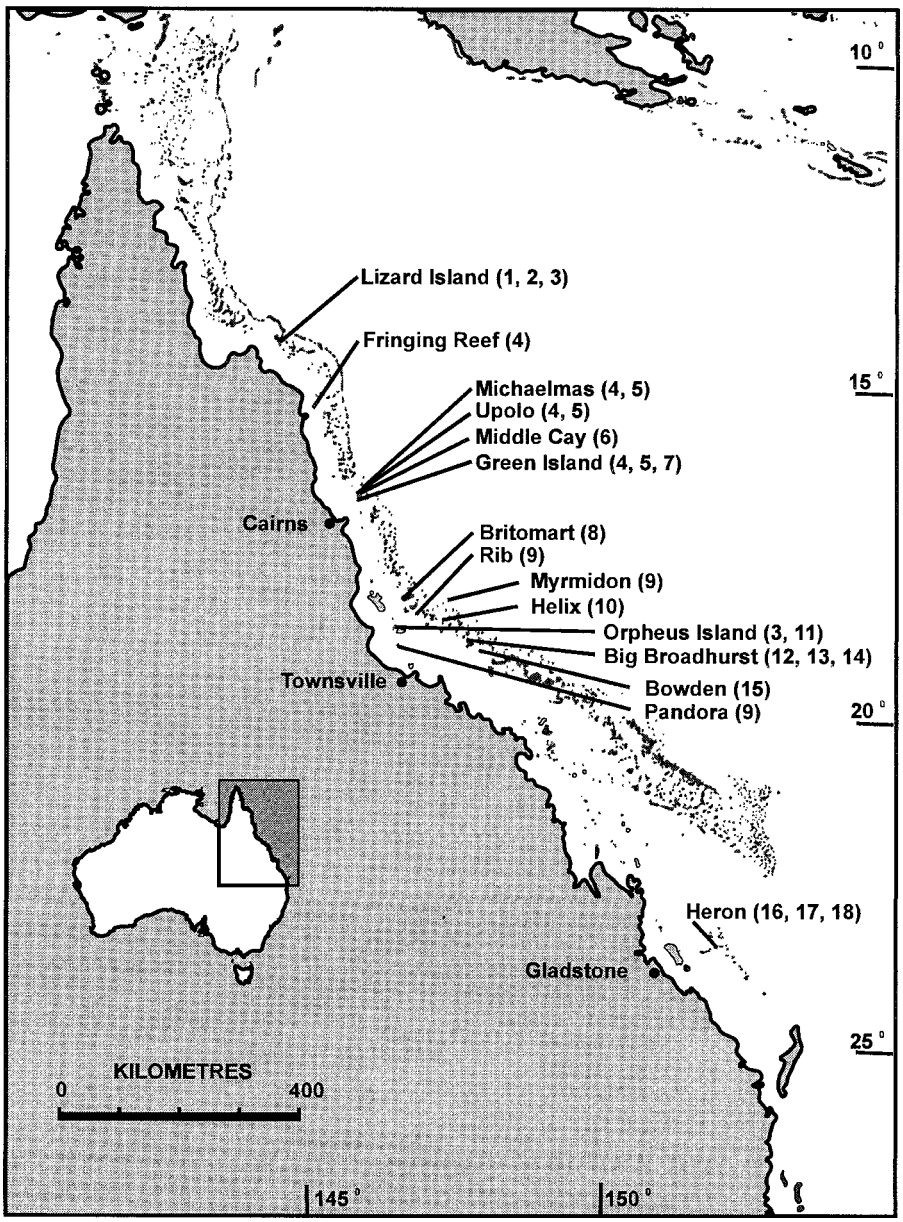


FIG. 2. Map of the Great Barrier Reef (GBR), indicating the location of reefs where recruitment of corals has been measured in 18 independent studies. A further three studies (not shown) conducted on islands to the south of the GBR at 26°–31° S were also included in the meta-analysis (see Table 1 for details of the 21 studies).

generally undertaken in more recent years, using larger panels that were deployed further offshore and in deeper water, with deployments beginning later in the year and lasting on average for a shorter period. We first examined the effects of each of the 11 independent variables separately, and then entered them sequentially into multiple regression models.

In the meta-analysis, latitude on its own explained less variation than the reef-scale regressions presented earlier (Fig. 3) because of the considerable within-reef scatter in the data. Consequently, only 5.7% of the variation in total recruitment (i.e., all taxa combined, $F_{1, 248} = 15.02$, $P < 0.001$) and 7.3% of the variation in spawners ($F_{4, 164} = 3.22$, $P = 0.014$) was explained by latitude. Moreover, there was no effect of latitude on the density of brooders ($F_{4, 155} = 1.50$, $P = 0.20$). Therefore, a significant effect of latitude on the proportion of spawners to brooders (accounting for 11.9% of the variation, $P = 0.027$) is attributable to a decline in spawners at southern sites rather than an increase in brooders. Recruitment was often related more strongly to individual variables other than latitude, particularly to those that measure temporal aspects of the deployment of panels. In contrast, three of the independent

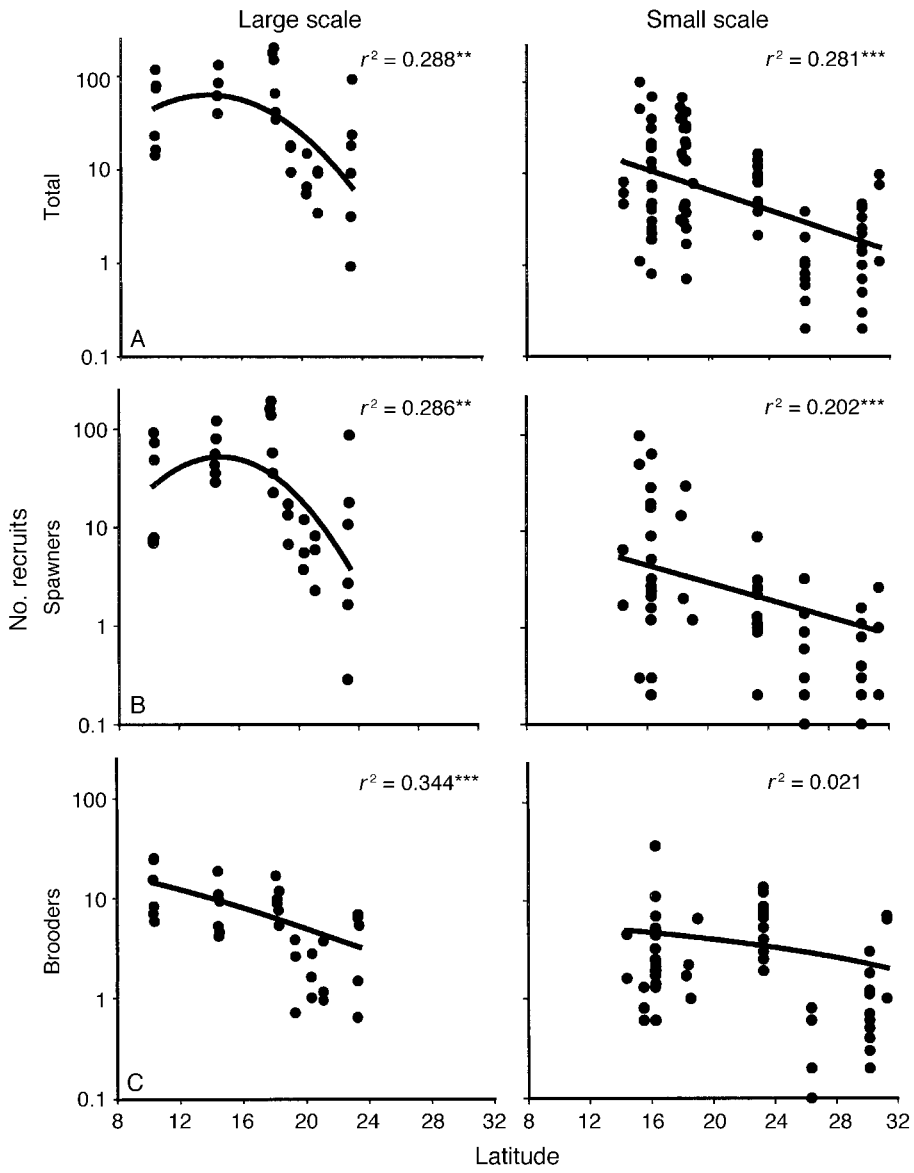


FIG. 3. Recruitment of corals vs. latitude along the east Australian coastline. Data from the large-scale study (left) and from 21 published studies (right, see Table 1). Recruitment (A) by all coral taxa; (B) by spawning corals; and (C) by brooders. Each point represents the mean number of recruits per panel on a single reef (all sites combined).
** $P < 0.01$, *** $P < 0.001$.

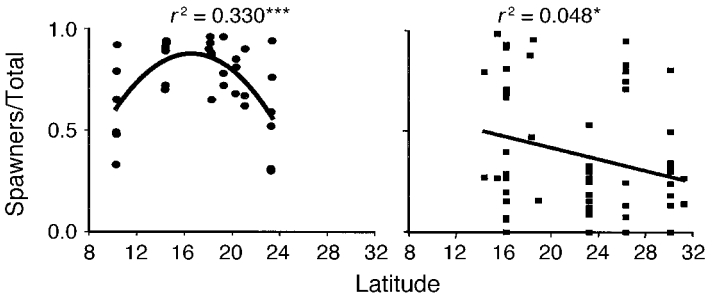


FIG. 4. The proportion of recruitment due to spawners vs. latitude, in the large-scale study (left) and in the 21 published studies (right). Zero on the *y*-axis represents 100% recruitment by brooders.
* $P < 0.05$, *** $P < 0.001$.

TABLE 2. Pearson correlations among the continuous variables used in the meta-analysis of small-scale studies.

Variable	Latitude	Year	Panel size	Distance from shore	Depth	Month initiated
Year	0.497***					
Panel size	0.372***	0.543**				
Distance from shore	0.372***	0.045	0.038			
Depth	0.299***	0.043	0.036	0.103		
Month initated	0.205***	0.131	0.227**	0.074	0.149*	
Duration	−0.194**	−0.041	−0.142*	−0.061	−0.015	−0.087

Note: Based on 250 records of mean number of recruits per panel, i.e., spawners and brooders combined.
* $P < 0.05$, ** $P < 0.01$, *** $P < 0.001$. Tests are two tailed.

variables we examined (the panel composition, method of deployment, and habitat) had no explanatory power for any recruitment measure (each accounting for <2% of the variation, with P values usually >0.5), and were not considered further.

Total recruitment was significantly correlated with the month and year of deployment, whether the deployment included the month of mass spawning (spawning), depth, distance from shore, duration and habitat, and latitude (Table 3a). Each of these variables on their own explained 3–10% of the variation in overall recruitment. Entering latitude first into our GAM analysis provided the best model. Once latitude was accounted for, spawning and then distance from shore explained an additional 7.4% and 10.0% of the variation, respectively (giving a total of 23.1%). These different percentages are both higher than the variation attributable to each variable on its own, because of the correlations between them (see Table 2). No other variable added significantly to the model beyond these three. The pattern for recruitment by spawners was broadly similar, reflecting their overall numerical dominance. The same three variables were important (distance from shore, latitude, and spawning), although their sequence in the model was different (Table 4). Each one explained slightly more variation than for the total recruits model, accounting for a combined total of 29.9% of the variation in spawners. Thus, the meta-analysis confirms the latitudinal trends that were also detected by the large-scale study.

In contrast, recruitment by brooders was correlated only with distance from shore and panel size (Table 5a). Distance from shore became nonsignificant when panel size was entered first into the multiple regression model (because it had the larger F value), and year became significant ($F_{13, 154} = 1.95$, $P = 0.029$). Together, panel size and year explained 18.2% of the variation in number of brooders (Table 5b). There was no effect of latitude on recruitment by brooders, either on its own ($F_{1, 167} = 1.9$, $P = 0.11$), or in combination with the other independent variables. In marked contrast, the large-scale study found a significant north–south decline in recruitment by brooders (Fig. 3c).

Finally, variation in the ratio of brooders to spawners in the small-scale studies was explained most by the month of panel deployment, and by spawning (whether the deployment included the month of mass-spawning; Table 6). Obviously, these two are strongly correlated. Distance from the shore ($P = 0.008$), latitude ($P = 0.027$), panel size ($P = 0.029$), and year ($P = 0.035$), each accounted for 11–15% of the variation. None of these variables remained significant when the month of deployment was entered first into the additive regression model. This single variable accounted for almost

TABLE 3. Generalized additive regression models (GAM) showing the percentage variation in the total number of coral recruits in the small-scale studies that is attributable (A) to individual variables and (B) to sequential combinations of variables.

Variable	Type	df	F	P	Variation (%)
A) Individual variables					
Month initiated	discrete	9, 240	3.04	0.002	10.24
Year	continuous	4, 245	4.65	0.001	7.06
Latitude	continuous	1, 248	15.02	<0.001	5.71
Spawning	discrete	1, 248	13.79	<0.001	5.33
Depth	continuous	4, 245	3.41	0.010	5.27
Distance from shore	continuous	4, 245	3.13	0.016	4.86
Duration	continuous	4, 245	2.85	0.024	4.45
Habitat	discrete	2, 247	4.04	0.019	3.17
B) Sequential combination of variables					
Latitude	continuous	1, 248	15.02	<0.001	5.71
Spawning	discrete	1, 247	21.17	<0.001	7.44
Distance from shore	continuous	4, 243	7.88	<0.001	9.98
Total					23.13

TABLE 4. Regression models showing the percentage variation in the number of spawned recruits in the small-scale studies which is attributable (A) to individual independent variables, and (B) to sequential combinations of variables.

Variable	Type	df	*F*	*P*	Variation (%)
A) Independent variables					
Month initiated	discrete	8, 160	3.95	<0.001	16.48
Distance from shore	continuous	4, 164	6.78	<0.001	14.19
Latitude	continuous	4, 164	3.22	0.014	7.28
Year	continuous	4, 164	2.63	0.036	6.03
Spawning	discrete	1, 167	8.46	0.004	4.82
B) Sequential combination of variables					
Distance from shore	continuous	4, 164	6.78	<0.001	14.19
Latitude	continuous	1, 163	19.93	<0.001	9.35
Spawning	discrete	1, 162	14.75	<0.001	6.38
Total					29.92

half (47%) of the variation in the proportion of spawners in the small-scale studies.

DISCUSSION

Meta-analysis of large-scale patterns

Meta-analysis is a developing method for quantitatively synthesizing results across studies. Recent reviews and applications have emphasized its great potential, particularly in relation to experimental data, where studies can be compared in terms of a common metric of effect size (e.g., Gurevitch and Hedges 1993, Osenberg et al. 1999). Meta-analysis also has the potential to reveal large-scale patterns in space or time from smaller-scale descriptive data (e.g., Stehli and Wells 1969, O'Connor 1985, Taylor 1987). Our study is unusual because we have the capacity to compare such a meta-analysis with a large-scale study of the same system.

The two approaches we used (large-scale sampling and meta-analysis) both have their strengths and weaknesses. One general conclusion revealed by our comparison is that ''missing data'' is likely to be a significant impediment to meta-analysis of regional-scale patterns. In many cases, the spatial extent and/or distribution of the small-scale studies will be unsuitable for detecting latitudinal trends, since individual studies are not designed for this purpose. For example, a strong clustering of studies near centers of research would result in regression analysis of regional gradients being heavily influenced by a handful of points from poorly studied regions. In our study, the uneven distribution of reefs in the small-scale data set (Fig. 2) undoubtedly reflects the easier access to study sites that are located close to maritime centers (Townsville and Cairns), or to two major field research stations on the GBR (on Lizard and Heron Islands at 14° and 23° S, respectively). Furthermore, the hump-shaped pattern in recruitment by all taxa and by spawners from 10° to 23° S on the GBR (Fig. 3) could not be confirmed by the meta-analysis of small-scale studies because none of them extended further north than 14° S. Large-scale studies are more likely to have an evenly distributed spatial arrangement, and a hierarchical design and analysis will reveal small- and medium-scale variation as well as the overall, regional trend (e.g., Hughes et al. 1999, 2000). Undoubtedly, much of the unexplained variation in the small-scale studies is due to local differences from site to site within reefs, which cannot be partitioned out because the overall sampling ''design'' is not nested or balanced.

TABLE 5. Regression models showing the percentage variation in the number of brooded recruits in the small-scale studies which is attributable (A) to individual independent variables, and (B) to sequential combinations of variables.

Variable	Type	df	*F*	*P*	Variation (%)
A) Independent variables					
Year	discrete	13, 155	1.45	0.140	10.81
Distance from shore	continuous	4, 164	3.04	0.019	6.91
Panel size	continuous	4, 164	8.43	0.004	4.80
B) Sequential combination of variables					
Panel size	continuous	1, 167	8.43	0.004	4.80
Year	discrete	13, 154	1.95	0.029	13.43
Total					18.23

Note: Recruitment by brooders was not significantly correlated with latitude. Year became significant once the effects of panel size were accounted for.

TABLE 6. Regression models showing the percentage variation in the ratio of spawners to brooders in the small-scale studies that is attributable to individual variables.

Variable	Type	df	*P*	Variation (%)
Month initiated	discrete	8, 151	<0.001	47.02
Spawning	discrete	1, 158	<0.001	23.26
Distance from shore	continuous	4, 155	0.008	15.02
Latitude	continuous	4, 155	0.027	11.86
Panel size	continuous	4, 155	0.029	11.75
Year	continuous	4, 155	0.035	11.17

Note: Once the effects of month initiated was accounted for in multiple regression models no other variable remained significant.

The choice of which studies to include in a meta-analysis often has a critical effect on the patterns that emerge (e.g., Englund et al. 1999). Selection of studies is often subjective (e.g., based on a perception of the quality of the data, the amount of replication, experience of the authors, etc.), and there is an unfortunate tendency for bias towards choosing a subset of the available information which support a preconceived outcome (Mahoney 1977). Accordingly, we chose every available study of coral recruitment from the geographic region of interest. Another source of error in meta-analyses is the tendency for authors not to publish negative results (e.g., a nonsignificant experimental outcome), the so-called ''file drawer effect.'' This could also happen with descriptive data, e.g., if estimates of abundance that were zero or very low were not reported. We canvassed our colleagues working on recruitment of corals in Australia to rule out this possibility. It is no accident, however, that much of the literature on coral recruitment comes from the Great Barrier Reef, because rates of recruitment reported from elsewhere (e.g., in the Caribbean) are often much lower (e.g., Birkeland 1988, Richmond and Hunter 1990, Hughes et al. 1999, and references therein). Consequently, a paucity of published data from locations with very low recruitment would make a global meta-analysis problematical. In general, meta-analysis is unlikely to be fruitful where the range of the whole data set is small or where mean data values are close to zero. In our study, for example, meta-analysis detected the 20-fold latitudinal decline in the density of spawned recruits, but failed to resolve the more subtle regional-scale variation in brooders.

The inclusion of multiple years (14 separate years over a 16-yr period) in the small-scale data set potentially provides a major advantage over the large-scale study, because a longer time-scale can reveal spatial patterns that are not wholly consistent among years. Furthermore, a multi-year meta-analysis can explicitly examine longer term temporal variation. In our analysis, the year of each study had no significant effect on total recruitment, recruitment by spawners, or the ratio of brooders to spawners (Tables 3a, 3b, 5), indicting that the regional-scale patterns are consistent over time. However, recruitment by brooders did vary significantly from year to year (Table 3b), which may account for the failure of the meta-analysis to detect a consistent regional pattern. In contrast to the meta-analysis, the large-scale study was conducted only twice, in two consecutive years, one or both of which conceivably could have been unrepresentative. However, the patterns of recruitment in both years of the large-scale study were very similar (see Hughes et al. 1999, 2000), although this may well have been sheer good luck. Generally, the cost of large-scale sampling is substantial, which makes it difficult to repeat. Meta-analysis, on the other hand, by definition involves no new field costs since multi-year data can be derived from the literature.

The main drawback of using small-scale investigations to detect regional patterns is the noise in the data due to differences in methods among published studies. In our analysis, these methodological differences (e.g., distance from shore, panel size, depth, etc.) had surprisingly modest effects (Tables 3–5), but this is unlikely to be generally true, especially where regional-scale trends are more subtle than the order of magnitude variation in recruitment that we examined. Furthermore, latitude was positively or negatively correlated with most of these variables (Table 2). This is likely to be a general (and undesirable) property of meta-analysis: a nonrandom spatial distribution of methodologies arising from different research teams in different locations. These correlations raise the possibility that any large-scale pattern detectable in small-scale studies could simply be due to regional variation in methodology rather than biology. Alternatively, regional patterns could be partially obscured by confounding methodologies. In our study, we explicitly accounted for differences in methodologies, and the concordance between the small- and large-scale data sets gives us some confidence in concluding that the latitudinal patterns in recruitment are indeed real. Moreover, we are beginning to understand some of the processes that are responsible for the regional trends (see *Discussion: Mechanisms of large-scale recruitment variation*).

In summary, a large-scale study has numerous advantages over meta-analysis in terms of the comparability of data from different locations or census intervals. A single regional-scale study is also more likely to be developed in conjunction with predetermined sta-

tistical procedures, based on a uniform sampling design. The methods are invariably more homogeneous, the results are always expressed as the same metric, and the involvement of fewer people with similar training means that there is less likelihood of bias between observers or between research groups. Most importantly, the data are likely to be less noisy, unencumbered by extraneous methodological factors which often differ among individual small-scale studies. Of course, the downside is the expense, time, and effort associated with a larger sampling regime compared to an analysis of previously published studies (although we strongly suspect that our single-regional study of recruitment by corals was cheaper than the combined cost of the 21 individual field studies). Furthermore, sampling or experiments conducted at larger spatial scales are more difficult to repeat, and the limited temporal window could be unrepresentative.

Mechanisms of large-scale recruitment variation

The results from meta-analysis are particularly useful because they can be used to generate hypotheses addressing the mechanisms underlying large-scale patterns. The meta-analysis presented here demonstrates that the timing of deployment of panels had a critical impact on the amount and species composition of recruits. Intuitively, we would expect low rates of recruitment when and where few larvae are produced. Conversely, higher recruitment should result in time periods (seasons or years) or regions that have higher rates of production of larvae. Elsewhere, (as a component of the large-scale study) we tested the hypothesis that variation in recruitment by spawning acroporids (Fig. 3) was related to temporal and regional variation in their fecundity. We found large differences among reefs in the proportion of adult corals that underwent mass spawning in each of two years of the study, which accounted for a huge proportion (72%) of the variation in their recruitment among the 33 reefs that we sampled (Hughes et al. 2000). Moreover, once regional variation in the intensity of spawning was accounted for statistically, there was no further effect of latitude on large-scale patterns of recruitment by spawning corals. Consequently, we suggest that latitudinal patterns of recruitment on the GBR (Figs. 3 and 4) are driven by regional-scale gradients in the number of larvae produced each season, with reefs and sectors in the central GBR having higher recruitment by spawning corals (Fig. 3) because this region produces more larvae than elsewhere. Similarly, the continued decline in recruitment south of the GBR is probably due to a dwindling larval pool, as populations of breeding adults become smaller and more isolated.

The mechanisms of recruitment could also account for some of the disparities between the large- and small-scale studies. Specifically, the large-scale study had a higher density of recruits, and a greater proportion of spawners (Figs. 3 and 4), almost certainly because of differences in the timing and duration of the panel deployments in the two data sets. The large-scale deployments in late 1995 and 1996 were initiated 9–11 d before the predicted annual mass spawning of corals in November/December. This narrow timing was designed to allow the development of chemical or physical cues from bacteria or algae on the panels, which facilitate the settlement and metamorphosis of many corals (e.g., Morse et al. 1994). The annual peak settlement of spawning corals would have occurred 3–7 d after the release of gametes, ~2 wk after the panels were placed in position. In contrast, half of the small-scale studies missed entirely the annual mass spawning event, which obviously reduced the abundance of spawners and increased the proportion of brooders (that are released over a more protracted [lunar] breeding cycle, see Harrison and Wallace 1990, Tanner 1996). In addition, 20 of the 21 small-scale studies had longer durations than the large-scale data set (Table 1). Longer submergence times are likely to favor the accumulation of multiple cohorts of brooders, while a single annual cohort of spawners should rapidly decline due to mortality (Dunstan and Johnson 1998, Baird and Hughes 2000). Note, however, that the latitudinal decline in recruitment and the relative increase in brooders to the south cannot be explained by variation in the timing or duration of panel deployments among the small-scale studies. The southernmost studies were conducted closer to spawning (later in the year) and they were shorter (i.e., latitude was positively correlated with the month of initiation, and negatively correlated with duration, see Table 2). This should have produced an increase in numbers and proportion of spawners, the opposite of the pattern detected in both the large- and small-scale analyses. Thus, the large-scale pattern is not an artifact of methodology.

Large-scale hydrodynamics does not appear to play a major role in determining regional patterns of recruitment by corals along the Great Barrier Reef. Although the peak recruitment by spawners at 14°–18° S coincides with the predominant westward-flowing current which flows from the Coral Sea to the outer Great Barrier Reef, it is unlikely that substantial transport of coral larvae occurs at this scale, for several reasons. First, the area of reefs (a proxy for reproductive output) declines precipitously eastwards from the GBR. Consequently, the production of larvae by isolated oceanic reefs is unlikely to be a significant input onto the vast expanse of the Great Barrier Reef. Second, the GBR is much more speciose than reefs to the east, having ~100 more species than on New Caledonia, the nearest large reef system, 1100 km to the east (see Veron 1993). This biogeographic distinction implies that the Coral Sea is a significant barrier to dispersal, at least from west to east. Clearly, for the species found only on the GBR (and further north), the Coral Sea cannot be a source of larvae. Third, the strong concordance between sector-scale patterns of spawning and recruit-

ment by corals on the Great Barrier Reef suggests that dispersal by most species is limited, and that areas with high fecundity do not act as a source for downstream reefs that have lower reproductive outputs (Hughes et al. 2000). Fourth, recent estimates of the genetic variability of nine species of corals along the length of the Great Barrier Reef indicate that most successful recruitment is localized (Ayre and Hughes 2000). Accordingly, the very substantial latitudinal decline in larval recruitment we recorded (Fig. 3) occurs despite the potential for southerly transport by the East Australia Current, which implies that significant regional-scale transport of corals is prevented by early settlement (on natal or neighboring reefs), local entrapment of larvae due to reef-scale hydrodynamics (e.g., Black et al. 1991), and by the depletion of larval cohorts caused by mortality in the plankton.

In conclusion, our results show that that the dynamics of coral reefs vary substantially at regional scales. The latitudinal changes in the rate and composition of recruitment that we documented undoubtedly contribute to broad-scale biogeographic shifts in the community structure and diversity of coral assemblages. Similar regional-scale pattern in recruitment may occur on Pacific coral reefs in the northern hemisphere, where diversity and adult coral abundances decline from south to north along the length of the Ryukyu Island chain (24°–32° N). High diversity reefs to the south are dominated by spawners (Hayashibara et al. 1993, Morse et al. 1996), compared to depauperate northern locations that have lower rates of recruitment, mainly by brooders (S. Nojima, *personal comment*). Similar regional-scale patterns in recruitment are becoming apparent in other intertidal and subtidal marine systems (e.g., along the western coast of North America; see Connolly and Roughgarden 1998, 1999, Ebert and Russell 1988). As demonstrated here, meta-analysis provides a powerful approach for elucidating large-scale phenomena such as these, and for generating testable hypotheses about their causes and consequences.

Acknowledgments

We are very grateful to the authors listed in Table 1, whose published data allowed us to undertake the meta-analysis, and to 51 graduate student volunteers from James Cook University who provided crucial assistance with the large-scale fieldwork in 1995–1997. We thank D. Ayre, J. Caley, H. Choat, and P. Doherty for comments on the manuscript. Research was funded by grants to T. Hughes from the Australian Research Council. This is contribution No. 183 of the Coral Ecology Group at James Cook University.

Literature Cited

Arnqvist, G., and D. Wooster. 1995. Meta-analysis: synthesizing research findings in ecology and evolution. Trends in Ecology and Evolution **10**:236–240.

Ayre, D. J., and T. P. Hughes. 2000. Genotypic diversity and gene flow in brooding and spawning corals along the Great Barrier Reef, Australia. Evolution **54**:1590–1605.

Babcock, R. C. 1988. Fine-scale spatial and temporal patterns in coral settlement. Pages 635–639 *in* J. H. Choat, et al., editors. Proceedings of the Sixth International Coral Reef Symposium, Townsville, Australia. Volume 2.

Babcock, R. C., G. D. Bull, P. L. Harrison, A. J. Heyward, J. K. Oliver, C. C. Wallace, and B. L. Willis. 1986. Synchronous spawnings of 105 scleractinian coral species on the Great Barrier Reef. Marine Biology **90**:379–394.

Baird, A. H., and T. P. Hughes. 1997. Spatial variation in coral recruitment around Lizard Island, Australia. Pages 1207–1211 *in* H. Lessios and I. G. Macintyre, editors. Proceedings of the Eighth International Coral Reef Symposium, Panama. Smithsonian Tropical Research Institute, Balboa, Republic of Panama.

Baird, A. H., and T. P. Hughes. 2000. Competitive dominance by tabular corals: an experimental analysis. Journal of Experimental Marine Biology and Ecology **251**:117–132.

Banks, S. A., and V. J. Harriott. 1996. Patterns of coral recruitment at the Gneering Shoals, southeast Queensland, Australia. Coral Reefs **15**:225–230.

Birkeland, C. 1988. Geographic comparisons of coral-reef community process. Pages 211–220 *in* J. H. Choat, et al., editors. Proceedings of the Sixth International Coral Reef Symposium, Townsville, Australia. Volume 2.

Black, K. P., P. J. Moran, and L. S. Hammond. 1991. Numerical models show coral reefs can be self-seeding. Marine Ecology Progress Series **74**:1–11.

Bothwell, A. M. 1981. Fragmentation, a means of asexual reproduction and dispersal in the coral genus *Acropora* (Scleractinia: Astrocoeniidae: Acroporidae)—a preliminary report. Pages 137–144 *in* E. D. Gomez, C. E. Birkeland, R. W. Buddemeier, R. E. Johannes, J. A. Marsh, Jr., and R. T. Tsuda, editors. Proceedings of the Fourth International Coral Reef Symposium, Manila. Marine Sciences Center, University of the Philippines, Quezon City, Philippines.

Caley, M. J., M. H. Carr, M. A. Hixon, T. P. Hughes, G. P. Jones, and B. A. Menge. 1996. Recruitment and the local dynamics of open marine populations. Annual Reviews of Ecology and Systematics **27**:477–498.

Carpenter, S. R. 1990. Large-scale perturbations: opportunities for innovation. Ecology **70**:1142–1152.

Connell, J. H., T. P. Hughes, and C. C. Wallace. 1997. A 30-yr study of coral abundance, recruitment, and disturbance at several scales in space and time. Ecological Monographs **67**:461–488.

Connolly, S. R., and J. Roughgarden. 1998. A latitudinal gradient in intertidal community structure: evidence for an oceanographically-based synthesis of marine community theory. American Naturalist **151**:311–326.

Connolly, S. R., and J. Roughgarden. 1999. Theory of marine communities: competition, predation, and recruitment-dependent interaction strength. Ecological Monographs **69**: 277–296.

Crossland, C. 1988. Latitudinal comparisons of coral reef structure and function. Pages 221–266 *in* J. H. Choat, et al., editors. Proceedings of the Sixth International Coral Reef Symposium, Townsville, Australia. Volume 2.

Dayton, P. K., and M. J. Tegner. 1984. The importance of scale in community ecology. Pages 457–481 *in* P. W. Price, C. N. Slobodchikoff, and W. S. Gaud, editors. A new ecology. John Wiley and Sons, London, UK.

Downing, J. A., C. W. Osenberg, and O. Sarnelle. 1999. Meta-analysis of marine nutrient-enrichment experiments: variation in the magnitude of nutrient limitation. Ecology **80**:1157–1167.

Dunstan, P. K., and C. R. Johnson. 1998. Spatio-temporal variation in coral recruitment at different scales on Heron Reef, southern Great Barrier Reef. Coral Reefs **17**:71–81.

Eberhardt, L. L., and J. M. Thomas. 1991. Designing environmental field studies. Ecological Monographs **61**:53–73.

Ebert, T. A., and M. P. Russell. 1988. Latitudinal variation

in size structure of the west coast purple sea urchin: a correlation with headlands. Limnology and Oceanography **33**:286–354.

Edwards, P. J., R. M. May, and N. R. Webb, editors. 1994. Large-scale ecology and conservation biology. Blackwell Science, Oxford, UK.

Englund, G., O. Sarnelle, and S. D. Cooper. 1999. The importance of data-selection criteria: meta-analysis of stream predation experiments. Ecology **80**:1132–1142.

Fisk, D. A., and V. J. Harriott. 1990. Spatial and temporal variation in coral recruitment on the Great Barrier Reef: implications for dispersal hypotheses. Marine Biology **107**: 485–490.

Fisk, D., and V. J. Harriott. 1993. Are understorey corals recruitment limited? Pages 513–516 *in* Proceedings of the Seventh International Coral Reef Symposium. Volume 1. University of Guam Press, Mangilao, Guam.

Gaines, S. D., and J. Roughgarden. 1985. Larval settlement rates: a leading determinant of structure in an ecological community of the marine intertidal zone. Proceedings of the National Academy of Science, USA **82**:3707–3701.

Glass, G. V., B. McGraw, and M. L. Smith. 1981. Meta-analysis in social research. Sage Publications, Beverly Hills, California, USA.

Goldberg, D. E., T. Rajaneimi, J. Gurevitch, and A. Stewart-Oaten. 1999. Empirical approaches to quantifying interaction intensity: competition and facilitation along productivity gradients. Ecology **80**:1118–1132.

Gurevitch, J., and L. V. Hedges. 1993. Meta-analysis: combining the results of independent experiments. Pages 378–398 *in* S. M. Scheiner and J. Gurevitch, editors. Design and analysis of ecological experiments. Chapman and Hall, New York, New York, USA.

Harriott, V. J. 1985. Recruitment patterns of scleractinian corals at Lizard Island, Great Barrier Reef. Pages 367–372 *in* C. Gabrie and M. Harmelin Vivien, editors. Proceedings of the Fifth International Coral Reef Congress, Tahiti. Antenne Museum-Ephe, Moorea, French Polynesia.

Harriott, V. J. 1992. Recruitment patterns of scleractinian corals in an isolated sub-tropical reef system. Coral Reefs **11**:215–219.

Harriott, V. J., and S. A. Banks. 1995. Recruitment of scleractinian corals in the Solitary Islands Marine Reserve, a high latitude coral-dominated community in eastern Australia. Marine Ecology Progress Series **123**:155–161.

Harriott, V. J., and D. A. Fisk. 1987. A comparison of settlement plate types for experiments on the recruitment of scleractinian corals. Marine Ecology Progress Series **37**: 201–208.

Harriott, V. J., and D. A. Fisk. 1988. Recruitment patterns of scleractinian corals: a study of three reefs. Australian Journal of Marine and Freshwater Research **39**:409–416.

Harriott, V. J., P. L. Harrison, and S. A. Banks. 1995. The coral comunities of Lord Howe Island. Australian Journal of Marine and Freshwater Research **46**:457–465.

Harriott, V. J., and C. J. Simpson. 1996. Coral recruitment on tropical and subtropical reefs in Western Australia. Pages 1191–1196 *in* H. Lessios and I. G. Macintyre, editors. Proceedings of the Eighth International Coral Reef Symposium, Panama. Volume 2. Smithsonian Tropical Research Institute, Balboa, Republic of Panama.

Harrison, P. L., R. C. Babcock, G. D. Bull, J. K. Oliver, C. C. Wallace, and B. L. Willis. 1984. Mass-spawning in tropical reef corals. Science **223**:1186–1189.

Harrison, P. L., and C. C. Wallace. 1990. Reproduction, dispersal and recruitment of scleractinian corals. Pages 133–207 *in* Z. Dubinsky, editor. Ecosystems of the world. Volume 25. Elsevier, New York, New York, USA.

Hastie, T. J., and R. Tibshirani. 1990. Generalized additive models. Chapman and Hall, London, UK.

Hayashibara, T., K. Shimoike, T. Kimura, S. Hosaka, A. Heyward, P. Harrison, K. Kudo, and M. Omori. 1993. Patterns of coral spawning at Akajima Island, Okinawa, Japan. Marine Ecology Progress Series **101**:253–262.

Hedges, L. V., and I. Olkin. 1985. Statistical methods for meta-analysis. Academic Press, New York, New York, USA.

Hughes, T. P. 1990. Recruitment limitation, mortality and population regulation in a sessile invertebrate, *Cellepora pumicosa* (Bryozoa, Anasca). Ecology **71**:12–20.

Hughes, T. P., A. H. Baird, E. A. Dinsdale, N. A. Molchaniwskyj, M. S. Pratchett, J. E. Tanner, and B. L. Willis. 1999. Patterns of recruitment and abundance of corals along the Great Barrier Reef. Nature **397**:59–63.

Hughes, T. P., A. H. Baird, E. A. Dinsdale, N. A. Molchaniwskyj, M. S. Pratchett, J. E. Tanner, and B. L. Willis. 2000. Supply-side ecology works both ways: the link between benthic adults, fecundity, and larval recruits. Ecology **81**: 2241–2249.

Hughes, T. P., and J. E. Tanner. 2000. Recruitment failure, life histories, and long-term decline of Caribbean corals. Ecology **81**:2250–2263.

Karlson, R. H., and D. R. Levitan. 1990. Recruitment limitation in open populations of *Diadema antillarum*: an evaluation. Oecologia **82**:40–44.

Mahoney, M. J. 1977. Publication prejudices: an experimental study of confirmatory bias in the peer review systems. Cognitive Therapy and Research **1**:161–175.

Maida, M., P. W. Sammarco, and J. C. Coll. 1995*a*. Effects of soft corals on scleractinian coral recruitment. 1: directional allelopathy and inhibition of settlement. Marine Ecology Progress Series **121**:191–202.

Maida, M., P. W. Sammarco, and J. C. Coll. 1995*b*. Preliminary evidence for directional allelopathic effects of the soft coral *Sinularia flexibilis* (Alcyonacea: Octocorallia) on scleractinian coral recruitment. Bulletin of Marine Science **56**:303–311.

Menge, B. A., B. A. Daley, J. Lubchenko, E. Sanford, E. Dahlhoff, P. M. Halpin, G. Hudson, and J. L. Burnaford. 1999. Top-down and bottom-up regulation of New Zealand rocky intertidal communities. Ecological Monographs **69**: 297–330.

Milicich, M. J. 1994. Dynamic coupling of reef fish replenishment and oceanographic processes. Marine Ecology Progress Series **110**:135–144.

Morse, A. N. C., K. Iwao, M. Baba, K. Shiroike, T. Hayashibara, and M. Omori. 1994. An ancient chemosensory mechanism brings new life to coral reefs. Biological Bulletin **191**:149–154.

Mundy, C. N. 2000. An appraisal of methods used in coral recruitment studies. Coral Reefs **19**:124–131.

O'Connor, R. J. 1985. Long-term monitoring of British bird populations. Ornis Fennica **62**:73–79.

Osenberg, C. W., O. Sarnelle, S. D. Cooper, and R. D. Holt. 1999. Resolving ecological questions through meta-analysis: goals, metrics, and models. Ecology **80**:1105–1117.

Rastetter, E. B., A. W. King, B. J. Cosby, G. M. Hornberger, R. V. O'Neill, and J. E. Hobbie. 1992. Aggregating fine scale ecological knowledge to model coarse-scale attributes of ecosystems. Ecological Applications **2**:55–70.

Richmond, R. H. 1987. Energetics, competency and long-distance dispersal of planula larvae of the coral *Pocillopora damicornis*. Marine Biology **93**:527–533.

Richmond, R. H., and C. L. Hunter. 1990. Reproduction and recruitment of corals: comparisons among the Caribbean, the tropical Pacific, and the Red Sea. Marine Ecology Progress Series **60**:185–203.

Rosenzweig, M. L. 1995. Species diversity in space and time. Cambridge University Press, Cambridge, UK.

Sachs, H. S., J. Berrier, D. Reitman, V. A. Ancona-Berk, and

T. C. Chalmer. 1987. Meta-analysis of randomized controlled trials. New England Journal of Medicine **316**:450–455.

Sammarco, P. W. 1991. Geographically specific recruitment and post settlement mortality as influences on coral communities: the cross continental shelf transplant experiments. Limnology and Oceanography. **36**:496–514.

Sammarco, P. W., and J. C. Andrews. 1988. Localized dispersal and recruitment patterns in Great Barrier Reef corals. Limnology and Oceanography **34**:896–912.

Sammarco, P. W., and J. H. Carleton. 1981. Damselfish territoriality and coral community structure: reduced grazing, coral recruitment, and effects on coral spat. Pages 525–535 *in* E. D. Gomez, C. E. Birkeland, R. W. Buddemeier, R. E. Johannes, J. A. Marsh, Jr., and R. T. Tsuda, editors. Proceedings of the Fourth International Coral Reef Symposium, Manila. Marine Sciences Center, University of the Philippines, Quezon City, Philippines.

Schneider, D. C. 1994. Quantitative ecology: spatial and temporal scaling. Academic Press, San Diego, California, USA.

Schneider, D. C., R. Walters, S. Thrush, and P. Dayton. 1997. Scale-up of ecological experiments: density variation in the mobile bivalve *Macomona liliana*. Journal of Experimental Marine Biology and Ecology **216**:129–152.

Stehli, F. G., and J. W. Wells. 1969. Diversity and age patterns in hermatypic corals. Systematic Zoology **20**:115–126.

Tanner, J. E. 1996. Seasonal and lunar periodicity in the reproduction of pocilloporid corals. Coral Reefs **15**:59–66.

Taylor, L. R. 1987. Objective and experiment in long-term research. Pages 20–70 *in* G. E. Likens, editor. Long-term studies in ecology: approaches and alternatives. Springer-Verlag, New York, New York, USA.

Thrush, S. F., et al. 1997. Scaling-up from experiments to complex ecological systems: Where to next? Journal of Experimental Marine Biology and Ecology **216**:243–254.

Veron, J. E. N. 1993. A biogeographic database of hermatypic corals. Australian Institute of Marine Science Monograph Series. Volume 10. University of Hawaii Press, Honolulu.

Veron, J. E. N., and T. J. Done. 1979. Corals and coral communities of Lord Howe Island. Australian Journal of Marine and Freshwater Research **30**:203–236.

Wallace, C. C. 1985*a*. Seasonal peaks and annual fluctuations in recruitment of juvenile scleractinian corals. Marine Ecology Progress Series **21**:289–298.

Wallace, C. C. 1985*b*. Reproduction, recruitment and fragmentation in nine sympatric species of the coral genus *Acropora*. Marine Biology **88**:217–233.

Wallace, C. C., and G. D. Bull. 1981. Patterns of juvenile coral recruitment on a reef front during a spring-summer spawning period. Pages 345–350 *in* E. D. Gomez, C. E. Birkeland, R. W. Buddemeier, R. E. Johannes, J. A. Marsh, Jr., and R. T. Tsuda, editors. Proceedings of the Fourth international coral reef symposium, Manila. Volume 2. Marine Sciences Center, University of the Philippines, Quezon City, Philippines.

Wiens, J. A. 1989. Spatial scaling in ecology. Functional Ecology **3**:385–397.

Wiens, J. A., N. C. Stenseth, B. Van Horne, and R. A. Ims. 1993. Ecological mechanisms and landscape ecology. Oikos **66**:369–380.

Wilson, J. R., and P. L. Harrison. 1998. Settlement-competency periods of larvae of three species of scleractinian corals. Marine Biology **131**:339–345.

Wolanski, E. 1994. Physical oceanographic processes of the Great Barrier Reef. CRC Press, Boca Raton, Florida, USA.

Ecology, 81(8), 2000, pp. 2241–2249

SUPPLY-SIDE ECOLOGY WORKS BOTH WAYS: THE LINK BETWEEN BENTHIC ADULTS, FECUNDITY, AND LARVAL RECRUITS

T. P. HUGHES,[1] A. H. BAIRD, E. A. DINSDALE, N. A. MOLTSCHANIWSKYJ,[2] M. S. PRATCHETT, J. E. TANNER,[3] AND B. L. WILLIS

Department of Marine Biology, James Cook University, Townsville, QLD 4811, Australia

Abstract. ''Supply-side'' ecology recognizes the potential role that recruitment plays in the local population dynamics of open systems. Apart from the applied fisheries literature, the converse link between adults and the production of cohorts of recruits has received much less attention. We used a hierarchical sampling design to investigate the relationships between adult abundance, fecundity, and rates of larval recruitment by acroporid corals on 33 reefs in five sectors (250–400 km apart) stretching from north to south along the length of the Great Barrier Reef, Australia. Our goal was to quantify patterns of recruitment at multiple scales, and to explore the underlying mechanisms. Specifically, we predicted that large-scale patterns of recruitment could be driven by changes in the abundance of adults and/or their fecundity, i.e., that corals exhibit a stock–recruitment relationship. The amount of recruitment by acroporids in each of two breeding seasons varied by more than 35-fold among the five sectors. Adult density varied only twofold among sectors and was not correlated with recruitment at the sector or reef scale. In contrast, fecundity levels (the proportion of colonies on each reef that contained ripe eggs) varied from 15% to 100%, depending on sector, year, and species. Spatial and temporal variation in the fecundity of each of three common *Acropora* species explained most of the variation (72%) in recruitment by acroporids, indicating that the production of larvae is a major determinant of levels of recruitment at large scales. Once fecundity was accounted for, none of the other variables we examined (sector, reef area, abundance of adults, or year) contributed significantly to variation in recruitment. The relationship between fecundity and recruitment was nonlinear, i.e., rates of recruitment increased disproportionately when and where the proportion of gravid colonies approached 100%. This pattern is consistent with the hypothesis that enhanced fertilization success and/or predator satiation occurs during mass-spawning events. Furthermore, it implies that small, sublethal changes in fecundity of corals could result in major reductions in recruitment.

Key words: coral reefs; dispersal; fecundity; gene flow; Great Barrier Reef, Australia; mass spawning; population dynamics; recruitment limitation; spatial scale; stock size; supply-side ecology.

INTRODUCTION

Dispersal plays a crucial role in the ecology and evolution of many organisms, particularly in marine systems where many animals and plants exhibit alternate benthic and planktonic life-history stages (e.g., Thorson 1950, Reed et al. 1988, Strathmann 1993). At the end of the dispersal phase, the abundance of larvae at settlement is often highly variable, both spatially and temporally (e.g., Barnes 1956, Milicich 1994). Recent attention has focussed on the causes of variation in settlement, particularly on larval mortality (e.g., Houde 1987, Underwood and Fairweather 1989), transport mechanisms (e.g., Gaines et al. 1985, Black et al. 1991, Milicich 1994) and larval behavior before and during settlement (e.g., Boicourt 1982, Grosberg 1982). ''Supply-side ecology'' recognizes the role that variable larval input plays in determining the size of local adult populations (e.g., Underwood and Denley 1984, Hughes 1984, 1990, Gaines and Roughgarden 1985, Roughgarden et al. 1985, Caley et al. 1996). However, the converse linkage between adult stocks and the production of larvae is much less clear (Grosberg and Levitan 1992, Eckman 1996). At small scales (less than a few meters), fertilization rate in sessile or sedentary broadcast spawners is often crucially dependent on adult density, i.e., on the distance traveled by sperm before they encounter an unfertilized egg (in echinoids, Pennington 1985, Levitan 1991; starfish, Babcock et al. 1992; and cnidarians, Yund 1990, Coma and Lasker 1997). At larger scales, the relationship between adult abundance (stock size), fecundity and recruitment in noncommercial species remains virtually unknown.

The issue of spatial scale is crucial for understanding stock-recruitment relationships. If larvae are widely dispersed, the local production of propagules by sessile or sedentary adults will not be correlated with local recruitment; locally derived larvae go elsewhere, and

Manuscript received 10 September 1998; revised 4 June 1999; accepted 8 June 1999; final version received 16 July 1999.

[1] E-mail: terry.hughes@jcu.edu.au

[2] Present address: School of Aquaculture, University of Tasmania, Launceston, P.O. Box 1214, TAS 7250, Australia.

[3] Present address: SARDI Aquatic Sciences, P.O. Box 120, Henley Beach, SA 5022, Australia.

recruits come from afar. Nonetheless, at larger spatial scales there may be some detectable relationship between the size of the spawning stock (i.e., the amount of larval production) and the amount of recruitment. Certainly, temporal correlations between spawning by adults and settlement are well established, with settlement peaks corresponding to earlier cycles of breeding (e.g., corals, Wallace 1985*a*, *b*; barnacles, Barnes 1956; crabs, Christy 1982; fishes, Doherty and Williams 1988). The fisheries literature highlights the importance of large-scale stock-recruitment relationships for understanding population dynamics and for management of marine resources, although in most cases ''recruit'' refers to harvestable adults (e.g., Lipcius and Van Engel 1990, Hilborn and Walters 1992, Peterson and Summerson 1992). However, for most marine organisms, the spatial scale at which adult stocks and recruitment are coupled is unknown. This lack of data most likely stems from the difficulties of measuring recruitment, adult abundances and fecundities at large spatial scales which approach or exceed the extent of larval dispersal (Hughes et al. 1999).

It is often tacitly assumed that stochastic variation in fertilization success, planktonic duration, or mortality rates will destroy any relationship between stock size, the production of larvae, and the number of propagules still alive at settlement (e.g., Houde 1987, Underwood and Fairweather 1989). However, this argument is not valid at larger scales, where global recruitment must diminish if stock sizes are greatly suppressed (e.g., due to disease, Karlson and Levitan 1990, Peterson and Summerson 1992; climatic variation, Cushing 1986; or overfishing, Hilborn and Walters 1992). Moreover, temporal variation in recruitment (for a given stock size) or density-dependence do not preclude a stock-recruitment relationship. Rather, the former will simply increase the variance about the relationship, while the latter will alter its shape. Thus, at larger scales stock-recruitment relationships must exist, although they may be statistically messy and difficult to detect (e.g., Ricker 1954, Lipcius and Van Engel 1990).

We demonstrate here that large-scale variation in the density of coral recruits on the Great Barrier Reef, Australia, is strongly associated with spatial and temporal changes in the fecundity of adults. This study investigates adult–juvenile relationships at a very large spatial scale (~1800 km), and provides a unique insight into the coupling of benthic and planktonic processes in sessile marine organisms.

Methods

We examined the relationship between the abundance of adults, fecundity, and recruitment of corals along the length of the Great Barrier Reef (GBR), in 1995/1996 and 1996/1997 (year 1 and 2). Corals are either brooders, which release internally fertilized planulae, or broadcast spawners, which release eggs and sperm (Harrison and Wallace 1990). We focus here on spawning species in the most abundant scleractinian family, the Acroporidae, hereafter termed ''acroporid''. We designed our study to take advantage of the predictable, annual mass spawning of corals on the Great Barrier Reef, where more than 130 scleractinian species release their eggs and sperm over a period of a few days in November/December (see Harrison et al. 1984, Harrison and Wallace 1990).

To examine spatial patterns at a hierarchy of scales, we partitioned the GBR into five adjacent sectors, each one 250–400 km long from north to south (Fig. 1, see also Hughes et al. 1999). Eighteen reefs were sampled in year 1 (three reefs per sector, except for sector 4 which had six), and 15 other reefs in year 2; i.e., reefs were independent of year. We deliberately chose a wide range of reef sizes in each sector because bigger reefs should have larger stocks of breeding adults (although settlement can also occur over a larger area). Four sites were established on each reef on the reef crest (1 m depth at low tide), ~1–4 km apart. The abundance of corals (counts of colonies >1 cm in diameter, and percent cover) was measured using ten 10-m line intercept transects a few meters apart at each of the 132 sites (33 reefs × 4 sites). On each of the 1320 transects, each colony lying underneath the tape was identified, and the intercept was measured to the nearest centimeter. More than 30 000 colonies were censused.

To assess fecundity, tissue samples were collected synchronously each year 10 d (±1 d) before the predicted annual mass spawning of corals. Three species that are abundant throughout the GBR were selected for fecundity analysis: *Acropora hyacinthus, A. cytherea,* and *A. millepora.* Two or three branches containing several hundred polyps were taken each year from 20 large colonies (>30 cm diameter) of each species on each reef, a total of 1980 colonies. Samples were decalcified and dissected and the proportion of colonies with mature (pigmented) eggs was scored for each species and reef (see Hall and Hughes 1996). Additional samples collected after the predicted spawning were empty of eggs. We assume here that the fecundity patterns (in space and time) of these three species are broadly representative of acroporids as a whole, and that they may be able to predict patterns of acroporid recruitment. It is not possible to compare fecundity and recruitment at the individual species level because of the limited taxonomic resolution of newly-settled recruits.

To assess recruitment by spawning acroporids, ten replicate recruitment panels (11 × 11 cm) were deployed each year on the reef crest at each of the four sites per reef (a total of 1320 panels), also 10 d (±1 d) before the predicted annual mass spawning of corals (e.g., Harrison et al. 1984, Harrison and Wallace 1990). The panels were unglazed clay tiles attached individually by a bolt drilled into the substratum. Panels were retrieved after eight weeks (86% were recovered un-

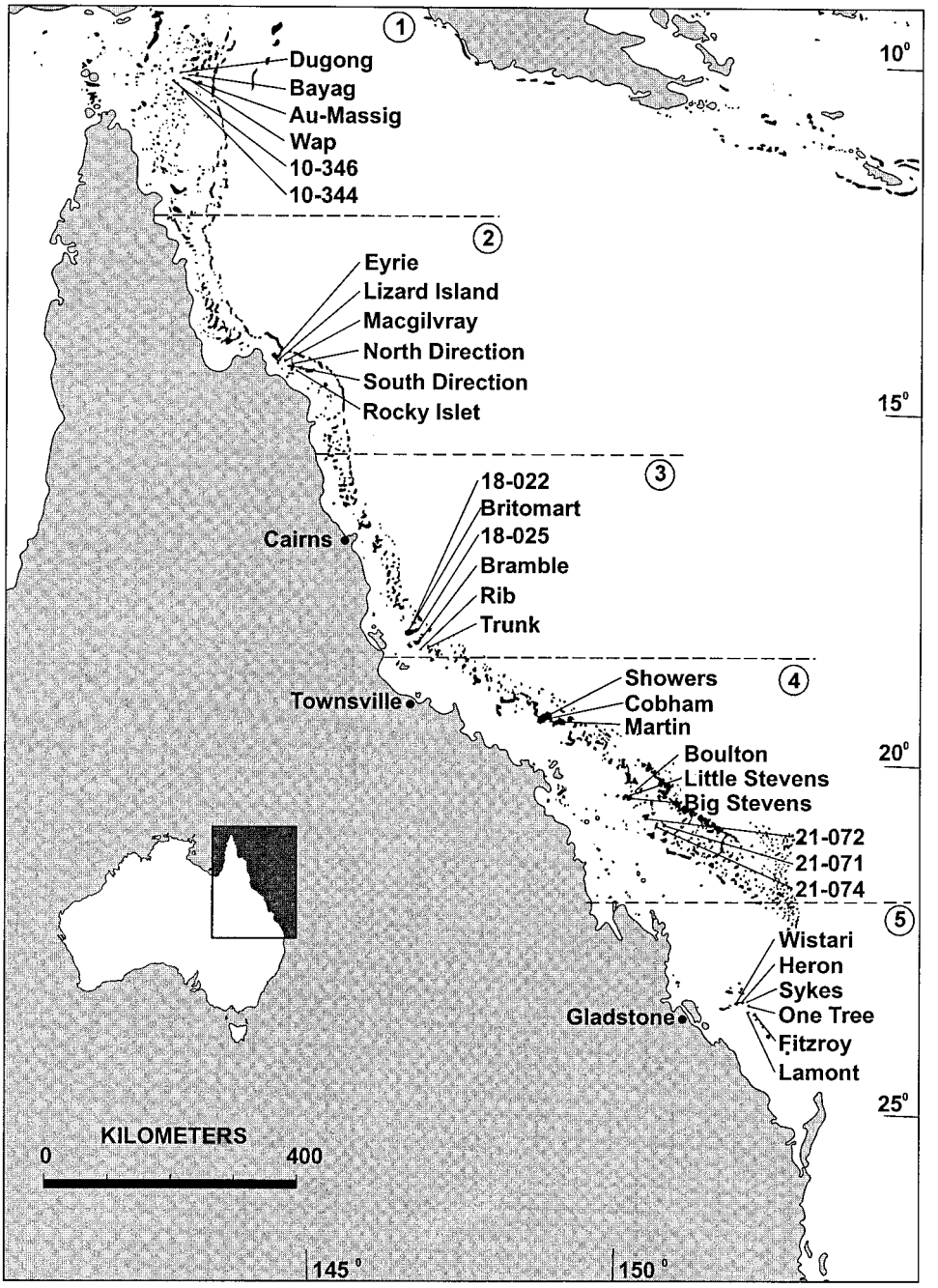


FIG. 1. Map of the Great Barrier Reef region of Australia, showing the locations of five sectors (numbers in circles) and 33 midshelf reefs where adult abundance, fecundity, and recruitment were measured.

damaged), and the coral recruits were counted and identified to family or genus. The synchronous deployment and retrieval of panels on large numbers of midshelf reefs (typically 30–80 km offshore) along nearly 2000 km of coastline was essential for ensuring that spatial patterns in recruitment were not confounded with (1) the timing of deployment and (2) the duration of exposure of the panels. Based on pilot studies, most of the recruits at the time of collection would have settled in the previous 5–6 wk; i.e., beginning ~2 wk after deployment of the panels and 3–5 d after the mass spawning. This relatively short duration was long enough to allow modest taxonomic resolution, while minimizing losses through early mortality. Acroprid colonies do not develop species-level morphological features until a minimum age of 2–3 yr.

Spatial variation in adult abundance, fecundity and recruitment were examined using hierarchical (nested)

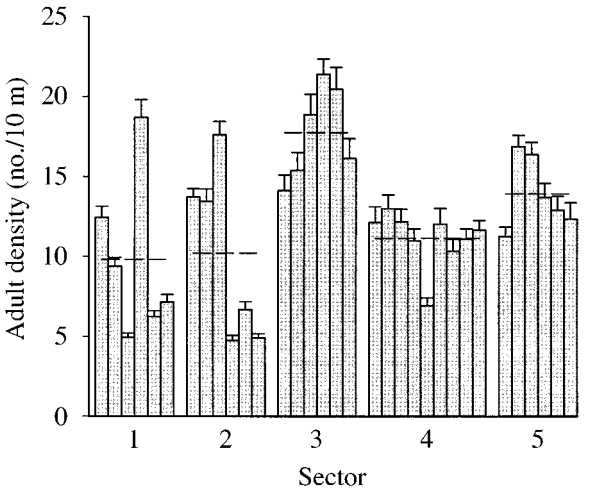


FIG. 2. Patterns of adult abundance of spawning acroporid corals on 33 reefs with five sectors of the Great Barrier Reef (arranged from north to south; see Fig. 1). Horizontal lines show the mean abundance for each sector. Error bars represent 1 SE (n = 4 sites per reef). See Table 1 for analysis.

analysis of variance, with appropriate transformations where necessary. We used nonlinear regression models (Generalized Additive Models, Hastie and Tibshirani 1990) to examine the relationship between recruitment by acroporids on each reef versus the abundance of established acroporids, fecundity of each of the three targeted *Acropora* species, and several other potentially important variables (reef size, sector, and year). The size of each reef (projected area in km^2) varied by 1–2 orders of magnitude in each sector, and was measured from GIS data. Sector (n = 5) and year (n = 2) were entered into the model sequentially as categorical variables to examine spatial and temporal variation that was independent of fecundity. All other variables were continuous, and preliminary analyses revealed, not surprisingly, that they were not linearly related to recruitment. Consequently, they were entered into the model as smoothed, nonlinear variables using spline functions with four degrees of freedom. (One degree of freedom would fit a straight line whereas n degrees would join all points. Four produces ''modest'' smoothing, see Hastie and Tibshirani 1990).

Results and Discussion

The mean abundance of adult acroporids on all reefs was 12.3 ± 1.3 (mean ± 1 SE) colonies per 10-m transect (or 20.56% ± 3.7% cover, n = 33 reefs). Adult density varied only twofold among sectors (Fig. 2), with most of the variation occurring at much smaller spatial scales (Table 1). A hierarchical analysis of variance indicated that 19% and 5% of the variation was attributable to sector and reef, respectively. The remaining 75% of the variation in adult density occurred within reefs (i.e., among sites on the same reef, and among adjacent transects). Similarly, 82% of the variation in the percent cover of spawning acroporids also occurred at very local scales, i.e., within reefs.

TABLE 1. Nested analysis of variance of adult acroporid densities (number of colonies per 10-m transect).

Source of variation	df	MS	F ratio	P	Percentage of variation
Sector	4	6.76	8.377	0.003	19
Reef(sector)	10	0.81	3.185	0.004	5
Site(sector × reef)	45	0.25	2.794	0.000	6
Residual	1126	0.09			69

Notes: Reefs are nested within sectors, and sites within reefs (see Fig. 1 for locations of sectors and reefs). Data were $\log(x + 1)$ transformed. Note that most of the variation occurred at the smallest scale, among replicate transects (see also Fig. 2).

In contrast, fecundity showed much more substantial large-scale variation among sectors, with different patterns occurring among years (yielding a significant sector × year interaction, P = 0.012; Table 2, Fig. 3). All three species exhibited a similar spatial and temporal pattern (Table 2), with markedly lower fecundities at both ends of the Great Barrier Reef in year 1, and in the northernmost sector in year 2. For the three species combined, the proportion of colonies containing eggs varied spatially by six-fold among sectors in year 1 and by twofold in year 2. The largest difference between years was in the far north and southern-most reefs (sectors 1 and 5), where the average proportion of colonies containing eggs was two to three times higher in year 2 (Fig. 3).

Recruitment by spawning acroporids also varied greatly in space and time (Fig. 4, Table 3). A total of 58 471 coral recruits were recorded on 1135 panels, for both years combined. Spawning corals comprised 83% of the recruits and brooders made up the remainder, with 96% of the spawners being juvenile acroporids. The mean number of acroporid recruits per panel varied by more than 100-fold among sectors in year 1, and by 35-fold in year 2 (Fig. 4). In year 1, fewer recruits were found at the northernmost and southern sectors (1, 4, and 5). In year 2, recruitment increased in all regions of the Barrier Reef (except for sector 4, which

TABLE 2. Three-way analysis of variance of acroporid fecundities, with reefs as replicates and percentage of individuals with eggs present as the response.

Source of variation	df	MS	F ratio	P
Sector	4	9376.3	4.545	0.056
Species	2	1419.7	2.272	0.165
Year	1	12 952.5	7.714	0.050
Sector × species	8	628.9	2.427	0.115
Species × year	2	834.1	3.229	0.093
Sector × year	4	1693.2	6.534	0.012
Sector × species × year	8	259.2	1.415	0.206
Residual	69	183.2		

Notes: Data were not transformed because the ANOVA assumptions were met. See also Fig. 3.

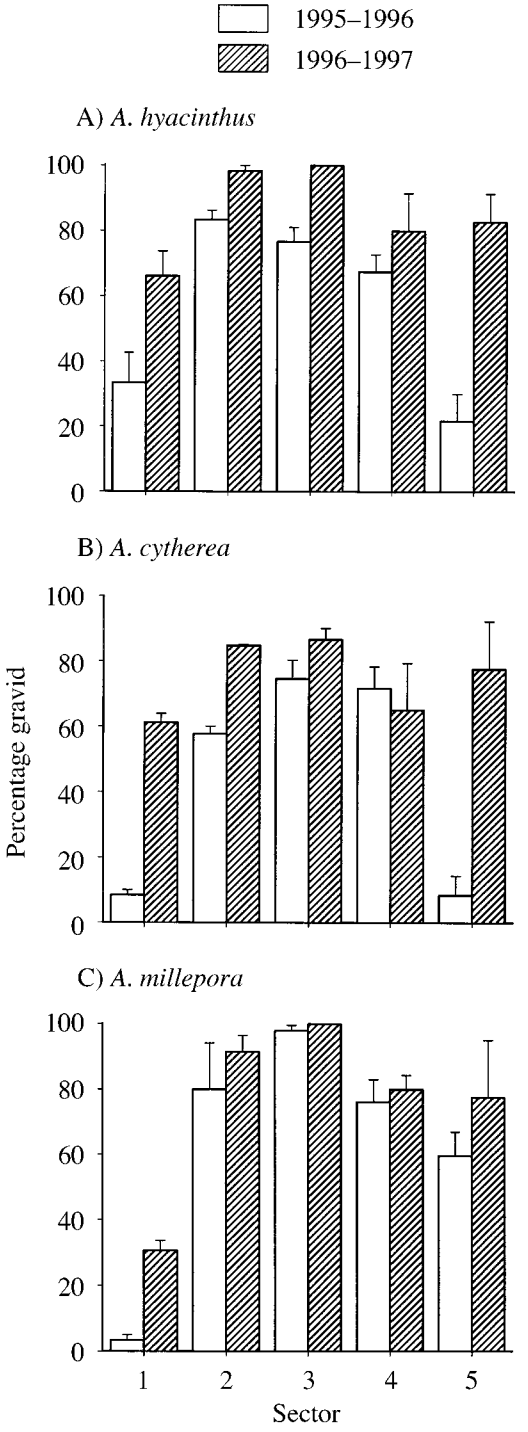


FIG. 3. Patterns of fecundity of spawning acroporid corals among five sectors of the Great Barrier Reef (numbered from north to south; see Fig. 1), and among years. Data shown are the mean percentage of colonies containing eggs in each sector for (a) *Acropora hyacinthus,* (b) *A. cytherea,* and (c) *A. millepora.* Error bars represent + 1 SE (n = 3 reefs·[sector]$^{-1}$·yr^{-1}, except for sector 4 in 1995/1996 which had six reefs). See Table 2 for analysis.

remained unchanged). The northernmost and southernmost sectors (1 and 5) exhibited the most marked increase, by a factor of about 10 and 100, respectively (yielding the highly significant sector × year interaction, $P < 0.001$; Table 3). For all sectors combined, the total amount of recruitment by acroporid corals along the Great Barrier Reef in year 2 was three times higher than in year 1.

The large-scale spatial and temporal variation in recruitment was clearly linked to patterns of variation in fecundity rather than adult abundance, i.e., when and where fecundity increased, so too did the density of recruits (Figs. 3 and 4). Among the variables we examined, the fecundities of *Acropora hyacinthus, A. millepora,* and *A. cytherea* on each reef were the three best predictors of recruitment by spawning acroporids, accounting for 49%, 45%, and 36% of the variation in recruitment among reefs, respectively (Table 4, Fig. 5). When the fecundities of each of the three species were entered sequentially into regression models, they collectively accounted for 72% of the variation in total recruitment by spawning acroporids (all species of recruits were combined, because they cannot be identified). This very high explanatory power implies that the fecundities of these three species are broadly representative of the reproductive output of acroporids in general, and that fecundity drives recruitment. A further 16% of the variation was attributable to the remaining variables (sector, 9.4%; reef area, 5.1%; adult abundance, 1.2%; and year, 0.2%), although none of these was statistically significant (Table 4). Only 12%

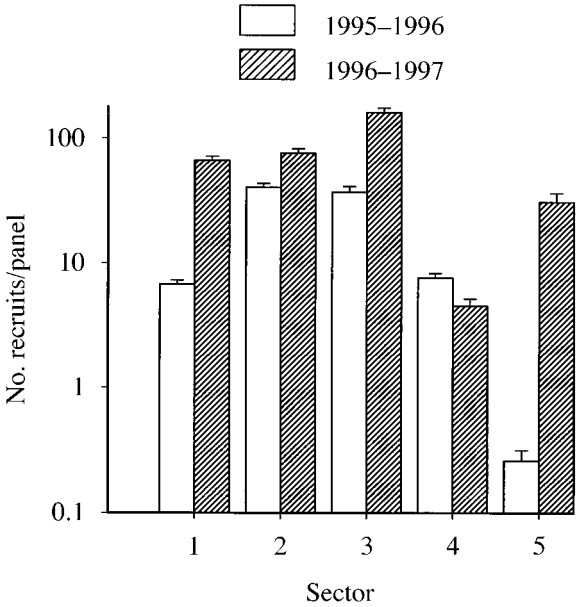


FIG. 4. Patterns of recruitment by spawning acroporid corals among sectors of the Great Barrier Reef (see Fig. 1), and among years. Data shown are the mean number of recruits per panel in each sector. Error bars represent + 1 SE (n = 3–6 reefs·[sector]$^{-1}$·yr^{-1}). See Table 3 for analysis.

TABLE 3. Two-way nested analysis of variance on density of acroporid recruits.

Source of variation	df	MS	*F* ratio	*P*
Year	1	69.273	5.525	0.780
Sector	4	48.542	3.623	0.120
Sector × year	4	13.364	10.321	0.000
Reef(sector × year)	20	1.433	1.801	0.033
Site(sector × year × reef)	85	0.826	8.958	0.000
Residual	1018	0.092		

Notes: Site is nested within reef and reef within sector (see Fig. 1). Data were $\log(x + 1)$ transformed. Year represents 1995/1996 and 1996/1997. See also Fig. 4.

of the variation in recruitment remained unexplained by our model.

The very high correlation between fecundity and recruitment of spawning corals at the scale of individual reefs (Table 4, Fig. 5), would seem to indicate that many of the recruits are of local origin. However, this conclusion is unlikely to be correct, since most of the variation in recruitment and in fecundity occurred at the largest spatial scale, i.e., among sectors rather than reefs. Depending on the year, 55–57% of the variation in recruitment was attributable to sector (see Fig. 4), while only 0–6% occurred among adjacent reefs (nested within sector). The remaining 39–43% of the variation occurred at smaller scales among sites and panels. Similarly, 91% of the variation in fecundity (for *A. hyacinthus, A. cytherea,* and *A. millepora* combined) occurred at the sector level in year 1, and 82% in year 2. Furthermore, regressions of fecundity in each sector versus the mean recruitment per sector (identical to Fig. 5, but with adjacent reefs combined) were also highly significant. We conclude therefore that the high correlation between fecundity and recruitment (Table 4) occurs at the scale of sectors rather than adjacent reefs (which show no significant differences in fecundity, Table 3).

A sector-level correlation between fecundity and recruitment implies that larvae are distributed relatively uniformly among adjacent reefs, but do not undergo larger-scale movements among sectors of the Great Barrier Reef (e.g., transported by the East Australian Current, see Wolanski 1994). Spawning acroporids are generally capable of settling ~3–7 d after spawning, although some may remain competent for much longer (Babcock and Heyward 1986, Wilson and Harrison 1998). After mass spawning, coral larvae are often aggregated into buoyant surface slicks that are usually blown or washed off a reef within a day or two, after which they dissipate (e.g., Willis and Oliver 1990, Oliver et al. 1992). Three-dimensional hydrodynamic models predict that a portion of larvae could be retained on natal reefs (e.g., 5% after 10 d; Black et al. 1991), but most are likely to be dispersed among neighboring reefs. Allozyme variation within and among reefs on the Great Barrier Reef points to modest amounts of gene-flow in spawning corals (Ayre and Hughes 2000) that approach the levels for fishes and other organisms with longer larval durations (e.g., Benzie 1994, Doherty et al. 1995). Brooding corals, in contrast, have a shorter precompetency periods (usually 1–2 d, see review by Harrison and Wallace 1990), and lower rates of gene flow (e.g., Ayre and Dufty 1994, Benzie et al. 1995, Ayre et al. 1997). Consequently, connectivity among reefs is likely to vary greatly among species, depending in part on their respective reproductive strat-

TABLE 4. (A) Analysis of the relationship between recruitment of spawning acroporids (all species combined) and fecundity of *Acropora hyacinthus, A. millepora,* and *A. cytherea* (see Fig. 3). Fecundity of each of the three species was first examined separately, and then entered sequentially (*A. hy.* + *A. m.* + *A. c.*) into multiple regression models. Once fecundity of each species was independently accounted for, we used a hierarchy of regression models (B) which successively incorporate additional explanatory variables (sector, area of each reef, cover of spawning acroporids, and year).

A.

Model	Residual deviance	df	Percentage variation explained	*P*
Null	78 477	32		
A. hyacinthus fecundity	39 706	28	49	<0.001
A. millepora fecundity	43 129	28	45	<0.01
A. cytherea fecundity	50 168	28	36	<0.01
Fecundity (*A. h.* + *A. m.* + *A. c.*)	21 976	20	72	<0.001

B.

Model	Residual deviance	df	Change in percentage variation explained	*F*	*P*
Fecundity	21 976	20			
Fecundity + sector	14 590	4	9.4	1.52	0.24
+ reef area	10 590	4	5.1	1.03	0.43
+ adult abundance	9 681	4	1.2	0.17	0.95
+ year	9 480	1	0.2	0.13	0.73

Note: The null model assumes that none of the variation in recruitment is explained by any of the variables we examined.

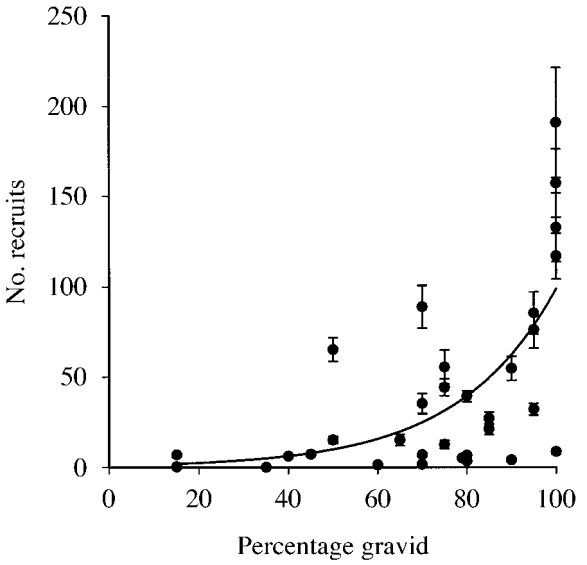


FIG. 5. Relationship between fecundity of *Acropora hyacinthus* (the percentage of colonies with eggs) and recruitment by spawning acroporids (mean number of recruits per panel ± 1 SE, all species combined). Each point represents a separate reef ($n = 33$) in either year 1 or 2. The fecundity of this one species accounted for 49% of the variance in total recruitment by acroporids. The fecundities of *A. hyacinthus*, *A. millepora*, and *A. cytherea* together explained 72% of the variance. (We cannot plot the three independent measures of fecundity against recruitment since it would require a four-dimensional graph; see Table 4 for additional analysis.)

egies and larval durations (e.g., Black et al. 1991, Milicich 1994, Ayre and Hughes 2000).

Our results suggest that the causes of large-scale spatial and temporal variation in fecundity of corals are crucial to understanding subsequent patterns of recruitment. This conclusion mirrors smaller-scale experimental studies that have demonstrated the effects of gamete dilution and aggregative spawning on rates of fertilization (Pennington 1985, Yund 1990, Levitan 1991, Babcock et al. 1992, Coma and Lasker 1997). Of necessity, our result is based on correlating large-scale spatial and temporal patterns, and the possibility remains that sectors and years which had higher fecundities also had higher rates of recruitment, but that there is no causal relationship between the two. However, we cannot formulate an alternative hypothesis that could explain large-scale variation in recruitment, independently of variation in fecundity. Conceivably, spatiotemporal variation in temperature could simultaneously enhance both egg production and reduce the length of larval life (resulting in more recruits). However, the temperature gradient along the Great Barrier Reef is not likely to explain lower fecundities or recruitment in both northern and southern regions (Fig. 3). Similarly, year to year variation in temperature is unlikely to have influenced the substantial temporal patterns of fecundity and recruitment documented in this study, since mean monthly sea surface temperatures were close to normal (<0.5°C anomalies) in both 1995/1996 and 1996/1997 (IGOSS 1998), throughout the 8–9 mo period which encompassed the gametogenic cycle and brief planktonic phase of spawning acroporids. If large-scale patterns of recruitment by spawners were primarily due to meteorological, climatic, or hydrodynamic variation rather than fecundities, we might expect that sectors or reefs with large numbers of spawning recruits should also have had greater than average recruitment of juvenile brooders. However, this was not the case, at any spatial scale: sectors, reefs, sites, and recruitment panels that had high numbers of spawners did not necessarily have high numbers of brooders (Hughes et al. 1999).

The most likely cause of spatial and temporal variation in fecundity in this study is ''split-spawning'' (sensu Willis et al. 1985), i.e., some corals may have lacked eggs when we collected them because they had released their gametes before the major mass spawning in early December. To test this hypothesis, we collected monthly samples of coral tissues in 1996/1997 on a subset of northern and southern reefs (three reefs each in sectors 2 and 5). These data indicate that a portion of adult corals did not release eggs at all in year 2. For example, up to 35% of large *A. cytherea* in sector 5 did not spawn that summer, nor did they contain immature eggs. Consequently, corals that belong to species which participate in multispecies mass spawning on the GBR may nonetheless miss one or more years, or release gametes in other months (see also Wallace 1985*b*, Willis et al. 1985). Based on two years of fecundity data on 33 reefs, we tentatively conclude that mass spawning is more synchronized within and among species in the central portions of the Great Barrier Reef than at the extremities (Fig. 3).

Our results indicate that corals which do not participate in mass spawning may be at a selective disadvantage. Our field evidence shows that as the proportion of gravid colonies approaches 100%, there is a disproportionate increase in recruitment (shown by the upward sloping curve in Fig. 5). We postulate that this pattern could arise if higher densities of eggs and sperm lead to greater levels of fertilization (and hence more larvae) after spawning (e.g., Pennington 1985), or if high numbers of larvae lead to satiation of predators (e.g., Westneat and Resing 1988). Both of these phenomena may be instrumental in the evolution of multispecies mass spawning (Harrison and Wallace 1990, Pearse 1990). Indeed, mass spawning of corals on the Great Barrier Reef occurs each year during a period of neap tides when dispersion of gametes prior to fertilization should be minimized, and at night when predation on eggs and larvae should be reduced (Harrison et al. 1984, Babcock et al. 1986).

In conclusion, this study demonstrates that reproductive processes occurring in the benthic phase of marine organisms (i.e., the production of eggs) may

have a fundamental impact on the distribution and abundance of recruits. The change in fecundities among years, which affected most of the Great Barrier Reef, indicates the potential impact of large-scale phenomena such as climate change on rates of recruitment and replenishment of coral reefs. Natural and human perturbations are normally measured in terms of their effects on adult abundances and rates of mortality of adults, while less obvious impacts on reproductive biology and regenerative processes are usually ignored (e.g., Richmond 1993, Hughes and Connell 1999). The linkage between benthic and larval stages, as demonstrated here, means that apparently localized changes that affect reproduction at one location may also have important effects on downstream populations. Moreover, small changes in fecundity could result in disproportionately larger changes in recruitment. Currently, it is often assumed that reductions in the size of open populations due to natural events or human impacts are readily reversible because of a virtually inexhaustible supply of recruits (but see, e.g., Karlson and Levitan 1990, Peterson and Summerson 1992, Hughes and Tanner 1999). Our results indicate that large-scale degradation of adult breeding stocks could also impinge on their ability to recover, potentially resulting in recruitment failure in areas that are in most need of replenishment. Understanding the dynamics of coral reefs and other open marine systems requires a better awareness of the two-way links between planktonic and benthic life history stages.

Acknowledgments

We are grateful to 41 graduate student volunteers from James Cook University who provided critical assistance with the large-scale fieldwork in 1995–1997. GIS data on reef area were generously provided by M. L. Puotinen, I. Poiner, the Great Barrier Reef Marine Park Authority, and the Commonwealth Scientific and Industrial Research Organization. We thank D. Ayre, J. Caley, H. Choat, P. Doherty, J. E. Duffy, and two anonymous reviewers for comments on the manuscript. Research was funded by a grant to T. Hughes from the Australian Research Council. This is contribution number 164 of the Coral Group at James Cook University.

Literature Cited

Ayre, D. J., and S. L. Dufty. 1994. Restricted gene flow in the viviparous coral *Seriatopora hystrix* on Australia's Great Barrier Reef. Evolution **48**:1183–1201.

Ayre, D. J., and T. P. Hughes. 2000. Genotypic diversity and gene flow in brooding and spawning corals along the Great Barrier Reef, Australia. Evolution, *in press*.

Ayre, D. J., T. P. Hughes, and R. J. Standish. 1997. Genetic differentiation, reproductive mode, and gene flow in the brooding coral *Pocillopora damicornis* along the Great Barrier Reef, Australia. Marine Ecology Progress Series **159**: 175–187.

Babcock, R. C., G. D. Bull, P. L. Harrison, A. J. Heyward, J. K. Oliver, C. C. Wallace, and B. L. Willis. 1986. Synchronous spawnings of 105 scleractinian coral species on the Great Barrier Reef. Marine Biology **90**:379–394.

Babcock, R. C., and A. J. Heyward. 1986. Larval development of certain gamete-spawning scleractinian corals. Coral Reefs **5**:111–116.

Babcock, R. C., C. N. Mundy, J. Keesing, and J. Oliver. 1992. Predictable and unpredictable spawning events: in situ behavioural data from free-spawning coral reef invertebrates. Invertebrate Reproduction and Development **22**:213–228.

Barnes, H. 1956. *Balanus balanoides* (L.) in the Firth of Clyde: the development and annual variation in the larval population and the causative factors. Journal of Animal Ecology **25**:72–84.

Benzie, J. A. H. 1994. Patterns of gene flow in the Great Barrier Reef and Coral Sea. Pages 67–79 *in* A. R. Beaumont, editor. Genetics and evolution of aquatic organisms. Chapman and Hall, London, UK.

Benzie, J. A. H., A. Haskell, and H. Lehman. 1995. Variation in the genetic composition of coral (*Pocillopora damicornis* and *Acropora palifera*) populations from different reef habitats. Marine Biology **121**:731–739.

Black, K. P., P. J. Moran, and L. S. Hammond. 1991. Numerical models show coral reefs can be self-seeding. Marine Ecology Progress Series **74**:1–11.

Boicourt, W. C. 1982. Estuarine larval retention mechanisms on two scales. Pages 45–56 *in* V. S. Kennedy, editor. Estuarine comparisons. Proceedings 6th Biennial International Estuarine Research Conference. Academic Press, New York, New York, USA.

Caley, M. J., M. H. Carr, M. A. Hixon, T. P. Hughes, G. P. Jones, and B. A. Menge. 1996. Recruitment and the local dynamics of open marine populations. Annual Reviews of Ecology and Systematics **27**:477–498.

Christy, J. H. 1982. Adaptive significance of semilunar cycles of larval release in fiddler crabs (Genus *Uca*): test of an hypothesis. Biological Bulletin **163**:251–263.

Coma, R., and H. R. Lasker. 1997. Effects of spatial distribution and reproductive biology on in situ fertilization rates of a broadcast-spawning invertebrate. Biological Bulletin **193**:20–29.

Cushing, D. H. 1986. Climate and fisheries. Academic Press, London, UK.

Doherty, P. J., and D. McB. Williams. 1988. The replenishment of coral reef fish populations. Oceanography and Marine Biology Annual Reviews **26**:487–551.

Doherty, P. J., S. Planes, and P. Mather. 1995. Gene flow and larval duration in seven species of fish from the Great Barrier Reef. Ecology **76**:2373–2391.

Eckman, J. E. 1996. Closing the larval loop: linking larval biology to the population dynamics of marine benthic invertebrates. Journal of Experimental Marine Biology and Ecology **200**:207–237.

Gaines, S., S. Brown, and J. Roughgarden. 1985. Spatial variation in larval concentrations as a cause of spatial variation in settlement for the barnacle, *Balanus glandula*. Oecologia **67**:267–272.

Gaines, S. D., and J. Roughgarden. 1985. Larval settlement rates: a leading determinant of structure in an ecological community of the marine intertidal zone. Proceedings of the National Academy of Science, USA **82**:3707–3711.

Grosberg, R. K. 1982. Intertidal zonation of barnacles: the influence of planktonic zonation of larvae on vertical distribution of adults. Ecology **63**:894–899.

Grosberg, R. K., and D. R. Levitan. 1992. For adults only? Supply-side ecology and the history of larval biology. Trends in Ecology and Evolution **7**:130–133.

Hall, V., and T. P. Hughes. 1996. Reproductive strategies of modular organisms: comparative studies of reef building corals. Ecology **77**:950–963.

Harrison, P. L., and C. C. Wallace. 1990. Reproduction, dispersal and recruitment of scleractinian corals. Pages 133–207 *in* Z. Dubinsky, editor. Ecosystems of the world. Volume 25. Elsevier, New York, New York, USA.

Harrison, P. L., R. C. Babcock, G. D. Bull, J. K. Oliver, C. C. Wallace, and B. L. Willis. 1984. Mass-spawning in tropical reef corals. Science **223**:1186–1189.

Hastie, T. J., and R. Tibshirani. 1990. Generalized additive models. Chapman & Hall, London, UK.

Hilborn, R., and C. J. Walters. 1992. Quantitative fisheries stock assessment. Chapman & Hall, New York, New York, USA.

Houde, E. D. 1987. Fish early life dynamics and recruitment variability. American Fisheries Society Symposium **2**:17–29.

Hughes, T. P. 1984. Population dynamics based on size rather than age: a general model with reef coral example. American Naturalist **123**:778–795.

Hughes, T. P. 1990. Recruitment limitation, mortality, and population regulation in open systems: a case study. Ecology **71**:12–20.

Hughes, T. P., and J. H. Connell. 1999. Multiple stressors on coral reefs: a long-term perspective. Limnology and Oceanography **44**:932–940.

Hughes, T. P., A. H. Baird, E. A. Dinsdale, N. A. Molchaniwskyj, M. S. Pratchett, J. E. Tanner, and B. L. Willis. 1999. Patterns of recruitment and abundance of corals along the Great Barrier Reef. Nature **397**:59–63.

IGOSS 1998. The Climate Data Library of the International Research Institute for Climate Predictions/Lamont Doherty Earth Observatory. ⟨**http://ingrid.ldgo.columbia.edn/SOURCES/.IGOSS/**⟩.

Karlson, R. H., and D. R. Levitan. 1990. Recruitment limitation in open populations of *Diadema antillarum*: an evaluation. Oecologia **82**:40–44.

Levitan, D. R. 1991. Influence of body size and population density on fertilization success and reproductive output in a free-spawning invertebrate. Biological Bulletin **181**:261–268.

Lipcius, R. N., and W. A. Van Engel. 1990. Blue crab population dynamics in Chesapeake Bay: variation in abundance (York River, 1972–1988) and stock-recruit functions. Bulletin of Marine Sciences **46**:180–194.

Milicich, M. J. 1994. Dynamic coupling of reef fish replenishment and oceanographic processes. Marine Ecology Progress Series **110**:135–144.

Oliver, J. K., B. A. King, B. L. Willis, R. C. Babcock, and E. Wolanski. 1992. Dispersal of coral larvae from a lagoonal reef. II. Comparisons between model predictions and observed concentrations. Continental Shelf Research **12**:873–889.

Pearse, J. P. 1990. Lunar reproductive rhythms in marine invertebrates: maximizing fertilization? Advances in Invertebrate Reproduction **5**:311–316.

Pennington, J. T. 1985. The ecology of fertilization of echinoid eggs: the consequences of sperm dilution, adult aggregation, and synchronous spawning. Biological Bulletin **169**:417–430.

Peterson, C. H., and H. C. Summerson. 1992. Basin-scale coherence of population dynamics of an exploited marine invertebrate, the bay scallop: implications of recruitment limitation. Marine Ecology Progress Series. **90**:257–272.

Reed, D. C., D. L. Laur, and A. W. Ebling. 1988. Variation on algal dispersal and recruitment: the importance of episodic events. Ecological Monographs **58**:321–335.

Richmond, R. H. 1993. Coral reefs: present problems and future concerns resulting from anthropogenic disturbance. American Zoologist **33**:524–536.

Ricker, W. E. 1954. Stock and recruitment. Journal of the Fisheries Board of Canada **11**:559–623.

Roughgarden, J., Y. Iwasa, and C. Baxter. 1985. Demographic theory for an open marine population with space-limited recruitment. Ecology **66**:54–67.

Strathmann, R. R. 1993. Hypotheses on the origins of marine larvae. Annual Reviews of Ecology and Systematics **24**: 89–117.

Thorson, G. 1950. Reproductive and larval ecology of marine bottom invertebrates. Biological Reviews **25**:1–45.

Underwood, A. J., and E. J. Denley. 1984. Paradigms, explanations, and generalizations in models for the structure of intertidal communities on rocky shores. Pages 151–180 *in* D. R. Strong, D. Simberloff, L. G. Abele, and A. B. Thistle, editors. Ecological communities: conceptual issues and the evidence. Princeton University Press, Princeton, New Jersey, USA.

Underwood, A. J., and P. G. Fairweather. 1989. Supply-side ecology and benthic marine ecology. Trends in Ecology and Evolution **4**:16–20.

Wallace, C. C. 1985*a*. Seasonal peaks and annual fluctuations in recruitment of juvenile scleractinian corals. Marine Ecology Progress Series **21**:289–298.

Wallace, C. C. 1985*b*. Reproduction, recruitment and fragmentation in nine sympatric species of the coral genus *Acropora*. Marine Biology **88**:217–233.

Westneat, M. W., and J. M. Resing. 1988. Predation on coral spawn by planktivorous fish. Coral Reefs **7**:89–92.

Willis, B. L., R. C. Babcock, P. L. Harrison, J. K. Oliver, and C. C. Wallace. 1985. Patterns in the mass spawning of corals on the Great Barrier Reef from 1981 to 1984. Proceedings of the 5th International Coral Reef Congress, Tahiti, **4**:343–348.

Willis, B. L., and J. K. Oliver. 1990. Direct tracking of coral larvae: implications for dispersal studies of planktonic larvae in topographically complex environments. Ophelia **32**: 145–162.

Wilson, J. R., and P. L. Harrison. 1998. Settlement-competency periods of larvae of three species of scleractinian corals. Marine Biology **131**:339–345.

Wolanski, E. 1994. Physical oceanographic processes of the Great Barrier Reef. CRC Press, Boca Raton, Florida, USA.

Yund, P. O. 1990. An in situ measurement of sperm dispersal in a colonial marine hydroid. Journal of Experimental Zoology **253**:102–106.

3 Coral Disease on the Great Barrier Reef

Bette L. Willis, Cathie A. Page, Elizabeth A. Dinsdale

3.1 Introduction

Coral disease is one of the most recent in a series of threats that is challenging the resilience of coral reef communities and is of particular concern because it may interact with and augment the impacts of other commonly recognised threats to coral health (e.g. bleaching, over-exploitation of fish stocks, destructive fishing practices and coastal developments). Since the first report of coral disease by Antonius in 1973, the rate of discovery of new diseases has increased dramatically with more than 29 coral diseases now described (Green and Bruckner 2000, Weil, this Vol.). Although coral disease is emerging as one of the major causes of coral reef deterioration in the Caribbean (Hayes and Goreau 1998; Harvell et al. 2002; Weil et al. 2002), at present we know very little about the ecology or pathology of coral disease on Indo-Pacific reefs. The comparatively few reports of coral disease from Indo-Pacific reefs, despite the region encompassing more than 80% of reefs worldwide (Bryant et al. 1998) is in contrast to the high proportion (>65%) of records in the Global Disease Database from the Caribbean reef region, now widely considered to be a coral disease hotspot (Green and Bruckner 2000; Weil, this Vol.). Such comparisons suggest that either disease is genuinely more prevalent in the Caribbean or lack of studies in other reef regions is underestimating its distribution and abundance. Distinguishing between these two alternatives represents an important step in advancing global epizootiological studies.

The rising incidence of marine diseases worldwide in the past few decades (Harvell et al. 1999), and particularly of coral diseases in the Caribbean, underscores the need for assessment of the status of disease on a region-by-region basis. Such assessments will help to identify the origins and reservoirs of pathogens and vectors involved in disease transmission. The Great Barrier Reef (GBR) stretches over 2000 km along the eastern coastline of Australia, representing the largest reef tract under management worldwide. Its unique status as one of the few reef systems under government jurisdiction for timescales that have preceded recent increases in the prevalence of coral disease has the potential to provide important insights into factors influencing disease occurrence and the underlying causes of escalating disease incidence. In this chapter, we summarise the current state of knowledge of coral disease on the Great Barrier Reef by (1) describing syndromes and diseases observed in our studies on GBR reefs and interpreted in the light of published literature

and (2) presenting the results of a 5-year, large-scale study in conjunction with a regional disease prevalence study that together provide an overview of the current status of disease occurring on reefs extending over 1200 km of the Great Barrier Reef.

3.2 Overview of Diseases Infecting Great Barrier Reef and Indo-Pacific Corals

Until recently, it has been tacitly assumed that disease has had little impact on the population dynamics or community structure of coral assemblages on the Great Barrier Reef (GBR). However, there have been only two detailed studies of coral diseases on the GBR, both at Lizard Island in the northern sector: one on black band disease (BBD; Dinsdale 2002) and the other on skeletal eroding band (SEB; Antonius 1999; Antonius and Lipscomb 2001). A few additional sightings of coral diseases have been reported in anecdotal notes, i.e. BBD (Miller 1996) and white band disease (WBD; Baird 2000), although the report of WBD must be viewed with caution since a number of diseases are now known to produce white band-like symptoms (e.g. WBDI, WBDII, white plague I and II). Fungal pathogens have also been reported in gorgonians (Morrison-Gardiner 2001) and tumours in scleractinian corals (Loya et al. 1984). However, in general, there have been few studies specifically targeting coral disease, a factor likely to have contributed to the current paradigm of apparently low occurrence of coral disease on the GBR.

Elsewhere in the Indo-Pacific, in addition to BBD, SEB and WBD (Antonius 1985), there are isolated reports of diseases generally not yet described from the Caribbean. For example, yellow band disease (YBD) affected ten species primarily from the families Acroporidae and Poritidae in the Arabian Gulf (Korrubel and Riegl 1998); the encysting stage of a trematode has infected *Porites compressa* in Hawaii causing enlarged pink polyps (Aeby 1991); and *Porites* ulcerative white spot disease (PUWSD) infected more than 20% of *Porites* colonies on 8 out of 10 reefs surveyed in the Philippines (Raymundo et al. 2003). In addition, fungal-algal associations have affected *Porites lobata* in French Polynesia (Le Champion-Alsumard et al. 1995), cyanobacteria have affected *Porites luta-* in the Indian Ocean (Ravindran and Raghukumar 2002), and a bacterial pathogen has infected coralline algae [coralline lethal orange disease (CLOD)] throughout a large part of the South Pacific (Cook Islands, Fiji, Solomon Islands and Papua New Guinea, GBR; Littler and Littler 1995; C. Page, pers. observ.). Thus, despite the paucity of studies of coral disease in the Indo-Pacific region, the occurrence of the more common and infectious Caribbean diseases, in combination with reports of diseases unique to the region, suggest that infectious pathogens are a common component of Indo-Pacific reef communities and that disease may have a greater role in structuring coral communities in the region than previously thought.

3.2.1
Black Band Disease on the Great Barrier Reef

Black-band disease was first observed on GBR reefs in 1994 (Dinsdale 1994), more than two decades after the first Caribbean record (Antonius 1973) and a decade after the first Indo-Pacific record (Antonius 1985). Initial Indo-Pacific records were for two massive faviid species, *Goniastrea pectinata* and *Platygyra lamellina*, from the Philippines and a further seven massive faviids from the Red Sea (ibid), but BBD has subsequently been recorded on 21 species in five families from Lizard Island, GBR (Dinsdale 2002). Unlike in the Caribbean, where BBD primarily infects massive species (Kuta and Richardson 1996), branching pocilloporid and acroporid corals are important host species on the GBR (6.0 and 3.6% of corals in the families Pocilloporidae and Acroporidae, compared to 3.0 and 2.1% in the primarily massive families Faviidae and Poritidae; Dinsdale 2002; Fig. 3.1a, b). Dinsdale (2002) found a mean prevalence of 2.8% (and range of 1.3–4.9%) on Lizard Island reefs in the summer of 1994, which is comparable to the prevalence of BBD on most Caribbean reefs (Green and Bruckner 2000, Weil 2003). Seasonal increases in the prevalence of BBD on reefs in the Caribbean region are related to high summer seawater temperatures, but may also be related to water depth, coral diversity, population density and elevated nutrients (Kuta and Richardson 1996, 2002; Bruckner and Bruckner 1997; Bruckner et al. 1997). However, there are no studies of factors associated with the prevalence of BBD on GBR reefs, so the global generality of these patterns, particularly the associations between high temperatures and nutrients and increased abundance of BBD, remains unclear.

BBD isolated from Caribbean corals was originally described as a consortium of microorganisms dominated by *Phormidium corallyticum*, a gliding filamentous cyanobacteria, but including heterotrophic bacteria, marine fungus, sulphide-oxidising bacteria (*Beggiatoa*) and sulphate-reducing bacteria (*Desulfovibrio*; Ducklow and Mitchell 1979; Richardson 1996). However, recent molecular studies have identified anomalies in the identification of the cyanobacteria suspected to be the causative agent and a range of additional microorganisms associated with BBD mats of corals from St Croix, US Virgin Islands, Curacao, Netherlands Antilles and New Britain, Papua New Guinea (Cooney et al. 2002; Frias-Lopez et al. 2002, 2003). rDNA sequence analysis of microorganisms isolated from BBD mats have revealed the presence of up to three unidentified taxa of cyanobacteria, whereas *P. corallyticum*, the previously identified causative agent, was not detected (Cooney et al. 2002; Frias-Lopez et al. 2002, 2003). The lack of concordance in the cyanobacterial species associated with BBD mats between these and earlier studies and the differences in cyanobacterial taxa between Caribbean and Indo-Pacific (PNG) corals (Frias-Lopez et al. 2003) raise questions about the causative agent. They also highlight the need for further microbial and molecular studies of BBD from different coral species and from different reef regions. There have been no studies of microorganisms associated with BBD mats on GBR corals and it is possible that cyano-

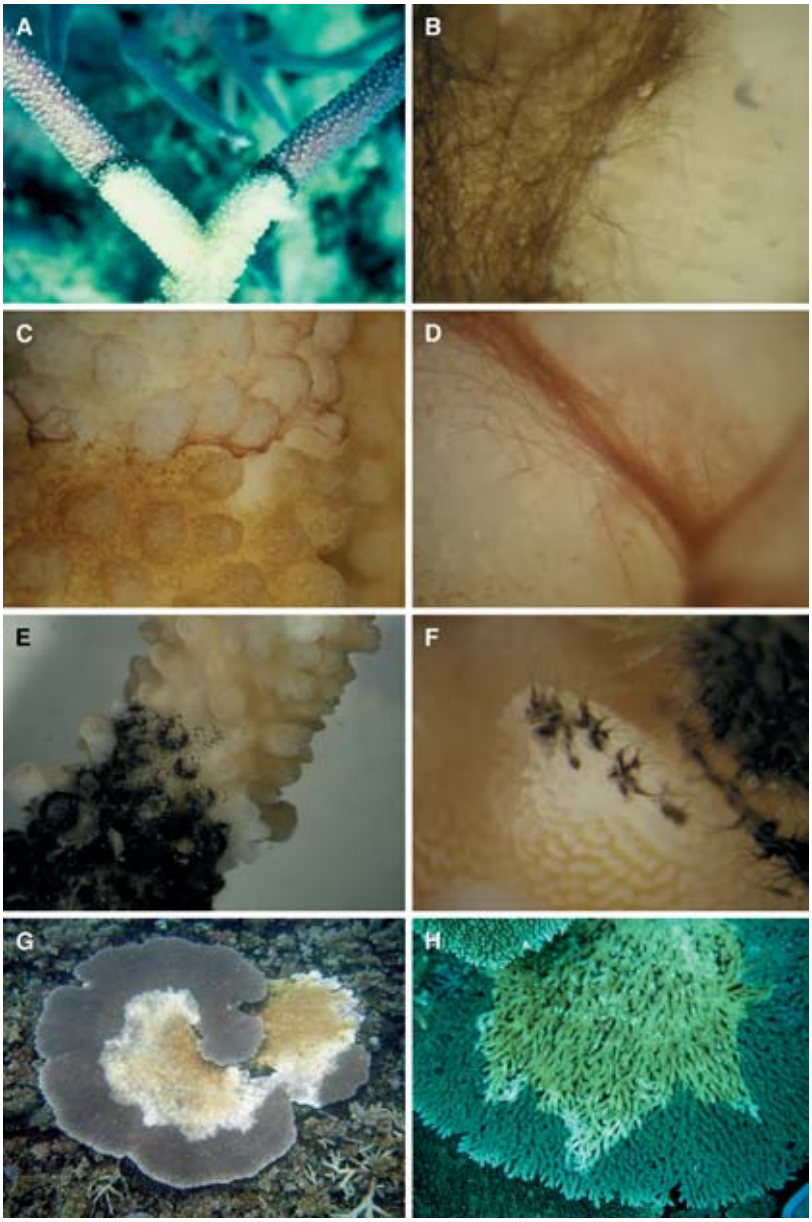


Fig. 3.1A–H. Field appearance of diseases and syndromes infecting corals and gorgonians on the Great Barrier Reef: **a** black band disease (BBD) on *Acropora intermedia,* **b** cyanobacterial mat, responsible for the black colouration of BBD, **c** unknown cyanobacteria forming a mat at tissue-skeleton interface, **d** unknown red-brown cyanobacteria forming BBD-like mat, **e** skeletal eroding band (SEB) on *A. intermedia* showing speckled appearance of band near tissue interface, **f** clusters of the ciliate, *Halofolliculina corallasia*, on live coral tissue ahead of the main SEB front, **g** white syndrome (WS) on *Acropora hyacinthus* in the Capricorn Bunker sector (photo AIMS LTMP), **h** WS on *Acropora clathrata* in the Lizard Is./Cooktown sector (photo AIMS LTMP)

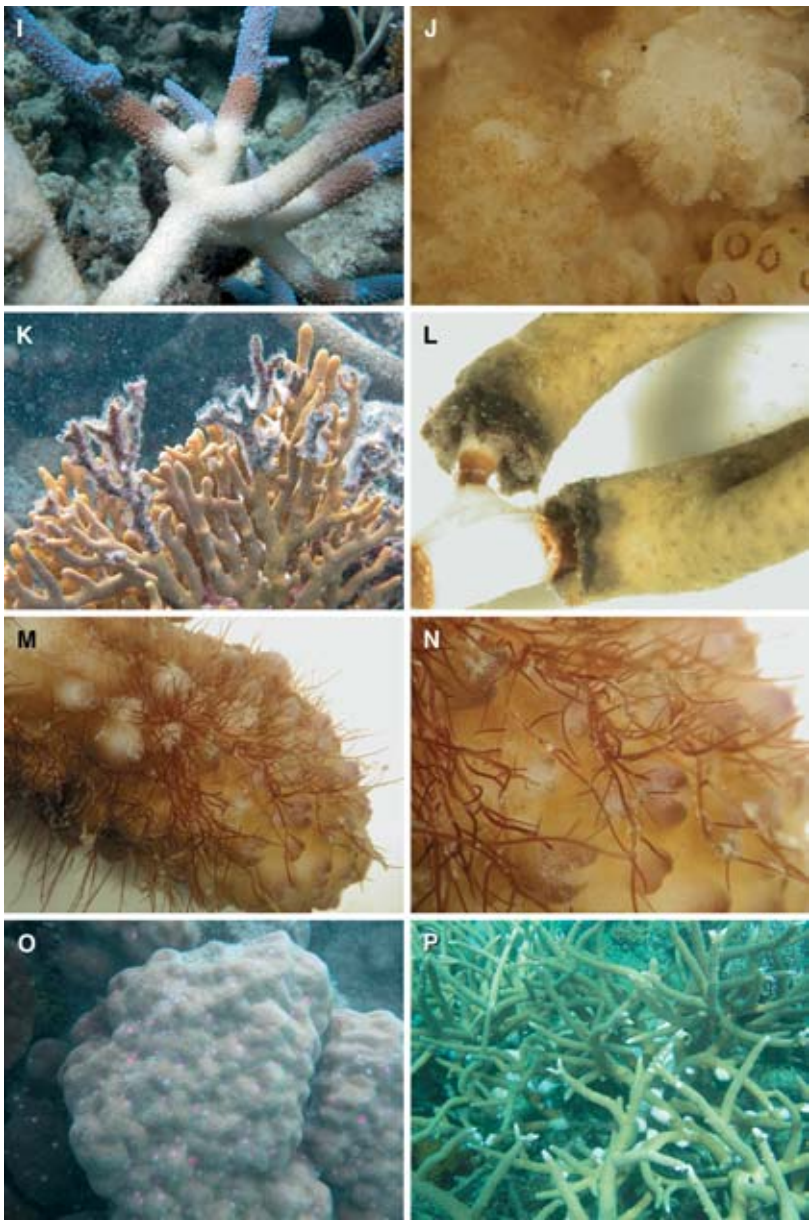


Fig. 3.1I–P.i brown band (BrB) on *A. muricata*, **j** clusters of the ciliates that cause the distinctive colouration of BrB, **k** black necrosing syndrome (BNS) on the gorgonian, *Isis* sp., **l** skeletal axis of *Isis* sp. exposed by BNS, **m** coral-algal interactions, **n** detail of filamentous algae overgrowing live coral tissue, **o** pink pigmented spots (PS) on massive *Porites* sp., **p** coral tumours on *Acropora* (photo L. Vail). All photographs were taken on Lizard Is. reefs by authors unless otherwise indicated

bacteria associated with GBR infections may differ from those isolated from Caribbean and even PNG corals. During our regional disease prevalence surveys (see Sects. 3.3.1.2, 3.3.3), we identified more than one type of cyanobacteria associated with coral disease states that resembled BBD (Fig. 3.1c, d). Therefore, in our analysis we have included unidentified cyanobacterial syndromes in the BBD category.

3.2.2 Skeletal Eroding Band : an Indo-Pacific Coral Disease?

Skeletal eroding band (SEB) is the only disease condition other than BBD for which there are more than anecdotal reports on the Great Barrier Reef. SEB is caused by the protozoan, *Halofolliculina corallasia* (Fig. 3.1e, f), which erodes the tissue and skeleton of corals as it produces a black lorica or test (Antonius 1999). Tissue damage occurs when the ciliates mechanically disrupt and lyse coral tissues through spinning and secretion of chemicals in the process of embedding their loricae within the coral skeletal matrix. Clusters of ciliates along the tissue-skeleton interface produce a black band (Fig. 3.1e) similar in appearance to black band disease, but the skeleton behind the advancing SEB is speckled with the remains of empty black loricae (Antonius and Lipscomb 2001), unlike the uniformly white skeleton exposed as BBD advances. Antonius and Libscomb (2001) report that the progression of SEB can be relatively slow, approximately 1 mm per week, further distinguishing it from BBD, but that it may also advance at rates up to 1 mm per day, comparable to BBD.

SEB affects at least 24 species of corals on reefs throughout the Indo-Pacific, but despite searching, there are no records from the Caribbean or the Atlantic Ocean (Antonius and Lipscomb 2001). A qualitative, 6-point scale was used to measure the prevalence of SEB on Indo-Pacific reefs, scoring the abundance of disease from rare (1–3 cases of SEB/30-min swim) to catastrophic (>100 cases per 30-min swim) (ibid). Prevalence of SEB increased in all reef regions revisited; from rare to moderate (4–12 cases/30-min swim) in the 10 years between visits to Lizard Island, GBR (1988–1998), and from rare to frequent (13–25 cases) in the 8 years between visits to Mauritius (1990–1998) and in the 3 years between visits to the Sinai (1994–1997) (ibid). Apart from these records at Lizard Island in the northern sector, the geographic extent of SEB on the GBR is currently unknown (but see Sect. 3.3.3).

3.2.3 White Syndrome – a Collective Term for Conditions Producing White Symptoms on the Great Barrier Reef

A proliferation of names for coral diseases that produce white symptoms in Caribbean corals presents challenges for relating Indo-Pacific white syndromes to the Caribbean white diseases based on macroscopic field characters. Rather than attempt to identify features such as the variable zone of bleached

tissue that distinguishes white band II (WBII) from white band I (WBI), or differences in the rates of movement that distinguish the faster moving white plague II (WPII) from white plague I (WPI; reviewed in Richardson 1998), we have chosen to use the collective term white syndrome (WS) to describe conditions resulting in white bands of tissue and/or skeleton on GBR corals (Fig. 3.1g, h). In addition to WBI/II and WPI/II, white syndrome could potentially encompass white pox (Patterson et al. 2002), patchy necrosis (Bruckner and Bruckner 1997; Rodriguez-Martinez et al. 2001), and even shut down reaction (Antonius 1977). However, WS is distinguished from feeding scars by the narrow width of the zone of recently exposed, white skeleton and the relatively regular appearance of the tissue front. These features are in contrast to the wide zone of white skeleton commonly exposed following *Acanthaster planci* predation and the scalloped or irregular tissue front produced by *Drupella* spp.

Determining the relationship(s) between the Caribbean white diseases and WS and applying the appropriate name(s) will not be possible until pathogens infecting GBR corals are isolated and compared to those producing white symptoms in Caribbean corals. It is thus difficult to determine the accuracy of records of white band disease on the GBR (Baird 2000; Antonius and Lipscomb 2001) and of records of WBD infecting 20 coral species in the Philippines (Antonius 1985). However, since white band disease and white plague have caused major changes to coral communities in the Caribbean region (Aronson et al. 1998; Green and Bruckner 2000; Aronson and Precht 2001), the potential for their presence and impact on coral communities on the GBR should be viewed with concern (see Sect. 3.3.2 for current distribution and abundance of WS on the GBR).

3.2.4
Brown Band: a New Syndrome on the Great Barrier Reef

Brown band (BrB) is a new syndrome that we have recorded for the first time infecting corals on surveys in the northern and southern sectors of the GBR (see Sect. 3.3.3). The distinctive macroscopic field symptom of corals infected with BrB is a brown zone of variable width, flanked by healthy tissue at the advancing front and exposed white skeleton at the trailing edge as the band progresses over the surface of the colony (Fig. 3.1i). There is often a white zone between the healthy tissue and brown band, which may comprise bleached tissue and/or denuded skeleton. Dense populations of ciliates, packed with zooxanthellae from engulfed coral tissue, cause the brown coloration of the band (Fig. 3.1j). As densities of ciliates decrease, the zone becomes lighter and may appear white at very low ciliate densities. In these latter cases, the condition would be assigned to the WS category based solely on field observations. It is possible that BrB is caused by the ciliate, *Helicostoma nonatum*, which is thought to produce a brown jelly-like condition on corals grown in aquaria (Borneman 2001), but to our knowledge, this ciliate infestation has not been reported previously from in situ corals. Note that an earlier report of a brown

band on a colony of *Acropora formosa* (Dinsdale 1994) referred to a different, but unknown syndrome, and has subsequently been mistakenly quoted as affecting 20 coral species on the GBR (Santavy and Peters 1997; Borneman 2001). While it is possible that the unknown syndrome was caused by a cyanobacterium similar to the one causing red-band disease in the Caribbean, as suggested by Santavy and Peters (1997), in the absence of the specimen it is not useful to speculate further about this isolated observation; it is not to be considered a record of BrB as described here.

3.2.5
Gorgonian Infections on the Great Barrier Reef: Black Necrosing Syndrome

Gorgonians are highly susceptible to disease in the Caribbean, where the fungal disease Aspergillosis has infected 12–90% of gorgonians on reefs in 13 countries (Nagelkerken et al. 1997a, b; Smith 2003) and black band disease has infected 13.8% of some species in the Florida Keys (Fengold 1988). However, little is known about gorgonian diseases on the GBR. The only study of GBR gorgonians to date reports that 10% of populations of *Isis hippuris* on Davies Reef were infected with a fungal disease that manifested as black necrotic areas and led to loss of both tissues and skeleton (Morrison-Gardiner 2001). Although two species of *Penicillium* isolated from infected gorgonians were able to infect healthy colonies of *I. hippuris* and *Pinnigorgia* sp., and could be re-isolated, they did not produce the typical symptoms of the disease (Morrison-Gardiner 2001). We have also observed black necrotic patches on many gorgonians at Lizard Island during our regional disease prevalence surveys (see Sect. 3.3.3) and will refer to the disease state as black necrosing syndrome (BNS; Fig. 3.1k, l). Whether gorgonian species on the GBR produce antifungal compounds similar to those produced by Caribbean gorgonians (Kim et al. 2000a, b), or vary in their susceptibility to fungal infections (Nagelkerken et al. 1997a) is unknown, but merits further study.

3.2.6
Coral-Algal Interactions: Algal Infections?

The impacts of coral-algal interactions may be positive, neutral or negative for the coral (reviewed in McCook et al. 2001), with negative interactions generally being discussed in the context of competition. However, when interactions that negatively affect corals (1) result in net positive benefits for algae and (2) impede the functioning and growth of coral polyps (e.g. through direct overgrowth and/or invasion of coral tissue), they take on the character of a disease. On reefs in the central GBR, examples that appear to cross the boundary between a competitive interaction and disease include overgrowth of coral by (1) the filamentous algae, *Coralliophila hurysmansii* causing tissue swelling, and (2) by *Anotrichium tenue*, which traps mucus, sediments and possibly microbes

damaging the underlying tissues (McCook et al. 2001). We also found filamentous algae overgrowing live coral tissue in both the southern and northern GBR (Fig. 3.1m, n). What is unclear at this stage is whether some other stress or pathogen had previously weakened the corals' resistance allowing algae to invade their tissues. Therefore, rather than attribute coral mortality solely to algal overgrowth in our disease prevalence surveys (Sect. 3.3.3), we assigned such cases to an unidentified syndrome category. However, reports of a coralline red alga, *Pneophyllum conicum*, overgrowing and killing up to 100% of colonies of nearly all coral species present on a patch of reef in Mauritius (Antonius and Afonso-Carillo 2001) suggest that algal overgrowth can reach epizootic status. Controlled experimental studies on the ability of algal species to infect healthy coral tissues will clarify the pathogenic nature these coral-algae interactions.

3.2.7 Pigmentation Response in *Porites:* A symptom with a variety of causes?

The reef coral, *Porites,* appears to respond to a variety of competitive, invasive and parasitic challenges by producing pink or purple pigmentation in polyps adjacent to interaction sites (Fig. 3.1o). Hence pink lines, rings or spots are often visible in coral tissue bordering the margins of competing or boring organisms. The pigmentation appears to be a symptom of a response mounted by the coral to contain invading or competing organisms such as cyanobacteria (Ravindran and Raghukumar 2002), polychaetes, molluscs, and the intermediate metacercariae stage of the digenetic trematode, *Podocotyloides stenometra* (Aeby 1991, 1998). The trematode has been reported to encyst in tissues of the massive coral, *Porites compressa,* on Hawaiian reefs causing coral polyps to appear swollen and pink in colour (Aeby 1998). Infected polyps are unable to retract, reducing their function and increasing their vulnerability to predation by butterflyfish, the final host for the trematode. On Hawaiian reefs, the pink spots represent a parasitic infection, which reduces growth of heavily infected colonies by up to 50% (Aeby 1991). When the cysts were removed (through fish predation), healthy coral polyps were regenerated. We recorded the presence of pigmented spots (PS) on *Porites* colonies as a potential indicator of a parasitic infection in our GBR disease prevalence studies (see section 3.3.3). The pigmented spots appeared as small raised pink areas surrounded by healthy tissue, however the presence of trematodes has not been confirmed. Their location in the midst of healthy tissue is more consistent with a parasitic infection than a competitive interaction, unlike a variety of pink lines or rings that were commonly seen bordering dead patches and could generally be interpreted as a response to competitive interactions.

3.2.8
Coral Tumours

Coral tumours, manifesting as raised roughly spherical masses projecting about 4.5 cm above the surface of the colony, were reported to affect 18–24% of populations of *Platygyra pini* and *P. sinensis* on Magnetic Island, central GBR (Loya et al. 1984). Tumours were associated with increased growth rates of polyps and a general proliferation of all cell types, some atrophied and others normal, but in all cases macroscopic polyp structures were discernible and tissues remained pigmented (Loya et al. 1984). This type of abnormal growth has been termed a hyperplasia, in contrast to the bleached neoplasms that have been classified as calicoblastic epitheliomas. The latter appear as white, globular masses of skeleton raised above the surface of the colony and have few discernible polyp structures (reviewed in Peters et al. 1986). Tumours identified in our disease prevalence surveys were similar to the latter bleached neoplasms (Fig. 3.1p; see Sect. 3.3.3). Such tumours tend to be largest and most concentrated in the centre of colonies of table acroporids in the Gulf of Oman, whereas they tend to be similar in size along the length of branches in arborescent species (Coles and Seapy 1998). In high densities, tumours may reduce UV absorption rates (Coles and Seapy 1998), lipid storage capacity (Yamashiro et al. 2001) and linear growth rates of colonies (Bak 1983). Bleached neoplasms occur mainly on corals in the family Acroporidae and have been reported from throughout the Indo-Pacific, i.e. from Guam and Enewetak (Cheney 1975), French Polynesia (Le Champion-Alsumard et al. 1995), Japan (Yamashiro et al. 2001) and the Gulf of Oman (Coles and Seapy 1998).

3.3
Coral Disease Surveys on the Great Barrier Reef

The diversity of diseases and syndromes infecting GBR corals as described above highlights the need for targeted surveys of coral disease in the region. Here, we present the results of two types of studies designed to redress this need: (1) a large-scale study comprising rapid annual surveys of coral disease abundance (# cases per site) on 48 reefs as part of the Australian Institute of Marine Science (AIMS) long-term monitoring program (LTMP; Sweatman et al. 2001), and (2) a regional study comprising belt transect surveys to estimate disease prevalence (i.e. the total number of cases of disease expressed as a proportion of the total number of colonies examined per reef, site, family/order or disease category as appropriate) at selected sites in the northern and southern GBR. The large-scale AIMS LTMP surveys provide a broad overview of the abundance of two coral diseases (WS and BBD) on reefs throughout the Great Barrier Reef and follow changes in the number of cases of each disease over the last 5 years. The regional disease prevalence surveys are designed to detect all diseases and syndromes present at selected GBR sites, to determine their prev-

alence with respect to species and family groups, and to determine changes in prevalence associated with season, coral cover and wave exposure.

3.3.1 Survey Protocols

3.3.1.1 Large-scale Australian Institute of Marine Science Long-Term Monitoring Program Surveys

Forty-eight reefs spanning 1200 km of the Great Barrier Reef were surveyed for coral disease annually between 1998 and 2003 (Sweatman et al. 2001). Reefs were partitioned into six latitudinal sectors (i.e. Cooktown/Lizard Is., Cairns, Townsville, Whitsundays, Swains and Capricorn Bunkers sectors; Fig. 3.2) and three cross-shelf locations (inner, mid and outer-shelf). Within each sector, generally three reefs were surveyed in each of the three cross-shelf locations

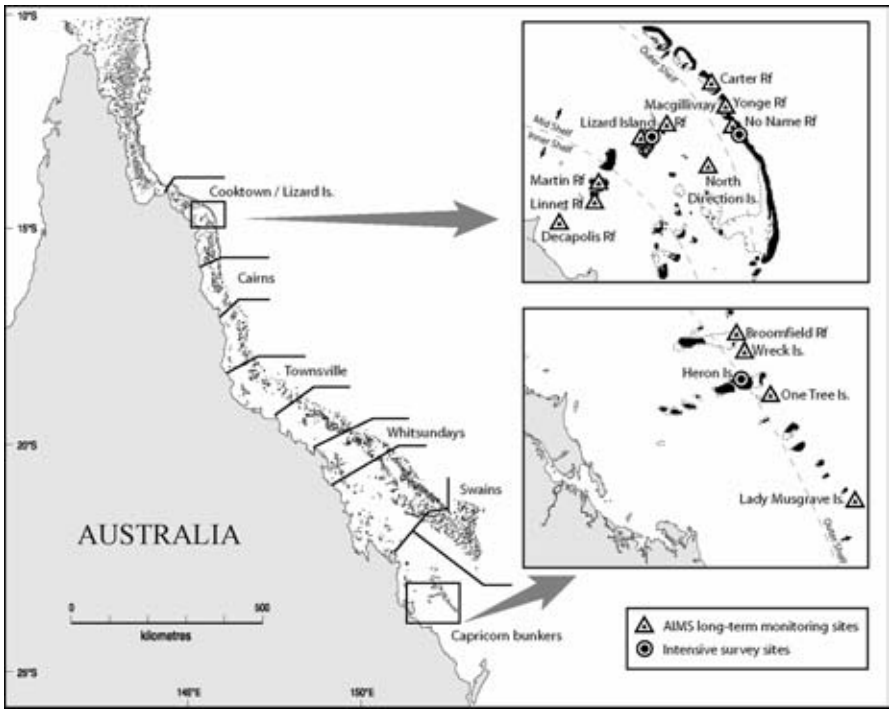


Fig. 3.2. Map of the Great Barrier Reef showing (1) the six sectors, and (2) the inner-, mid- and outer-shelf reefs in the Cooktown/Lizard Island sector and the outer-shelf reefs in the Capricorn Bunker sector that were surveyed as part of the Australian Institute of Marine Science Long Term Monitoring Program (AIMS LTMP), and (3) sites for the detailed surveys at Lizard Island and No Name Reef in the Cooktown/Lizard Is. sector and Heron Island in the Capricorn Bunker sector

(full methods in Sweatman et al. 2001). In total, there were 15 cross-shelf/sector combinations, which we will refer to as regions. Five 50-m transects were surveyed at each of three sites on the northeast flank of each reef. Transects were permanently marked and followed depth contours on the reef slope at 6–9 m. Surveys in the first 2 years (1998/1999, 1999/2000) were spread over the warmer months (September–May), whereas in the last 3 years, surveys in some sectors included the austral winter months of July and August. Changes in the timing of the surveys are discussed further in the context of their impact on disease prevalence in Section 3.3.2.2.

Coral mortality attributable to disease (BBD, WS), predation (*Acanthaster planci*, *Drupella*) and unknown sources was recorded in visual censuses (as per Bass and Miller 1996) of 2-m belts along each 50-m transect; thus an area of 1500 m^2 was surveyed on each reef. Diseases were identified from macroscopic field symptoms as outlined in Sections 3.2.1 for BBD and 3.2.3 for WS. Counts of the number of coral colonies manifesting symptoms of the two disease states on each transect are hereafter referred to as the number of cases of BBD or WS. It is likely that some cases of skeletal eroding band (SEB) and brown band (BrB) are included in the WS category because both can appear as white zones when ciliate densities are low (discussed in Sect. 3.2.4). Mortality was attributed to *A. planci* or *Drupella* when white zones were consistent with the appearance of feeding scars (see Sect. 3.2.3) and/or these predators were visible in the vicinity of white zones adjacent to healthy coral tissue. If coral mortality could not be clearly attributable to disease or predation, it was recorded in the unknown category. Percent cover estimates of benthic groups were determined from video transects (further details in Page et al. 2001).

3.3.1.2
Regional Disease Prevalence Surveys

To determine the prevalence of coral disease in summer, we surveyed eight sites in January 2003 in the northern and southern sectors of the GBR, where the AIMS LTMP found the highest number of cases of disease (see Sect. 3.3.2.2). The eight sites comprised: four mid-shelf sites at Lizard Island [two exposed (Bird Is., Lizard Head) and two sheltered (Vicki's and Horseshoe Reefs)] and two outer-shelf sites at No Name Reef (the exposed NE front and sheltered NW back reef) in the northernmost sector; and two sites [one exposed (Coral Gardens) and one sheltered (Little Bay)] at Heron Island in the southernmost sector of the GBR (Fig. 3.2). The two sheltered Lizard Island sites were also surveyed in winter (July 2002) to initiate seasonal comparisons of disease prevalence. At each site, three random 20×2 m belt transects were surveyed along depth contours at 3–6 m and all hard corals, soft corals and gorgonians were identified to the lowest taxonomic level recognised or morphological group as appropriate. Each colony was then categorised as healthy, bleached, or assigned to one of eight disease categories: BBD (including BBD-like mats associated with a number of different cyanobacteria), SEB, WS, BrB, tumour, BNS, PS (pigmented spots on *Porites*), or

to an unidentified syndrome category. The unidentified syndrome category included filamentous algae overgrowing live coral tissue and unidentified syndromes causing deterioration in soft corals. Samples of diseased colonies were collected and examined microscopically to identify associated microorganisms and verify field identifications of disease states. To enable comparisons of disease prevalence with coral cover, we used line intercept surveys to record percent cover of the major benthic categories along the first 10 m of each transect.

3.3.1.3 Statistical Analysis

Differences in the abundance of WS detected in the AIMS LTMP surveys among shelf positions, sectors and years were tested using split-plot ANOVA. The total number of diseased colonies were summed over transects on each reef. Data were log transformed [log (X+0.1)] to satisfy assumptions of normality and homogeneity of variances. Where significant changes in disease abundance over time among sectors and shelf positions were identified, available degrees of freedom were partitioned into single degree of freedom contrasts to determine the specific years in which changes occurred within each sector by shelf combination. The abundances of BBD were too low to allow formal analysis of change.

Differences in distribution of WS among shelf positions, sectors and years were also examined by comparing changes in the proportion of transects on which WS was recorded using split-plot ANOVA. The number of transects with disease present was summed on each reef and divided by the number of transects sampled. The data were square root transformed to satisfy assumptions of normality and homogeneity of variances. As for WS abundance above, when significant changes over time in the proportion of transects with disease were identified among sectors and shelf positions, contrasts were used to determine the specific years in which changes occurred.

The relationships between WS abundance and (1) hard coral cover and (2) *Drupella* spp. abundance were examined by including hard coral cover and abundance of *Drupella* as covariates in a split-plot ANOVA model. Interaction terms in the model were used to estimate how consistent differences in relationships with WS abundance were among sectors and shelf positions. The abundances of WS and *Drupella* were log (X+0.1) transformed for analysis as described above. Similarly, single degree of freedom contrasts were used to determine when the relationship between disease abundance and coral cover or *Drupella* abundances differed among sectors and shelf positions.

The relationship between change in percent hard coral cover and change in WS abundance was also examined by including the change in cover of hard corals between years as a covariate in an additional split-plot ANOVA model.

Variations in disease prevalence detected in the regional disease prevalence surveys were compared among reefs (Lizard Is., No Name, Heron Is.) and among seasons (winter vs. summer) and exposures (sheltered vs. exposed) on

Lizard Is. reefs using separate 1-way ANOVAs. When Levene's test determined that variances were heterogeneous, data were arcsine transformed. Differences in the distribution of the number of diseased vs. healthy colonies, pooled for the two sheltered and two exposed sites at Lizard Is., among the five scleractinian families in summer 2003 were tested using a χ^2 homogeneity test.

3.3.2 Results of Large-Scale AIMS LTMP Surveys

3.3.2.1 Patterns in the Distribution and Abundance of Black Band Disease

BBD is widespread throughout the GBR, occurring in all six sectors and all three cross-shelf locations. There were only three regions (mid-shelf Cooktown/Lizard Is., inner-shelf Cairns, and outer-shelf Townsville), of the 15 surveyed, in which BBD was not detected in any of the surveys. However, in any one year, BBD was recorded on a maximum of 2.5% of transects (n=720) from a maximum of 47% of regions (n=15).

The abundances of BBD were too low to allow formal analysis of change, however, the number of colonies infected by BBD did not appear to change markedly between 1998 and 2003 (Fig. 3.3), infections occurring on 0.04–0.47 colonies per reef in any given year. The highest occurrence of BBD was a total of 22

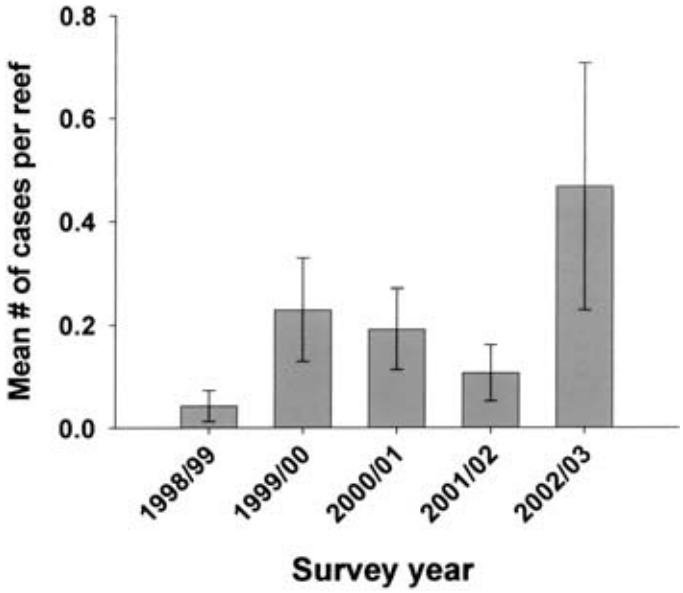


Fig. 3.3. Mean abundance (±SE) of black band disease (BBD) in survey years between 1998 and 2003. Histograms represent the mean of the total number of cases of BBD (±SE) in the 1500-m^2 area surveyed on each of the n=48 reefs per survey season

cases across all reefs in 2002/2003. Thus, despite its widespread distribution, the general abundance of BBD has been very low and stable, for the last 5 years.

3.3.2.2 Patterns in the Distribution and Abundance of White Syndrome

Abundance of WS

In contrast to the stable abundance of BBD over the past 5 years, white syndrome has increased 20-fold (F_{year}=52.12, *df*=4, *P*<0.001), from a mean of 1.7±0.58 cases in 1998 to 47.7±12.60 cases in 2002/2003 (Fig. 3.4). Mean occurrence of WS has increased at all three cross-shelf locations (Fig. 3.5), with significantly greater increases occurring on outer-shelf reefs, where there was a mean of 85±29.5 cases per reef in 2002/2003 (F_{shelf}=13.28, *df*=2, *P*<0.001). Overall, there is a pattern of increasing occurrence of WS with increasing distance from the coast over the 5 years ($F_{year \times shelf}$=1.36, *df*= 8, *P*=0.221), a pattern that is particularly pronounced in the last two survey years (Fig. 3.5). However, the pattern breaks down when variances due to the sector level are factored in ($F_{year \times shelf \times sector}$=1.91, *df*=28, *P*=0.008) because of the comparatively constant abundance of WS on all cross-shelf transects (within each year) in the Townsville, Whitsundays and Swains sectors and the higher abundance of WS on transects on the mid-shelf reefs in the Cairns sector in 2002/2003.

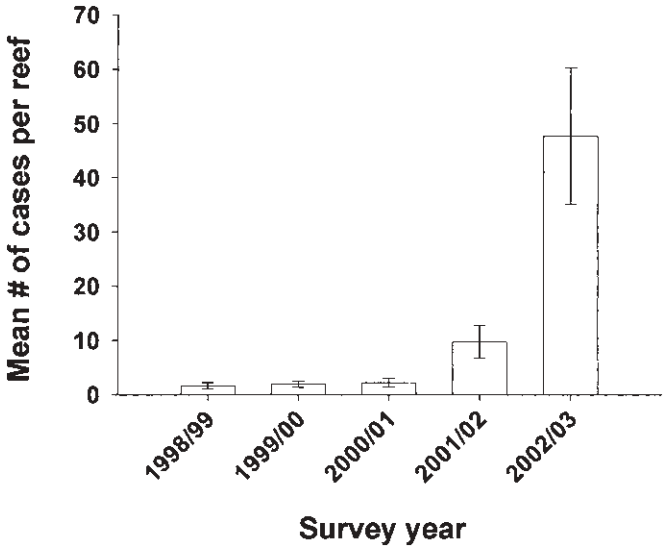


Fig. 3.4. Mean abundance (±SE) of white syndrome (WS) in survey years between 1998 and 2003. Histograms represent the mean (±SE) of the total number of cases of WS in the 1500-m^2 area surveyed on each of the reefs (n=48) per survey year

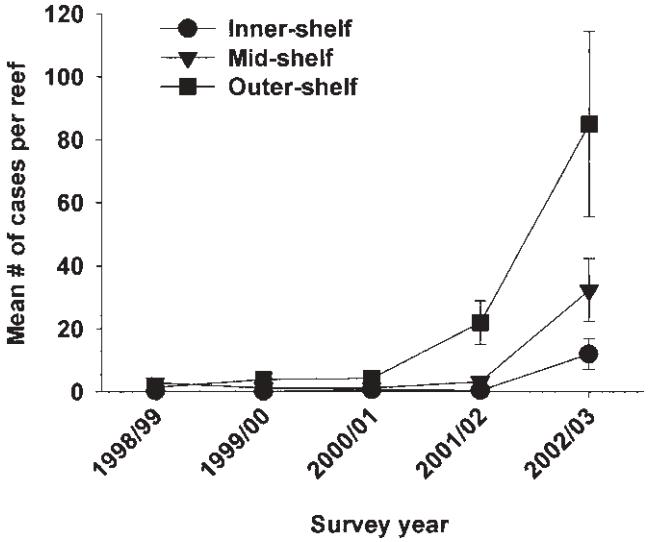


Fig. 3.5. Comparison of patterns in the mean (±SE) number of white syndrome (WS) cases per reef throughout the 5-year study period among reefs in three cross-shelf locations [i.e. inner-shelf (n=12 reefs), mid-shelf (n=18 reefs), and outer-shelf (n=18 reefs)]

The increase in WS abundance through time occurred in every sector except Townsville, where it has remained at low levels (≤11 cases per reef) since 1998/1999 (Fig. 3.6). Increases were first detected on outer-shelf reefs in the northernmost Cooktown/Lizard Is. sector in 2001/2002, when a more than 20-fold increase in the number of cases was recorded (an increase from a mean of 3 to 67 cases per reef). Smaller increases were also detected on both mid- and outer-shelf reefs from the central Whitsundays sector south to the southernmost sector (i.e. on outer-shelf reefs in the Whitsundays, mid-shelf reefs in the Swains and outer-shelf reefs in the Capricorn Bunker sectors). A dramatic, 30-fold increase in WS to a mean of 304 cases per reef occurred in the following year (2002/2003) on outer-shelf reefs in the southernmost Capricorn Bunker sector. The greatest increases in WS also occurred this year in regions representing all cross-shelf locations and all sectors except Townsville (i.e. on inner-shelf reefs of Cooktown/Lizard Is., mid-shelf reefs of Cairns, inner- and mid-shelf reefs of the Whitsundays, and mid- and outer-shelf reefs of the Swains sectors; Fig. 3.6). In summary, mean occurrence of WS has either increased (9 regions) or remained constant (6 regions) in all regions surveyed (n=15) on the GBR between 1998 and 2003 ($F_{year \times sector \times shelf}$ =1.92, df=28, P=0.008).

Given that prevalence of coral diseases like BBD and white pox increase with high summer temperatures (Rodriguez-Martinez et al. 2001; Kuta and Richardson 2002), changes in the timing of survey seasons from warmer to cooler months in the Cooktown/Lizard Is and Capricorn Bunker sectors in 2000/2001 would be predicted to have underestimated the potential magnitude of changes

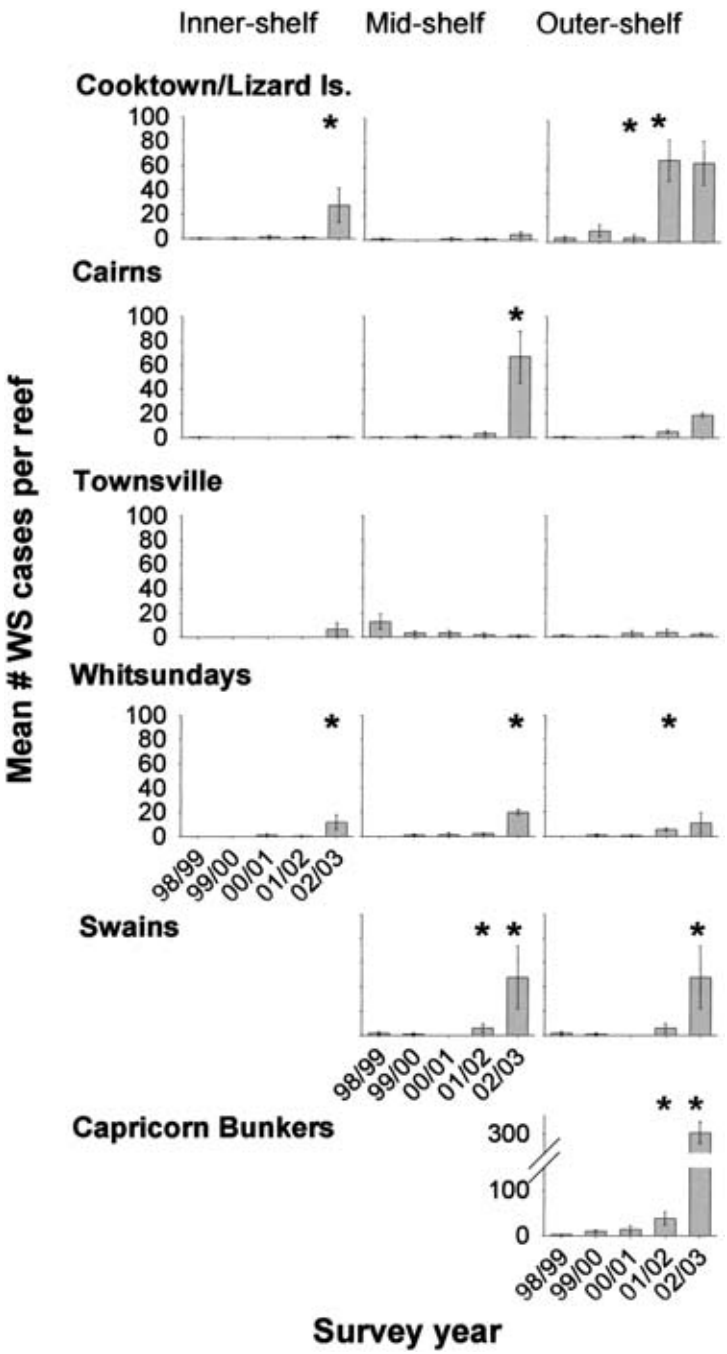


Fig. 3.6. Mean (±SE) of the total number of white syndrome (WS) cases per 1500-m^2 area surveyed on each reef compared among the 15 regions (i.e. combinations of the 6 sectors and 3 cross-shelf locations). Significant change from previous year is denoted by *

in the distribution and abundance patterns for WS. Thus, despite the striking 22- and 150-fold increases in the abundance of WS in outer-shelf reefs in these two sectors over the five years (Fig. 3.6), their magnitude might have been even greater if reefs in these sectors had been surveyed during summer in the last 3 years. In contrast, despite surveying the Cairns and Townsville sectors in the warmer months from 2000/2001 onwards, there was no increase in the abundance of WS. In addition, since WS was erected as a category representing a new source of mortality 6 years after the AIMS LTMP began, it is conceivable that researchers were changing the categorisation of colonies from unknown to WS. However, despite the continuously increasing abundance of WS, records in the unknown category remained relatively constant, suggesting that the rise in WS abundance is not accounted for by a decrease in the unknown category (data not shown).

Distribution of WS

In addition to WS becoming more abundant, infections have increased in distribution over the 5 years. In 1998, WS was distributed across approximately 75% of regions (11 of 15 regions) and 45% of reefs (22 of 48 reefs). However, by 2002/2003, WS had spread to all regions and 89% of reefs. Furthermore, the number of transects with WS increased more than ten-fold, from 3% of transects in 1998/1999 to 39% of transects in 2002/2003 (F_{year}=57.05, *df*=4, *P*<0.001). Patterns of increasing distribution of WS across sectors and regions are similar to those described above for abundance. In particular, we found the same pattern of consistently increasing occurrence of WS through time on transects in all cross-shelf locations (F_{shelf}=12.07, *df*=2, *P*<0.001; $F_{shelf \times year}$=1.83, *df*=8, *P*=0.077), but patterns of occurrence through time differed among sectors ($F_{sector \times year}$=3.57, *df*=20, *P*<0.001) and regions ($F_{sector \times shelf \times year}$=2.47, *df*=28, *P*<0.001). In summary, the number of transects with WS increased with increasing distance of cross-shelf location from the coast (when transects at each shelf location were combined across sectors), from lows of <1–5% of transects in 1998/1999 in all cross-shelf locations to maxima which differed with cross-shelf location in 2002/2003 (i.e. from 17% of transects on inner-shelf reefs to 45% of transects on mid- and 51% of transects on outer-shelf reefs). However, this pattern broke down because there were six regions spread across all shelf locations in which the percent of transects with WS did not increase in at least 1 year.

3.3.2.3
Relationship Between Percent Coral Cover and Abundance of White Syndrome

Given the profound increases of WS on reefs in some sectors of the GBR (up to 150-fold on outer-shelf reefs in the Capricorn Bunker sector, Fig. 3.6), we analysed WS abundance in relation to both percent coral cover and abundance of *Drupella* spp. to (1) provide insights into factors promoting the spread of WS

and (2) evaluate the possible effects of WS on coral abundance. We found a significant relationship between mean percent cover of scleractinian corals and abundance of white syndrome (F_{cover}=14.55, *df*=1, *P*<0.001), which was consistent among shelf locations ($F_{cover \times shelf}$=2.11, *df*=2, *P*=0.127) and regions ($F_{cover \times sector \times shelf}$=1.70, *df*=7, *P*=0.117), and only marginally inconsistent between sectors ($F_{cover \times sector}$=2.38, *df*=5, *P*=0.044). However, single degree of freedom contrasts revealed that, although there were positive trends between percent coral cover and WS abundance in all sectors but Cairns, the relationship was only significant on reefs within the Capricorn Bunkers sector (*P*=0.012). After accounting for the association between percent cover of hard corals and WS abundance, we found no relationship between the abundance of *Drupella* spp. and WS (F=2.45, *df*=1, *P*=0.121). In summary, there is a general trend for abundance of WS to be greatest on reefs with the highest percent hard coral cover that was most pronounced in the Capricorn Bunkers sector.

Mean percent cover of scleractinian corals has fluctuated between about 20–40% in the past decade in all, except four of the regions surveyed (see Sweatman et al. 2001). On outer-shelf reefs in the Cooktown/Lizard Is. and Capricorn Bunker sectors, percent hard coral cover has increased continuously between 1995 and 1998 to approximately 60%, but this pattern altered in 1998, with percent coral cover remaining about the same or declining slightly during the last 5 years (Fig. 3.7). This decline in the rate of change of coral cover coincides roughly with the rising incidence of WS (Fig. 3.7a, b), which reached its highest abundances on outer-shelf reefs in these two sectors (Fig. 3.6). Thus, in the northern Cooktown/Lizard Is. sector, the mean number of WS cases has been highest for the last 2 years on Carter and No Name Reefs, where there is a declining, but non-significant, trend in coral cover (Fig. 3.7a). In the southern Capricorn Bunker Sector, there has been no change in coral cover in the past 5 years since percent cover has stabilised, but WS infections have only risen dramatically in the last year (Fig. 3.7b).

Given that large increases in WS appeared to have occurred concurrently with increases in hard coral cover on some reefs, we examined the relationship between changes in hard coral cover and changes in WS abundance between survey years. Thus we asked: "Does an increase or decrease in hard coral cover correlate with a corresponding increase or decrease in WS?". We found that change in hard coral cover did not always coincide with a similar change in WS abundance ($F_{\Delta cover \times \Delta WS}$=5.50, *df*=1, *P*=0.022); in particular, it was variable across cross-shelf positions ($F_{\Delta cover \times \Delta WS \times shelf}$=3.49, *df*=2, *P*=0.035). Single degree of freedom contrasts indicated that there was a significant association between changes in WS abundance and changes in coral cover only on outer-shelf reefs (*P*=0.025).

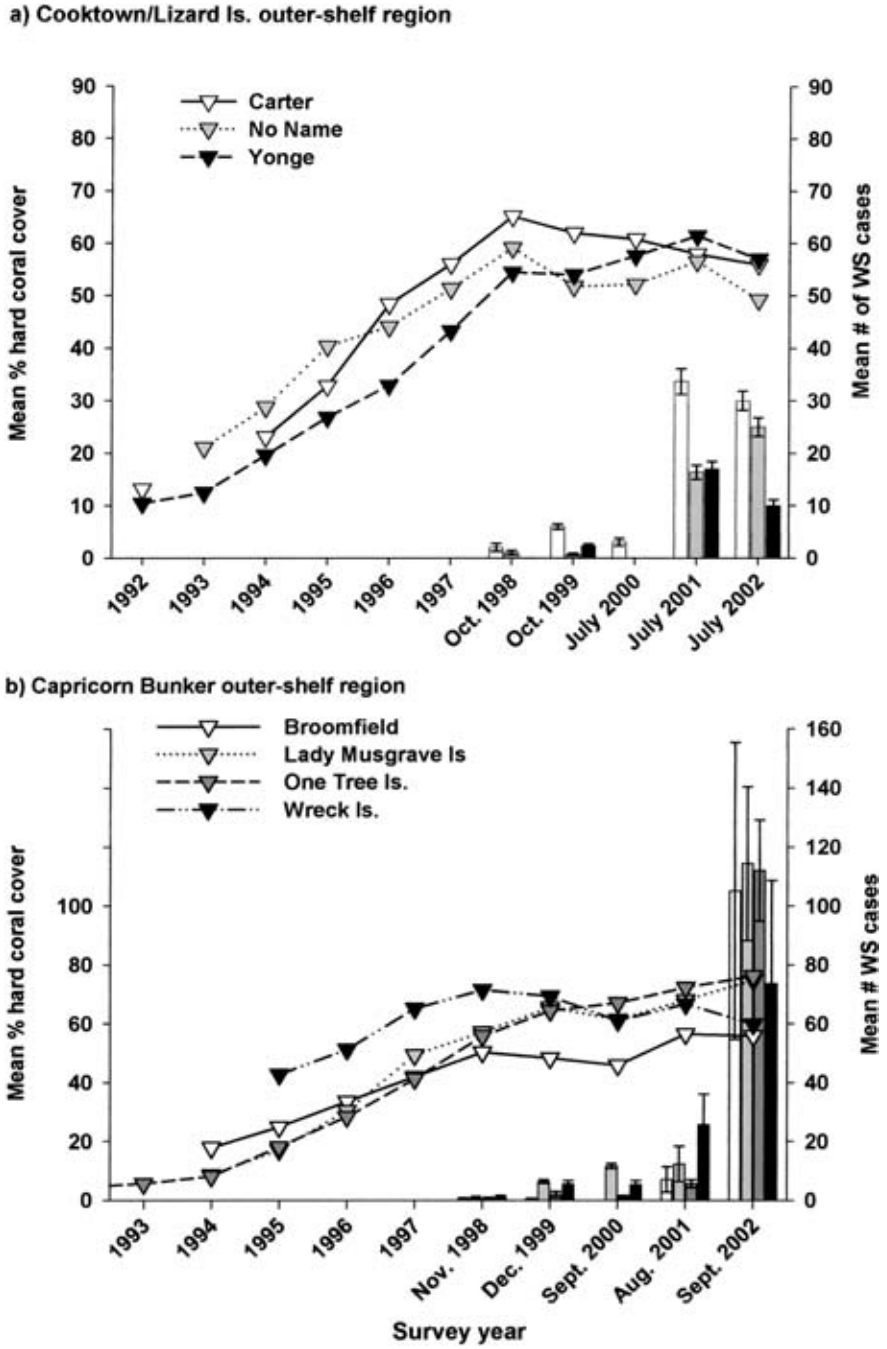


Fig. 3.7. Comparison of the mean percent hard coral cover (n=15 transects per reef) compared to mean (±SE) number of WS cases (n=15 transects per reef) in **a** Cooktown/Lizard Is. outer-shelf region, and **b** Capricorn Bunker outer-shelf region

3.3.3
Results of Regional Disease Prevalence Surveys

3.3.3.1
Comparison of Disease Prevalence Between the Northern and Southern Sectors of the Great Barrier Reef

Overall, symptoms of disease were detected in 8.97±0.79% of colonies (n=27,842) examined in the northern Cooktown/Lizard Is. and southern Capricorn Bunker sectors in summer 2003. Combining records for all scleractinians, alcyonaceans and gorgonians, mean disease prevalence was similar on the northern Lizard Is. (9.4±0.53%) and southern Heron Is. (10.4±2.07%) reefs, but marginally lower on the northern outer-shelf No Name reef (7.2±1.13%; Fig. 3.8). Patterns in overall mean prevalence of disease were

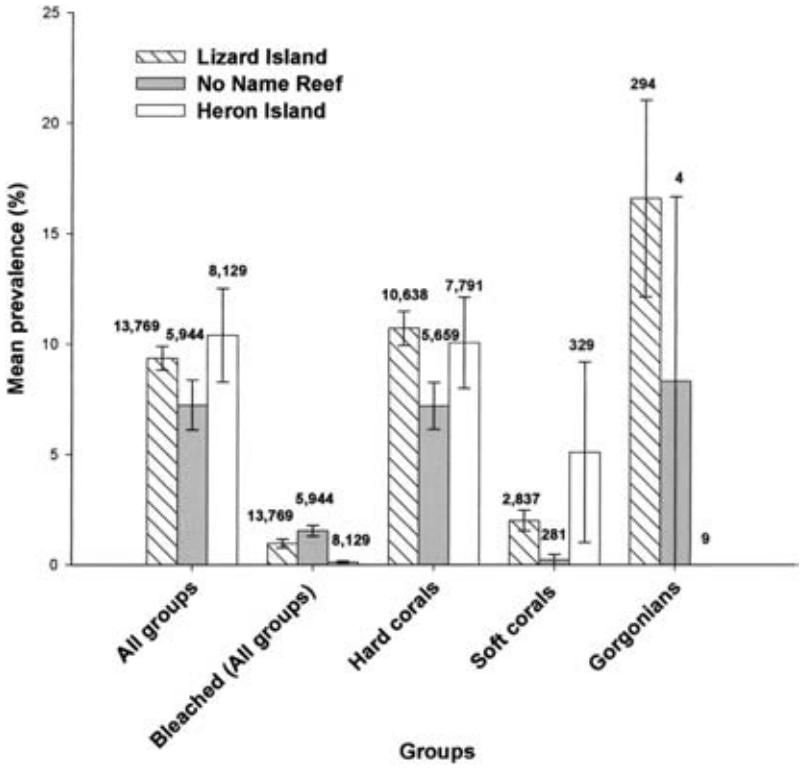


Fig. 3.8. Mean (±SE) prevalence of disease per reef for all taxonomic groups (hard and soft corals and gorgonians) combined compared to mean (±SE) prevalence of disease in hard corals, soft corals, gorgonians and bleached colonies (from all groups) at Lizard Is. (n=4 sites × 3 transects), No Name Reef (n=2 sites × 3 transects), and Heron Is. (n=2 sites × 3 transects). Disease prevalence (per taxonomic group) is calculated relative to the total number of colonies examined in each group, at each reef, as shown above the appropriate histogram

driven by patterns of disease prevalence in hard corals, which dominate cnidarian communities on these reefs (Fig. 3.8). Disease prevalence in hard corals ranged from a minimum of 7.2±1.06% at No Name reef to a maximum of 10.7±0.76% on Lizard Is. reefs, both in the northern sector. Gorgonian assemblages on Lizard Is. reefs were most affected by disease, with a mean of 16.6±4.50% of colonies infected on these reefs (Fig. 3.8). Disease was least prevalent amongst soft coral assemblages. Bleaching affected less than 1.7% of colonies from all three cnidarian groups.

3.3.3.2 Patterns in Prevalence of Disease Categories Among Sectors and Coral Families

The prevalence of four (SEB, BBD, BNS and PS) of the seven major disease categories was greatest at Lizard Is. in the northern sector, whereas WS was most prevalent at Heron Is. in the southern sector (Fig. 3.9). BrB and tumours were

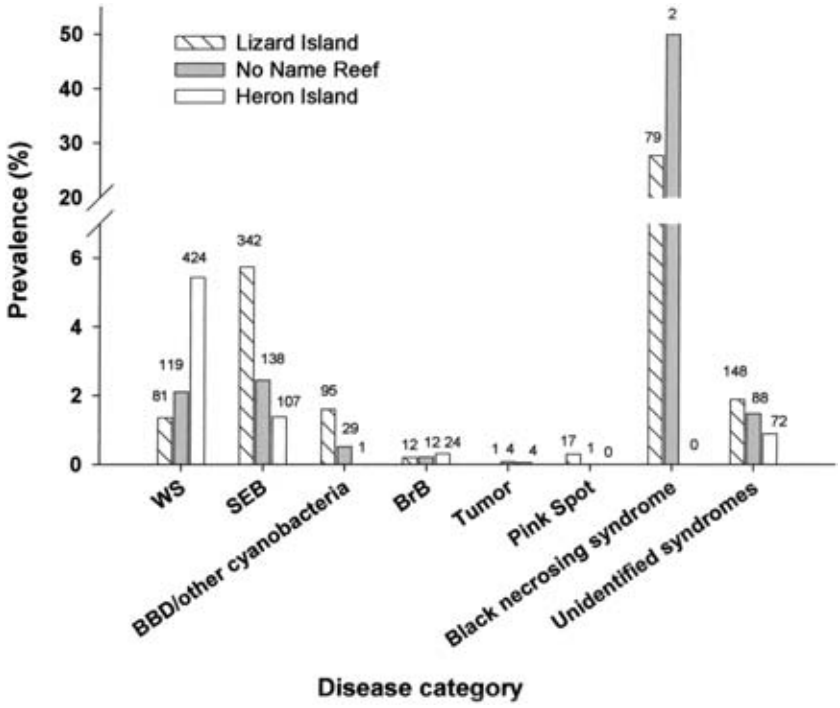


Fig. 3.9. Prevalence of each disease category at Lizard Is., No Name Reef and Heron Island in summer 2003, based on surveys of two sites per reef. Prevalence (per disease category) is calculated relative to the number of hard coral colonies examined at each reef for all disease categories except BNS (calculated relative to total number of gorgonian colonies) and unidentified syndromes (calculated relative to number of scleractinian and alcyonacean colonies combined). Total number of cases of each disease category recorded at each reef is shown above the appropriate histogram

uncommon on all reefs, although twice as many cases of BrB were recorded at Heron Is. in the southern sector compared to the two reefs in the northern sector. Among disease states, black necrosing syndrome (BNS) had the highest prevalence, but the syndrome was restricted to gorgonians, which represent only a minor component of the community. Among hard corals, white syndrome (WS) attained the highest prevalence of any of the disease categories at Heron Is., where 424 cases were recorded (survey area = 240 m^2; Fig. 3.9). Although WS was the most prevalent hard coral disease on Heron Is. reefs, skeletal eroding band (SEB) was the most prevalent on Lizard Is. reefs. Overall, WS and SEB were the two most common diseases on all reefs. Black band disease

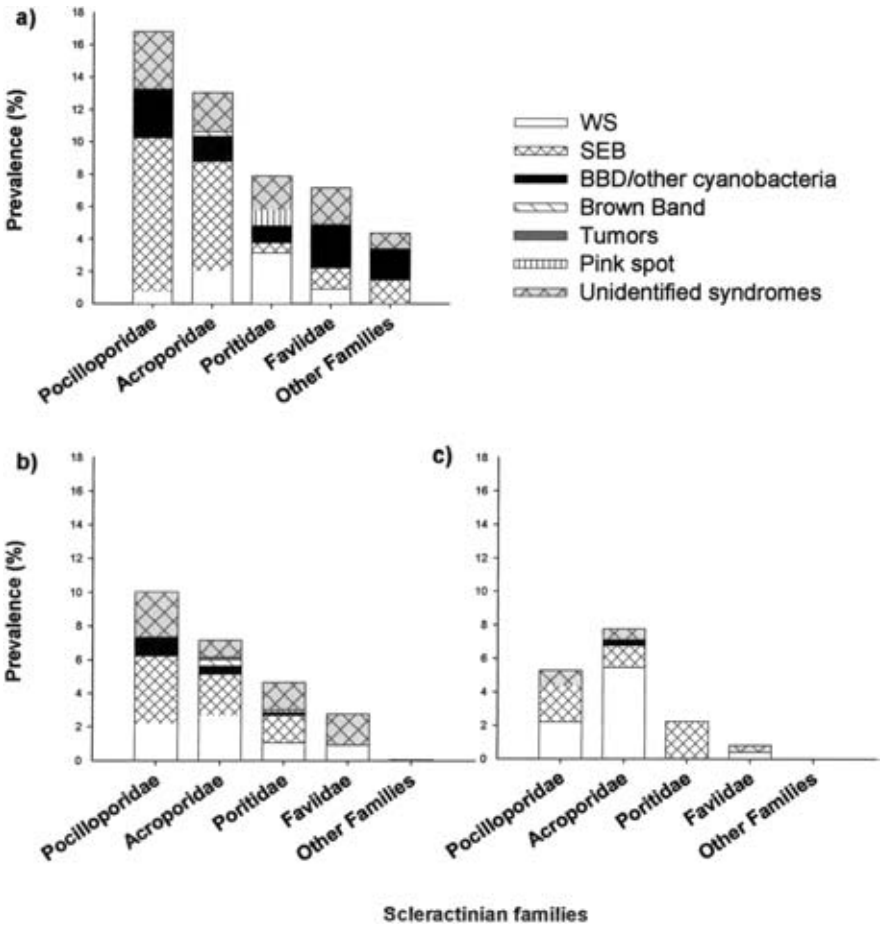


Fig. 3.10. Prevalence of seven disease categories in scleractinian families at **a** Lizard Is. (n=4 sites × 3 transects), **b** No Name reef (n=2 sites × 3 transects) and **c** Heron Is. (n=2 sites × 3 transects) in summer 2003. Prevalence (per family) is calculated relative to the total number of colonies examined in the respective family at each site

(BBD), which was grouped with unidentified cyanobacterial syndromes, tended to be the third most common disease and was present on all three reefs, although it affected only a very low proportion of colonies on Heron Is. reefs. Brown band (BrB) was also present on all three reefs, but generally with lower prevalence than BBD, although the pattern was reversed on Heron Is. reefs. In summary, five of the seven disease categories were present on reefs in all three locations (i.e. WS, SEB, BBD, BrB, and tumours), the exceptions being pigmented spots on *Porites* (PS) and black necrosing syndrome (BNS), which were not recorded from southern sector reefs (Fig. 3.9).

Disease prevalence varied among scleractinian families (χ^2=130.460, *df*=4, *P*<0.001), being greatest in the Pocilloporidae and Acroporidae at all three reefs (Fig. 3.10). When all disease categories were combined, disease prevalence in the northern sector was greatest (16.8%) in the family Pocilloporidae, but in the southern sector, it was greatest (7.8%) in the family Acroporidae. Otherwise, patterns of disease prevalence were consistent at all three reefs, decreasing in the Poritidae and further still in the Faviidae to a minimum prevalence of 0.8% in

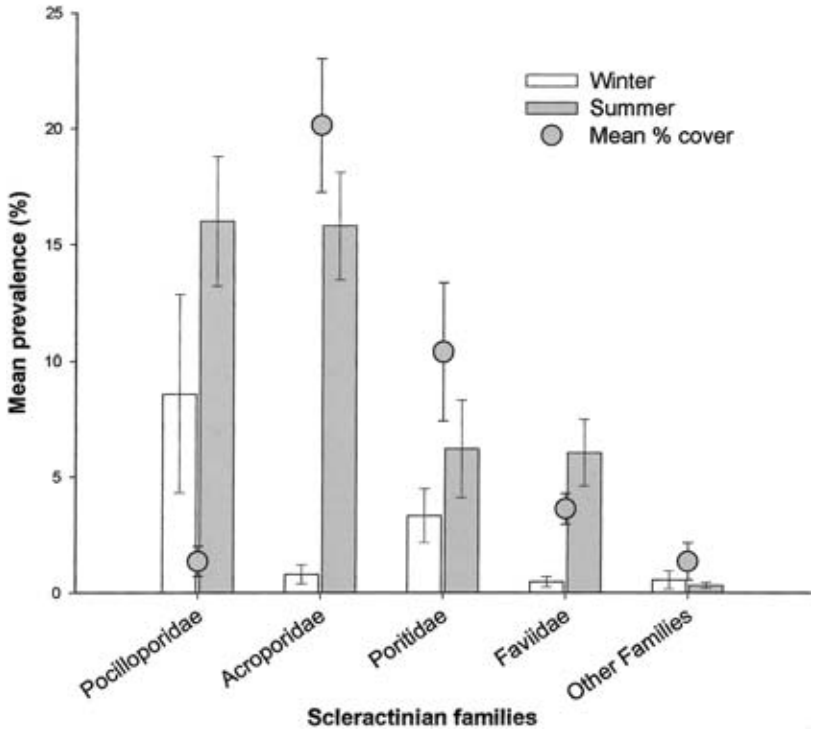


Fig. 3.11. Mean prevalence (±SE) of all diseases in scleractinian families at Lizard Island in winter 2002 vs. summer 2003 (*n*=2 sites × 3 transects) compared to mean (±SE) percent cover of each family in summer 2003 (*n*=2 sites × 3 transects). Prevalence (per family) is calculated relative to the total number of colonies examined in the respective family in each season

faviids on Heron Is. reefs. Interestingly, the high prevalence of disease in the pocilloporids on Lizard Is. reefs was despite the mean percent cover of this family being the lowest of the five family groups on these reefs (Fig. 3.11).

The major families (Pocilloporidae, Acroporidae, Poritidae and Faviidae) were each host to four to five diseases, with WS, SEB and BBD infecting corals in all four families (Fig. 3.10). SEB and WS were generally the most common diseases affecting pocilloporids and acroporids in both sectors, followed by BBD and BrB. The high prevalence of WS on Heron Is. sites (Fig. 3.9) is mostly explained by its high prevalence in acroporids; the proportion of colonies affected by WS being two times greater in acroporids than in pocilloporids at these sites. SEB was the dominant disease affecting pocilloporids and acroporids on Lizard Is. and No Name reefs. BBD showed highest prevalence at Lizard Is., where it disproportionately affected faviid corals. BrB affected a low proportion of corals (≤0.31%) on all three reefs, targeting particularly acroporids, but also pocilloporids and faviids. Poritids were host to WS, SEB, BBD and PS on both Lizard Is. and No Name reefs in the northern sector, but were only affected by SEB on Heron Is. in the southern sector. In the northern sector, pink pigmented spots (PS) were found on 0.97% of poritids on Lizard Is. reefs and 0.17% on No Name Reef, the only syndrome other than tumours (in this study) that was restricted to one family. Tumours were found only on acroporids, and only on a low proportion (≤0.13%) of colonies on each reef.

Within the Acroporidae and Pocilloporidae, WS affected at least 9 and 4 species respectively, and SEB at least 18 and 5 species respectively (Table 3.1). Colonies of other scleractinian families were also observed to be diseased, notably SEB affected fungid and merulinid colonies and BBD and unidentified cyanobacterial syndromes affected pectinid, mussid, dendrophylliid and siderastreid colonies (Table 3.1).

Table 3.1. Species and growth forms or taxonomic groups (if species not identified) of cnidarians displaying symptoms of seven potential disease states during regional prevalence surveys on Lizard Is. (L), No Name (N) and Heron Is. (H) reefs in January 2003. Total number of 1) scleractinian families or alcyonarian orders and 2) minimum number of species affected by each of the potential disease states are shown

		Disease state						
Family or order	Species/ growth form	WS	SEB	BrB	BNS	BBD	Tumour	Unidentified
Acroporidae	*Acropora hyacinthus*	N L	N H	H N L	–	L	–	H L
	A. cytherea	–	H L	L	–	–	–	H
	A. nasuta	–	L	–	–	L	–	–
	A. millepora	–	L	–	–	L	–	L
	A. subulata	–	–	H	–	–	–	–
	A. tenuis	–	–	–	–	–	–	H
	A. latistella	L	–	–	–	L	–	L

Family or order	Species/ growth form	Disease state WS	SEB	BrB	BNS	BBD	Tumour	Unidentified
	A. cerealis	–	–	N	–	–	–	–
	A. valida	–	H	H	–	H	–	H
	A. secale	–	–	N	–	–	–	–
	A. humilis	–	N	–	–	N	–	–
	A. gemmifera	–	N L	–	–	L	–	–
	A. monticulosa	–	L	N	–	–	–	L
	A. elseyi	–	–	–	–	L	–	–
	A. microphthalma	–	H L	–	–	L	–	–
	A. loripes	–	L	–	–	L	–	–
	A. austera	–	H	–	–	–	–	H
	A. yongei	–	L	L	–	L	–	–
	A. muricata	–	H N L	H N L	–	H L	–	H L
	A. grandis	–	L	L	–	–	–	–
	A. florida	–	–	–	–	L	–	–
	A. intermedia	–	H L	H L	–	–	–	H
	A. palifera	–	H	L	–	–	–	H
	A. cuneata	–	–	–	–	N	–	–
	A. cuneata/palifera	–	–	–	–	–	–	N
	A. brueggemanni	–	–	–	–	–	–	L
	Tabular *Acropora*	N L	N L	N L	–	N L	N	N
	Staghorn *Acropora*	H N L	H N L	N L	–	L	–	H L
	Corymbose Acropora	H N L	H N L	H N	–	L	–	H N L
	Digitate *Acropora*	H N L	H N L	N	–	N L	–	N L
	Bottlebrush *Acropora*	H L	H N L	–	–	H L	–	N L
	Bushy *Acropora*	N L	N L	–	–	L	L	L
	Isoporan *Acropora*	H N	H N L	H	–	N L	H	H N L
	Astreopora spp.	L	H L	–	–	L	–	L
	Montipora spp.	L	H NL	H	–	H N L	L	H L
Pocilloporidae	*Pocillopora meandrina/verrucosa*	–	N	N	–	–	–	–
	P. verrucosa	–	L	–	–	L	–	–
	P. eydouxi	–	N L	–	–	L	–	N
	P. damicornis	H N L	H N L	L	–	N L	–	H N L
	Seriatopora hystrix	–	L	–	–	L	–	–
	Seriatopora spp.	H N	H L	–	–	N L	–	L
	Stylophora pistillata	HN	H N L	–	–	N L	–	N L
	Other pocilloporids	N L	H N L	N	–	N L	–	H N L
Poritidae	*Porites* spp.	N L	N L	–	–	N L	–	N L
Fungiidae		–	H	–	–	–	–	–
Pectiniidae		–	–	–	–	L	–	L
Mussidae		–	–	–	–	L	–	N L

Family or order	Species/ growth form	Disease state WS	SEB	BrB	BNS	BBD	Tumour	Unidentified
Merulinidae	*Hydnophora rigida*	–	L	–	–	–	–	L
	H. microconos	–	–	–	–	–	–	L
Faviidae	*Favia, Favites* or *montastrea* spp.	H N L	L	–	–	L	–	H N L
	Favia stelligera	N	–	–	–	–	–	–
	Goniastrea or *Platygyra* spp.	N L	L	–	–	L	–	H N L
	Echinopora sp.	–	L	L	–	L	–	–
	E. horrida	–	L	–	–	–	–	–
	E. mammiformis	L	–	–	–	–	–	–
	E. lamellosa	–	L	–	–	–	–	L
	Other faviids	N L	L	L	–	L	–	N L
Dendrophylliidae	*Turbinaria* sp.	–	–	–	–	L	–	L
Siderastreidae	*Psammocora digitata*	–	–	–	–	L	–	–
O: Alcyonacea	*Sinularia* sp.	–	–	–	–	H	–	–
	Lobophytum sp.	–	–	–	–	L	–	–
	Other alcyonaceans	–	–	–	–	L	–	N H L
O: Gorgonacea	*Isis* sp.	–	–	–	N L	–	–	L
O:Hydrocorallina	*Millepora* sp.	–	–	–	–	L	–	L
	Number of families/ orders affected	4	6	3	1	10	1	11
	Minimum number of species affected	17	31	16	1	32	4	30

3.3.3.3
Seasonal and Habitat (Wave Exposure) Patterns in Disease Prevalence

Disease prevalence was higher in summer than in winter on sheltered Lizard Is. reefs ($F=78.13$, $df=1$, $P<0.001$; Fig. 3.11). In particular, mean disease prevalence in summer (January 2003) was more than 15-fold greater in acroporids, more than 12-fold greater in faviids and approximately doubled in pocilloporids compared to the preceding austral winter (July 2002). Seasonal patterns of increased disease prevalence in summer in most coral families corresponded to striking increases in the number of cases of disease in all categories except tumours and pigmented spots on *Porites* (Fig. 3.12). In particular, disease incidence was high for WS (increased from 0 to 81 cases), SEB (~20-fold increase to 342 cases), BBD and unidentified cyanobacterial syndromes (~8-fold increase to 95 cases), but moderate for BrB (increase from 1 to 12 cases) (Fig. 3.12). In addition, the number of cases of BNS on gorgonians and unidentified syndromes increased in summer (the latter 14-fold).

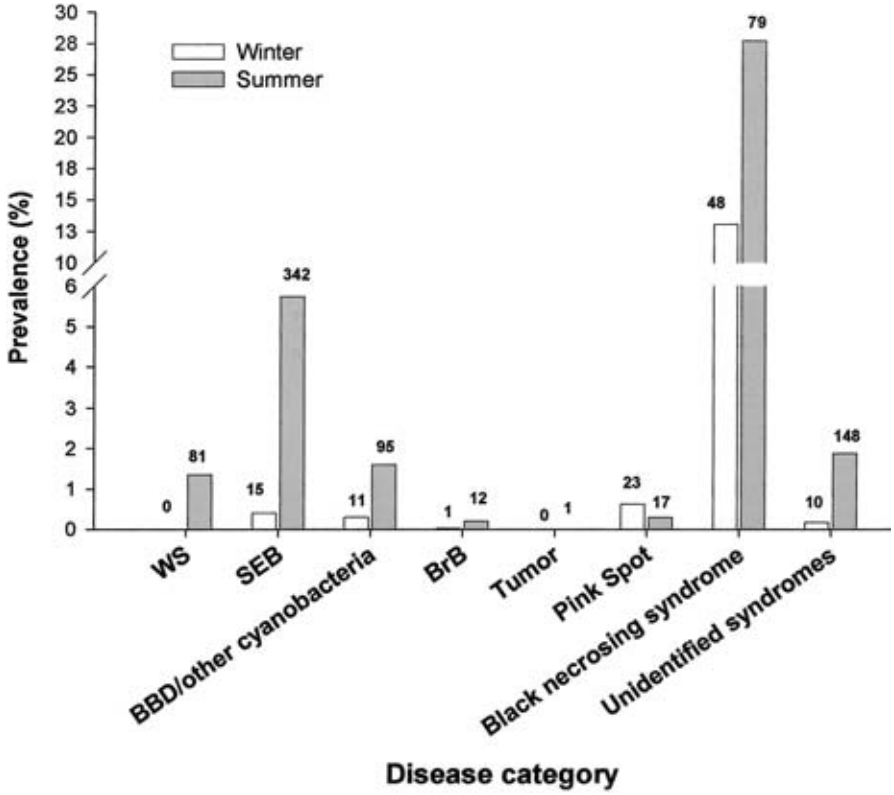


Fig. 3.12. Prevalence of seven disease categories of hard corals at Lizard Island in winter 2002 compared to summer 2003, based on surveys of two sites. Prevalence (per disease category) calculated as in Fig. 3.9. Total number of cases of each disease category is shown above the appropriate histogram

Among hard corals, disease prevalence was greater on sheltered than exposed sites at Lizard Is. ($F=298.23$, $df=1$, $P=0.003$). This pattern was largely the result of the more than two-fold greater disease prevalence on acroporids at sheltered compared to exposed sites ($F=31.22$, $df=1$, $P=0.031$). Disease prevalence did not differ significantly between the two exposure habitats for the three other major families (Pocilloporidae, Poritidae, Faviidae).

3.3.3.4
Relationship Between Percent Coral Cover and Disease Prevalence

There was no correlation between mean disease prevalence and mean percent cover of all groups (scleractinian, alcyonaceans and gorgonians combined) at the eight sites surveyed (Fig. 3.13). There was more than a two-fold range in both mean disease prevalence (varying between 5.2±0.98% on the reef front at No Name Reef in the northern sector to 12.8±3.02% at Little Bay, Heron Is. in

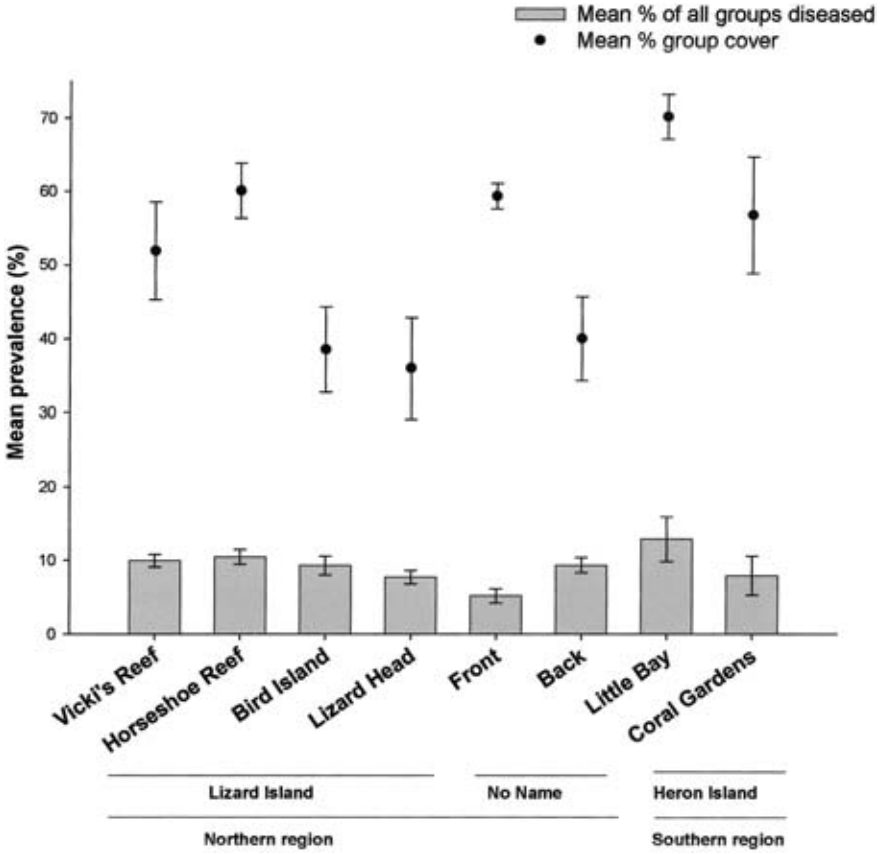


Fig. 3.13. Geographic patterns in mean disease prevalence (±SE) in all groups (hard corals, soft corals and gorgonians combined) compared to mean percent cover (±SE) of all groups at each reef in summer (January) 2003 (n=3 transects per site). Prevalence (per site) is calculated relative to the total number of scleractinian, gorgonian and alcyonacean colonies examined at each site

the southern sector) and mean percent cover (varying between 36.0±6.84% at Lizard Head to 70.2±3.05% at Little Bay) over the eight sites. However, despite mean disease prevalence being highest where mean percent cover was highest, it was also lowest where mean percent cover was second highest.

3.3.4 Discussion and Conclusions

Our review of disease records to date reveals the presence of at least eight disease states on the Great Barrier Reef (GBR) and at least another four elsewhere in the Indo-Pacific. Although this approximates to only half of the 22 diseases so far recorded from the Caribbean, the global coral disease hot spot (Green and Bruckner 2000; Weil, this Vol.), most of these records are from the last

10 years and as a result of comparatively minimal research effort. Given that the first record of coral disease in the Caribbean was 30 years ago (Antonius 1973) and that observations have been escalating ever since (Harvell et al. 1999), the rate of discovery of new syndromes and diseases may accelerate in the Indo-Pacific as research becomes more focused and reefs come under increasing pressure from a plethora of environmental issues. In particular, with more than 80% of reefs in SE Asia under medium to high threat from activities like over-exploitation of resources and coastal developments (Bryant et al. 1998) and with predicted increased sea temperatures associated with global climate change (IPCC 2002) likely to augment pathogen virulence (Harvell et al. 2002; Rosenberg and Ben-Haim 2002), environmental conditions in the next few decades are poised to foster increasing incidence and spread of disease on Indo-Pacific reefs. The presence of a number of pathogens on the Great Barrier Reef that have had major impacts on the structure of Caribbean coral communities, such as black band disease and potentially one or more of the white band or plague diseases within the white syndrome category (Gladfelter 1982; Bruckner and Bruckner 1997; Richardson 1998; Richardson and Aronson 2002), emphasises the gravity of the threat posed by predicted environmental changes for coral reefs in this region.

One of the greatest causes for concern is the 22- to 150-fold increases in the abundance of white syndrome on outer-shelf reefs in the northern and southern sectors of the GBR that have been detected over the last 5 years by the Australian Institute of Marine Science Long Term Monitoring Program (AIMS LTMP). The occurrence of these striking increases on reefs separated by 1200 km indicates that conditions promoting the spread of the syndrome are widespread and extend from the northern to southern sectors of the GBR. The lack of association between the abundance of *Drupella* spp. and the abundance of WS suggests that GBR *Drupella* species are not major vectors in the transmission of WS, in contrast to previous positive correlations between *D. cornus* and abundance of white diseases found in the Red Sea (Antonius and Riegl 1997, 1998). Curiously, patterns of increasing abundance of WS are correlated with increasing distance of reefs from the coast and are associated with high mean percent cover of hard corals. Furthermore, the significant and positive relationship between changes in percent coral cover and changes in WS abundance on outer-shelf reefs, where the greatest increases in WS abundance have been recorded, further implicates increases in coral cover as playing a role in the spread of WS. The pattern of greatest occurrence of WS on outer-shelf reefs, where anthropogenic impacts are least, indicates that WS abundance is unlikely to be directly caused by human activities or terrestrial sources of pollution. The increasing abundance of WS with increasing percent coral cover could reflect either increased pathogen transmission or host vulnerability as coral assemblages become more crowded and approach carrying capacity, or it could reflect increased pathogen susceptibility as colonies age.

In the absence of other identifiable disturbances, the association between rising WS abundance and declining rates of increase or negative trends in percent

coral cover on some reefs, suggests high WS abundance may be contributing to the lower than predicted (from rates of change prior to the rise of WS) percent covers attained on these reefs. Given that we found the prevalence of WS to be higher on two of the three reefs surveyed in the regional disease prevalence surveys (5.44% on Heron Is. Reef and 2.11% on No Name Reef) than prevalences recorded for most of the white band (WB) and white plague (WP) diseases that have been so destructive in the Caribbean [range: 0.01–1.85%, except for 3.62% prevalence of WPI in Florida (Dustan 1977); reviewed in Weil, this Vol.], establishing the causative agent(s) of white syndrome on the GBR must be considered an urgent priority.

The regional disease prevalence surveys revealed equally disturbing patterns in disease occurrence on the GBR. The overall mean disease prevalence of 8.97 ±0.79% at eight sites in the northern and southern sectors of the GBR in summer 2003 is higher than the 5.38±1.2% mean disease prevalence that has been recorded in comparable surveys of 28 Caribbean sites in the past 4 years (Weil, this Vol.). Although we surveyed sites in regions identified as having the highest abundance of WS in the large-scale surveys, nevertheless the Caribbean surveys included some of the most impacted sites within the biogeographic reef region, for example Jamaica, which had a mean disease prevalence of 16.21±1.55% in 1999, and Mexico, which had mean prevalence of 10.91±1.57% in 2002 (Weil, this Vol.). The lowest mean prevalence on GBR sites (5.16±0.98% at the reef front site at No Name Reef, an outer-shelf reef in the northern sector) was similar to the overall mean prevalence of the Caribbean sites, although the highest GBR disease prevalence (12.89±3.02% at the Little Bay site on Heron Is. in the southern sector) was somewhat less than the highest Caribbean prevalence (16.21±1.55% in Jamaica; Weil, this Vol.). The lower disease prevalence at No Name than at Lizard Is. sites is contrary to the patterns of WS abundance recorded on the same reefs in the large-scale AIMS LTMP surveys, but may reflect the shallower depth of transects and, hence, the higher wave energy habitats that were surveyed in the disease prevalence study. The lower disease prevalence found on the shallower transects accords with the pattern of lower disease prevalence on exposed sites found for hard corals on Lizard Is. reefs, particularly for the family Acroporidae. Overall, the increasing abundance of WS recorded by the AIMS LTMP surveys in all GBR sectors but one (Townsville sector) and in all cross-shelf locations (inner-, mid- and outer-shelf) in the past 5 years highlights the need for a co-ordinated, large-scale program to establish baseline levels of disease prevalence at key sites throughout the GBR, against which to judge whether disease incidence is increasing.

Until more is known about the etiology of GBR and Indo-Pacific coral diseases, it is difficult to compare prevalence of specific diseases reported here with those in the Caribbean. Black band disease (BBD) is the only disease that is common to the two reef regions, although it appears that there are further cyanobacterial species associated with BBD-type infections in both the GBR and the Caribbean (see Sect. 3.2.1). Assuming that BBD records in both regions may encompass a variety of cyanobacterial agents and are thus comparable,

the BBD prevalence we found on Lizard Is. reefs (1.7%) is similar to BBD prevalence on Caribbean reefs (0.2–6.0%; reviewed in Weil, this Vol.), however, BBD prevalence was lower on the two outer-shelf GBR reefs (0.01% at Heron Is., 0.51% at No Name Reef) than was generally found on Caribbean reefs. The very low and stable abundance of BBD throughout the past 5 years in the AIMS large-scale survey program, in combination with the higher prevalence found a decade ago at the Lizard Is. study sites (Dinsdale 2002), suggests that BBD is a common component of pathogenic assemblages on GBR corals but, as in the Caribbean, it rarely reaches outbreak proportions.

Prevalences of other diseases on the GBR were generally low (<1%), with the exception of skeletal eroding band (SEB) on hard corals and black necrosing syndrome (BNS) on gorgonians. The occurrence of SEB at all sites in the southern and northern sectors (1.4–5.7% prevalence) and the range of hosts (at least 32 species in 6 scleractinian families) suggest that it is a widespread, generalist pathogen. Although direct comparisons of the prevalence of SEB found in our surveys at Lizard Is. (5.7%) with previous records of SEB at the same reef in 1998 (season unknown) are not possible given the semi-quantitative nature of the latter surveys (Antonius and Lipscomb 2001), it is likely that the 344 SEB cases in 240 m^2 represents, if anything, an increase in abundance over the 13–25 cases/30-min swim recorded in the previous study. However, the nearly eight-fold increase in prevalence of SEB that we found between winter and summer on Lizard Is. reefs suggests that comparisons are only valid if reefs are surveyed in the same season. Prevalence of BNS also increased in summer on Lizard Is. reefs, infecting more than 25% of gorgonian populations compared to 13% in winter. The year-round high prevalence of BNS suggests that it may have a major impact on gorgonian populations on Lizard Is. reefs.

Patterns in disease prevalence among families suggest the faster growing corals in the families Acroporidae and Pocilloporidae are disproportionately targeted by pathogenic microorganisms, including cyanobacteria and protozoans. Although acroporids dominated coral benthic cover at Lizard Is. sites (and at all other sites), the pattern of very low percent cover of pocilloporids despite high disease prevalence in both winter and summer, indicates that pathogens are not necessarily keying into the most abundant or spatially dominant corals. In contrast, disease prevalence in the two slow-growing massive families, Poritidae and Faviidae, was less than half that of pocilloporids, despite their 2.5–7.5 times greater percent cover. It is possible that corals with fast growth have less well-developed disease resistance strategies as a consequence of life histories that channel resources into growth for space monopolisation rather than into maintenance activities, whereas massive corals that tend to be more committed to confrontational strategies (Jackson 1979), may have evolved greater disease resistance. The tendency for WBD epizootics in the Caribbean to disproportionately affect acroporids (e.g. Gladfelter 1982) supports the hypothesis that faster growing corals may have decreased disease resistance. However, more extensive testing of patterns in host susceptibility among coral families is required before life history patterns in disease resistance can be identified.

3.3.5 Some Unresolved Questions and Future Research

There are a myriad of unresolved questions that should be tackled with some urgency to begin to address questions concerning the impact of coral disease on the Great Barrier Reef and the wider Indo-Pacific. Foremost, surveys of disease prevalence on reefs representative of the major habitats and community types throughout the region are required to better document the full range of pathogens and establish a baseline against which to judge whether disease incidence is increasing. Although rapid surveys of the number of cases of disease identify general trends, more detailed disease prevalence surveys are preferred to accurately estimate the impact of disease on coral populations and assemblages. A key objective will be to determine rates of mortality caused by disease and to put them into context with mortality caused by other disturbance agents on Indo-Pacific reefs, such as bleaching events, cyclones and *Acanthaster planci* outbreaks.

The modular nature of corals raises another set of issues regarding the impact of disease on coral populations that should be addressed concurrently. Whereas disease impacts the whole animal in unitary organisms, modular organisms may suffer partial mortality, which compromises colony fecundity, but does not reduce population size. Thus, in addition to establishing disease prevalence and rates of mortality attributable to disease within populations, it will be equally important to determine rates of disease spread and tissue loss within colonies and their associated impacts on colony fecundity and growth, to fully understand the impact of diseases on coral populations. The limited nature of current knowledge of the etiology of Indo-Pacific coral diseases is a major impediment to determining reservoirs and vectors involved in disease transmission, both of which are keys to the management of potential epizootics. Therefore, another critical focus for future research is molecular and microbiological studies to characterise and identify pathogens associated with currently uncharacterised disease states, particularly white syndrome. There is some urgency to initiate research in each of these areas given that impacts of global climate change are likely to include decreased resistance and increased susceptibility of coral hosts, potentially in combination with increased virulence of pathogens. In conclusion, without a concerted effort to characterise the impacts of coral disease on GBR coral communities as well as the pathogens associated with coral diseases, including their patterns of spread, origins, reservoirs and vectors, our ability to develop effective strategies to manage disease on the Great Barrier Reef is limited.

Acknowledgements. We would like to thank past and present members of the AIMS LTMP for their assistance with data collection in the large-scale surveys. We are particularly grateful to Hugh Sweatman for management of the AIMS LTMP and for permission to publish these data, Greg Coleman for assistance with data management, Steve Delean for assistance with statistical analysis, Lyle Vail and Anne Hoggett for logistical support at Lizard Is. Research Station, and Heron Is. Research Station staff for logistical support at Heron Is. We thank E. Rosenberg and Y. Loya for their invitation to write this chapter and for their comments, and also S. Connolly, S. Neale, A. Thompson and R. Willis for comments that improved it. This research was supported by the Australian Institute of Marine Science and by grants from the World Bank and James Cook University. This is publication number 210 from the Coral Group at James Cook University.

References

Aeby GS (1991) Behavioural and ecological relationship of a parasite and its hosts within a coral reef system. Pac Sci 45:263–269

Aeby GS (1998) A digenean metacercaria from the reef coral, *Porites compressa*, experimentally identified as *Podocotyloides stenometra*. J Parasitol 84:1259–1261

Antonius A (1973) New observations on coral destruction in reefs. Abs Ass Isl Mar Lab Carib 10:3

Antonius A (1977) Coral mortality in reefs: a problem for science and management. Proc 3rd Int Coral Reef Symp 2:3–6

Antonius A (1985) Coral disease in the Indo-Pacific: a first recording. PSZNI Mar Ecol 6:197–218

Antonius A (1999) *Halofolliculina corallasia*, a new coral-killing cilicate on Indo-Pacific reefs. Coral Reefs 18:300

Antonius A, Riegl B (1997) A possible link between coral diseases and a corallivorous snail (*Drupella cornus*) outbreak in the Red Sea. Atoll Res Bull 47:1–9

Antonius A, Riegl B (1998) Coral diseases and *Drupella cornus* invasion in the Red Sea. Coral Reefs 17:48

Antonius A, Afonso-Carillo J (2001) *Pneophyllum conicum* killing reef-corals in Mauritius: a new Indo-Pacific syndrome? Bull Mar Sci 69:613–618

Antonius A, Lipscomb D (2001) First protozoan coral-killer identified in the Indo-Pacific. Atoll Res Bull 481–493:1–21

Aronson RB, Precht WF (2001) White-band disease and the changing face of Caribbean reefs. In: Porter JW (ed) The ecology and etiology of newly emerging marine diseases. Kluwer, London, pp 25–38

Aronson RB, Precht WF, Macintyre I (1998) Extrinsic control of species replacement on a Holocene reef in Belize: the role of coral disease. Coral Reefs 17:223–230

Baird A (2000) Microbial menace. CRC Reef Research note, 2 pp

Bak R (1983) Neoplasia, regeneration and growth in the reef-building coral *Acropora palmata*. Mar Biol 77:221–227

Bass DK, Miller IR (1996) Crown-of-thorns starfish and coral surveys using manta tow and SCUBA search techniques. Long-term monitoring of the GBR, Standard Operational Procedure no 1. Aust Inst Mar Sci, Townsville, 42 pp

Borneman EH (2001) Aquarium corals: selection, husbandry, and natural history. TFH Publishing, Neptune City, NJ, USA

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 A, Bruckner J, Wiliams E Jr (1997) Spread of a black-band disease epizootic though the coral reef system in St Ann's Bay, Jamaica. Bull Mar Sci 61:919–928

Bryant D, Burke L, McManus J, Spalding M (1998) Reefs at risk: a map-based indicator of threats to the World's coral reefs. World Resource Institute, Washington, DC, 56 pp

Cheney D (1975) Hard tissue tumors of scleractinian corals. In: Hildemann W, Benedict A (eds) Immunologic phylogeny. Plenum Press, New York, pp 77–87

Coles S, Seapy D (1998) Ultra-violet absorbing compounds and tumorous growths on acroporid corals from Bandar Khayran, Gulf of Oman, Indian Ocean. Coral Reefs 17:195–198
Cooney RP, Pantos O, Tissier MDAL, Barer MR, O'Donnell AG, Bythell JC (2002) Characterization of the bacterial consortium associated with black band disease in coral using molecular microbiological techniques. Environ Microbiol 4:401–413
Dinsdale EA (1994) Coral disease on the Great Barrier Reef. Joint scientific conference on science, management and sustainability of marine habitats in the 21st century. Abstract
Dinsdale EA (2002) Abundance of black-band disease on corals from one location on the Great Barrier Reef: a comparison with abundance in the Caribbean region. Proc 9th Int Coral Reef Symp 2:1239–1243
Ducklow HW, Mitchell R (1979) Observations on naturally and artificially diseased tropical corals: a scanning electron microscope study. Microbial Ecol 5:215–223
Dustan P (1977) Vitality of reef coral populations of Key Largo, Florida: recruitment and mortality. Environ Geol 2:51–58
Fengold JS (1988) Ecological studies of a cyanobacterial infection on the Caribbean sea plum *Pseudopterogorgia acerosa* (Coelenterata: Octocorallia). Proc 6th Int Coral Reef Symp 3:157–162
Frias-Lopez J, Zerkle AL, Bonheyo GT, Fouke BW (2002) Partitioning of bacterial communities between seawater and healthy, black-band diseased, and dead coral surfaces. Appl Environ Microbiol 68:2214–2228
Frias-Lopez J, Bonheyo GT, Jin Q, Fouke BW (2003) Cyanobacteria associated with coral black band disease in Caribbean and Indo-Pacific reefs. Appl Environ Microbiol 69:2409–2413
Gladfelter W (1982) White-band disease in *Acropora palmata*, implications for structure and growth of shallow reefs. Bull Mar Sci 32:639–643
Green E, Bruckner A (2000) The significance of coral disease epizootiology for coral reef conservation. Biol Conser 96:347–361
Harvell CD, Kim K, Burkholder J, Colwell RR, Epstein PR, Grimes J, Hofmann EE, Lipp EK, Osterhaus ADME, Overstreet R, Porter JW, Smith GW, Vasta GR (1999) Emerging marine diseases – climate links and anthropogenic factors. Science 285:1505–1510
Harvell CD, Mitchell CE, Ward JR, Altizer S, Dobson AP, Ostfeld RS, Samuel MD (2002) Climate warming and disease risk for terrestrial and marine biota. Science 296:2158–2162
Hayes R, Goreau T (1998) The significance of emerging diseases in the tropical reef ecosystem. Rev Biol Trop 46 [Suppl 5]:173–185
IPCC (2002) Climate change and biodiversity. In: Gitay H, Suarez A, Watson R, Dokden D (eds) Intergovernmental panel on climate change. Technical Paper V, 86 pp
Jackson JBC (1979) Morphological strategies of sessile animals. In: Larwood G, Rosen BR (eds) Biology and systematics of colonial organisms. Academic Press, New York, pp 499–555
Kim K, Harvell CD, Kim PD, Smith GW, Merkel SM (2000a) Fungal disease resistance of Caribbean sea fan corals. Mar Biol 136:259–267
Kim K, Kim PD, Alker AP, Harvell CD (2000b) Chemical resistance of gorgonian corals against fungal infections. Mar Biol 137:393–401
Korrubel J, Riegl B (1998) A new coral disease from the southern Arabian Gulf. Coral Reefs 17:22
Kuta KG, Richardson LL (1996) Abundance and distribution of black band disease on coral reefs in the northern Florida Keys. Coral Reefs 15:219–223
Kuta KG, Richardson LL (2002) Ecological aspects of black band disease of corals: relationships between disease incidence and environmental factors. Coral Reefs 21:393–398
Le Champion-Alsumard T, Golubic T, Priess K (1995) Fungi in corals: symbiosis or disease? Interactions between polyps and fungi causes pearl-like skeleton biomineralization. Mar Ecol Progr Ser 117:137–147
Littler M, Littler D (1995) Impact of CLOD pathogen on Pacific coral reefs. Science 267:1356–1360
Loya Y, Bull G, Pichon M (1984) Tumor formations in scleractinian corals. Helgol Meer 37:99–112
McCook LJ, Jompa J, Diaz-Pulido G (2001) Competition between corals and algae on coral reefs: a review of evidence and mechanisms. Coral Reefs 19:400–417

Miller I (1996) Black-band disease on the Great Barrier Reef. Coral Reefs 15:58
Morrison-Gardiner S (2001) Studies on the morphology and ecology of fungi associated with the Australian marine environment. PhD Thesis, James Cook University, Townsville, 246 pp
Nagelkerken I, Buchan K, Smith G, Boniar K, Bush P, Garzon-Ferreira J, Botero L, Gayle P, Harvell CD, Heberer C, Kim K, Petrovic C, Pors L, Yoshioka P (1997a) Widespread disease in Caribbean Sea Fans. II. Patterns of infection and tissue loss. Mar Ecol Progr Ser 160:255–263
Nagelkerken I, Buchan K, Smith G, Boniar K, Bush P, Garzon-Ferreira J, Botero L, Gayle P, Heberer C, Petrovic C, Pors L, Yoshioka P (1997b) Widespread disease in Caribbean Sea fans. I. Spreading and general characteristics. Proc 8th Int Coral Reef Symp 1:679–682
Page C, Coleman G, Ninio R, Osborne K (2001) Surveys of sessile benthic communities using underwater video. Standard operational procedure no 7. Aust Inst Mar Sci, Townsville, 49 pp
Patterson K, Porter JW, Ritchie KB, Polson SW, Mueller E, Peters E, Santavy D, Smith GW (2002) Etiology of white pox, a lethal disease of the Caribbean elkhorn coral, *Acropora palmata*. Proc Natl Acad Sci USA 99:8725–8730
Peters E, Halas J, McCarty H (1986) Calicoblastic neoplasms in *Acropora palmata* with a review of reports on anomalies of growth and form in corals. J Natl Cancer Inst 76:895–912
Ravindran J, Raghukumar C (2002) Pink line syndrome (PLS) in the scleractinian coral *Porites lutea*. Coral Reefs 21:252
Raymundo L, Harvell CD, Reynolds T (2003) Porites ulcerative white spot disease: description, prevalence, and host range: a new disease impacting Indo-pacific reefs. Dis Aquat Org 56(2):95–104
Richardson LL (1996) Horizontal and vertical migration patterns of *Phormidium corallyticum* and *Beggiatoa* spp. associated with black-band disease. Microbial Ecol 32:323–335
Richardson LL (1998) Coral diseases: what is really known. Trends Evol Ecol 13:438–443
Richardson LL, Aronson RB (2002) Infectious diseases of reef corals. Proc 9th Int Coral Reef Symp 2:1225–1230
Rodriguez-Martinez RE, Banaszak A, Jordan-Dahlgren E (2001) Necrotic patches affect *Acropora palmata* (Scleractinia: Acroporidae) in the Mexican Caribbean. Dis Aquat Org 47:229–234
Rosenberg E, Ben-Haim Y (2002) Microbial diseases of corals and global warming. Environ Microbiol 4:318–326
Santavy D, Peters E (1997) Microbial pests: coral disease in the Western Atlantic. Proc 8th Int Coral Reef Symp 1:607–612
Sweatman H, Cheal A, Coleman G, Delean S, Fitzpatrick B, Miller I, Ninio R, Osborne K, Page C, Thompson A (2001) Long–term monitoring of the Great Barrier Reef. Status Report no 5, 106 pp
Weil E, Urreiztieta I, Garzón-Ferreira J (2002) Geographic variability in the incidence of coral and octocoral disease in the wider Caribbean. Proc 9th Int Coral Reef Symp 2:1231–1238
Yamashiro H, Oku H, Onaga K, Iwasaki H, Takara K (2001) Coral tumors store reduced levels of lipids. J Exp Mar Biol Ecol 265:171–179