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-

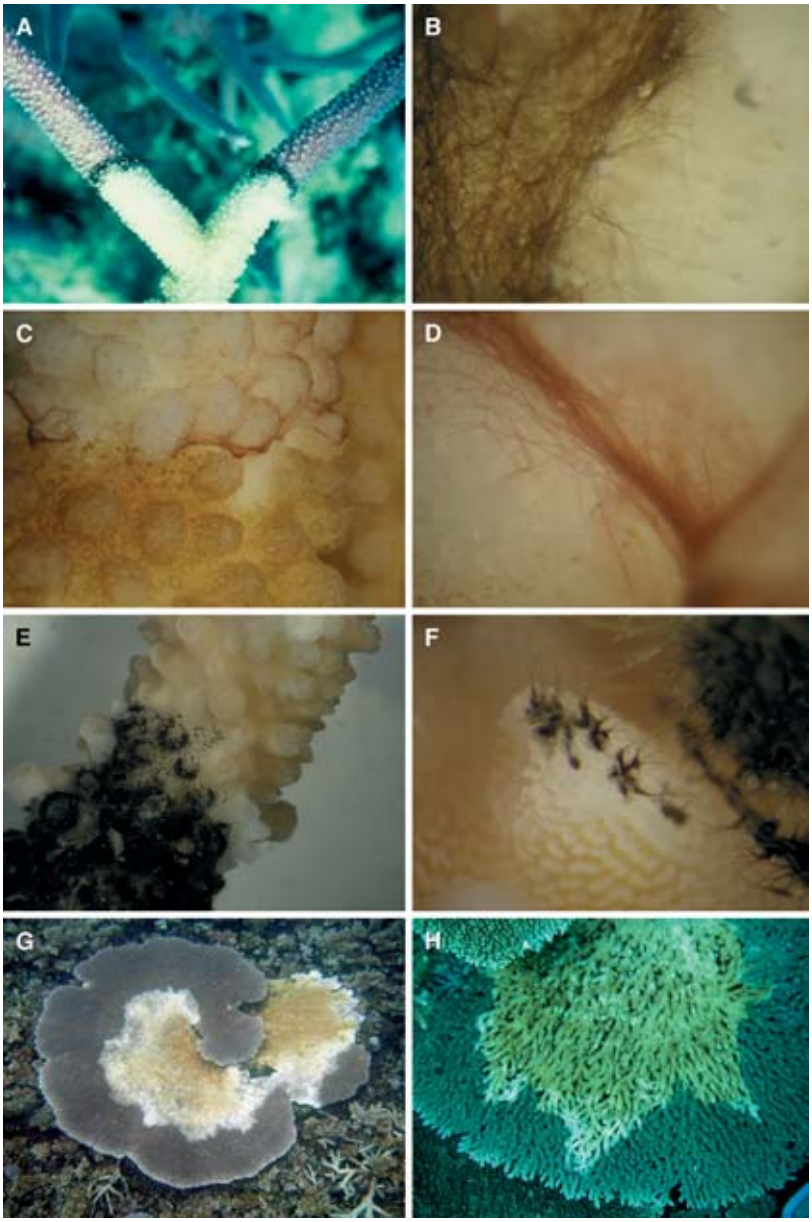

Fig. 3.1A–H. Field appearance of diseases and syndromes infecting corals and gorgonians on the Great Barrier Reef: **a** black band disease (BBD) on *Acropora intermedia,* **b** cyanobacterial mat, responsible for the black colouration of BBD, **c** unknown cyanobacteria forming a mat at tissue-skeleton 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)

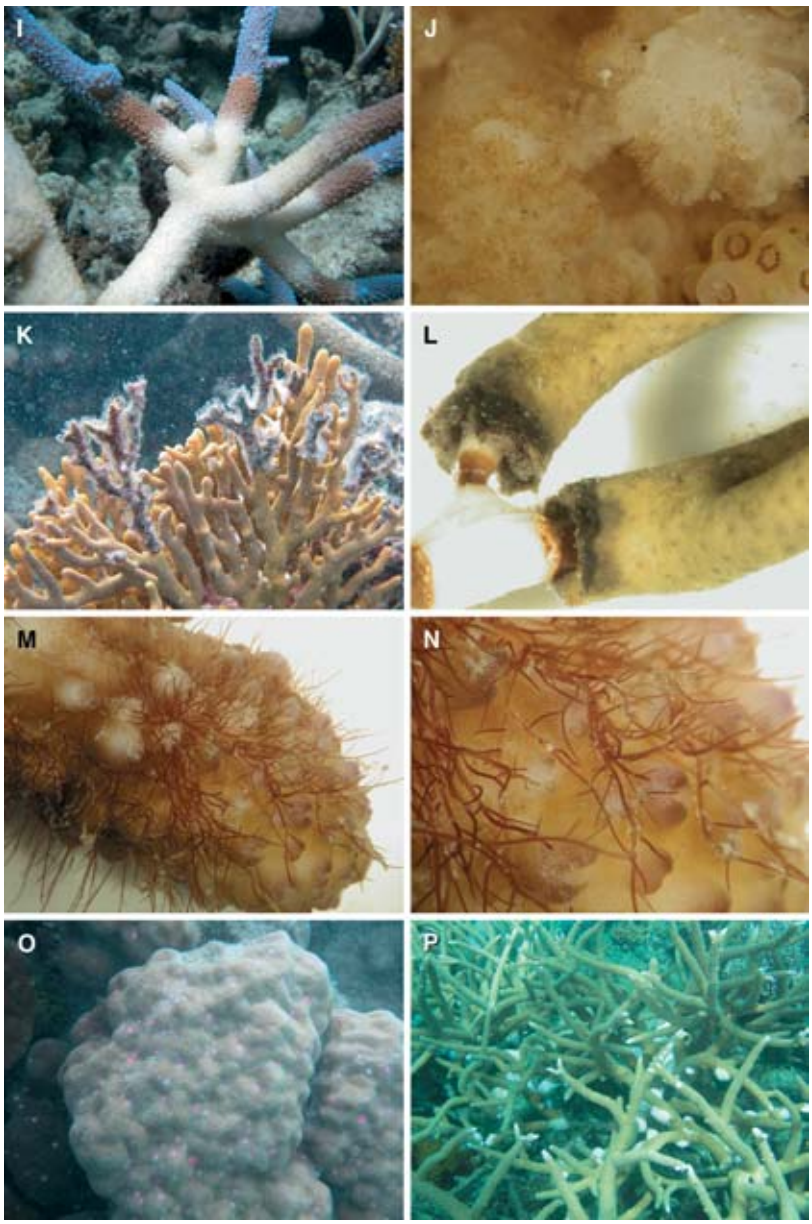

Fig. 3.1I–P.i brown band (BrB) on *A. muricata*, **j** clusters of the ciliates that cause the distinctive colouration of BrB, **k** black necrosing syndrome (BNS) on the gorgonian, *Isis* sp., **l** skeletal axis of *Isis* sp. exposed by BNS, **m** coral-algal interactions, **n** detail of filamentous 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

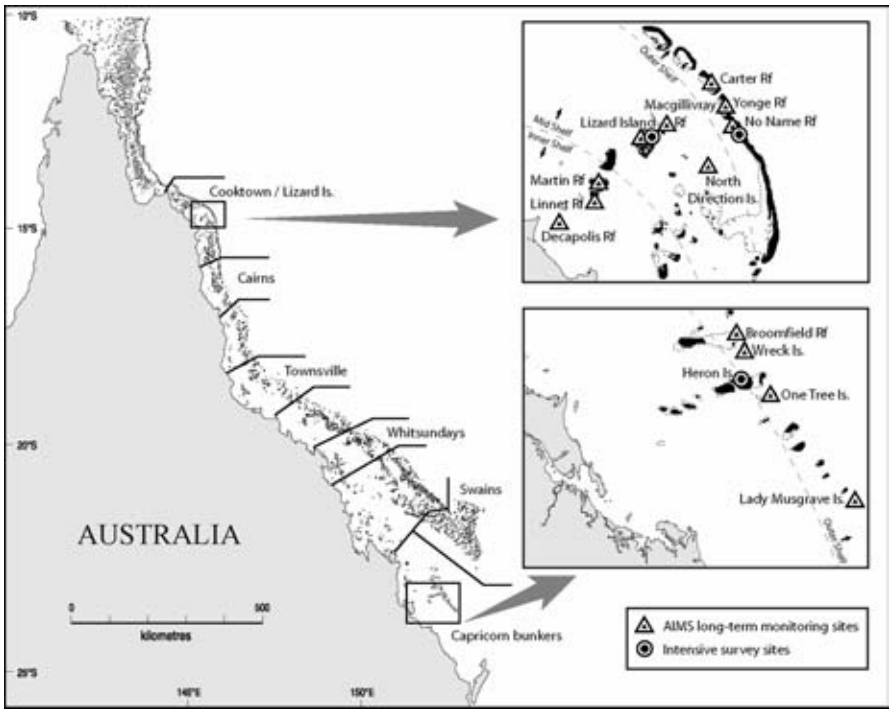

Fig. 3.2. Map of the Great Barrier Reef showing (1) the six sectors, and (2) the inner-, mid- and outer-shelf reefs in the Cooktown/Lizard Island sector and the outer-shelf reefs in the Capricorn Bunker sector that were surveyed as part of the Australian Institute of Marine Science Long Term Monitoring 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

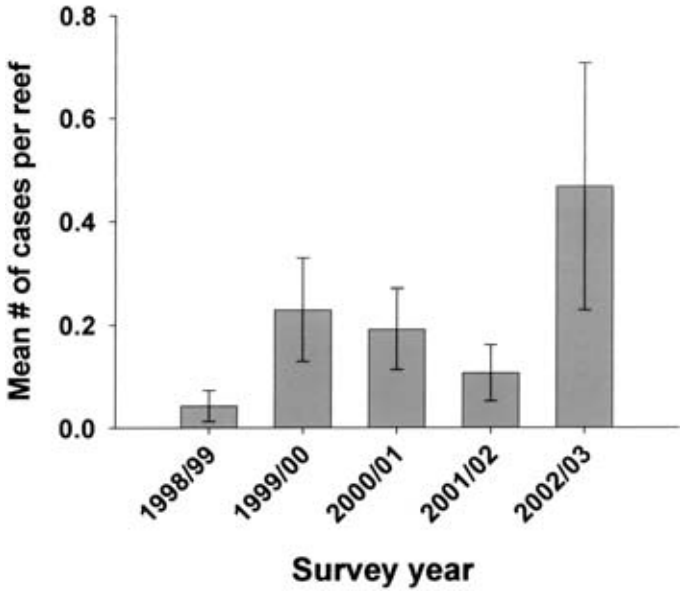

Fig. 3.3. Mean abundance (±SE) of black band disease (BBD) in survey years between 1998 and 2003. Histograms represent the mean of the total number of cases of BBD (±SE) in the 1500-m^2 area surveyed on each of the n=48 reefs per survey season

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.

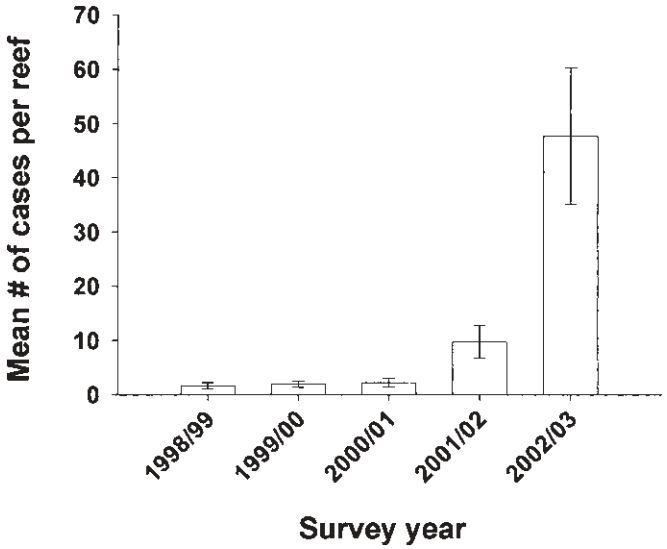

Fig. 3.4. Mean abundance (±SE) of white syndrome (WS) in survey years between 1998 and 2003. Histograms represent the mean (±SE) of the total number of cases of WS in the 1500-m^2 area surveyed on each of the reefs (n=48) per survey year

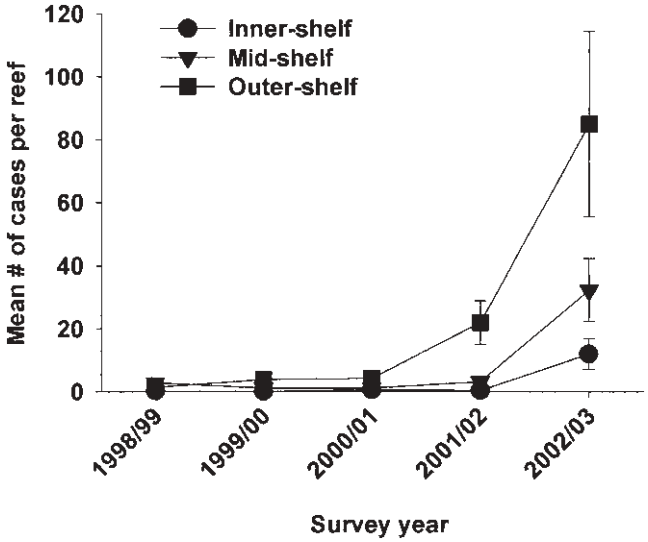

Fig. 3.5. Comparison of patterns in the mean (±SE) number of white syndrome (WS) cases per reef throughout the 5-year study period among reefs in three cross-shelf locations [i.e. inner-shelf (n=12 reefs), mid-shelf (n=18 reefs), and outer-shelf (n=18 reefs)]

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

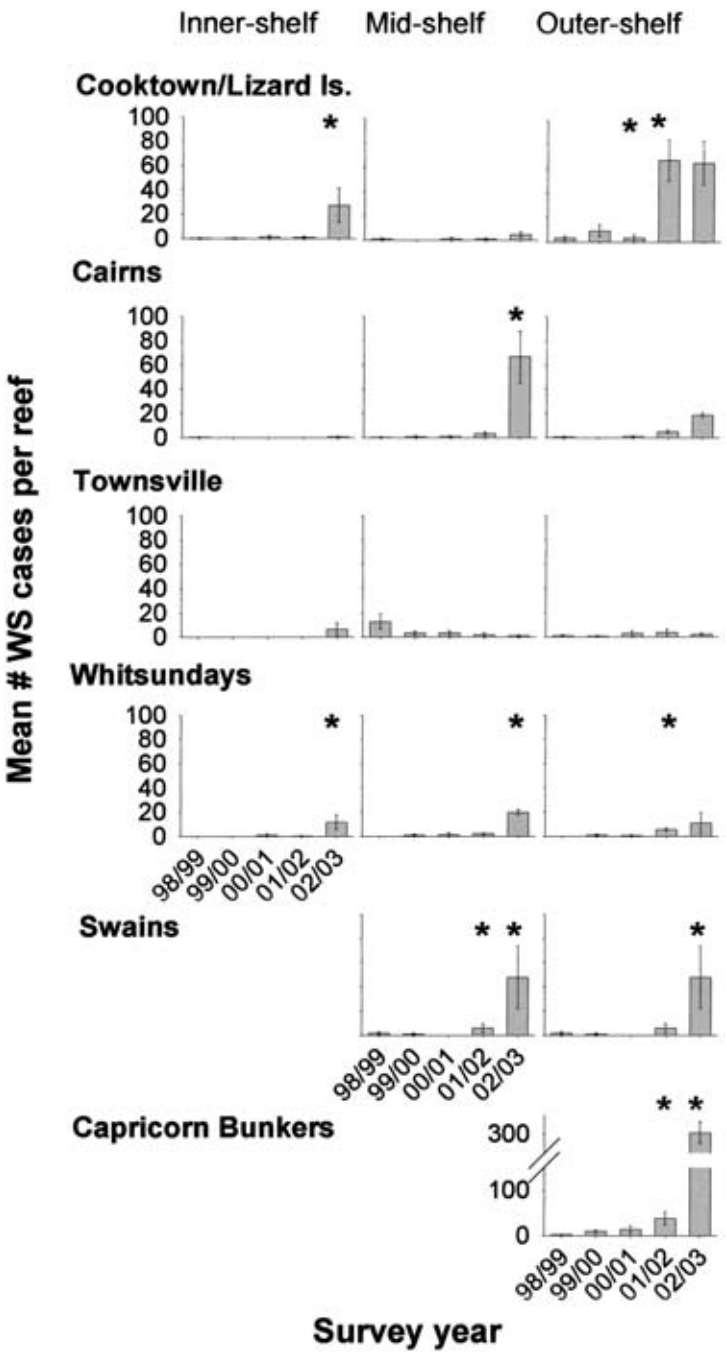

Fig. 3.6. Mean (±SE) of the total number of white syndrome (WS) cases per 1500-m^2 area surveyed on each reef compared among the 15 regions (i.e. combinations of the 6 sectors and 3 cross-shelf locations). Significant change from previous year is denoted by *

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).

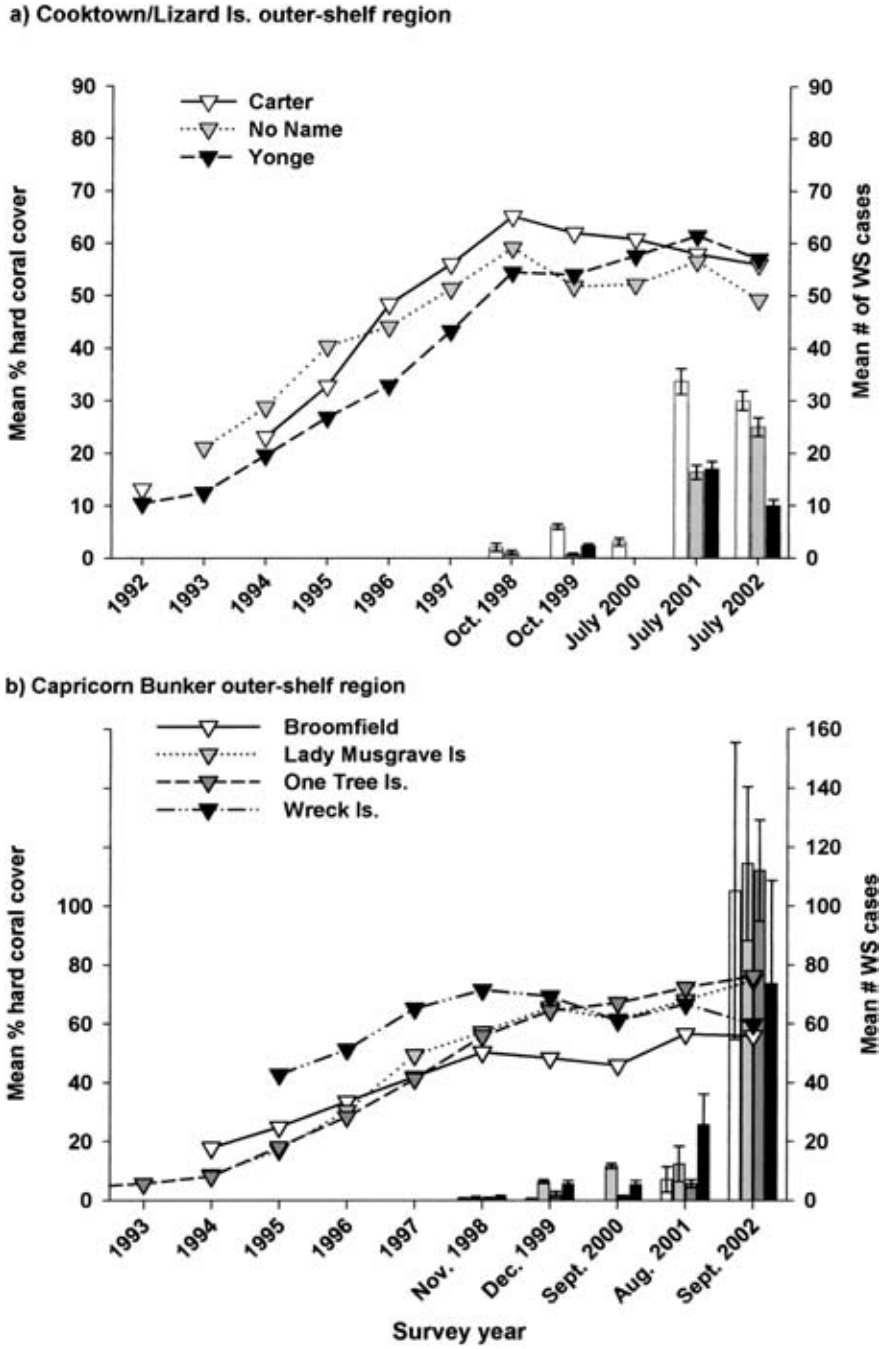

Fig. 3.7. Comparison of the mean percent hard coral cover (n=15 transects per reef) compared to mean (±SE) number of WS cases (n=15 transects per reef) in **a** Cooktown/Lizard Is. outer-shelf region, and **b** Capricorn Bunker outer-shelf region

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

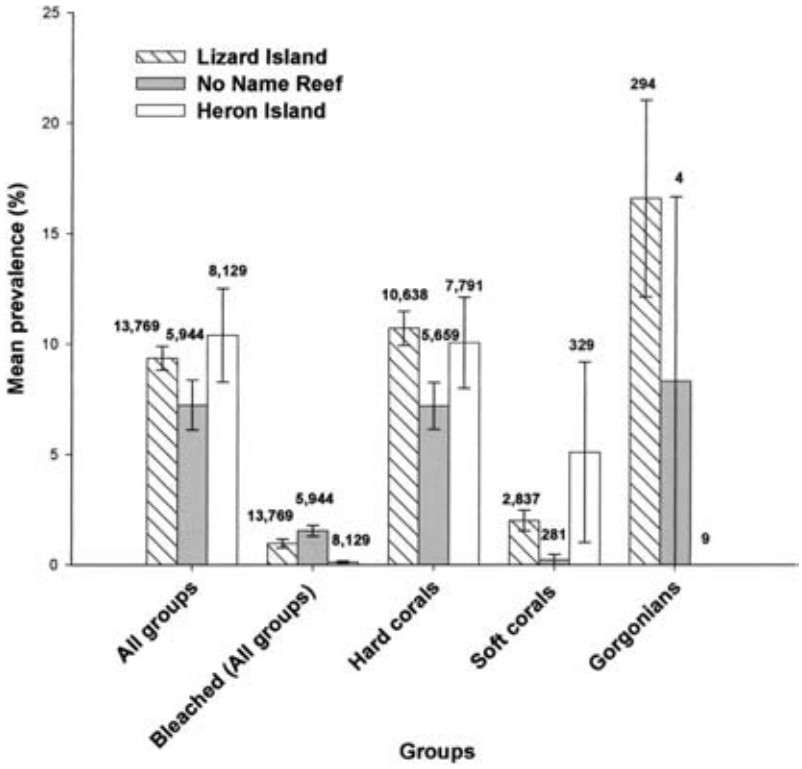

Fig. 3.8. Mean (±SE) prevalence of disease per reef for all taxonomic groups (hard and soft corals and gorgonians) combined compared to mean (±SE) prevalence of disease in hard corals, soft corals, gorgonians and bleached colonies (from all groups) at Lizard Is. (n=4 sites × 3 transects), No Name Reef (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

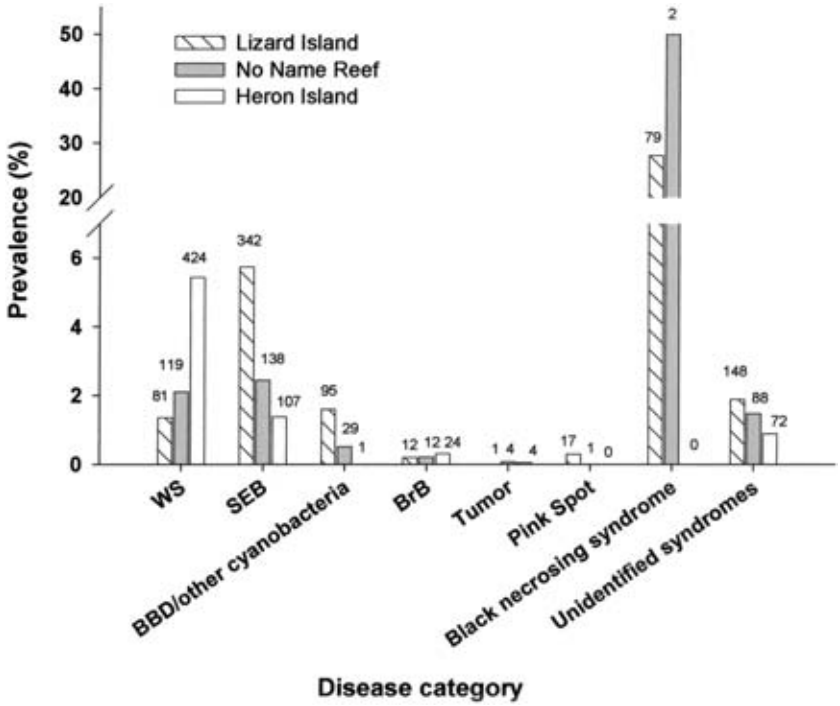

Fig. 3.9. Prevalence of each disease category at Lizard Is., No Name Reef and Heron Island in summer 2003, based on surveys of two sites per reef. Prevalence (per disease category) is calculated relative to the number of hard coral colonies examined at each reef for all disease categories except BNS (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

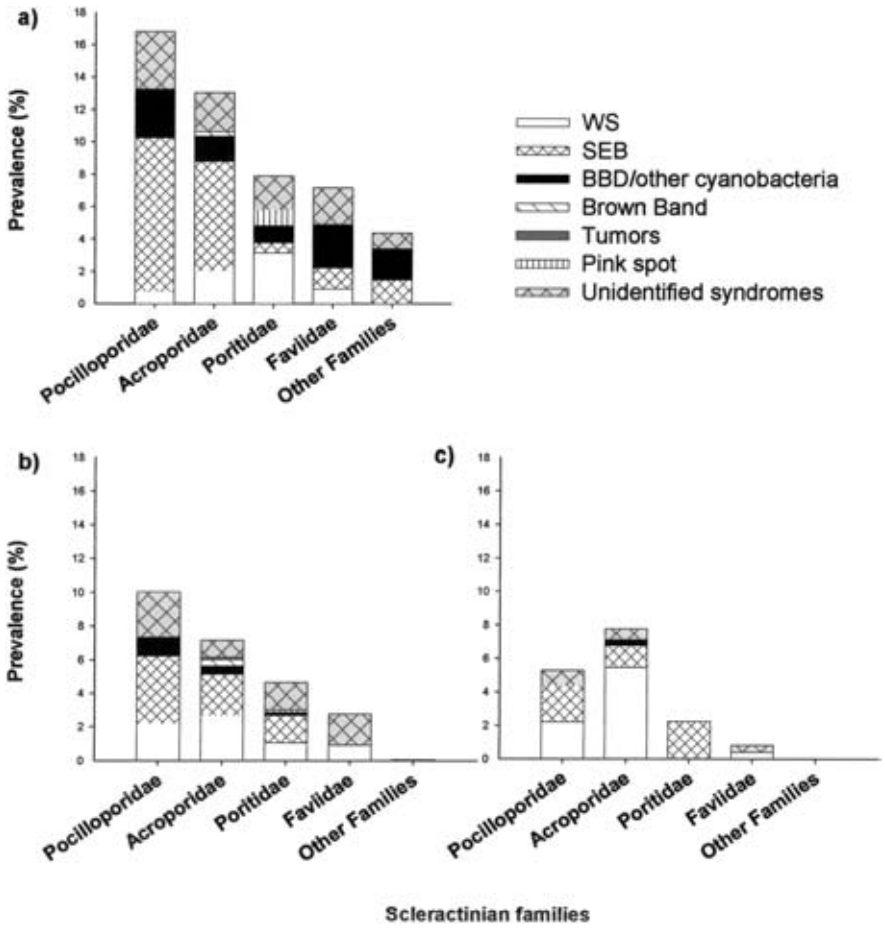

Fig. 3.10. Prevalence of seven disease categories in scleractinian families at **a** Lizard Is. (n=4 sites × 3 transects), **b** No Name reef (n=2 sites × 3 transects) and **c** Heron Is. (n=2 sites × 3 transects) in summer 2003. Prevalence (per family) is calculated relative to the total number of colonies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

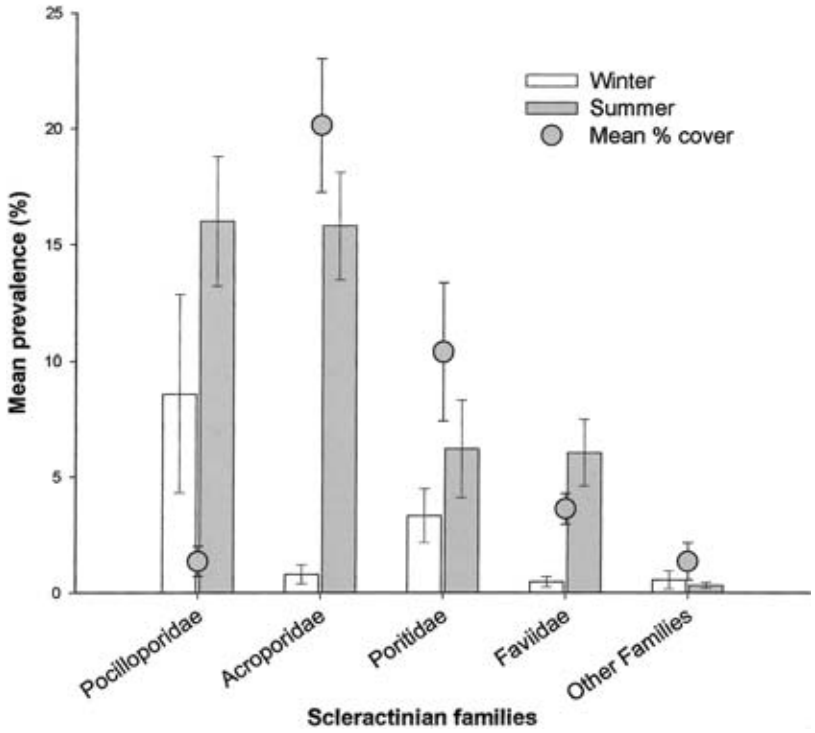

Fig. 3.11. Mean prevalence (±SE) of all diseases in scleractinian families at Lizard Island in winter 2002 vs. summer 2003 (*n*=2 sites × 3 transects) compared to mean (±SE) percent cover of each family in summer 2003 (*n*=2 sites × 3 transects). Prevalence (per family) is calculated relative to the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

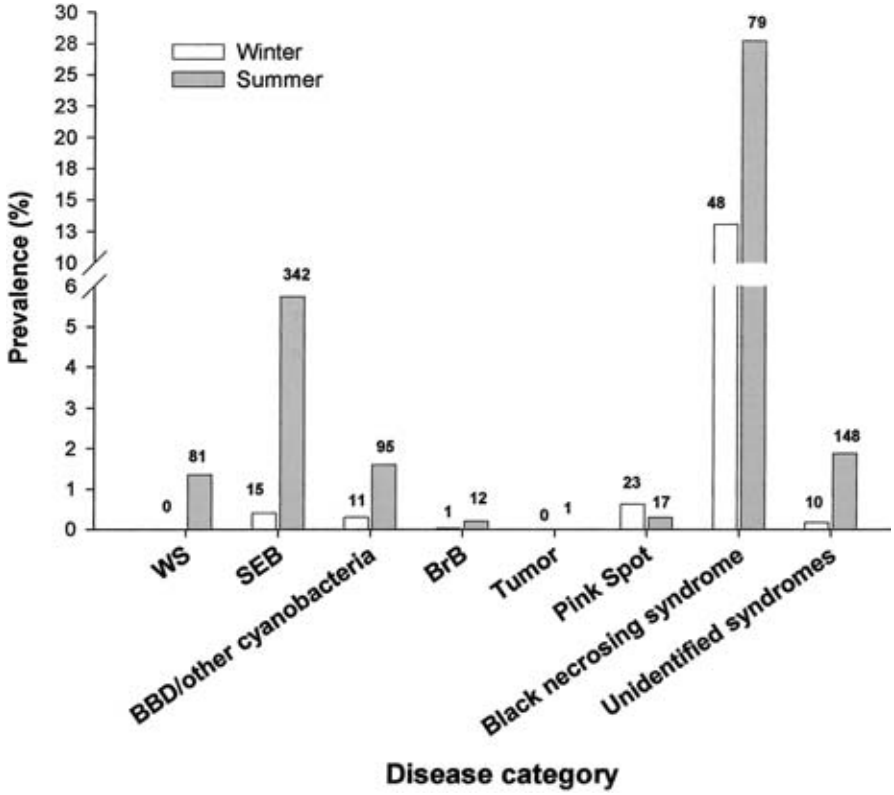

Fig. 3.12. Prevalence of seven disease categories of hard corals at Lizard Island in winter 2002 compared to summer 2003, based on surveys of two sites. Prevalence (per disease category) calculated as in Fig. 3.9. Total number of cases of each disease category is shown above the appropriate histogram

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

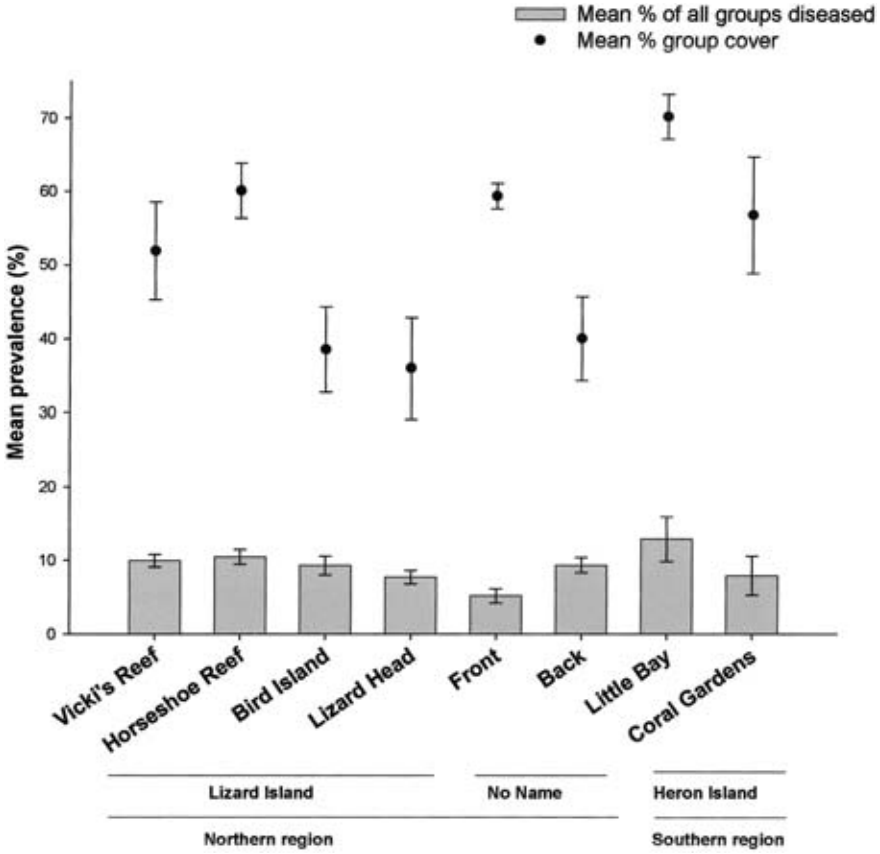

Fig. 3.13. Geographic patterns in mean disease prevalence (±SE) in all groups (hard corals, soft corals and gorgonians combined) compared to mean percent cover (±SE) of all groups at each reef in summer (January) 2003 (n=3 transects per site). Prevalence (per site) is calculated relative to the total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

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