Influence of marine-based industries on coral health and disease

Thesis submitted by
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This thesis is dedicated to my family, friends, and to all of those that share my passion for conserving coral reefs

ABSTRACT

The recent global emergence of coral disease outbreaks and subsequent coral mortality is commonly linked with human activities, however almost nothing is known about the influence of marine-based industries on coral disease. Given the growing demand for coastal development and natural resource extraction in locations that overlap with coral reefs, and growth of industries that rely on coral reefs, particularly tourism and fishing, resource managers need tools to combat coral disease epizootics and prevent future outbreaks. Research presented in this thesis identifies factors associated with industries that influence coral disease and evaluates existing and potential management tools for mitigating their impacts.

Concentrating tourism activities can be an effective way to closely manage high-use parks and minimise the effects of visitors on plants and animals, however, the effects of reef-based tourist facilities on coral health have not been assessed. In partnership with reef managers and the tourism industry, in **Chapter 2**, I test the effectiveness of concentrating tourism activities on reefs with and without permanent tourist platforms as a strategy for managing tourism on coral reefs in the Great Barrier Reef Marine Park. Coral diseases were 15 times more prevalent at reefs with offshore tourism platforms than at nearby reefs without platforms. The maximum prevalence and maximum number of cases of each disease type occurred at reefs with permanently moored tourism platforms. Diseases affected 10 coral genera from 7 families at reefs with platforms, but only 4 coral genera from 3 families at reefs without platforms. The greatest number of disease cases occurred within the spatially dominant acroporid corals, which exhibited 18-fold greater disease prevalence at reefs with versus without platforms. Neither the percent cover of acroporids nor overall coral cover differed significantly between reefs with and without platforms, which suggests that neither factor was responsible for the elevated levels of disease. Identifying how tourism activities facilitate coral disease will help ensure ongoing conservation of coral assemblages and tourism.

Recreational scuba diving on coral reefs is one of the fastest growing tourism sectors globally. Although physical injury and sedimentation associated with intensive dive tourism has been documented extensively, other impacts on coral health are unknown. In **Chapter 3,** I compare the prevalence of 4 coral diseases and 8 other indicators of compromised health at five of the highest and lowest used dive sites around the small community-managed island of Koh Tao, Thailand. The mean prevalence of healthy corals at low-use sites (79%) was twice that at high-use sites (45%). I found a 3-fold increase in coral disease prevalence at high-use sites, and significant increases in sponge overgrowth, physical injury, tissue necrosis from sediment, and non-normally pigmented coral tissues. Sediment necrosis was strongly associated with white syndrome prevalence across all sites. Injured corals were more susceptible to skeletal eroding band disease only at high-use sites, suggesting that additional stressors associated with use intensity facilitate disease development. Unexpectedly, I observed 113 corals entangled in derelict fishing line, of which 87% had ciliates associated with skeletal eroding band disease initiating from lesion boundaries, increasing disease susceptibility 5-fold compared to non-entangled corals, an unreported mechanism of coral mortality associated with fishing gear. Use of numerous indicators of coral health increases understanding of impacts associated with rapid tourism growth. Identifying practical management strategies, such as spatially separating multiple reef-based activities, is necessary to balance the expansion of tourism and maintenance of coral health.

The rapid pace of coastal development near sensitive coral reef ecosystems necessitates a comprehensive understanding of the impacts that development activities have on all aspects of coral health. While elevated sedimentation and turbidity are often cited as drivers of reef decline, their influence on coral disease prevalence has never been investigated *in situ*. In **Chapter 4**, coral health surveys were conducted along a dredging-associated sediment plume gradient to assess the relationship between sedimentation, turbidity and coral health near Montebello and Barrow Islands, Western Australia. Reefs exposed to the highest number of days under the sediment plume (296 to 347 days) had 2-fold higher levels of disease, largely driven by increases in white syndromes, and a 6-fold increase in other signs of compromised coral health, relative to reefs with little or no plume exposure (0 to 9 days). Multivariate modeling and ordination incorporating sediment exposure level, coral community composition and

cover, predation and multiple thermal stress indices provided further confirmation that the level of sediment plume exposure was the main driver of elevated disease and other indicators of compromised coral health. This study provides the first empirical evidence linking sedimentation and turbidity with elevated coral disease prevalence *in situ*. Minimising sedimentation and turbidity associated with coastal development will provide an important management tool for controlling coral disease epizootics.

A limited number of options are available for directly managing diseases in marine environments. In **Chapter 5**, the utility of marine reserves for mitigating coral disease was assessed for the first time in the Great Barrier Reef Marine Park. Comparisons of coral disease assemblages and the prevalence of six individual diseases among sites with protection versus sites with fishing revealed that no-take reserves resulted in a 3-fold reduction in pooled coral disease prevalence. Of the 31 explanatory factors tested, including habitat and environmental characteristics, fish assemblages, and differences in fishing gear restrictions, a multivariate regression demonstrated that protection from fishing was the primary factor explaining variability in coral disease assemblages. Further, significant partial correlations with coral damage and the abundance of derelict fishing line indicate that direct damage associated with line fishing is the primary driver of differences between protection levels. Gear restrictions within fished zones did not improve coral health, instead I found significantly greater levels of skeletal eroding band disease, white syndromes, coral damage, and derelict fishing line when gear was restricted, compared to unrestricted. Moreover, within fished zones, the prevalence of skeletal eroding band disease, coral damage, and fishing line increased with increasing proximity to the nearest reserve boundary, signifying that fishers target areas just outside of reserve boundaries due to ease of accessibility from boat moorings located within reserves or perceptions that fish stocks are less depleted near reserve boundaries. This study concludes that both protection from fishing and spatially managing use-intensity within fished areas are important strategies to improve coral health.

This thesis consistently demonstrates that reducing stressors associated with marine-based industries can ameliorate coral health and alleviate the impacts of disease. Identifying and implementing effective management strategies to improve coral health represent practical tools for increasing the resilience of vulnerable reef ecosystems in a changing climate and developing world.

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I declare that this thesis is my own work and has not been submitted in any form for another degree or diploma at any university or other institution of tertiary education. Information derived from the published or unpublished work of others has been acknowledged in the text and a list of references is given.

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CONTRIBUTION OF OTHERS

This thesis resulted from an immense number of collaborations among scientists, managers, students, friends, government organisations, and industry partners that I would like to acknowledge and sincerely thank for making this research possible. The chapters of this thesis are also manuscripts that have been published or submitted for publication, therefore it is necessary to recognise individual contributions accordingly.

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CHAPTER 1. General Introduction

Global deterioration of coral reef ecosystems is of critical conservation concern, not only for numerous reef-associated species, but also for one-eighth of the world's populations who reside within 100 km of a coral reef and benefit from the essential ecosystem services they provide (Moberg & Folke 1999; Bellwood et al. 2004; Burke et al. 2011). Over the last 30 years, coral cover has decreased, on average, by 50% on Indo-Pacific reefs and 80% on Caribbean reefs (Gardener et al. 2003; Bruno & Selig 2007). While a number of factors have contributed to these declines, including water pollution, habitat destruction, overfishing, invasive species, and global climate change (Pandolfi et al. 2003; Bellwood et al. 2004; Hoegh-Guldberg et al. 2007; De'ath & Fabricius 2010), outbreaks of disease have recently emerged as a significant driver of global coral reef degradation and a major threat to reef sustainability (Harvell et al. 1999, 2007). The destructive potential of coral disease is most clearly exemplified in the Caribbean, where successive disease outbreaks from 1986 to 1993 decreased populations of two significant reef-building acroporid corals by 95% and contributed substantially to observed ecological phase shifts from coral to algal-dominated reefs (Aronson & Precht 2002; Sutherland et al. 2004; Weil et al. 2006).

Evidence from paleontological and ecological monitoring suggests that the number and geographic distribution of coral disease epizootics have increased in recent years (Richardson 1998; Aronson & Precht 2002; Sutherland et al. 2004; Harvell et al. 2007), with existing reviews of the literature indicating that over 27 different diseases affect corals globally, many of which are occur across regions (Figure 1.1). Despite reports of disease significantly impacting coral reefs worldwide, knowledge underlying the distributions, causative agents and environmental drivers of marine diseases affecting coral populations is lacking. One probable explanation for the rise in disease

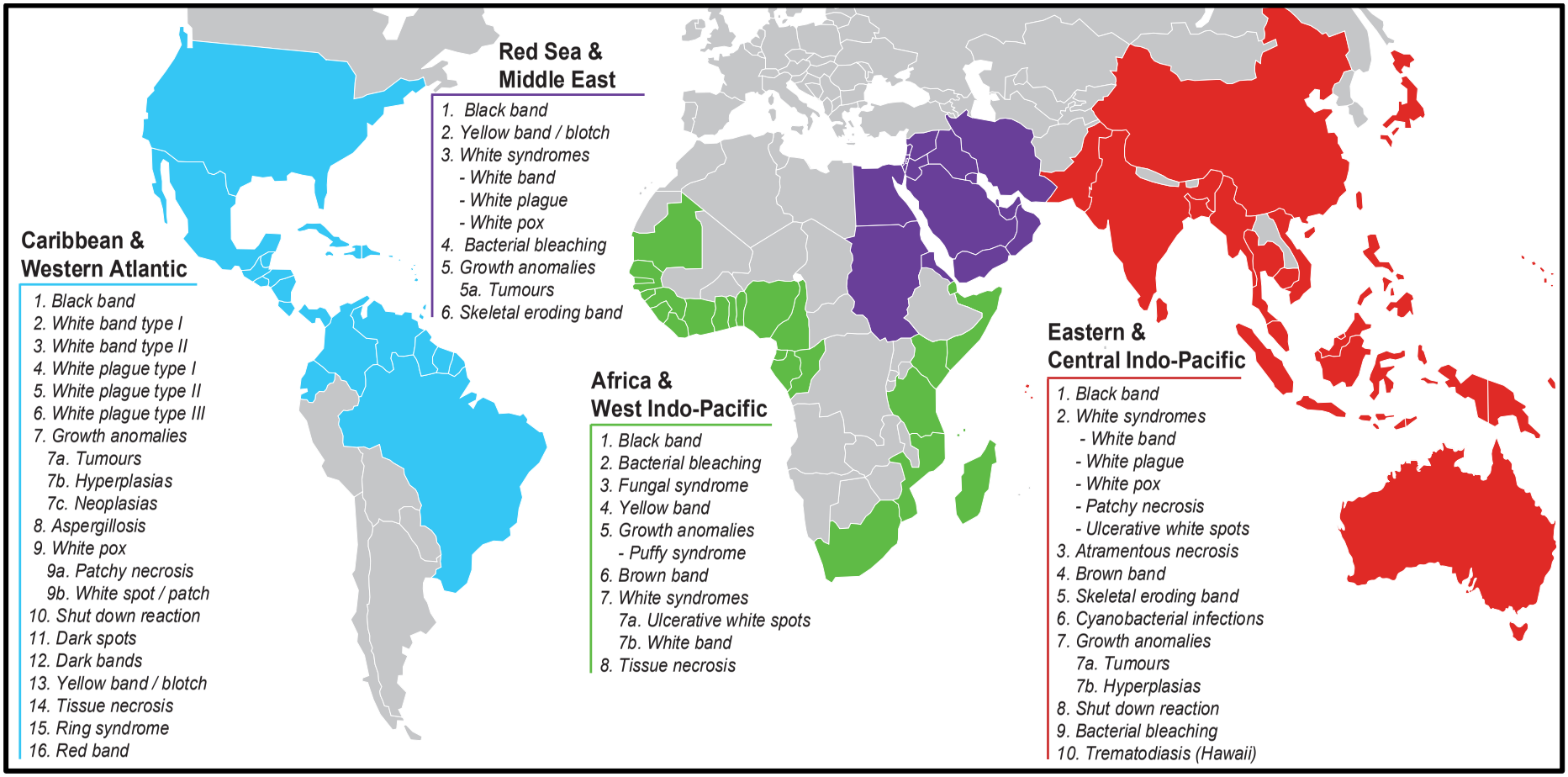

Figure 1.1 Worldwide distributions of diseases, syndromes, abnormal tissue conditions and parasitic infestations reported for scleractinian corals and gorgonians. Nomenclature may vary by region (as listed under disease types). Colours denote countries with tropical coral reefs within each region. Sources used by region: **Caribbean & Western Atlantic:** Bruckner 2002, Sutherland et al. 2004, Weil 2004, Raymundo et al. 2008; **Red Sea & Middle East:** Rosenberg & Loya 2004, Raymundo et al. 2008; **Africa & West Indo-Pacific:** Rosenberg & Loya 2004, McClanahan et al. 2004, 2009; **Eastern and Central Indo-Pacific:** Bruckner 2002, Willis et al. 2004, Aeby et al. 2007, Raymundo et al. 2008.

epizootics is that environmental conditions have been altered by a range of human-related activities, potentially compromising coral immune defences and/or enhancing the virulence of pathogens (Hayes et al. 2001; Harvell et al. 2002). Therefore, understanding the relationships between coral disease and a variety of environmental parameters that are influenced by anthropogenic activities is fundamental to identifying and managing outbreaks (Green & Bruckner 2000; Kuta & Richardson 2002; Ward & Lafferty 2004; Sutherland et al. 2010; Altizer et al. 2013).

Many coral diseases have been associated with environmental factors linked to human activities, but evidence for such links is generally limited or correlative. For example, sewage outfalls containing a human gut microbe have been associated with white plague on reefs along the coast of Florida (Patterson et al. 2002; Kaczmarsky et al. 2005; Sutherland et al. 2010). Elevated nutrients and eutrophication have been correlated with increased occurrence of yellow band, black band, and dark spots disease in various experimental studies in the Caribbean (Kuta & Richardson 2002; Bruno et al. 2003; Vega Thurber et al. 2013). Terrestrial run-off has been associated with increases in the incidence of both atramentous necrosis and black band disease in the Indo-Pacific and Western Atlantic regions (Littler & Littler 1996; Bruckner et al. 1997; Haapkyla et al. 2011). Other anthropogenic activities implicated in disease outbreaks include proximity to human population centres and coastal land alteration (Aeby et al. 2010; Guilherme Becker et al. 2012), aquaculture and fish farms (Harvell et al. 1999), overfishing and the reduction in the diversity of reef fish assemblages (Pandolfi et al. 2005; Raymundo et al. 2009), agricultural herbicides (Owen et al. 2002), and even sunscreens, which have been shown to enhance virus reproduction in symbiotic zooxanthellae and result in coral bleaching (Danovaro et al. 2008). Despite the common assumption that human-caused environmental perturbations are associated with higher levels of coral disease, quantitative evidence to unequivocally support such

hypotheses is scarce and links to activities commonly proposed to compromise coral health are generally unclear (Williams & Bunkley-Williams 2000; Bruckner 2002; Kuta & Richardson 2002; Harvell et al. 2007). Further understanding of human-related activities that undermine coral health is a critical research priority and an overarching goal of this thesis. Such knowledge will enable predictions of the regions where coral reefs are most vulnerable to degradation from infectious disease and how these pressures are likely to translate into changes in global human health and security.

1.1 Impacts of industries near coral reefs – drivers of disease?

With the global population estimated to reach 8.9 billion by 2050 (UN 2004), there is widespread concern that increasing coastal development and reef dependence is leading to progressive degradation of coral reef health (Worm et al. 2006; Halpern et al. 2008), however almost nothing is known about the influence of marine-based industries on coral disease.

1.1.1 Coastal development and dredging near coral reefs

One-quarter of coral reefs are threatened by rapid coastal development to accommodate expanding urban activities (Burke et al. 2011), with land clearing estimated to expose 1% of the world's surface to eroding processes annually (Fabricius 2006; UNEP/GPA 2006). Intense coastal development often requires the excavation, transportation and disposal of hard and soft bottom material, a process collectively known as dredging (Erftemeijer et al. 2012). Marine ecosystems, including coral reefs, located near dredging projects are subjected to a suite of pressures ranging from direct removal or burial of reef habitat (Newell et al. 1998, Thrush & Dayton 2002) to lethal and sublethal stress from elevated turbidity (suspended particulate matter) and sedimentation (deposition of particulate matter) (Bak 1978). Elevated turbidity reduces

the amount and quality of ambient light available for photosynthesis by the corals' endosymbiotic algae (*Symbiodinium*) and excess sedimentation inhibits the heterotrophic feeding efficiency of corals, reducing the energy intake of both symbiotic and asymbiotic corals (Falkowski et al. 1990). While corals are able to shed some sediment through mucus production and ciliary action, these mechanisms are energetically expensive and further burden the corals' already reduced energy budgets (Peters & Pilson 1985; Riegl & Branch 1995).

Although high or sustained sediment exposure is a major stressor that can lead to significant coral mortality, resilient coral reefs can recover from isolated sediment pulses (Browne et al. 2010). For example, Brown et al. (1990) reported a 30% reduction in living coral cover one year following the beginning of dredging operations in Thailand. After the dredging event had ceased, the reef recovered rapidly, with coral cover values and diversity indices restored to former levels within two years. In addition, Wesseling et al. (1999) noted that the recovery time of corals following experimental short-term burial varied among coral species and depended on the duration of the sedimentation event. Also, coral responses to sedimentation differ considerably between sediment types, for example nutrient content and grain size (Fabricius 2005; Weber et al. 2006). On the other hand, sedimentation can protect corals from mortality caused by higher temperature and high light conditions that lead to bleaching (Anthony et al. 2007). This may be due to particles in the water providing corals with more food under highly turbulent conditions by facilitating tissue growth and lipid levels (Anthony & Fabricius 2000; Anthony et al. 2002). Accordingly, the impacts of elevated sedimentation and turbidity associated with activities like dredging on coral community structure will depend on its species composition and the intensity of the activity.

The effects of elevated sedimentation and turbidity levels on coral health have

the potential to add a new dimension to current understanding of the overall impacts of activities, like dredging, on coral reefs. By stressing corals, sediments may make corals more susceptible to infection by microbial pathogens and may also act as disease reservoirs (Voss & Richardson 2006; Brandt et al. 2013). For example, dredging may transfer pathogens from the sediment onto nearby corals. Hodgson (1990) identified silt-associated bacteria as a possible cause of necrosis in sediment-damaged corals, as antibiotic-treated water reduced the amount of tissue damage in experimentally silted corals. In field-based observations, Haapkylä et al. (2011) noted a correlation between seasonal coastal runoff, including associated increased sedimentation and turbidity, and the prevalence of coral disease (i.e., the number of cases of a disease in a given population at a specific time) on inshore reefs of the Great Barrier Reef. Terrigenous sediment stress has also been linked to black band disease in the Indo-Pacific, Red Sea and Jamaica (Antonius 1985; Littler & Littler 1996; Bruckner & Bruckner 1997; Al-Moghrabi 2001). Despite a wealth of circumstantial evidence to suggest that exposure to elevated levels of sediment and turbidity could lead to outbreaks of disease, there have been no studies have linked dredge-plume exposure with coral disease in the field.

1.1.2 Reef-based tourism

Coral reef-based tourism is one of the fastest growing tourism sectors worldwide (Ong & Musa 2011), often providing a positive alternative to destructive and extractive uses of marine resources (Birkeland 1997; Hodgson 1997). Approximately 94 countries and territories benefit from tourism associated with coral reefs, totalling an estimated annual global net worth of US$11.5 billion (Cesar et al. 2003, estimate adjusted to US$ in 2010 by Burke et al. 2011). However, because the majority of coral reefs are located in developing and frequently unmanaged island and coastal regions (Donner & Portere 2007), the unrestricted growth and rapid development of reef-based tourism often

undermine conservation priorities necessary to sustain the industry.

Until recently, recreational reef-based tourist activities, such as diving and snorkeling were thought to have little direct impact on coral assemblages. However, numerous studies on the impacts and management of tourism on coral reefs worldwide have concluded that diving and snorkeling adversely affect coral assemblages through direct physical injury (e.g. Hawkins & Roberts, 1992, 1993; Davis & Tisdell 1995; Hawkins et al. 1999, 2005; Plathong et al. 2000; Dinsdale et al. 2004) and sediment deposition (Zakai & Chadwick-Furman 2002; Barker & Roberts 2004). Other findings suggest that damage and increased sediment and turbidity associated with the construction of permanent platforms and moorings (Smith et al. 2005), increased boat traffic (Yousef et al 1980; Jones 2011), and the movement of anchor chains (Schafer & Inglis 2000), constitute additional impacts on reefs adjacent to reef-based tourist sites. In two studies, coral disease has been noted near popular tourist locations (Hawkins et al. 1999; Winkler et al. 2004), however there have been no quantitative studies examining the links between tourist activities and coral disease.

In addition to impacts associated with sediment and turbidity (see Section 1.1.1), physical injury to corals may facilitate disease development by providing a primary site for the invasion of pathogens or by reducing immune system function. Direct links between physical stress and impaired immunity have been demonstrated in the mollusc *Haliotis turberculata* (Malham et al. 2003). Moreover, the availability of resources for allorecognition (the ability of an individual organism to distinguish its own tissues from those of another) and cell-mediated immune responses are known to be depleted during regeneration of wounds in corals, sponges and other invertebrates (Henry & Hart 2005). Thus the capacity of immune cells to resist pathogens may be limited during active regeneration of wounds (Mydlarz et al. 2006). If so, coral colonies that survive breakage

or wounding as a consequence of tourist activities may undergo reductions in the capacity to develop an immune response following exposure to a foreign substance, such as pollutants, bacteria, or viruses, which may increase their subsequent susceptibility to disease. Experimental studies indicate that physical wounding to corals may allow ciliates to become established, which has been observed for both skeletal eroding band disease (Page & Willis 2008) and brown band disease (Nugues & Bak 2009; Nicolet et al. 2013). With tourism expected to grow globally (Burke et al. 2011), the capacity to identify mechanisms linked with reef-based tourist activities that enhance either pathogen virulence or susceptibility of corals to disease is becoming increasingly crucial. This will aid in the development of strategies to mitigate disease impacts on coral assemblages and ensure the continuation of tourism as a source of income for millions of people and as a reef conservation strategy.

1.2 Strategies for managing disease in marine environments

The extent and severity of threats to reefs, in combination with the critically important ecosystem services they provide, point to an urgent need for management action. Mitigating the effects of coral decline as a result of disease represents a major management challenge (see Bruckner 2002; Harvell et al. 2004; McCallum et al. 2005; Beeden et al. 2013), and resource managers need tools to combat coral disease epizootics and prevent future outbreaks. Managers faced with controlling terrestrial disease outbreaks have several tools available, including quarantine and culling to restrict contact of infected individuals with rest of the population, vaccination, applications of chemical and biological controls, elimination and control of vectors, modification of nutrition, and even genetic breeding for resistance or tolerance (Anderson & May 1991; Scheffer 1997; Wobeser 2006; Matthews 2009). However, the

inherent difficulty of implementing terrestrial disease control methods in marine environments means that these techniques are largely impractical for coral reef managers (Bruckner 2002; McCallum et al. 2003).

Marine protected areas (MPAs) have been suggested as a potential tool for mitigating coral disease. Several lines of reasoning suggest that MPAs are likely to influence levels of disease in coral populations, although it could be argued that influences could be either beneficial or detrimental to coral health. Areas that exclude activities that damage corals may reduce disease prevalence by limiting injury. For example, many of the fishing methods and gear types used to catch coral reef fishes cause direct physical damage to corals (Bavestrello et al. 1997; Schleyer & Tomalin 2000; Yoshikawa & Asoh 2004; Mangi & Roberts 2006; Asoh et al. 2006).

In addition to reducing habitat complexity as a consequence of fishing gear impacts (Roberts 1995), the removal of targeted reef fish species has the potential to affect coral health and disease through indirect shifts in reef fish community structure (Bohnsack 1982; Russ & Alcala 1989) and disrupt the balance between corals, competitors and organisms that act as potential vectors or reservoirs of pathogens. For example, line fishing has been shown to target piscivores, important in structuring coral reef fish assemblages (Mumby et al. 2006) and indirectly, coral reef communities (McClanahan & Muthiga 1988; Roberts 1995; Graham et al. 2003). Results from several studies have found that protection in reserves increased the abundance and biomass of grazers that consume macroalgae (Mumby et al. 2006; Newman et al. 2006; Hughes et al. 2007) and have been implicated as reservoirs of pathogens in Caribbean corals (Nugues et al. 2004; Smith et al. 2006). On the other hand, greater predatory fish abundance from protection in marine reserves could actually result in decreased abundance of herbivorous fish (Hixon & Beets 1993; Graham et al. 2003). Certain fish

species may also have negative effects on corals. For example, some species of parrotfish can impede coral recovery from disturbance events by grazing on coral (Rotjan et al. 2006).

Despite the potential for marine reserves to ameliorate coral health, few studies have assessed the utility of MPAs to mitigate epizootics in coral populations. Effectively managed MPAs in the Philippines significantly reduced total coral disease, with the mechanism purportedly related to the maintenance of taxonomically diverse fish assemblages (Raymundo et al. 2009). However, Page et al. (2010) found no evidence that MPAs enhanced coral health on reefs in Palau, although an inverse correlation between fish diversity and coral disease prevalence suggested that diverse fish assemblages are important for coral health. Additional studies in Kenya (McClanahan et al. 2009) and Little Cayman Island in the Caribbean (Coelho & Manfrino 2007) did not find evidence that MPAs are effective in reducing coral disease, although most authors concluded that their results are potentially confounded by poor compliance with site restrictions or by influences that permeate reserve borders, such as terrestrial pollutants or thermal anomalies (McClanahan et al. 2009). There is a clear need to assess well-managed MPAs to evaluate the efficacy of mitigating epizootics in coral populations. Coral reefs in Australia represent approximately one-third of the reefs encompassed by MPAs worldwide (Burke et al. 2011) and recent evidence suggests that no-take reserves located in the Great Barrier Reef Marine Park are extremely effective for increasing densities of fish targeted by commercial and recreational fishers (Williamson et al. 2004; Russ et al. 2008). To date, the efficacy of marine reserves for mitigating coral disease has not been assessed in the Great Barrier Reef Marine Park.

Within both terrestrial and marine parks, resource impacts caused by increasing numbers of recreationists and tourists have become a conservation concern, prompting managers to implement a wide variety of strategies and actions, many of which are spatial (Dixon 1993; Leung & Marion 1999; Newsome et al. 2012). Spatial segregation in the form of site closures or zoning are common approaches, yet complete restrictions inhibit potentially profitable sources of income and obstruct public awareness of local conservation issues (Dixon 1993; Leung & Marion 1999). Therefore, spatial containment strategies are often adopted to confine the aggregate extent of visitor impacts, particularly at remote tourist attractions, backcountry campsites and along trails (Leung & Marion 1999; Marion & Farrell 2002). However, this strategy may require substantial use of infrastructure. Although visitor facilities, such as recreation centres or large campsites with plumbing and electricity may make the outdoor tourist experience more convenient and comfortable (Leung & Marion 1999), they can cause substantial changes to surrounding areas (Higginbottom et al. 2002; Marion & Ferrell 2002). In contrast, managers of some protected areas have sought to minimise the permanent resource impacts by dispersing visitors over extensive areas (Leung & Ferrell 1999). While the effects of such management strategies have been evaluated for many terrestrial parks and nature reserves (e.g. Leung & Marion 2000; Cole & Monz 2004; Monz et al. 2010; Newsome & Moore 2012), the effects of implementing spatial strategies to manage reef-based tourism impacts have not been examined in in relation to coral disease.

1.3 Using multiple measures of coral health to assess human impacts on reefs

Health is defined as the state of an organism when it functions optimally without evidence of disease or abnormality (Stedman 2006). For corals, linking field-based indicators of stress with potential causes is necessary for management actions to be

initiated prior to irrevocable declines in coral health from disease outbreaks. Coral degradation is principally indicated by loss of cover, however there is an emerging consensus that estimates of total coral cover fail to detect areas affected by human activities from those unaffected (Muthiga & McClanahan 1997; Hawkins et al. 1999; Dinsdale & Harriott 2003; Darling et al. 2013). Therefore, classifying numerous visual signs of coral health, such as coral partial mortality and deviations from normal coloration or morphology using standardised protocols (Figure 1.2), can be useful for associating specific lesions or damage from anthropogenic impacts with specific drivers of coral loss (Santavy & Peters 1997; Beeden et al. 2008). To date, the concurrent use of multiple field-based signs of disease and other indicators of compromised health to classify stress associated with human activities on reef corals has not been undertaken.

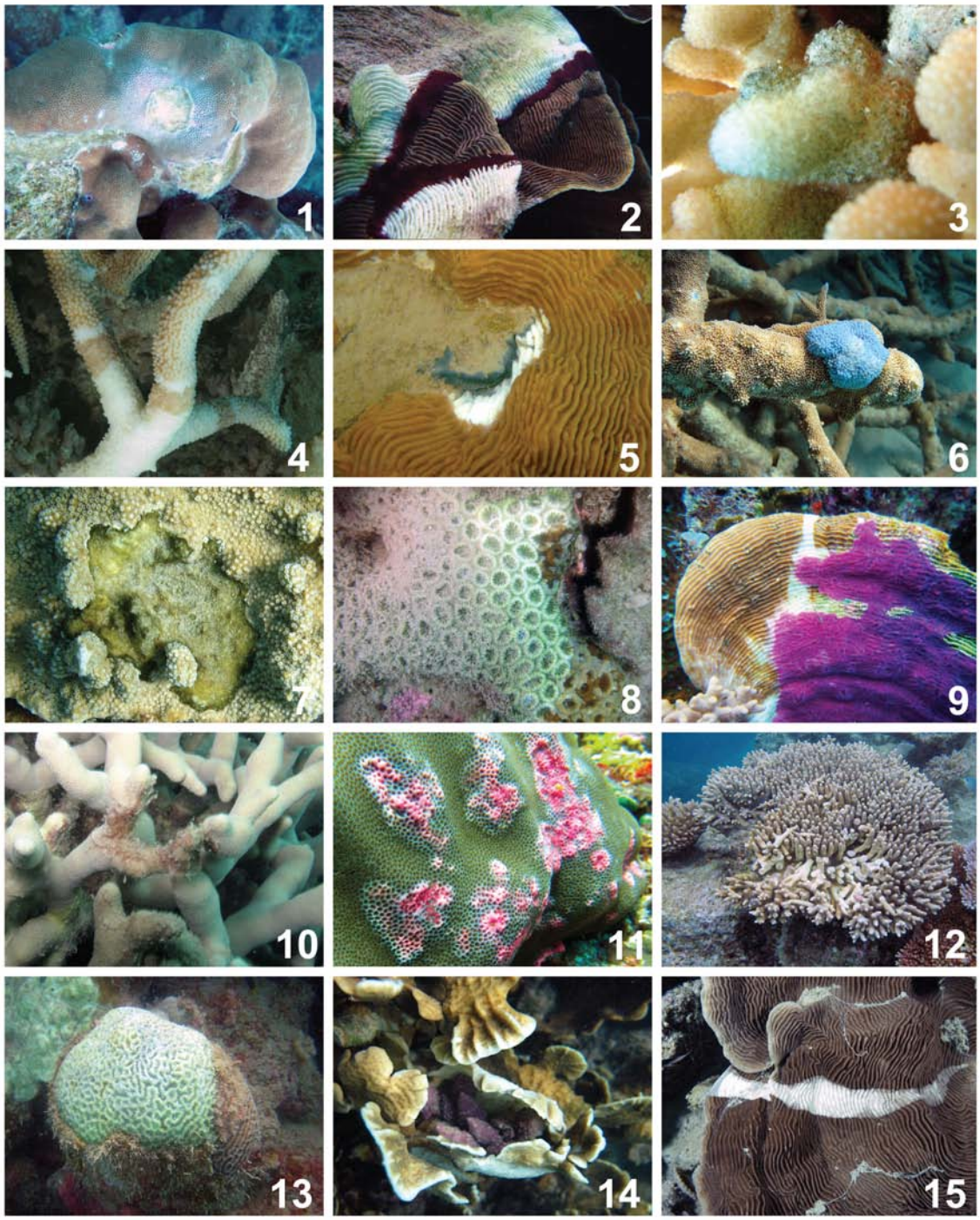

Figure 1.2 Field photographs exhibiting signs of coral disease and other indicators of compromised health frequently observed affecting scleractinian corals in the Indo-Pacific[a]. Coral diseases: **(1)** white syndrome, **(2)** black band disease, **(3)** skeletal eroding band disease, **(4)** brown band disease, **(5)** atramentous necrosis (photo: Y. Sato), **(6)** growth anomaly, **(7)** other cyanobacteria overgrowth (photo: Y. Sato). Other indicators of compromised coral health: **(8)** sediment necrosis, **(9)** sponge overgrowth, **(10)** red algae overgrowth, **(11)** pigmentation response, **(12)** physical damage, **(13)** bleaching, **(14)** predation from *Drupella* spp., **(15)** unusual bleaching. [a]Standardised signs of disease and compromised coral health as per Beeden et al. (2008), an output of the Global Environment Facility and World Bank Coral Disease Working Group.

1.4 Thesis objectives

The overall aim of this study is to identify the influence of marine-based industries on coral health, and to evaluate if current management approaches are useful tools for mitigating disease impacts on reef corals. Identifying the roles that human activities play in facilitating outbreaks of coral diseases is the critical first step in developing management strategies to ensure the ongoing conservation of coral reefs and persistence of dependent industries. The principal objectives of my research were to:

A. Identify the effects of marine-based industries on coral disease prevalence. By comparing coral disease prevalence and diversity between reefs located near industry-related impacts and reefs without these impacts, I will evaluate the extent to which marine-based industries are affecting the health of reef corals. I use this comparative approach to identify coral health impacts caused by concentrating tourist activities to offshore platforms on the Great Barrier Reef (Chapter 2), intensive reef-based tourism on community managed coastal reefs in Thailand (Chapter 3), dredging associated with Australia's largest natural gas project based offshore in the Indian Ocean (Chapter 4), and recreational fishing on inshore reefs of the Great Barrier Reef Marine Park (Chapter 5). Understanding the implications of industry for coral health will enable a critique of existing management strategies and lead to the development of improved management practices for ameliorating coral disease.

B. Elucidate factors associated with marine-based industries that compromise coral health. Identifying factors associated with industries near reefs that influence coral disease prevalence and compromise coral health is vital for developing appropriate management strategies for alleviating these specific impacts, therefore I use a combination of field-based surveys, remotely sensed environmental data, and multivariate modeling to evaluate variation in coral disease assemblages and individual disease types with physical injury and sediment damage (Chapter 3) and length of exposure to elevated levels of sediment and turbidity (Chapter 4).

C. Assess the efficacy of marine reserves as a tool to mitigate coral disease in the Great Barrier Reef Marine Park. Examining the effects of existing no-take areas on both reef fish and coral communities will help determine if well-established marine protected areas are useful management tools for moderating coral disease (Chapter 5).

CHAPTER 2.

Using coral disease prevalence to assess the effects of concentrating tourism on offshore reefs in the Great Barrier Reef Marine Park[†]

[†]**Lamb, J.B.** and B.L. Willis. 2011. Using coral disease prevalence to assess the effect of concentrating tourism activities on offshore reefs in a tropical marine park. Conservation Biology 25(5): 1044–1052.

2.1 Introduction

Infectious diseases are emerging as an important issue in the conservation of terrestrial and marine species (Harvell et al. 2002). Disease is now recognised as a major factor in the accelerating degradation of coral reefs in many regions of the world (Harvell et al. 1999, 2007). The causes of most diseases of corals are largely unknown (Richardson 1998; Harvell et al. 2007), but it is assumed that a variety of human activities may alter environmental conditions on reefs and potentially reduce coral resistance to microbial infections or increase pathogen virulence (Harvell et al. 2002). For example, coral diseases are associated with elevated nutrient concentrations (Bruno et al. 2003; Voss & Richardson 2006) from terrestrial runoff (Littler & Littler 1996) and sewage outfalls containing human enteric microorganisms (Patterson et al. 2002). Other human activities implicated in rising disease prevalence in corals include aquaculture (Harvell et al. 1999), unsustainable levels of fishing (Pandolfi et al. 2005), and introduced chemicals (Owen et al. 2002; Danovaro et al. 2008).

Although first-hand experience of local flora and fauna is one of the best ways to promote public awareness of conservation issues (Dixon 1993), achieving the dual objectives of providing recreational opportunities and preserving natural environments is challenging (Higginbottom et al. 2003). Management actions implemented to direct the location of tourist activities have minimised the aggregate extent of visitor effects

on animals and plants in many terrestrial parks and protected areas (Leung & Marion 1999), particularly by concentrating visitor effects at remote tourist attractions, backcountry campsites and along trails (Marion & Farrell 2002). However, this strategy may require substantial use of infrastructure. Although installations of permanent buildings and trails may make the tourism experience more convenient and comfortable for visitors and are often desired features for safety and social reasons (Leung & Marion 1999), they can cause substantial changes to surrounding areas (Higginbottom et al. 2003). Thus, managers of some protected areas have sought to minimise the effects of infrastructure by dispersing visitor numbers over extensive areas. The effects of such management strategies have been evaluated for many terrestrial nature reserves (Leung & Marion 1999), but not for marine parks.

Tourism on the Great Barrier Reef is one of the most economically important industries in Australia and is geographically concentrated in the Cairns and Whitsunday Island sections of the Great Barrier Reef Marine Park (Harriott 2002). Approximately half of the 1.4 million visitors to these 2 regions each year take a day trip to 1 of 4 reefs with permanently moored offshore tourism platforms (Harriott 2002; Smith et al. 2005). Platforms are in shallow, sheltered waters adjacent to offshore reefs and provide visitors with easy access to reefs for viewing fish and coral communities. Since the first platforms were moored in the early 1980s, they have developed from small, simple platforms to large platforms with multiple levels (averaging 45 m x 12 m) that can each accommodate roughly 400 visitors. There is a growing demand for offshore tourism platforms to facilitate and enhance reef visitor experience; thus, the number and size of tourism platforms are forecast to increase (Smith et al. 2005).

Much of what is currently known about the effects of tourism activities on coral reefs comes from studies of changes in percent coral cover in response to direct physical

contact, for example, coral breakage due to activity of divers (Hawkins & Roberts 1992) and swimmers along snorkeling trails (Plathong et al. 2000), construction of permanent platforms and moorings (Smith et al. 2005), and movement of anchor chains (Schafer & Inglis 2000). The results of previous studies show that the effects of tourism platforms on coral cover are few and isolated (Smith et al. 2005). However, ongoing tissue loss caused by slowly progressing diseases could cause greater levels of coral mortality than immediate but short-term effects associated with breakage or localised shading. For example, in the Caribbean, 2 dominant reef-building corals, *Acropora cervicornis* and *A. palmata,* have been nearly extirpated on some reefs by an outbreak of white band disease that caused tissue loss of 0.5 cm/day on average (Patterson et al. 2002). On the Great Barrier Reef, reported rates of tissue loss vary from 1 cm/day for black band disease to 10 cm/day for brown band disease (Page & Willis 2006; Boyett et al. 2007).

Human activity on coral reefs may stress the ecosystem and reduce coral health at reefs in close proximity to offshore tourism platforms. Near these platforms, nutrient levels may increase from seabird guano that accumulates on platforms and is washed onto the reef by rain or by cleaning of the platform, from visitors and tourism operators feeding fish, and from tourists entering the water. Tourists also introduce pollutants and may physically damage the coral while snorkeling and diving. The platform infrastructure itself (e.g., chains used for anchoring the platform, snorkeling trail boundaries, and reef viewing stations) may also physically damage corals exposed by low and varying tides. However, there are no published studies on the prevalence of coral diseases as a measure of coral health in relation to tourism activities. Here I compare coral disease prevalence among reefs with permanently moored tourism platforms and adjacent reefs without platforms.

2.2 Methods

2.2.1 Study sites and data collection

I conducted surveys in the Great Barrier Reef Marine Park off the northeast coast of Australia during late June and early July of 2009 (Figure 2.1). I selected 8 reefs located within 2 adjacent management sections that are the most frequently visited in the Great Barrier Reef Marine Park (Central and Cairns sections in the central-northern region of the park). I selected 180-m^2 survey sites on the sheltered sides of mid-shelf reefs, which were located 40–50 km offshore and between latitudes 16º11'S and 16º44'S in the Central section and latitudes 19º32'S and 19º48'S in the Cairns section. I surveyed 2 reefs with permanently moored tourism platforms in each of the 2 sections. I selected 4 reefs without permanent platforms and lower levels of reef-based tourism (i.e., at most a single boat mooring with a maximum of 40 in-water visitors/site/day) on the basis of their proximity to reefs with tourism platforms (within 10–25 km of the nearest reef with a platform) and taxonomic composition of the coral assemblage. Thus, 3 reefs were selected in the Cairns section and 1 reef was selected in the Central section.

At each reef, I used scuba to examine corals for disease. I surveyed along 6 randomly placed 15 m x 2 m belt transects, except at Milln Reef, where I surveyed 3 transects. I randomly placed transects along depth contours of 2–6 m and 5 m apart on upper reef slopes close to the main entry point of in-water visitors. Within each 30-m^2 belt transect, I identified each coral colony over 5 cm in diameter to genus and further classified it as either healthy (no disease observed) or affected by one or more of the following: black band disease (and other cyanobacterial mats), brown band disease, white syndromes, which are among the most virulent diseases, growth anomalies, and skeletal eroding band (Willis et al. 2004). I estimated how much coral cover was present for each genus using standard line-intercept surveys along each 15-m transect by recording the extent of each coral to the nearest centimeter.

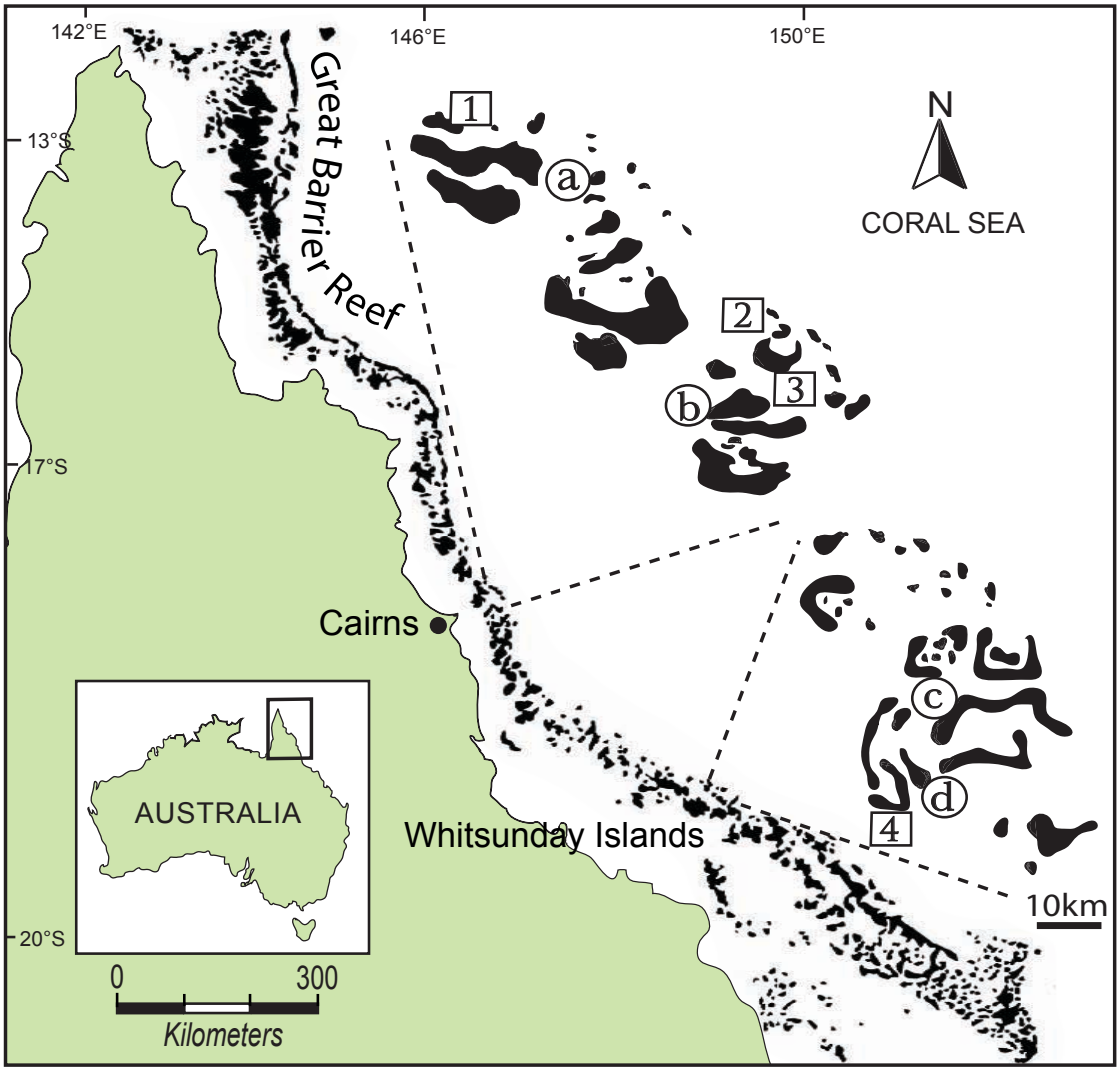

Figure 2.1 Locations of 8 reefs surveyed for coral disease within 2 latitudinal sectors (dashed lines) of the Great Barrier Reef Marine Park off the northeast coast of Australia (reefs with permanent tourism platforms, a–d circles; reefs without tourism platforms, 1–4 squares).

2.2.2 Data Analyses

Disease prevalence was calculated within each 30-m^2 belt transect by dividing the number of colonies in the 5 disease classes by the total number of colonies present (24 prevalence values at reefs with platforms; 21 prevalence values at reefs without platforms). To analyse broad taxonomic patterns in disease prevalence, I assigned coral families to 1 of 3 groups on the basis of spatial abundance on the Great Barrier Reef (Willis et al. 2004): Acroporidae, the spatially dominant family; common reef-building families (Pocilloporidae, Poritidae, and Faviidae); and less common families (Agariciidae, Fungiidae, Merulinidae, Mussidae, Oculinidae, Pectiniidae, and Siderastreidae).

I compared differences in mean disease prevalence and coral cover among platform and control reefs with a 3-factor nested analysis of variance. I classified effect (reefs with versus without platforms) and location (Central vs. Cairns sections) as fixed factors and nested reef within both effect and location. To assess individual variation among reefs, I treated reef as a random factor. Prior to analyses, I tested assumptions of normality (Shapiro-Wilks) and homogeneity of variance (Levene's test of homogeneity). I transformed data to the square root to meet assumptions of normality. We tested associations between disease prevalence and both total hard coral and acroporid cover with Pearson product-moment correlations. All analyses were performed in Statistica 9 (StatSoft, Tulsa, Oklahoma).

2.3 Results

2.3.1 Disease prevalence relative to tourism platforms

Mean disease prevalence was 15-fold greater at reefs with tourism platforms (mean ± SE = 3.27% ± 0.62) than at reefs without platforms (0.21% ± 0.07; Figure 2.2 & Table 2.1). The mean of minimum disease prevalence values recorded at reefs with

platforms was 4 times greater than the mean of maximum prevalence values at reefs without platforms. Disease prevalence ranged from 0.2% to 12.0% (median = 2.5%) at individual reefs with tourism platforms, whereas prevalence at individual reefs without platforms ranged from 0% to 1.1% (median = 0%). At each of the 4 reefs with tourism platforms, corals surveyed (*n* = 7043) exhibited 5 of the 7 diseases typically recorded on the Great Barrier Reef (172 disease cases at reefs with platforms; Table 2.1 & Figure 2.2). The virulent diseases, white syndromes, brown band disease, and black band disease, were most prevalent. Prevalence values for skeletal eroding band and growth anomalies were approximately 4 times lower than virulent diseases (Table 2.1). The maximum number of cases and prevalence of each disease were recorded at reefs with tourism platforms.

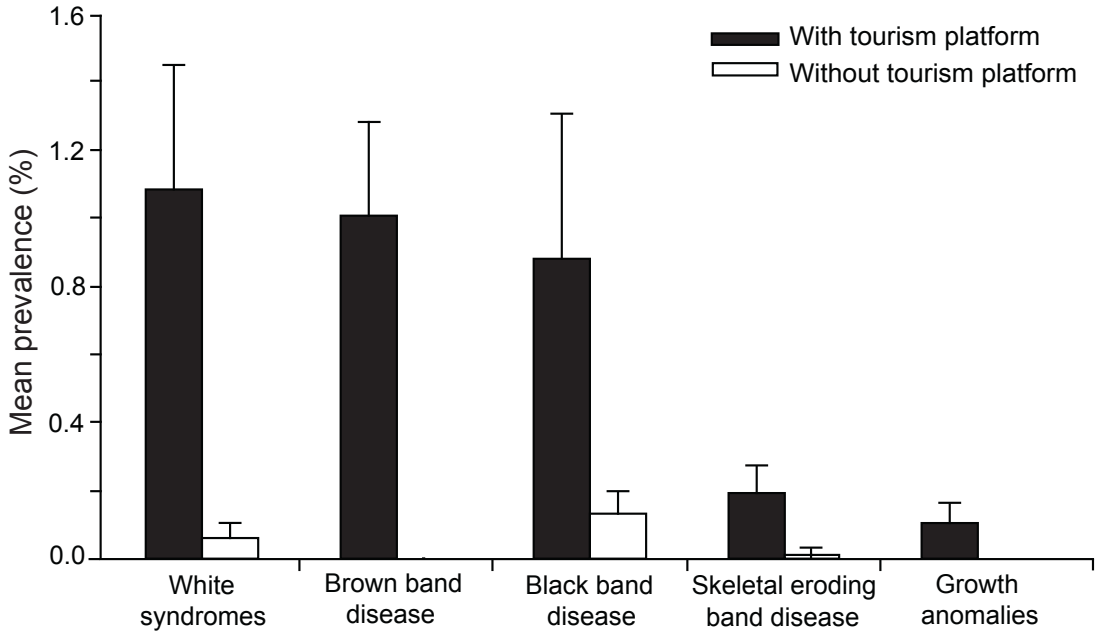

Figure 2.2 Mean (SE) disease prevalence between reefs with tourism platforms (black bars, 24 transects) and without tourism platforms (white bars, 21 transects) for the 5 disease classes recorded in surveys.

Table 2.1 Abundance and mean prevalence[a] of coral disease at reefs with and without tourism platforms and results of 3-level nested analysis of variance (ANOVA).

	With platform (n = 24 transects)		Without platform (n = 21 transects)		ANOVA			
Variable	No. cases	Mean (SE) prevalence (%)	No. cases	Mean (SE) prevalence (%)	Effect F, *P*	Location F, *P*	Effect x Location F, *P*	Reef (Effect x Location) F, *P*
Total disease	172	3.27 (0.62)	14	0.21 (0.07)	25.4, <0.009*	0.5, < 0.51	0.1, <0.77	2.1, <0.10
White syndromes	55	1.08 (0.37)	7	0.06 (0.04)	32.4, <0.008*	3.4, <0.15	1.9, <0.26	0.8, <0.51
Brown band	64	1.01 (0.27)	0	-	13.3, <0.02*	0.02, <0.91	0.02, <0.92	1.5, <0.23
Black band	35	0.88 (0.42)	6	0.13 (0.06)	47.7, <0.04*	0.4, <0.54	0.6, <0.48	2.6, <0.06
Skeletal eroding band	10	0.19 (0.08)	1	0.13 (0.01)	53.3, <0.02*	47.7, <0.04*	24.1, <0.06	0.2, <0.92
Growth anomalies	8	0.18 (0.06)	0	-	19.3, <0.01*	0.001, <0.97	0.05, <0.83	2.2, <0.09
Family group[b]								
Acroporidae	137	2.63 (0.57)	6	0.08 (0.05)	20.8, <0.01*	0.3, <0.58	0.007, <0.94	2.3, <0.08
Common	13	0.23 (0.10)	8	0.13 (0.06)	2.0, <0.25	0.1, <0.73	3.5, <0.16	0.7, <0.60
Other	22	0.41 (0.33)	0	-	0.9, <0.40	2.0, <0.23	1.5, <0.30	2.8, <0.04*

[a] Mean prevalence calculated as the percentage of colonies with disease for each disease type or family group as a percentage of the total number of corals per transect. Analyses performed on data transformed to the square root (*significant difference for $\alpha = 0.05$).

[b] Families grouped on the basis of spatial abundance on the Great Barrier Reef: Acroporidae, the spatially dominant family; common families, Pocilliporidae, Poritidae, and Faviidae; and less common families, Agariciidae, Fungiidae, Merulinidae, Mussidae, Oculinidae, Pectiniidae and Siderastreidae.

In contrast, 14 cases of disease were recorded at reefs without platforms (n = 9468 colonies surveyed), where black band disease and white syndromes were the most prevalent diseases. I observed one case of skeletal eroding band and no cases of brown band diseases at reefs without tourism platforms. Disease prevalence for each of the 5 diseases recorded was significantly higher at reefs with tourism platforms than at reefs without platforms (white syndromes, $P < 0.01$; brown band disease, $P < 0.05$; black band disease, $P < 0.05$; skeletal eroding band, $P < 0.05$; growth anomalies, $P < 0.01$; Table 2.1).

2.3.2 Patterns in disease prevalence among coral families

Diseases affected a 2.5-fold greater range of corals on reefs with tourism platforms than on reefs without such platforms. Diseases were present in 10 genera from 7 families of reef-building corals at reefs with tourism platforms and in 4 genera from 3 families at reefs without nearby platforms. The difference in the number of coral taxa present between effect groups was not statistically significant.

On average, corals in the family Acroporidae accounted for the largest proportion of coral cover at reefs with and without platforms (Figure 2.3a). Acroporid corals accounted for 76% of all disease cases at reefs with tourism platforms (Figure 2.3b). Approximately 4% of acroporid corals at the 4 reefs with platforms, particularly the staghorn (branching) species, were affected by at least one disease, whereas 0.2% of acroporids at reefs without platforms were affected by disease. Thus, disease prevalence on acroporid corals was 18-fold greater at reefs with platforms. All 5 of the diseases I recorded were observed on acroporid corals at all 4 reefs with tourism platforms, whereas a maximum of 2 diseases was recorded on acroporid corals at 1 reef without a platform.

The prevalence of disease within the group of common coral families (Pocilloporidae, Poritidae, and Faviidae) was approximately 0.1% and not significantly different between reefs with and without platforms (Table 2.1). White syndromes, black band disease, and skeletal eroding band affected these families at reefs both with and without platforms (Figure 2.3b).

All 22 cases of disease at reefs with tourism platforms affected hard corals in the less common families Agariciidae, Merulinidae, and Siderastreidae (Figure 2.3b). The prevalence of black band disease and other cyanobacterial mats in the Agariciidae and Merulinidae was 10.6% and 7.2%, respectively; however, these cases were observed at a single reef in the group with platforms. Of all other reef-building corals, 1.4% had disease, and prevalence values did not differ significantly between platform and control reefs (Table 2.1).

2.3.3 Relation between hard coral cover and disease prevalence

The total hard coral cover did not differ significantly between reefs with and without platforms (mean ± SE = 46.2% ± 2.4 and 45.7% ± 3.8, respectively; $F = 0.007$, $P = 0.94$). Moreover, disease prevalence was not correlated with hard coral cover, either at sites with ($r = 0.31$, $P = 0.14$; Figure 2.4a) or without tourism platforms ($r = -0.72$, $P = 0.76$; Figure 2.4b).

The mean percent cover of acroporid corals was slightly higher at reefs with tourism platforms (mean ± SE = 29.2% ± 3.3 vs. 22.5% ± 4.8; Figure 2.3a); however, the difference was not statistically significant ($F = 1.7$, $P = 0.26$). Although the majority of disease cases occurred within the acroporid family (see above), there was no association between disease prevalence and percent acroporid cover at reefs either with ($r = 0.29$, $P = 0.16$; Figure 2.4c) or without tourism platforms ($r = 0.26$, $P = 0.26$; Figure 2.4d).

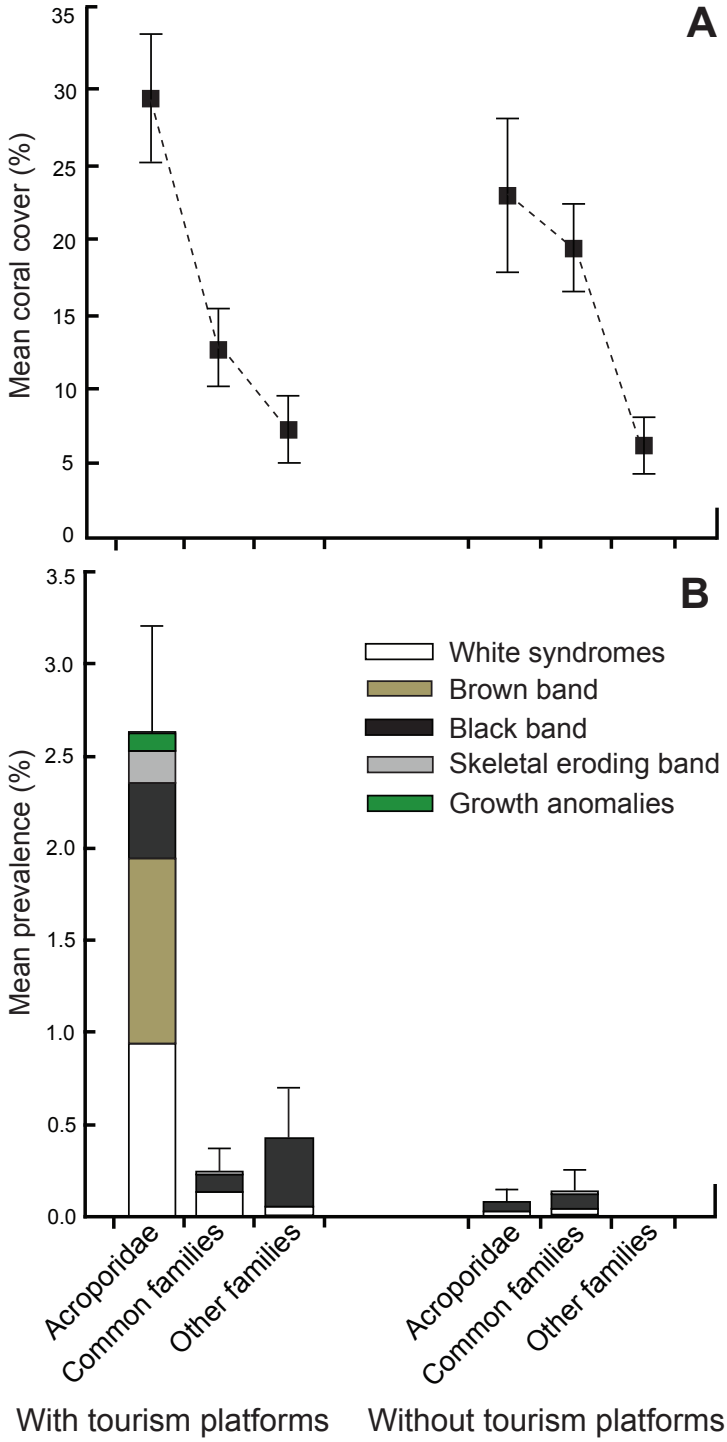

Figure 2.3 Taxonomic patterns of (a) mean (SE) coral cover and (b) mean (SE) coral disease prevalence between reefs with (24 transects) and without (21 transects) tourism platforms. (Acroporidae, spatially dominant family; common reef-building families, Poritidae, Pocilloporidae, and Faviidae; less common families, Agariciidae, Fungiidae, Merulinidae, Mussidae, Oculinidae, Pectiniidae, and Siderastreidae).

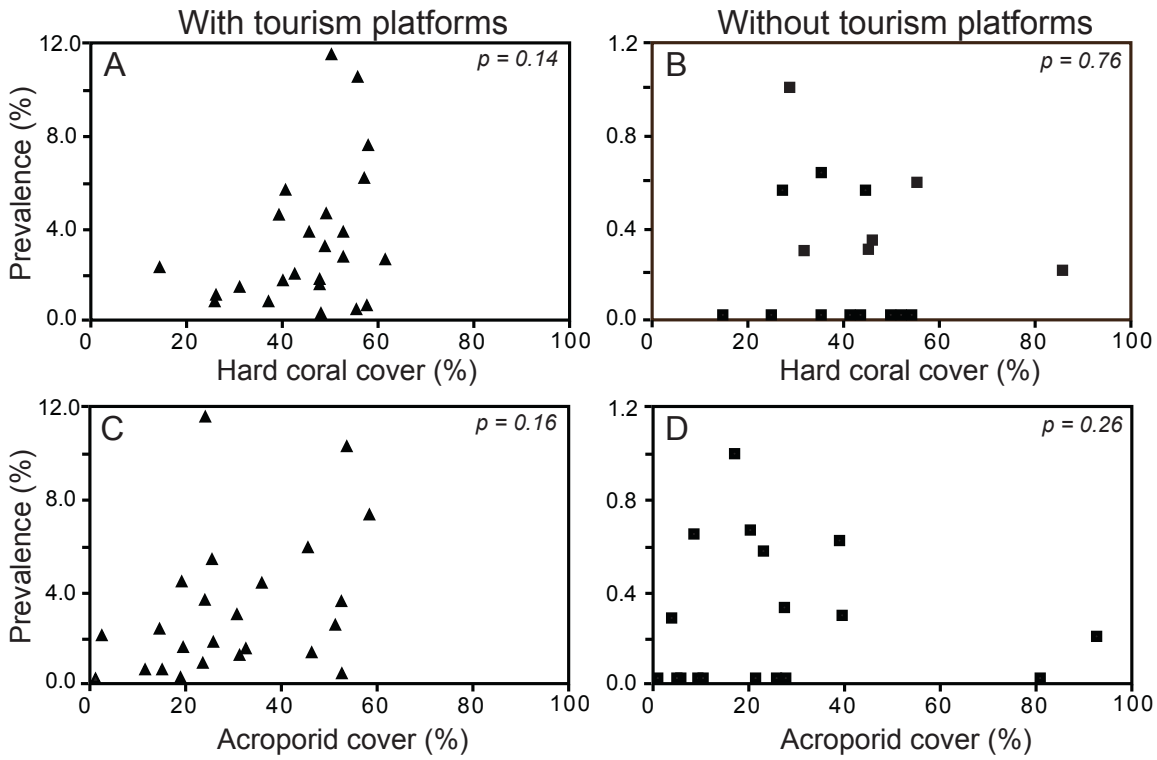

Figure 2.4 Associations between prevalence of all coral diseases and total hard coral cover at reefs (a) with tourism platforms (24 transects) and (b) without tourism platforms (21 transects) and associations between disease prevalence and acroporid cover at reefs (c) with tourism platforms and (d) without tourism platforms. Y-axis values in (a) and (c) are larger by a factor of 10 than those in (b) and (d).

2.4 Discussion

The consistently elevated prevalence of coral disease on reefs with tourism platforms compared to reefs without such platforms over an extent of 600 km suggests that either offshore tourism platforms or activities associated with them reduce resistance of reef corals to disease. Because I found no significant differences in percent cover of all corals or of the dominant, disease susceptible Acroporidae among reefs with and without platforms, I believe differences in host density or family composition are unlikely to have caused the difference in disease prevalence. Models link increases in the abundance of corals with diseases with increases in host density (Bruno et al. 2007),

presumably reflecting transmission of pathogens via direct colony-to-colony contact (Riegl 2002). However, I detected no associations between disease prevalence and cover of all scleractinian corals or of acroporid corals. Increased susceptibility to infection from normally nonpathogenic local microbial communities, as a consequence of proximity to tourism platforms (cf. Ritchie 2006), could have played a role in the prevalence of coral diseases at these reefs. Thus, coral disease prevalence may represent a useful metric of human disturbance on coral reefs.

Identifying and managing potential disease drivers

Pathogens may spread rapidly in marine systems (McCallum et al. 2003). For example, the coral disease white plague spreads along the coast of Florida at rates of approximating 200 km/year (Richardson et al. 1998). Tracing the origins and halting known environmental inputs that influence the abundance and severity of coral disease is the most viable option for alleviating the effects of coral diseases (Harvell et al. 2007). However, the overall increase in coral disease on reefs with tourism platforms may represent the cumulative effect of a number of factors that might otherwise not negatively affect corals, which may have disproportionate, long-term effects when they occur in combination with other stressors.

Pollutants

Tourist platforms and in-water viewing stations are often used as resting sites by sea birds, and their guano may increase levels of nitrogen and phosphorus near these platforms (Bosman & Hockey 1986). Bird guano may also contain toxins, including dichlorodiphenyltrichloroethane (DDT), mercury, and hexachlorobenene (HCB) (Blais et al. 2005). Even moderate nutrient enrichment can significantly increase the severity of both aspergillosis on sea fans and yellow band disease on corals in situ (Bruno et al.

2003), and the abundance of black band disease is positively correlated with concentration of nitrogen (Kuta & Richardson 2002). I suggest removal of any platforms that are not used regularly by tourists and washing guano into gutters placed around the edges of platforms that drain into wastewater tanks already in place. Nitrogen isotope analysis, which can separate nitrogen inputs originating from wastewater versus other anthropogenic sources (Baker et al. 2007), may prove useful for assessing potential sources of nutrients at platform sites, potentially including human waste.

Chemical compounds contained in sunscreens and other such products can reach detectable levels in both freshwater and seawater (Daughton & Ternes 1999; Giokas et al. 2007). Danovaro et al. (2008) estimate 4000–6000 tons of sunscreen may be released per year into tropical reef areas. In laboratory studies, organic ultraviolet filters from sunscreens induce lytic viral cycles in symbiotic zooxanthellae, causing bleaching in acroporid corals (Danovaro et al. 2008). Although the degree to which pollutants come into contact with reef corals is unknown, a precautionary approach that limits the entry of nutrients and chemicals into the water could include enforcement of alternative measures of sun protection (e.g., hats and full-body sun suits) and increased tourist education.

Physical damage

A major challenge for managers of coral reefs is control of activities in heavily used areas that could severely damage corals, particularly branching species of *Acropora* (Plathong et al. 2000). The availability of energy for allorecognition and cell-mediated immune responses declines during regeneration of damaged tissue in corals, sponges, and other invertebrates (Mydlarz et al. 2006). Therefore, even if coral colonies survive breakage or damage from recreational activities, reductions in

immunocompetence may increase their subsequent susceptibility to disease.

The ciliate diseases brown band and skeletal eroding band occurred only at reefs with tourism platforms. These are the only two diseases known to be associated with ciliates on the Great Barrier Reef (Willis et al. 2004). The ciliate that causes brown band disease, the most prevalent coral disease at reefs with tourism platforms, may be transmitted via the water column and spread through human activity around tourism platforms. Physical damage to corals may allow ciliates to become established, which may lead to skeletal eroding band lesions (Page &Willis 2008). Thus, increased injury to corals near platforms may be contributing to increased disease prevalence and diversity. Injured colonies can become infected with black band disease after being transplanted downstream from diseased corals (Rutzler & Santavy 1983). Thus, dislodged black band mats, which comprise primarily cyanobacteria, may transmit the disease as they are transported by water currents and divers' fins (Bruckner et al. 1997). Tourists themselves could serve as vectors of coral disease.

Although it has been suggested that more than 5000 visitors per year damages reefs (Hawkins & Roberts 1997), each of the 4 tourism platform operators in this study reported over 40,000 visitors per year, although not all visitors enter the water. Boundaries limiting snorkeling activities are in place at all tourism platforms in our study, but much of the physical contact with corals is a result of uninformed or careless behavior. Managers can educate and compel visitors to reduce high-impact behavior (e.g., standing on and touching corals) and to engage in low-impact behavior (e.g., use of personal flotation devices when resting). Large groups of visitors have greater potential to damage coral than the same number of individuals in smaller groups (Higginbottom et al. 2003). Therefore, probability of disease may be reduced by limiting group sizes, extending the length of snorkeling trails to reduce crowding, and

varying trail location according to tides to standardise distance to the reef throughout the day.

Different growth forms and species of coral vary in their response to trampling (Plathong et al. 2000; Marion & Farrell 2002). Locating viewing sites and moorings away from more susceptible families and growth forms may reduce disease.

Longer-term effects of increased disease prevalence

Although the mean disease prevalence at platform reefs in autumn and winter was low, increases in prevalence are typical in summer (Willis et al. 2004), and it is likely that increases in ocean temperature associated with climate change will further increase the abundance and severity of coral diseases (Harvell et al. 1999, 2002; Bruno et al. 2007). Summer increases in disease prevalence in all Great Barrier Reef coral families, which are up to 15-fold higher for acroporid corals during summer months than in winter months (Willis et al. 2004), suggest that high summer temperatures and thermal anomalies may stress corals and reduce their immunity to disease, potentially concurrent with increased growth of pathogens or pathogen virulence as temperatures increase (Harvell et al. 1999; Mydlarz et al. 2006). Increasing distances of snorkeling trail boundaries to the reef or reducing visitor numbers in summer could reduce stress to corals. However, peak tourist season is during summer on the Great Barrier Reef; therefore, enforcing limits on reef visitor numbers during peak periods would severely affect the local economy.

I suggest that measuring and monitoring coral disease near popular tourism destinations is necessary to inform strategies for controlling visitor use. Results of studies of visitor effects on terrestrial trails and campsites (Marion & Farrell 2002) and marine snorkeling trails (Plathong et al. 2000) and dive sites (Hawkins & Roberts 1992)

show that most negative effects on natural resources have a curvilinear relation to visitor-use levels (i.e., the majority of damage accumulates rapidly during initial use of the visitor area and subsequent use causes little additional change) (Higginbottom et al. 2003). Lower coral disease prevalence at our control sites, which were used by fewer than 5000 recreational divers per year (levels recommended by Hawkins & Roberts 1997), suggests that dispersing visitors and creating low-use sites without permanent platforms may benefit coral health.

The status of corals and fishes influences the satisfaction of day visitors to coral reefs (Schafer & Inglis 2000). If visitor activities degrade local environments, the financial benefits of tourism may not be sustainable and conservation objectives will not be met (Dixon 1993; Higginbottom et al. 2003). Quantifying spatio-temporal coral disease prevalence to establish reference points for future comparisons may help evaluate the success or failure of management actions.

CHAPTER 3.

Impacts of intensive reef-based tourism and derelict fishing line on coral disease prevalence and susceptibility on community managed reefs in Thailand[†]

[†]Accepted for publication in Biological Conservation

3.1 Introduction

Global decline in coral reef health is a critical conservation concern, especially for the estimated 275 million people that live within 30 km of coral reefs and draw extensively on them for livelihood and food security (Bellwood et al. 2004; Burke et al. 2011). There is pressing demand to find income-generating alternatives to destructive and extractive uses of marine resources (Birkeland 1997). Tourism is generally considered a favourable alternative, typically providing an incentive to preserve natural areas, thereby contributing to environmental protection, sustainable use practices, and the restoration of biological diversity (Buckley 2012). Coral reef-based tourism is one of the fastest growing tourism sectors worldwide (Ong & Musa 2011). However, because the majority of coral reefs are located in developing and often undermanaged island and coastal regions (Donner & Portere 2007), the unrestricted growth and rapid development of reef-based tourism often undermines the conservation priorities necessary to sustain it.

Coral disease outbreaks are now recognized as a significant factor in the accelerating degradation of coral reefs, and it is commonly assumed that a variety of human-related activities have altered environmental conditions, potentially impairing coral resistance to microbial infections or increasing pathogen virulence (Altizer et al. 2013). Anthropogenic activities implicated in disease outbreaks and rising prevalence

levels (i.e., the number of cases of a disease in a given population at a specific time) include proximity to human population centres (Aeby et al. 2010), coastal land alteration and terrestrial runoff of sediment or agricultural herbicides (Owen et al. 2002; Haapkyla et al. 2011; Guilherme Becker et al. 2012; Chapter 4), sewage outfalls containing human enteric microorganisms (Patterson et al. 2002), increases in nutrient concentrations (Bruno et al. 2003), aquaculture and fish farms (Harvell et al. 1999; Garren et al. 2009), a reduction in the diversity of reef fish assemblages (Raymundo et al. 2009), and sunscreens (Danovaro et al. 2008).

Until recently, recreational reef-based activities, such as diving and snorkeling, were thought to have little direct impact on coral assemblages. However, over the past two decades, numerous studies have been conducted on the physical impacts and management of diving on coral reefs worldwide. Most concluded that diving could adversely affect coral assemblages through physical injury (e.g. Hawkins & Roberts 1992, 1993; Davis & Tisdell 1995; Dinsdale et al. 2004; Hawkins et al. 1999, 2005) or sediment deposition (Zakai &Chadwick-Furman 2002). In a few studies, coral disease has been associated with the presence of concentrated tourist activities (Hawkins et al. 1999; Winkler et al. 2004; Lamb & Willis 2011), however no studies have attempted to directly link coral susceptibility or disease prevalence with measures of dive site use intensity, such as levels of physical injury or sediment deposition. Minor damage and resuspension of sediment by most divers may seem trivial, but by compounding other reef stresses associated with tourism, they could undermine the resilience of local reef ecosystems (Nystrom et al. 2000) and reduce recovery rates following natural disturbances (Connell 1997). In addition, a variety of other factors could increase coral disease prevalence and reduce health at intensively dived tourist sites in rapidly developing regions, including possible increases in nutrients from vessel sewage and

wastewater and elevated levels of resuspended sediment associated with shoreline erosion from boat wakes and crowding.

The island of Koh Tao, located in the western Gulf of Thailand, has rapidly grown as a tourist and recreational destination, leading to the replacement of small-scale hook-and-line or traditional hand net fisheries by reef-related tourist activities (Yeemin et al. 2006). From 1992 to 2003, the number of tourists increased by 375% and now considered the hub of scuba diving certification in Southeast Asia, estimated to generate US$62 million per year to the local economy (Larpnun et al. 2011). At present, the island has approximately 50 dive operators that accommodate greater than 300,000 visitors per year to a total reef area of 2 km^2 (Weterings 2011; Larpnun et al. 2011), reaching intensities of use beyond levels seen even in regions heavily impacted by damage, such as the Red Sea (< 250 000 divers/year to 4km^2 of reef area: Zakai & Chadwick-Furman 2002).

Here, I use the prevalence of four coral diseases and eight additional indicators of compromised coral health to assess the effects of recreational diving intensity on coral reefs surrounding Koh Tao. To date, the concurrent use of multiple field-based signs of disease and other indicators of compromised health to classify stress associated with human activities on reef corals has not been undertaken. Using a multitude of indicators to assess coral health may, for the first time, will improve our capacity to identify and manage specific impacts of tourism on reef corals. In light of predicted increases in tourism and recreational activities globally, the results of this study will aid in the development of practical management strategies to mitigate the impacts of frequent visitation that increase the likelihood of coral disease outbreaks and ensure long-term persistence of corals reefs and livelihoods in developing coastal regions.

3.2 Methods

3.2.1 Data collection

I conducted surveys around the island of Koh Tao in September 2011, approximately 1 year after a bleaching event and subsequent wet season in the Gulf of Thailand (Figure 3.1). I selected a total of ten, 90m^2 sites distributed around the island and located approximately 100m from shore. Based on questionnaires from 23 of the largest dive operators on the island, Weterings (2011) found that most of the dive sites around Koh Tao were unevenly visited and a select number were often frequented by up to 10 dive operators in a single day. Due to ease of access, dive sites with the highest levels of use are often located nearest to the large number of operators located in the west and southwest regions of the island (Figure 3.1). I surveyed the top 5 dive sites that are heavily and constantly used by visitors throughout the year (i.e., more than 5 boat operators with a minimum of 50 in-water visitors/site/day) (high use sites), and 5 sites that had similar coral assemblages but had few to no in-water visitors each year (low use sites).

At each site, three 15 m x 2 m belt transects were laid randomly along depth contours at 2 - 6 m and approximately 5 m apart, consistent with standardised protocols developed by the Global Environment Facility (GEF) and World Bank Coral Disease Working Group (Beeden et al. 2008). Within each 30 m^2 belt transect, every scleractinian coral over 5 cm in diameter was identified to genus and further classified as either diseased (i.e., affected by one or more of the following disease classes recorded in the Indo-Pacific region: white syndromes, skeletal eroding band, black band disease, brown band disease, and/or growth anomalies); showing other signs of compromised health (i.e., affected by one or more of the following: tissue necrosis due to sediment, bleaching, non-normal pigmentation of tissue, overgrowth by sponges, red

or green algae, and cuts and scars from predation by crown-of-thorns starfish and corallivorous marine snails); physically damaged (recently exposed skeleton from breakage or severe abrasions); or healthy (i.e., no visible signs of disease lesions, other compromised health indicators or physical damage) (Willis et al. 2004; Lamb & Willis 2011). Standard line-intercept surveys were also used to determine coral cover and community composition by estimating the linear extent of each coral to the nearest centimeter along the central line of each 15 m transect. These standardised protocols allow the data from this study to be directly compared to other coral disease datasets collected globally.

3.2.2 Data analyses

The prevalence of coral disease and other signs of compromised health was calculated within each 30 m^2 belt transect by dividing the number of colonies with one of the four diseases or eight other compromised health categories recorded in this study by the total number of colonies present, i.e. 15 prevalence values per disease or other category, both for the group of high use and low use sites.

3.2.2.1 Multivariate analyses Differences in overall disease assemblages were investigated using multivariate community analyses. A nested permutational multivariate analysis of variance (PERMANOVA, Clark et al. 2006; Anderson et al. 2008) was used to test for differences between high and low use levels, with site (random factor) nested within use-level (fixed factor). The analysis was based on a zero-adjusted Bray-Curtis similarity matrix (Clark et al. 2006), type III partial sums of squares, and 999 random permutations of the residuals under the reduced model. To identify indicators of disease and other signs of compromised coral health between the

two use-levels (those contributing most to the patterns in multivariate space), I used a principal coordinates analysis (PCO) performed on a Bray-Curtis similarity matrix using square root transformed data due to strong linear pairs of variables (Anderson et al. 2008; Clarke and Gorley 2008). Pearson correlations of the ordination axes were calculated with the original disease and other compromised health data, where indicators with strong correlations (defined in this study as ≥ 0.6) were then overlaid as vectors on a bi-plot.

Similarities between coral communities at the family-level were illustrated using a non-metric multidimensional scaling plot (nMDS), with hierarchical clusters overlaid from dendrograms based on a Bray-Curtis similarity matrix from square-root transformed data at the transect level (Clark & Gorley 2008). I used a nested analysis of similarity (ANOSIM) to test differences in coral assemblages between use-levels, where I nested site (random factor) into use-level (fixed factor). All multivariate analyses were performed using PRIMER and PERMANOVA+ v6 (PRIMER-E Ltd, Plymouth, UK).

3.2.2.2 Univariate analyses To analyse patterns of coral disease among broad taxonomic groups, coral families were assigned to 1 of 3 disease susceptibility categories on the basis of previous studies of coral disease prevalence in the Indo-Pacific region (Willis et al. 2004; Kaczmarsky 2006; Aeby et al. 2011; Lamb & Willis 2011; Ruiz-Moreno et al. 2012): the highly disease susceptible and abundant Acroporidae; the disease susceptible Pocilloporidae and Poritidae; and the disease resistant Agariciidae, Faviidae, Fungiidae, Merulinidae, and Mussidae. Differences in mean prevalence of disease, other signs of compromised health and physical damage among high and low use sites were compared using a 2-factor nested analysis of variance (ANOVA), where site (random factor) was nested within use-level (fixed

factor). Associations between continuous variables were tested with Pearson Product-moment correlations. The occurrence of disease associated with fishing line entanglement and other signs of compromised coral health was examined using a Pearson's chi-square test. Prior to all univariate analyses, assumptions of normality (Shapiro-Wilks) and homogeneity of variance (Levene's test of homogeneity) were tested. Data were transformed to meet assumptions of normality where necessary. Univariate analyses were performed using Statistica 10 (StatSoft, Tulsa, Oklahoma, USA).

3.3 Results

3.3.1 Effects of intensive use on coral health and disease

Assemblages of disease and other compromised health signs differed significantly between use-level (*Pseudo-F = 3.63, P = 0.008*), with the prevalence of apparently healthy corals contributing most strongly to driving this separation (39.5% of variation on the PCO1 axis: Figure 3.2 & Table 3.2). The mean prevalence of healthy corals recorded at high use sites was 45.2% ± 6.2 SE (range = 31% to 63%, n = 5983 corals surveyed), approximately half the mean percentage of healthy corals recorded at low use sites (78.8% ± 2.5 SE, range = 71% to 84%, n = 4516 corals surveyed).

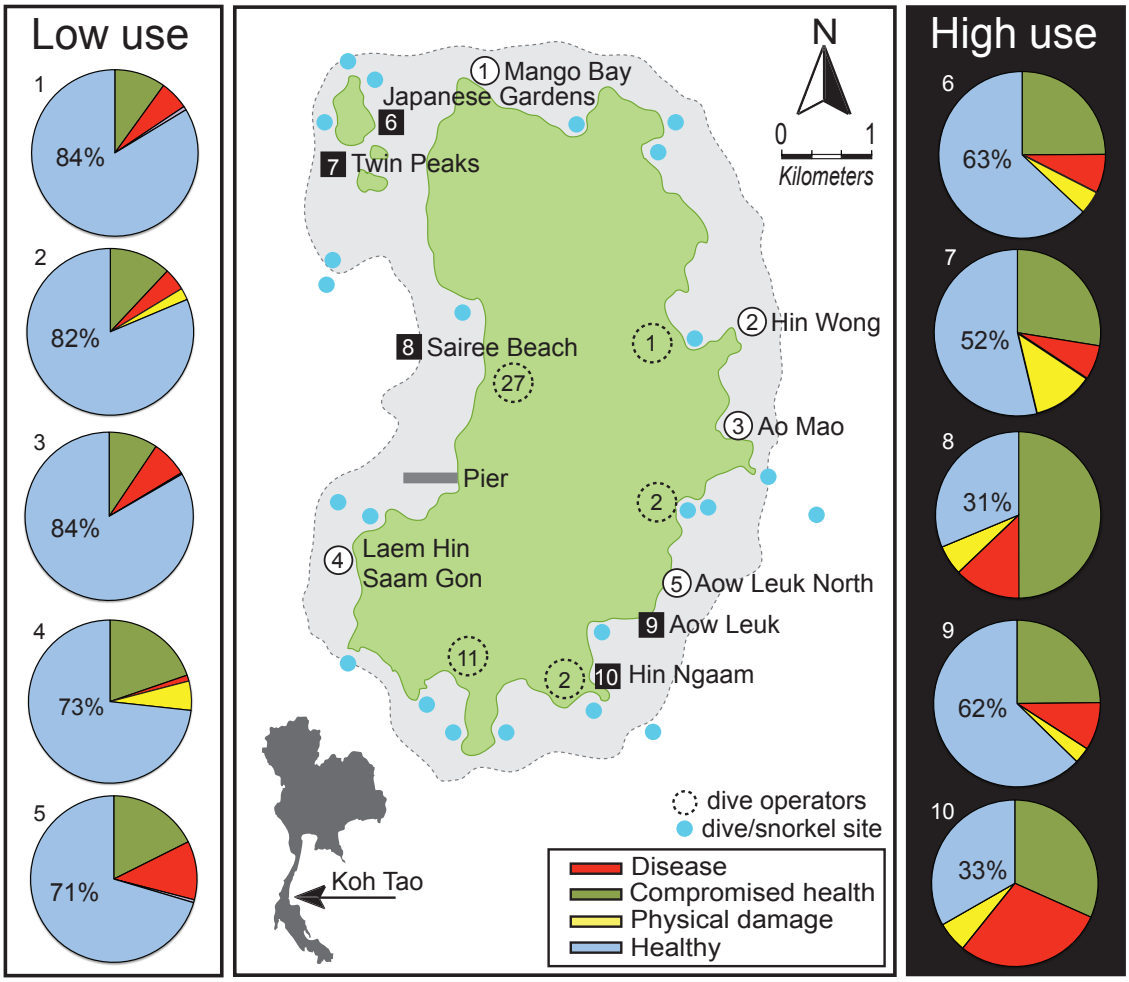

Figure 3.1 Locations of survey sites with low visitor use and without boat moorings (open circles, numbered 1 to 5), and high use with boat moorings for tourism operators (solid squares, numbered 6 to 10) around the island of Koh Tao, Thailand in September 2011. Individual pie charts represent the mean proportion of coral colonies at each site classified within 4 health status categories: disease (including skeletal eroding band disease, white syndromes, black band disease, and growth anomalies); other compromised health indicators (including bleaching, green and red algal overgrowth, sediment necrosis, sponge overgrowth, pigmentation responses and predation scars); physical damage (recently exposed skeleton); or healthy. Category means were calculated from 3 transects per site. Percentages indicated within each pie graph represent healthy colonies.

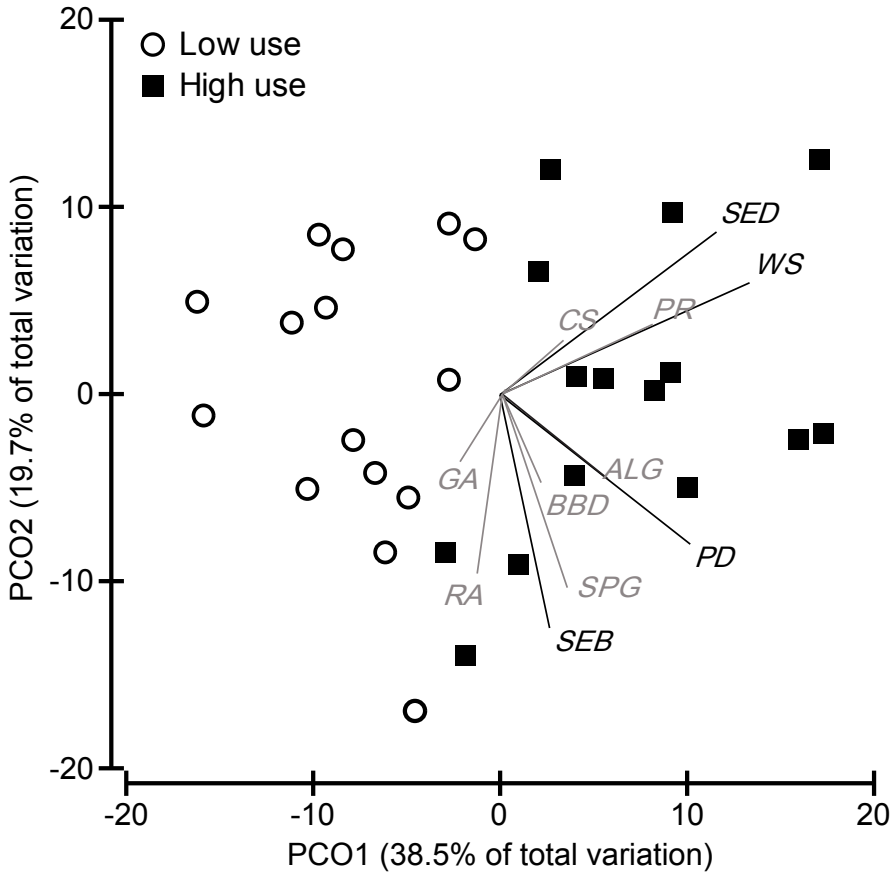

Figure 3.2 Principal coordinates analysis (PCO) of coral health and disease variables. Spatial variation in 4 coral disease and 8 other compromised coral health indicators at the transect level, for high use (solid squares, n = 15) and low use sites (open circles, n = 15) for the first two principal components. Analysis performed on a Bray-Curtis similarity matrix using square root-transformed data, with vectors depicting original variables and Pearson correlation values (grey vectors ≥ 0.2, black vectors ≥ 0.6) representing relative contributions of disease or other compromised coral health signs on the observed variation in use level. Coral diseases: SEB = skeletal eroding band, WS = white syndromes, BBD = black band disease, GA = growth anomalies; Other compromised coral health indicators: PD = physical damage, SED = sediment necrosis, SPG = sponge overgrowth, ALG = algal overgrowth, PR = pigmentation response, RA = red algal overgrowth, CS = cuts and scars from predation, and BL = bleaching.

3.3.1.1 Disease prevalence

Mean overall coral disease prevalence was approximately 3-fold greater at sites with high visitation (mean ± SE = 14.5% ± 4.0; 727 cases of disease) compared to low use sites (5.2% ± 1.3; 197 cases of disease; $F = 40.5$, $P < 0.001$; Table 3.1). At low use sites, disease prevalence ranged between 1.2% and 8.5% (median = 5.6%), whereas it ranged between 6.9% and 29.9% (median = 11.4%) at high use sites. Both the maximum prevalence and maximum number of cases of each of the four diseases were recorded at high use sites (Table 3.1). No cases of brown band disease, a common ciliate disease in the Indo-Pacific (Willis et al. 2004), were recorded during these surveys. The two most prevalent diseases, skeletal eroding band (SEB) and white syndromes (WS), were 2-fold and 4-fold greater, respectively, at high use sites (SEB = 9.0% ± 2.8 and WS = 4.4% ± 1.3) than at low use sites (SEB = 3.8% ± 0.9 and WS = 1.0% ± 0.5: Table 3.1 & Figure 3.3a). Mean black band disease (BBD) prevalence was low at all sites, however it was 9-times greater at high use sites than low use sites (0.8% ± 0.7 and 0.09% ± 0.06, respectively), although it did not differ significantly between use levels (Table 3.1 & Figure 3.3a). There was no difference in the mean prevalence of growth anomalies between the two use levels (0.4% ± 0.2 and 0.3% ± 0.2; Table 3.1 & Fig 3.3a).

3.3.1.2 Prevalence of other signs of compromised health and physical damage

When combined, overall mean prevalence of the 8 other compromised health categories was approximately 2 times greater at high use sites (mean ± SE = 32.3% ± 9.4; 1897 corals with other signs of compromised health) compared to low use sites (15.0% ± 4.1; 752 cases; Table 3.1). Four of these compromised health categories were significantly more prevalent at high use sites (Table 3.1 & Figure 3.3b). Specifically, there was a 12-fold increase in corals with sediment damage (tissue necrosis), and a 9-

fold increase in corals with exposed skeleton (physical damage) at sites with high use (Table 3.1). In addition, approximately 3 times as many corals at high use sites had non-normally pigmented tissue (pigmentation responses) or were actively overgrown by sponges (Table 3.1 & Figure 3.3b). There was no significant difference in the prevalence of bleaching, algal overgrowth or cuts and scars associated with predation between the two use levels (Table 3.1 & Figure 3.3b).

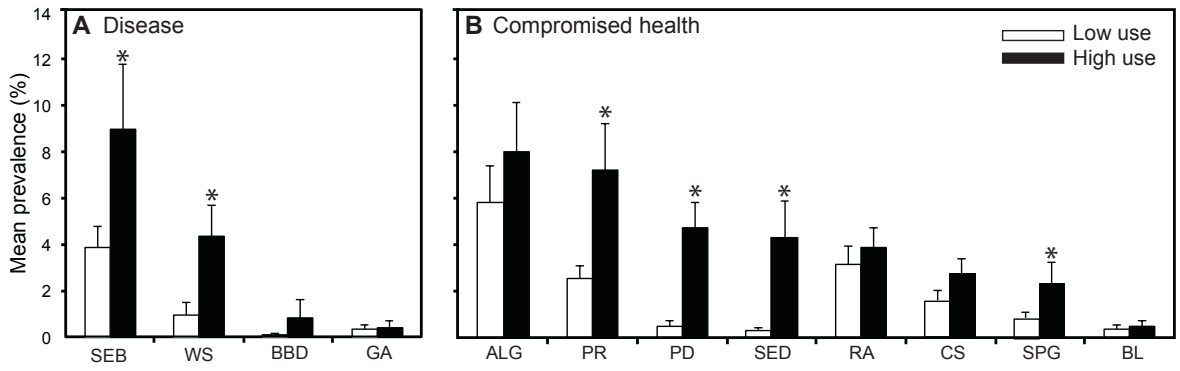

Figure 3.3 Effect of use-level on coral disease and other compromised health indicators. Prevalence (mean ± SE) of (a) coral disease (SEB = skeletal eroding band, WS = white syndromes, BBD = black band disease, GA = growth anomalies) and (b) other compromised coral health signs (ALG = algal overgrowth, PR = pigmentation response, PD = physical damage, SED = sediment necrosis, RA = red algal overgrowth, CS = cuts and scars from predation, SPG = sponge overgrowth, and BL = bleaching) at low use sites (open bars, n = 15 prevalence values; 4516 colonies surveyed) and high use visitor sites (solid bars, n = 15 prevalence values; 5983 colonies surveyed). Analyses performed on data transformed to the square root and asterisks indicate significant differences set at $\alpha = 0.05$ for each individual indicator.

Table 3.1 Mean prevalence[a] and number of cases of coral disease and other signs of compromised health at sites with low levels of recreational use (n = 15 transects, 4516 colonies surveyed) and high levels of recreational use (n =15 transects, 5983 colonies surveyed), and results of a nested analysis of variance (ANOVA) between use-level groups (main effect).

	Low use sites		High use sites		Main effect ANOVA	
Variable	No. of cases	Mean (SE) prevalence (%)	No. of cases	Mean (SE) prevalence (%)	*F*	*P*
Total disease	197	5.2 (1.3)	727	14.5 (4.0)	40.5	< 0.001*
Skeletal eroding band	153	3.8 (0.9)	464	9.0 (2.8)	17.6	< 0.001*
White syndromes	32	0.9 (0.5)	185	4.4 (1.3)	28.8	< 0.001*
Black band	4	0.09 (0.06)	51	0.8 (0.8)	0.3	< 0.57
Growth anomalies	8	0.3 (0.2)	27	0.4 (0.2)	< 0.1	< 0.82
Total other compromised health	752	15.0 (4.1)	1927	32.3 (9.4)	35.3	< 0.001*
Algal overgrowth	324	5.8 (1.5)	416	7.9 (2.1)	0.5	< 0.47
Pigmentation response	107	2.6 (0.5)	337	7.2 (2.0)	11.2	< 0.003*
Physical damage	31	0.5 (0.2)	339	4.7 (1.1)	23.9	< 0.001*
Sediment necrosis	13	0.3 (0.1)	192	4.2 (1.6)	36.9	< 0.001*
Red algal overgrowth	133	3.2 (0.8)	265	3.9 (0.9)	2.3	< 0.14
Sponge overgrowth	46	0.9 (0.2)	216	2.4 (0.8)	10.0	< 0.004*
Predation scars	84	1.4 (0.4)	136	2.6 (0.5)	3.6	< 0.09
Bleaching	14	0.4 (0.2)	26	0.5 (0.3)	< 0.1	< 0.92

[a] Mean prevalence calculated as the percentage of colonies with disease or other signs of compromised health as a percentage of the total number of corals per transect. Analyses performed on data transformed to the square root and asterisks indicate significant differences set at $\alpha = 0.05$.

3.3.1.3 Patterns in the assemblage of diseases and other signs of compromised coral health

Sites with a high prevalence of corals showing other signs of compromised health also had high levels of disease ($r = 0.54$; $P < 0.005$; Figure 3.4a). Patterns in the assemblages of diseases and other compromised health indicators differed among sites within use-level (*Pseudo-F = 4.073*, $P < 0.01$), although differences (19.7% of variation on the PCO2 axis: Figure 3.2 & Table 3.2) were largely driven by the prevalence of skeletal eroding band and physical damage, which were strongly correlated across all sites ($r = 0.78$, $P < 0.001$; Figure 3.4b), and the prevalence of white syndrome and sediment necrosis, which were also strongly correlated across all sites ($r = 0.67$, $P < 0.001$; Figure 3.4c). Coral colonies with recently exposed skeleton were more likely to also have skeletal eroding band disease (22%) than colonies without recent physical damage (6%) at high use sites ($\chi^2_1 = 136.1$, $P < 0.001$), but at low use sites, recent physical damage did not affect the susceptibility of corals to SEB (7% compared to 5%: $\chi^2_1 = 0.45$, $P = 0.51$). Colonies with tissue necrosis associated with sediment were also more likely to have white syndrome lesions than colonies without sediment damage at both high (26% compared to 3%: $\chi^2_1 = 256.0$, $P < 0.001$) and low use sites (31% compared to 4%: $\chi^2_1 = 28.2$, $P < 0.001$).

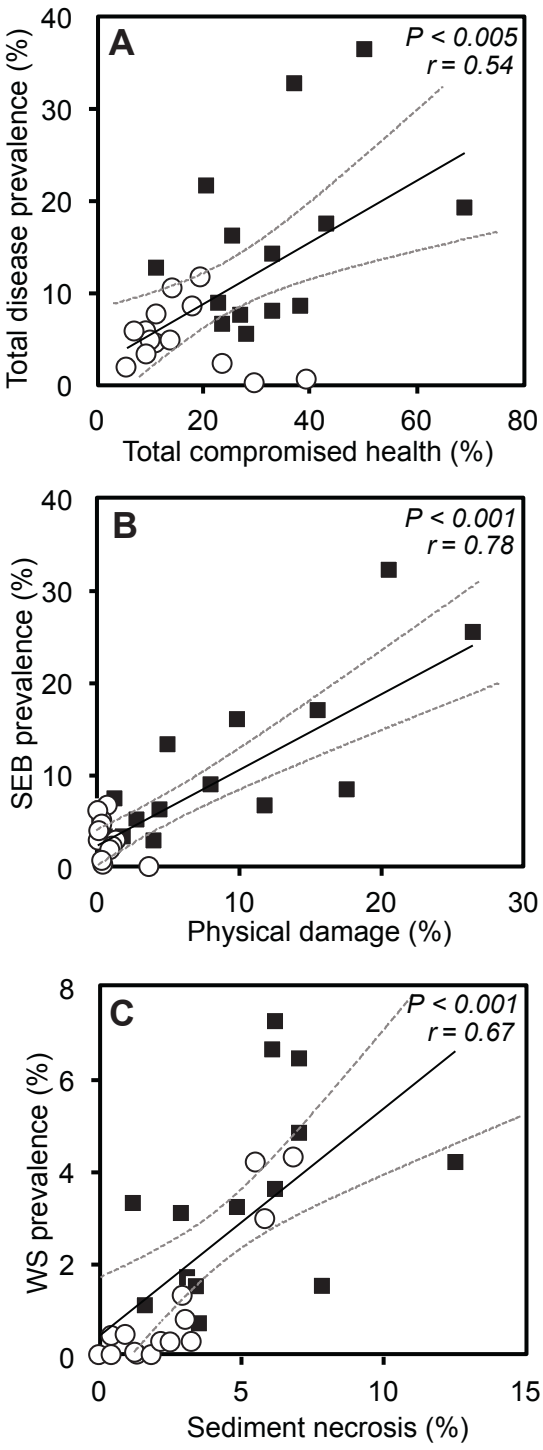

Figure 3.4 Associations between the prevalence of (a) total coral disease and other signs of compromised coral health, (b) recent physical damage and skeletal eroding band (SEB) disease, and (c) tissue necrosis due to sediment and white syndromes (WS). Open circles indicate low use sites (n = 15) and black squares indicate high use sites (n = 15) in each panel. Pearson product-moment correlations conducted on transects pooled from low and high use sites. Dashed lines represent 95% confidence intervals.

Table 3.2 Eigenvalues and cumulative variation of each individual axis of a principal coordinates analysis (PCO) performed on a Bray-Curtis similarity matrix using square root-transformed prevalence values (n = 30) of 4 coral disease and 8 other compromised coral health indicators (see Table 3.1).

Axis	Eigenvalue	Individual %	Cumulative %
1	5350.10	38.49	38.49
2	2738.70	19.71	58.20
3	2139.90	15.40	73.60
4	1599.90	11.51	85.11
5	918.60	6.61	91.72
6	667.64	4.80	96.52
7	504.73	3.63	100.15

3.3.2 Impact of derelict fishing line on coral disease susceptibility

Discarded monofilament fishing line was observed along 4 transects at high use sites but not at low use sites. Of the 113 surviving corals entangled in derelict fishing line (Figure 3.5a-b), approximately 87% also had the ciliate-associated disease, skeletal eroding band (SEB), initiating from the lesion boundary (Figure 3.5c-e). As a result, corals entangled in derelict fishing line were 5 times more likely to have SEB than corals without derelict fishing line (837 colonies without fishing line or other physical injury, n = 4 transects: $\chi^2_1 = 190.4$, $P < 0.001$; Figure 3.5e). Excluding injury or abrasion from fishing line, transects with discarded fishing line at high use sites had an approximately 3-fold greater mean prevalence of corals with physical injury (mean ± SE = 13.7% ± 5.8) compared to transects without fishing line (4.6% ± 1.4, n = 12). No other diseases were observed on corals with fishing line, however other diseases were recorded on corals without fishing line along these transects (mean prevalence = 6.5% ± 0.9; Figure 3.5e). Corals with branching growth forms were the most susceptible to fishing line entanglement (55%), followed by tabular (27%), massive (16%) and encrusting (2%) growth forms.

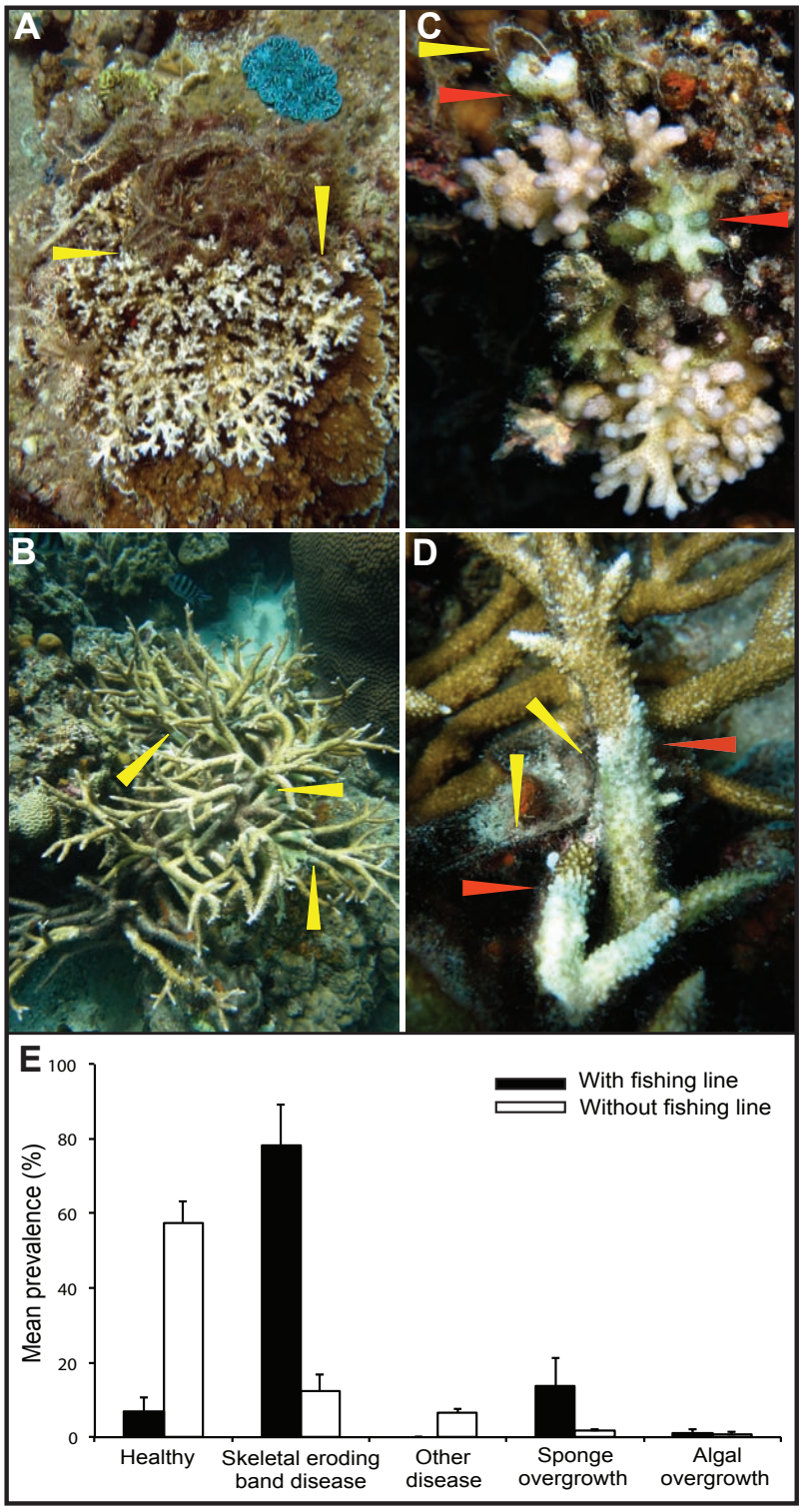

Figure 3.5 Impact of derelict fishing line on coral susceptibility to disease. Field photographs of derelict fishing line (yellow arrows) entangled on the branching corals (a) *Pocillopora damicornis* and (b) *Acropora* spp. and the subsequent tissue loss margins (red arrows) due to the ciliate-associated disease skeletal eroding band (c,d). The resulting health status (e) of coral colonies (mean prevalence ± SE; n = 4 transects) entangled in derelict fishing line (n = 113 colonies) and without fishing line (n = 837 colonies).

3.3.3 Host density, cover and composition as potential drivers of disease

Mean coral density (number of colonies per m^2) did not vary significantly between low (mean ± SE = 10.0/m^2 ± 1.1) and high use sites (13.3/m^2 ± 1.9; *F = 0.8, P = 0.40*). Moreover, the number of disease cases (see Table 3.1) was not associated with coral density at sites with low (*r = 0.30, P = 0.28*) or high recreational use (*r = 0.13, P = 0.63*).

The composition of coral assemblages was at least 60% similar among all transects surveyed in this study (Bray-Curtis similarity), and did not differ significantly between high and low use sites (*Global R = 0.11, P = 0.18;* Figure 3.6a). Community composition around Koh Tao was predominantly comprised of 3 families (Figure 3.6b). On average, corals in the disease resistant families (Agariciidae, Faviidae, Fungiidae, Merulinidae, and Mussidae) accounted for the largest percentage of coral cover at both low (mean ± SE = 42.1% ± 4.8) and high use recreational sites (33.9% ± 3.6; *F =0.5, P = 0.51*; Figure 3.7a). The two disease susceptible families (Pocilloporidae and Poritidae) represented the second most abundant coral cover group at both low and high use sites (12.5% ± 5.5 and 17.9% ± 7.8, respectively; *F = 0.7, P = 0.41*), while the family Acroporidae contributed to the lowest percentage of mean coral cover at both low (2.1% ± 1.4) and high use reef sites (4.2% ± 2.0; *F =1.7, P = 0.22*; Figure 3.7a).

Overall, total coral disease prevalence was not associated with total percent cover of all coral families combined at high use sites (*r = 0.45, P = 0.10*), however there was a significant positive correlation between total disease prevalence and total coral cover at low use sites (*r = 0.54, P = 0.04*; Figure 3.7b). Although percent cover of Acroporidae and the disease susceptible families was marginally higher at high use sites, disease prevalence was not correlated with cover of acroporid corals nor disease susceptible corals at low (*r = 0.12, P = 0.68* and *r = 0.11, P = 0.69*, respectively) or high use sites (*r = 0.09, P = 0.76* and *r = 0.14, P = 0.62*, respectively; Figure 4.3c-d).

Benthic cover of disease resistant coral families was not associated with coral disease prevalence at low (*r = 0.32, P = 0.24*) or high use recreational sites (*r = 0.04, P = 0.89*; Figure 3.7e).

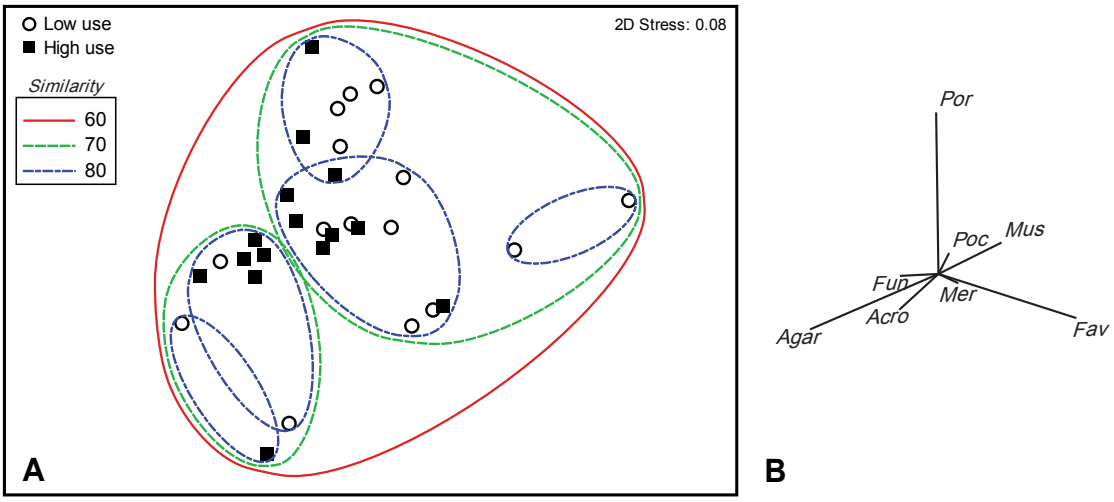

Figure 3.6 Non-metric multidimensional scaling analysis of coral community composition. (a) Spatial variation in the taxonomic composition of percent coral cover by family at the transect level (high use sites = solid squares, low use sites = open circles), assessed using a non-metric multidimensional scaling (nMDS) plot and hierarchical clusters overlaid from dendrograms based on a Bray-Curtis similarity matrix on square root-transformed data. Distances between transects signifies similarity of coral community composition and the similarity scale on clusters indicates the percentage of similarity between transects (range = 0 to 100). (b) The relative contribution (a proxy of vector length) of all 8 hard coral families to the observed variation at sites (Acro = Acroporidae, Agar = Agariciidae, Fav = Faviidae, Fun = Fungiidae, Mer = Merulinidae, Mus = Mussidae, Por = Poritidae, and Poc = Pocilloporidae).

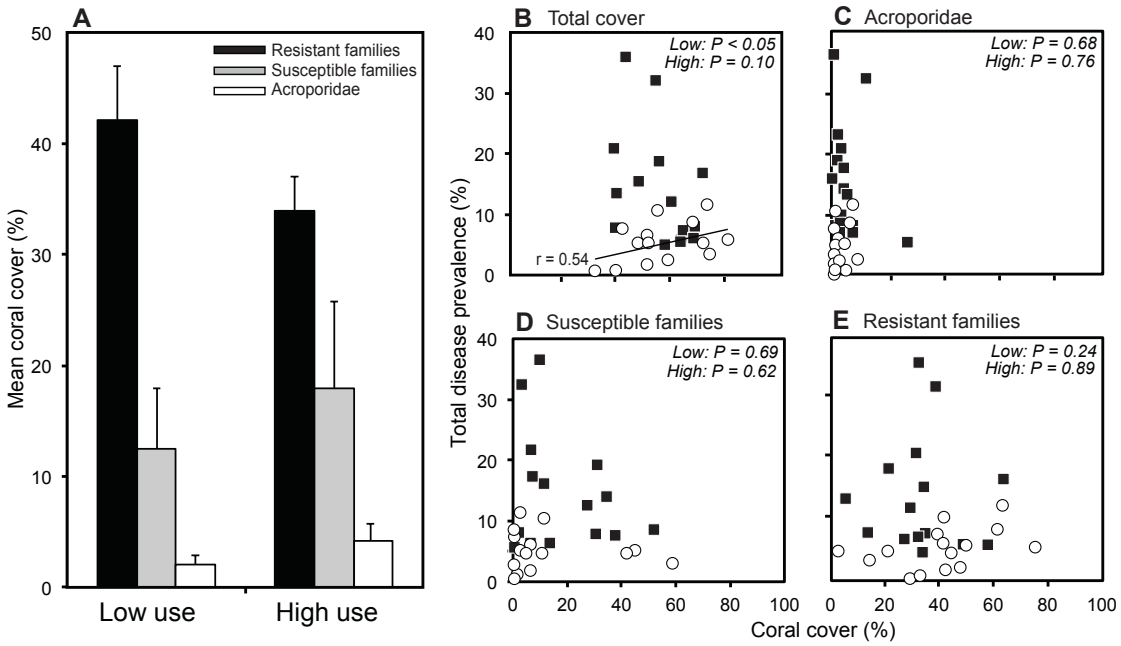

Figure 3.7 Taxonomic patterns of (a) mean coral cover (mean ± SE) between sites with low visitor use (Low: n = 15 transects) and high visitor use (High: n = 15 transects) and associations between prevalence of overall coral disease and percent coral cover of (b) total coral cover, (c) Acroporidae, (d) susceptible coral families, and (e) resistant coral families at sites with low use (open circles) and high use (black squares) recreational activities. Disease resistant families: Agariciidae, Faviidae, Fungiidae, Merulinidae, and Mussidae; disease susceptible families: Pocilloporidae and Poritidae; and the highly disease susceptible family Acroporidae.

3.4 Discussion

This study reveals that intensive site use associated with reef-based tourist activities significantly reduces the overall health of corals, undermining the value of the resource necessary for sustaining the growing nature-based tourism industry. Consistency in the pattern of substantially elevated levels of disease at high use sites highlights the urgent need to identify and mitigate potential causes of increased disease prevalence at these sites, particularly as additional impacts are anticipated with accelerated development of infrastructure along coastal regions to support tourism growth. Differences in coral cover, density or family composition are unlikely to have caused the striking differences in disease prevalence among sites, given that percent

cover of all corals and of disease-susceptible families did not differ among high and low use sites.

Similarities in coral cover, density and composition among sites that clearly differed in a range of coral health indicators contribute to the emerging consensus that percent cover of live coral is of limited value as an indicator of ecosystem health, typically failing to separate areas affected from those unaffected by human activities (Muthiga & McClanahan 1997; Hawkins et al. 1999; Dinsdale & Harriott 2003). I conclude that percent cover is not appropriate as the sole indicator of impacts when assessing reef-based activities, but is useful when used in conjunction with other indicators. I note, however, that in the group of low use site, disease prevalence was positively correlated with total cover, potentially reflecting transmission of pathogens via direct colony-to-colony contact (Riegl 2002). At high use sites, it is more likely that increased susceptibility to infection by microbial communities associated with localised environmental stressors led to higher prevalence of coral disease (cf. Ritchie 2006). It is also likely that a recent bleaching event had already caused extensive mortality of bleaching- and disease-susceptible families, as supported by the high prevalence of algal overgrowth (6-8%) and low prevalence of both bleaching (~0.5%) and cover of acroporid corals (2-4%) at all ten survey sites.

New approaches to identifying and managing stressors affecting coral health

Linking indicators of stress with potential causes, so that action can be initiated before irreversible declines in health occur, has been challenging for corals. Bleaching is one of the few readily identifiable signs of coral stress, but bleaching has been associated with a wide range of stressors, like changes in water temperature and light (Brown 1997), ocean acidification (Hoegh-Guldberg et al. 2007), bacterial infections (Kushmaro et a. 1997), herbicides (Jones et al. 2003), and sunscreen (Danovaro et al.

2008). This study of coral health impacts associated with the intensity of diving-related activities provides valuable insights for linking a range of compromised health indicators with potential stressors, and highlights the need for multiple metrics of coral health and disease to deduce sources of stress on coral reefs and aid in developing practical management strategies for mitigating them.

Tissue necrosis and white syndromes as indicators of sediment stress

The twelve-fold greater prevalence of sediment-associated tissue necrosis at high use sites represented one of the greatest differences in coral health indicators between sites exposed to high versus low intensity recreational diving. Recreational divers significantly increased turbidity and resuspended sediment at popular dive sites in the Red Sea, each causing approximately nine sediment clouds to settle back onto corals per dive (Zakai & Chadwick-Furman 2002). In addition, wakes generated by boat traffic can redistribute and increase turbidity from sediment resuspension and shoreline erosion, with turbidity taking between 4 and 24 hours to return to background levels following disturbance (Yousef et al. 1980; Jones 2011). Although corals possess mechanisms to actively remove sediment particles, such mechanisms are energetically costly (Hubbard & Pocock 1972; Rogers 1990; Philipp & Fabricius 2003), thus corals at intensively used sites suffer depleted energy budgets from even low levels of chronic sediment deposition (Rogers 1990; Philipp & Fabricius 2003), leading to localised bleaching and tissue necrosis.

The high correlation found between the prevalence of sediment-associated tissue necrosis and the prevalence of white syndromes, regardless of site use intensity, signifies that localised direct contact with sediment may be a primary factor contributing to this disease. Sediment could act as both a disease reservoir and potentially a vector when resuspended as a result of tourist-related activities, and could

also increase the likelihood of infection by stressing coral hosts (Lafferty & Holt 2003). On hurricane-damaged reefs, Brandt et al. (2013) reported that another tissue loss disease, white plague, occurred primarily on fragments in direct contact with sediment, and hypothesised a link with bacterial overgrowth. Evidence that sediment damage to corals is reduced following treatment with antibiotics (Hodgson 1990), and that growth rates of coral-associated microbes increased ten-fold and led to rapid tissue loss following exposure to elevated levels of carbon (Kline et al. 2006), further support this link. Whether sediment accumulation causes coral disease by introducing pathogens or is a general sign of coral stress to other environmental stressors warrants further study. Practical and readily-introduced solutions for reducing sedimentation include limiting boat traffic and site crowding, and the establishment of no-wake zones and speed limits when traveling within close proximity to reefs.

Black band disease prevalence as an indicator of sediment and nutrient stress

Although overall levels of black band disease (BBD) were low and not significantly different between high and low use sites, the nine-fold increase of BBD at high use sites further corroborative evidence that sediment accumulation plays a key role in diving-related disturbances. The biogeochemical microenvironment beneath BBD microbial mats, which represent complex and diverse polymicrobial consortia (Sutherland et al. 2004; Kaczmarsky 2006; Sato et al. 2010), is characterized by anoxia, high sulfide concentrations and low pH, conditions that are lethal to underlying coral tissues (Glas et al. 2012). These toxic conditions are most pronounced under low light conditions (Glas et al. 2012), therefore sediment accumulation on coral surfaces could provide an anaerobic microenvironment conducive to microbial mat formation, while increased turbidity (and associated decreased light levels) could facilitate the rapid establishment of conditions characteristic of the disease. Furthermore, enhanced BBD

progression rates when nutrients are enriched experimentally (Voss & Richardson 2006), suggests that increased nutrient availability in turbid waters may contribute to the trend for higher BBD prevalence at high use sites.

Sponge overgrowth and other indicators of nutrient enrichment

The marked increase in sponge overgrowth at high use sites further suggests that nutrient enrichment is a significant issue associated with intensive tourist use. Increased primary production associated with nutrient enrichment and sediment favours benthic filter-feeding organisms, particularly sponges, which then typically outcompete corals (Pastorok and Bilyard 1985). On Grand Cayman, a five-fold increase in the biomass of *Cliona delitrix*, a sponge overgrowing the coral *Montastrea cavernosa*, and a six-fold increase in bacterial biomass was recorded on fringing reefs exposed to discharges of untreated fecal sewage compared to a control site 1 km away (Rose and Risk 1985). In other studies, widespread overgrowth of corals by the cyanobacteriosponge *Terpios hoshinota* on Japanese reefs was particularly noteworthy in pollution-stressed zones (Rutzler and Muzik, 1993), and bacteria similar to those detected in black band disease were detected on sponge-covered but not on sponge-free corals (Tang et al. 2011), suggesting that *T. hoshinota* might benefit from the presence of bacteria associated with unhealthy corals.

Inputs of nutrients, pathogens, and other wastewater-derived pollution (Camargo and Alonso, 2006) have also been linked to several other coral diseases (Bruno et al., 2003). For example, sewage outfalls containing the human gut microbe *Serratia marscens* have been associated with a type of white syndrome infecting and decimating acroporid corals off the coast of Florida (Patterson et al. 2002). Nutrient enrichment from sewage and wastewater pollution is one of the few stressors that, with proper research, policy, and management, can be effectively mitigated. Fecal indicator bacteria,

such as *Enterococcus*, can be monitored (Gronewold et al. 2008) or alternatively, stable isotope analysis can detect the presence of sewage-derived nitrogen within an ecosystem. In Mexico, $\delta^{15}N$ values of the common sea fan were more variable near a developed tourist site than at an undeveloped site, with 84% of the observed variation explained by tourist visitations in the preceding year (Baker et al. 2013). Due to the unregulated and rapid expansion of dive tourism in many developing countries, most tourist vessels are not equipped with proper storage systems for wastewater and sewage. Tertiary treatment systems on fitted to vessels can remove up to 90% of nutrients (Judd 2010). Because pollutants cannot be isolated in open marine systems and may have implications beyond local coral assemblages (McCallum et al. 2004), the possibility of disease dispersing from sites with higher levels of environmental stress is concerning.

Skeletal eroding band disease as an indicator of physical injury

The nine-fold increase in the prevalence of recent coral damage at high use sites suggests that physical injury and lacerations from direct diver contact play an important role in increased disease prevalence at these sites. Moreover, corals with physical injury were four times more susceptible to skeletal eroding band disease compared to colonies without injury at high use sites. Ongoing chronic injuries could reduce immune function associated with the regeneration of coral tissue, resulting in increased susceptibility to disease (Mydlarz et al. 2006). In experimental studies, artificially-inflicted wounds enhanced the ability of ciliates associated with skeletal eroding band disease to form dense band-like aggregations that caused tissue loss of up to 0.3 cm day^{-1} on the GBR (Page & Willis 2008). Increased presence of this ciliate disease has been documented near other tourist locations (Winkler et al. 2004; Lamb & Willis 2011), however this study is the first to demonstrate a strong link between the prevalence of physical injury and the presence of skeletal eroding band disease. Repair of broken tips takes up to two

months (Kobayashi 1984), therefore physical injury may provide a primary site for the invasion of pathogens and ciliates or reduce immune system function, extending the impact timeframe well beyond the immediate time of injury.

Additional microbial or environmental factors at high use sites may be necessary for the development of the band-like ciliate aggregations that cause tissue loss characteristic of skeletal eroding band disease. In contrast to high use sites, injury did not appear to affect the likelihood of skeletal eroding band infections at low use sites. While mean levels of damage found in this study were two times higher than on frequently dived reefs of Saba and Bonaire in the Caribbean (Hawkins et al. 1999; Hawkins et al. 2005), they were markedly lower than on the more heavily dived reefs of Egypt and Israel, where approximately 10% of colonies were broken (Riegl & Velimirov 1991; Hawkins & Roberts 1992). Significant increases in loose fragments of coral at heavily dived sites (Hawkins & Roberts 1993) raises the possibility of colony-to-colony pathogen transmission if fragments are already infected (Brandt et al. 2013). Whilst marine-based tourist activities do not represent disease agents themselves, they nevertheless appear to cause lesions that compromise the health of corals.

Pigmentation response as a general indicator of compromised health

Non-normal pigmentation of coral tissue, or pigmentation response, has been characterised as a general immune response to a physical or pathogenic challenge (Willis et al. 2004; Bongiorni & Rinkevich 2005; Palmer et al. 2008). Pigmented tissues possess high levels of melanin, an important component of invertebrate innate immunity that can act as a defensive barrier against foreign bodies (Palmer et al. 2008), therefore the elevated prevalence of pigmented tissue recorded at high use sites may represent signs of a general immune response to a multitude of factors, including invading foreign

pathogens, physical injury or sediment accumulation, but analyses of further indicators are required to deduce the source(s) of stress.

Impacts of derelict fishing line on coral disease susceptibility

My surveys unexpectedly revealed that discarded or entangled fishing line significantly increases the susceptibility of corals to disease, a previously unreported mechanism of coral mortality associated with fishing gear. Fishing line has been shown to cause considerable coral mortality on coastal reefs, although the mechanism was unclear (Bavestrello et al. 1997; Yoshikawa & Asoh 2004). These results suggest that fishing line entangled on corals may continue to act as a substrate from which opportunistic ciliates, algae, sponges and pathogens directly invade wounded tissue or immunocompromised corals. In addition to direct effects of fishing line on coral disease susceptibility, transects with fishing line also had three times the amount of physical injury compared to transects at other high use sites, potentially representing further damage inflicted as a result of fishing itself.

Fishing gear can lead to major direct and indirect shifts in community structure, affecting corals, fishes and reef communities as a whole (Russ & Alcala 1989). Branching and tabular corals represented 82% of colonies affected by disease as a result of fishing line entanglement, potentially reducing vital habitat complexity for coral reef-associated fishes. The complex morphologies of many branching corals offer potential shelters from larger predatory fishes (Wilson 2008), while tabular corals are used significantly more by large reef fishes than other morphologies (Kerry & Bellwood 2012). Tabular corals also attract settling reef fish by providing shade (Hair et al. 1994). Consequently, the loss of reef habitat complexity associated with fishing gear also reduces fish species diversity (Roberts 1995). In the Philippines, Raymundo et al. (2009) found that fish taxonomic diversity may ameliorate coral disease prevalence

inside marine reserves. Therefore, both the extraction of fish and type of gear used for extraction could directly and indirectly influence the dynamics of diseases affecting corals.

In this study, fishing line is associated with increased susceptibility to disease, however line fishing may still be a preferred method for extracting fish on coral reefs. In artisanal fisheries, hand lines and monofilament fishing gear have been estimated to cause the least amount of coral contact per average kilogram of catch weight, whereas spear fishing captures the highest proportion of fish but causes the most coral damage per kilogram (Mangi et al. 2007). Also, line fishing potentially catches the lowest proportion of fish species belonging to key functional groups vulnerable to the effects of climate-induced coral bleaching (Cinner et al. 2009) and potentially to disease, given that most highly disease-susceptible coral families are also more susceptible to bleaching (Willis et al. 2004). Hawkins et al. (1997) reported that intensive recreational diving had no adverse effects on fish communities themselves, noting that the differences detected among dived and reserve sites were attributable mainly to habitat differences, such as the generally higher coral cover and greater structural complexity in reserves than in dived sites. Due to the potentially synergistic impacts of intensive dive site use and fishing, restricting fishing around heavily visited sites or the implementation of spatially-explicit zones for these activities is recommended.

Using multiple metrics of coral health may be a more suitable indicator for selecting appropriate management strategies and assessing their success and failure on reefs facing increasing levels of human and disturbance. When site access is unrestricted, individual users have little or no incentive to conserve it (Davis & Tisdell 1995), therefore alternative and practical management options that have greater potential for compliance in developing tropical countries are urgently required. The economic value of coral reef tourism for developing coastal communities highlights the

importance of improved management practices for conserving the coral reef resource underpinning the industry. Educating and involving local communities in sustainable practices that provide long-term revenues can decrease over-exploitation for short-term profits.

CHAPTER 4.

Sediment and turbidity associated with offshore dredging increase coral disease prevalence on nearby reefs[†]

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4.1 Introduction

Reduced water quality caused by explosive human population growth is often cited as an important factor driving coral disease epizootics (Hayes et al. 2001; Harvell et al. 2002; Bruno et al. 2003). Land clearing exposes 1% of the Earth's surface to eroding processes annually and urbanisation of coastal areas is expanding disproportionally to population growth (Fabricuius 2006; UNEP/GPA 2006). Consequently, coastal coral reefs, like many other marine ecosystems, are increasingly subjected to elevated levels of eutrophication, sedimentation and turbidity, factors proposed to compromise disease resistance of corals and/or increase pathogen virulence (Harvell et al. 2007). Coastal dredging for land reclamation, beach nourishment and port construction further exacerbates terrestrial nutrient and sediment influx by resuspending benthic sediments (PIANC 2010). The most conspicuous impact of dredging on coral reef communities is through the direct damage, removal and/or burial of reef habitat within or immediately adjacent to the dredge footprint, though the influence of dredging activities typically extends well beyond the immediate dredge area (Kutser et al. 2007; Erftemeijer et al. 2012). Field studies have demonstrated local scale degradation of coral reef ecosystems following prolonged dredging activities, which are thought to result mainly from increased turbidity and sedimentation caused by the resuspension of benthic sediments (Newcombe & Macdonald 1991; McArthur et al. 2003). Additionally, more frequent and intense storms associated with climate change amplify

water quality declines by promoting coastal runoff and sediment resuspension (Nearing et al. 2005).

Sedimentation and turbidity, associated with both weather events and anthropogenic activities, are also frequently proposed to contribute to increased coral disease prevalence (Harvell et al. 2007), although empirical evidence is lacking. Hodgson (1990) suggested sedimentation as a potential mechanism for the transmission of coral pathogens from marine or terrestrial substrates onto nearby corals. Silt-associated bacteria were identified as a possible cause of necrosis in sediment-damaged corals, since antibiotic-treated water reduced tissue damage in experimentally silted corals. In field-based observations, Haapklyä et al. (2011) noted a correlation between seasonal coastal runoff, including increased sedimentation and turbidity, and the prevalence of coral disease on inshore reefs. Elevated turbidity reduces the amount and quality of ambient light available for photosynthesis by the corals' endosymbiotic algae (*Symbiodinium*) and excess sedimentation inhibits the heterotrophic feeding efficiency of corals, reducing the energy intake of both symbiotic and asymbiotic corals (Falkowski et al. 1990). While corals are able to shed some sediment through mucus production and ciliary action, these mechanisms are energetically expensive and further burden the corals' already reduced energy budgets (Peters & Pilson 1985; Riegl & Branch 1995). Despite a wealth of circumstantial evidence indicating sedimentation and turbidity as potential coral disease drivers, no studies have directly linked sedimentation, turbidity and coral disease in the field. Given that 25% of coral reefs are threatened by rapid coastal development to accommodate expanding urban activities (Burke et al. 2011), effective coastal management will increasingly depend upon a comprehensive understanding of the impacts of sediment, turbidity and associated water quality decline, on all aspects of coral reef health.

Here, I describe the first *in situ* test of the hypothesis that elevated sedimentation and turbidity increase coral disease prevalence on reefs. I performed detailed coral health assessments along a gradient of exposure to a sediment-laden dredge plume within the Montebello and Barrow Islands off the northern coast of Western Australia. The otherwise pristine conditions of these offshore reefs enabled an empirical examination of the relationship between sedimentation, turbidity and coral disease prevalence in the absence of other confounding influences.

4.2 Methods

4.2.1 Study site

Montebello and Barrow Islands are situated in the Pilbara region of Northwest Australia, approximately 1,600 km north of Perth (Figure 4.1). The Montebello and Barrow Islands Marine Protected Areas (MBIMPA), incorporating the Montebello Islands Marine Park, Barrow Island Marine Park and the Barrow Island Marine Management Area, were gazetted in 2004. The environment within the MBIMPA is considered to be relatively pristine as a consequence of low human usage, minimal terrestrial influence and strict management controls on industrial developments in the area (Western Australia Department of Environment and Conservation 2007).

The Gorgon Project (GP), based on Barrow Island (20.80°S, 115.40°E), is one of the world's largest natural gas projects and the largest single resource natural gas project in Australia's history. The GP dredging program involved the removal and dumping of approximately 7.6 million tons of marine sediment over an 18-month period from 19 May 2010 to 7 November 2011.

4.2.2 Data collection

4.2.2.1 Satellite-derived assessment of dredge plume extent The area affected by the dredging-induced sediment plume area was quantified daily over the duration of the dredging program using Moderate Resolution Imaging Spectroradiometer (MODIS) satellite imagery, as described by Evans et al. (2012). Briefly, the sediment plume boundary was interpreted manually using one of two MODIS images captured daily. A 'hotspot' analysis was performed on the cumulative daily plume boundaries to provide a dataset describing the number of days the sediment plume was present at any position within the waters surrounding the Montebello and Barrow Islands. These data were used to determine sediment plume exposure days, which are defined here as the cumulative number of days a suspended sediment plume was visible in satellite images at a given location throughout the duration of dredging operations. One year of pre-dredging MODIS imagery was also analysed to identify a baseline for naturally occurring turbidity events.

4.2.1.2 Coral health and community composition surveys Coral health surveys were conducted in December 2011, one month after the completion of the 18-month GP dredging program. Eleven sites were selected, extending both north and south from the dredging site, representing a gradient of sediment plume exposure (Figure 4.1). At each site, three 15 m × 2 m belt transects, placed haphazardly at least 5 m apart, were surveyed along depth contours at 2 to 6 m, consistent with standardised protocols developed by the Global Environment Facility and World Bank Coral Disease Working Group (Beeden et al. 2008). Within each 30 m^2 belt transect, every scleractinian coral colony over 5 cm in diameter was identified to genus-level and classified as either diseased [i.e., affected by one or more of the following diseases classes: white

syndromes, brown band disease, skeletal eroding band, black band disease, and/or growth anomalies]; showing other signs of compromised health (i.e., tissue necrosis associated with sediment accummulations, bleaching, pigmentation response; and/or sponge, red algae, or green algae overgrowth); or healthy (i.e., no visible signs of disease lesions or other indicators of compromised health) using indicators described by Willis et al. (2004). Additionally, signs of coral predation by *Drupella* spp. and/or *Acanthaster planci* (crown-of-thorns seastar; COTS) were recorded. Standard line-intercept surveys were used to determine coral cover and coral community composition by estimating the linear extent of each coral to the nearest centimeter along the central line of each 15 m transect. These protocols allow the data collected in this study to be directly compared to other similar standardised coral disease datasets worldwide.

4.2.2.3 Assessment of temperature-based risk of disease To evaluate the role that thermal stress might have played in shaping the spatial patterns of coral disease and other signs of compromised coral health observed, temperature-based predictors of disease outbreak risk based on published empirical relationships between temperature metrics, coral cover and disease abundance were analysed (summarised in Heron et al. 2012). While temperature-disease relationships analysed in this study were derived for only one disease type (white syndromes) affecting one coral genus (*Acropora* spp.) on the Great Barrier Reef, these thermal stress metrics provide useful indicators of host susceptibility and potentially of pathogen loads (Heron et al. 2012). Briefly, retrospective satellite sea-surface temperature (SST) time-series for the period 1985 to 2009 were concatenated with NOAA's near real-time 11 km SST time-series (February 2009 to December 2011). The resulting dataset provided a SST time-series for each survey location throughout the dredging period (May 2010 to November 2011) and an

internally consistent climatological baseline for the calculation of thermal stress metrics. Five temperature-based stress metrics associated with disease likelihood were derived (see Table 4.1 for definitions): Hot Snap, Cold Snap and Winter Conditions (see Heron et al. 2010 for full details); and mean positive summer anomaly (MPSA) and predicted abundance (see Maynard et al. 2011 in Appendix 2). Predicted abundance of disease cases per 1,500 m^2 ($A_{disease}$) was calculated using MPSA and total hard coral cover for all species from the field surveys, following the model of Maynard et al. (2011):

$$A_{\text{disease}} = MPSA^{a}\ TCC^{b} \qquad (1)$$

where a = 1.07 and b = 1.59 (from Maynard et al. 2011). All temperature-based metrics were assessed at the site level (i.e., no replication at the transect level) due to the limited resolution of satellite-derived SST data.

4.2.3 Data analyses

Prevalence values for coral diseases and other signs of compromised health were calculated within each 30 m^2 belt transect by dividing the number of colonies with signs of 5 diseases or of 6 other indicators of compromised health by the total number of colonies present. To assess the effect of dredging on disease prevalence and on other indicators of compromised health along the plume gradient, sites were assigned to one of three exposure categories based on the number of days a dredging-associated suspended sediment plume was visible in MODIS satellite images (Figure 4.1):

- Low exposure (0-9 sediment plume exposure days; 18 transects),
- Moderate exposure (40-78 sediment plume exposure days; 9 transects) and
- High exposure (296-347 sediment plume exposure days; 6 transects).

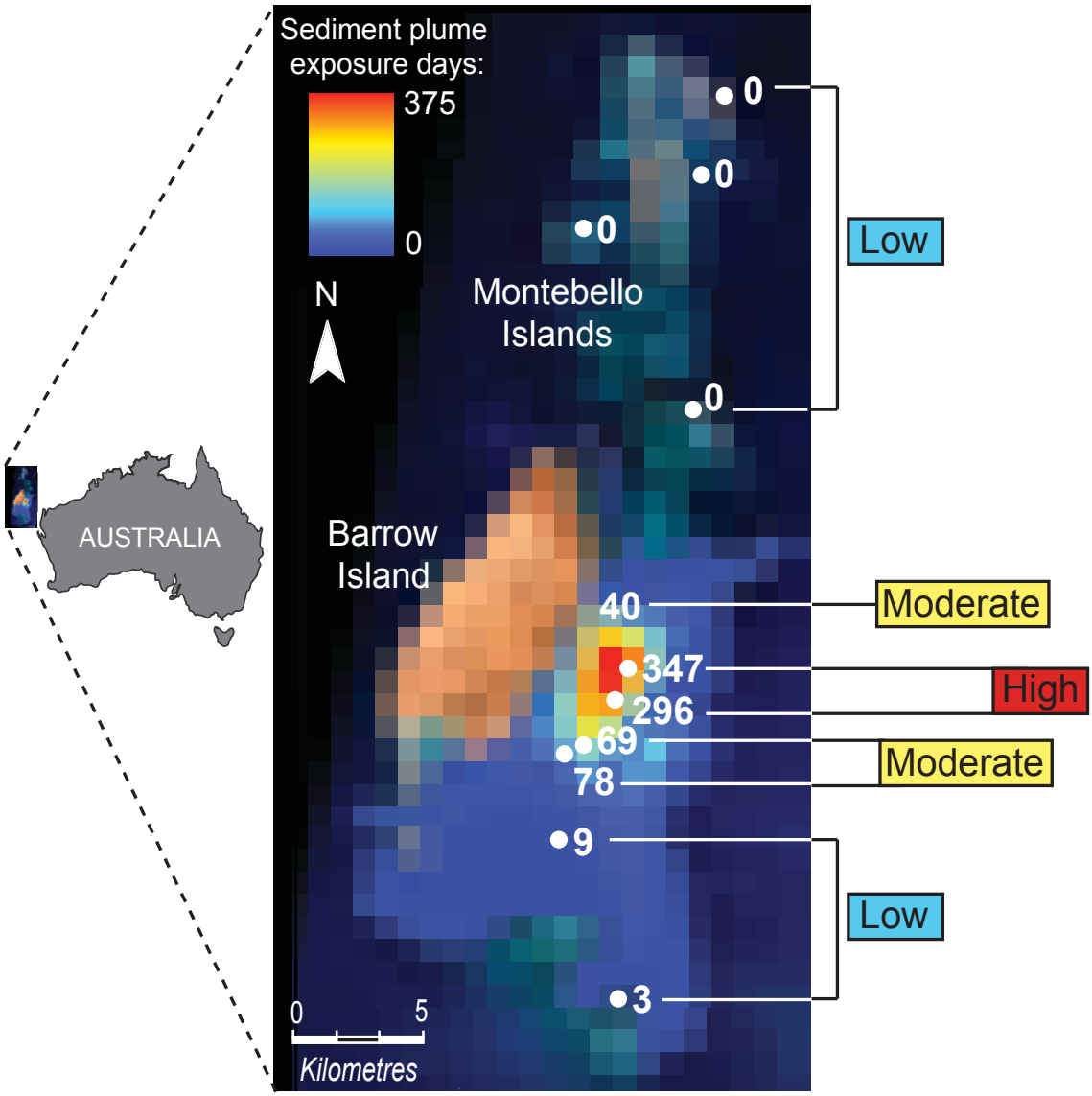


Figure 4.1 Study sites and coral health survey locations at Montebello and Barrow Islands, Western Australia. Coloured overlays (gradient from red to blue) indicate satellite-derived sediment plume exposure days determined by hot spot analysis of MODIS satellite imagery. Numbers indicate satellite-derived sediment plume exposure days at each site and coloured boxes represent sediment exposure categories used in statistical analyses: low (0 to 9 exposure days), moderate (40 to 78 exposure days), and high (296 to 347 exposure days).

To analyse patterns of coral disease and other signs of compromised health among broad coral growth forms within each sediment plume exposure category, coral genera were assigned to one of three growth form categories: (1) Massive (*Acanthastrea*, *Alveopora*, *Astreopora*, *Cyphastrea*, *Diploastrea*, *Favia*, *Favites*, *Fungiidae*, *Goniastrea*, *Goniopora*, *Leptastrea*, *Leptoria*, *Leptoseris*, *Lobophyllia*, *Montastrea*, *Moseleya*, *Oulophyllia*, *Platygyra*, massive *Porites* and *Symphyllia);* (2) Plating (tabular *Acropora*, *Echinophyllia*, plating *Echinopora*, *Galaxea*, *Merulina*, plating *Montipora*, *Mycedium*, *Oxypora*, *Pachyseris*, *Pectinia*, *Podabacia* and *Turbinaria*); or (3) Branching (bushy *Acropora*, digitate *Acropora*, staghorn *Acropora*, *Anacropora*, *Australogyra*, branching *Echinopora*, *Hydnophora*, *Isopora*, branching *Montipora*, *Palauastrea*, *Paraclavarina*, *Pavona*, branching *Pocillopora*, branching *Porites*, *Psammocora*, *Seriatopora* and *Stylophora*).

Associations between the prevalence of disease and other compromised coral health indicators and sediment plume exposure days were tested with Pearson product-moment correlations. Differences in mean prevalence levels among the three sediment plume exposure groups were analysed using two-way (sediment plume exposure category, site) nested analyses of variance (ANOVA), with site treated as a random factor that was nested within the fixed factor, plume exposure. Plume exposure days, coral predation by COTS and *Drupella*, and total hard coral cover were compared among plume exposure groups using the two-way ANOVA design described above. Differences in mean prevalence of disease and compromised health indicators were compared among growth forms within each sediment exposure category using one-way ANOVAs. Similarly, all temperature-based measures of disease likelihood were compared using one-way ANOVAs. Prior to analyses, assumptions of normality (Shapiro-Wilks) and homogeneity of variance (Levene's test of homogeneity) were

tested. Post-hoc comparisons between groups were performed using Tukey's HSD tests. All univariate statistical analyses were performed using Statistica 10 (StatSoft, Tulsa, Oklahoma).

To test for differences in the composition of coral communities among sediment plume exposure categories, a nested permutational analysis of variance (PERMANOVA) was used, in which site was treated as a random factor nested within the fixed factor, plume exposure (Anderson et al. 2008). Similarities among coral communities were illustrated using a non-metric multidimensional scaling plot (nMDS) at the transect level (Anderson et al. 2008).

A distance-based linear model (DISTLM) was used in combination with distance based redundancy ordination analysis (dbRDA) to explore the hypotheses that variability in patterns of disease and other compromised health indicators could be explained by environmental variables (i.e., sediment plume exposure days, hard coral cover, predation, and calculated thermal stress; see Table 4.1). Preliminary diagnostics to assess multi-collinearity among predictor variables using draftsman plots indicated that two thermal stress indicators, Peak SST and Peak SSTA, were highly correlated with Hot Snap ($r = 0.87$ and 0.79, respectively). To avoid redundancy, Peak SST and Peak SSTA were not included in the DISTLM or dbRDA. The DISTLM models the relationship between predictor variables and the multivariate data cloud based on a multiple regression. This routine finds the linear combination of variables that explains the greatest variation in the data cloud and examines the amount of variance explained by each variable, providing a pseudo-F statistical value. The best-fit model, based on corrected Akaike's Information Criterion (AICc), was then visualised in multidimensional space using dbRDA ordination (Anderson et al. 2008). Predictors that best explained the data were overlaid as biplots representing the strength (vector length)

and direction of influence (Anderson et al. 2008). All multivariate analyses were conducted in PRIMER v6 (Clarke and Gorley 2006) and PERMANOVA+ (Anderson et al. 2008) using Bray-Curtis similarity matrices based on fourth-root transformed data.

4.3 Results

4.3.1 Satellite-derived assessment of sediment plume extent

Satellite images of the GP sediment plume, of a quality suitable for deriving plume extent, were available for 411 of the 538 dredging days (i.e. 76% of days). Poor quality images (e.g., due to cloud cover or the sensor not capturing the study region) during the remaining 127 days (24% of days) were omitted from the analysis. Therefore, the number of sediment plume exposure days reported here is conservative and likely underestimate the true number of days sites spent under the dredge plume. Hotspot analysis of satellite imagery revealed that the sediment plume was most commonly detected around the dredge channel and sediment spoil dumping sites (Figure 4.1). Cumulative sediment plume exposure declined away from these sites, with the plume typically dispersing to the south of the dredge and spoil sites in response to prevailing wind and current patterns (Evans et al. 2012).

4.3.2 Impact of dredging on coral disease prevalence

A significant positive association was found between overall coral disease prevalence and exposure to dredging-associated suspended sediment ($r_{(9)} = 0.49$, $P < 0.05$). Mean disease prevalence (± SE) was nearly 2-fold higher at high exposure sites (7.26% ± 1.56) than at low (3.1% ± 0.6) and moderate exposure sites (4.7% ± 1.5) ($P < 0.002$; Figure 4.2a & Table 4.2). When results from all sites were combined, white syndromes (69%) and skeletal eroding band (17%) dominated the disease cases observed (n = 10,656 corals surveyed). At the high exposure sites, elevated disease

prevalence was largely the result of high white syndrome levels, which were 2.5-fold greater than at low and moderate exposure sites ($P < 0.001$; Figure 4.2a & Table 4.2). In contrast, the highest prevalence of brown band disease (BrB) was recorded at moderate exposure sites, where it was 9 times greater than at high or low exposure sites (*$P < 0.001$;* Figure 4.2a & Table 4.2). The prevalence of black band disease, growth anomalies and skeletal eroding band did not differ significantly between exposure categories (*$P > 0.05$;* Figure 4.2a & Table 4.2).

4.3.3 Impact of dredging on compromised coral health

There was a significant positive association between the prevalence of other compromised health indicators and exposure to dredging-associated suspended sediment ($r_{(9)} = 0.79$, $P < 0.001$). Mean prevalence of these indicators was more than 6-fold greater at high exposure sites (47.9% ± 11.2) than at low (8.0% ± 1.4) or moderate exposure sites (7.9% ± 0.9) (*$P < 0.001$*; Figure 4.2b & Table 4.2). Sediment-associated tissue necrosis was 57 times more prevalent at high exposure sites compared to low and moderate exposure sites (*$P < 0.001$;* Figure 4.2b & Table 4.2). Bleaching, sponge overgrowth and pigmentation responses were also significantly greater at high exposure sites relative to low or moderate exposure sites (all: *$P < 0.001$*; Figure 4.2b & Table 4.2,). The prevalence of red and green algae did not differ significantly between exposure categories (*$P > 0.05$;* Table 4.2).

Table 4.1 Environmental predictor variables assessed in a multivariate multiple regression analysis (DISTLM) at sites within three dredge-associated sediment plume exposure categories determined by MODIS satellite imagery: low (0 to 9 plume exposure days), moderate (40 to 68 plume exposure days) and high (296 to 347 plume exposure days); and results of an analysis of variance (ANOVA) for each predictor variable among sediment plume exposure groups.

		Sediment plume exposure category					
		Low (n = 6 sites)	**Moderate** (n = 3 sites)	**High** (n = 2 sites)	ANOVA	Model	
Environmental predictors		mean ± SE	mean (SE)	mean (SE)	*F, P*	inclusion	Source
Dredging:	Sediment plume exposure[a]	2.0 (1.5)	62.3 (11.5)	321.5 (25.5)	285.7, <0.001*	Yes	Evans et al. 2012
Predation:	COTS scars[b]	-	1.6 (1.1)	-	1.5, 0.29	Yes	This study
	Drupella scars[c]	3.6 (0.6)	11.7 (3.3)	1.4 (0.9)	2.9, 0.11	Yes	This study
Coral cover:	Total hard coral cover[d]	36.0 (15.0)	53.5 (21.1)	26.0 (20.4)	1.7, 0.24	Yes	This study
Thermal stress:	Peak SST[e]	30.7 (0.2)	30.8 (0.3)	30.9 (0.0)	0.2, 0.81	No[k]	Heron et al. 2010
	Peak SSTA[f]	2.8 (0.1)	2.7 (0.2)	2.7 (0.0)	0.07, 0.94	No[k]	Heron et al. 2010
	Hot Snap[g]	2.0 (1.7)	1.8 (1.4)	2.2 (0.4)	0.04, 0.96	Yes[k]	Heron et al. 2010
	Winter Conditions[h]	23.2 (0.6)	21.9 (0.7)	22.3 (0.4)	1.0, 0.41	Yes	Heron et al. 2010
	MPSA[i]	0.3 (0.1)	0.2 (0.03)	0.3 (0.01)	1.2, 0.35	Yes	Maynard et al. 2011
Modeled:	Predicted disease abundance[j]	101 (94)	88 (39)	52 (57)	0.3, 0.75	No	Maynard et al. 2011

[a] Number of days the sediment plume was recorded over a site for the duration of dredging operations (days)
[b] Prevalence of coral colonies with crown-of-thorns seastar lesions determined by *in situ* coral health surveys (%)
[c] Prevalence of coral colonies with *Drupella* lesions determined by *in situ* coral health surveys (%)
[d] Total hard coral cover on transects determined by line intercept method (%)
[e] Maximum sea surface temperature recorded during dredging operations (May 2010 - Nov 2011) (°C)
[f] Maximum excursion of sea surface temperature from the long-term climatological value during dredging operations (May 2010 - Nov 2011) (°C)
[g] Accumulation of thermal anomalies greater than the long-term summer mean temperature plus one standard deviation (°C-weeks)
[h] Accumulation of winter anomalies (+ and -) from the long-term winter mean temperature (°C-weeks)
[i] Average of summer temperature anomalies greater than zero calculated from the monthly mean temperature plus one monthly standard deviation
[j] Modeled numeric prediction of disease abundance based upon MPSA and total coral cover (disease cases per 1,500 m^2)
[k] Peak SST and Peak SSTA were excluded from the DISTLM due to a strong correlation with Hot Snap (r = 0.87 and 0.79, respectively)

Table 4.2 Mean prevalence of coral disease and compromised coral health indicators at sites classified into three sediment plume exposure categories determined by MODIS satellite imagery: low (0 to 9 plume exposure days), moderate (40 to 68 plume exposure days) and high (296 to 347 plume exposure days) and results of a 2-way nested analysis of variance (ANOVA). Mean prevalence calculated as the percentage of colonies with disease or compromised health as a percentage of the total number of corals per transect. Analyses performed on square root transformed data. Number of total corals surveyed = 10,656

	Sediment Plume Exposure Category				
	Low (n = 18 transects)	**Moderate** (n = 9 transects)	**High** (n = 6 transects)	**ANOVA**	
Variable	mean (SE) prevalence (%)	mean (SE) prevalence (%)	mean (SE) prevalence (%)	Exposure *F, P*	Site (exposure) *F, P*
Total disease	3.1 (0.6)	4.7 (1.5)	7.3 (1.6)	9.1, <0.002*	7.3, <0.001*
White syndromes	2.3 (0.5)	2.9 (1.2)	6.7 (1.2)	17.5, <0.001*	10.4, <0.001*
Brown band	0.07 (0.04)	0.8 (0.4)	0.09 (0.09)	0.9, <0.001*	5.7, <0.005*
Black band	0.03 (0.02)	0.5 (0.4)	0.0 (0.0)	2.7, <0.09	1.6, <0.17
Skeletal eroding band	0.7 (0.2)	0.5 (0.2)	0.4 (0.3)	1.4, <0.27	2.3, <0.06
Growth anomalies	0.0 (0.0)	0.03 (0.03)	0.2 (0.2)	1.6, <0.22	0.7, <0.66
Total compromised health	8.0 (1.4)	7.9 (0.9)	47.9 (11.2)	50.8, <0.001*	8.1, <0.001*
Sediment necrosis	0.5 (0.2)	0.3 (0.2)	22.7 (8.5)	154.9, <0.001*	16.0, <0.001*
Bleaching	1.4 (0.4)	0.7 (0.2)	9.5 (3.8)	20.1, <0.001*	3.0, <0.02*
Sponge overgrowth	1.0 (0.3)	1.2 (0.4)	2.8 (0.3)	6.9, <0.005*	3.3, <0.01*
Pigmentation response	0.3 (0.1)	1.6 (0.5)	2.4 (0.3)	24.8, <0.001*	2.5, <0.04*
Red algal overgrowth	0.9 (0.4)	1.2 (0.4)	0.4 (0.3)	1.8, <0.19	2.2, <0.07
Green algal overgrowth	2.5 (1.1)	1.5 (0.8)	0.5 (0.2)	2.3, <0.12	4.2, <0.003*

* denotes a significant difference for α = 0.05

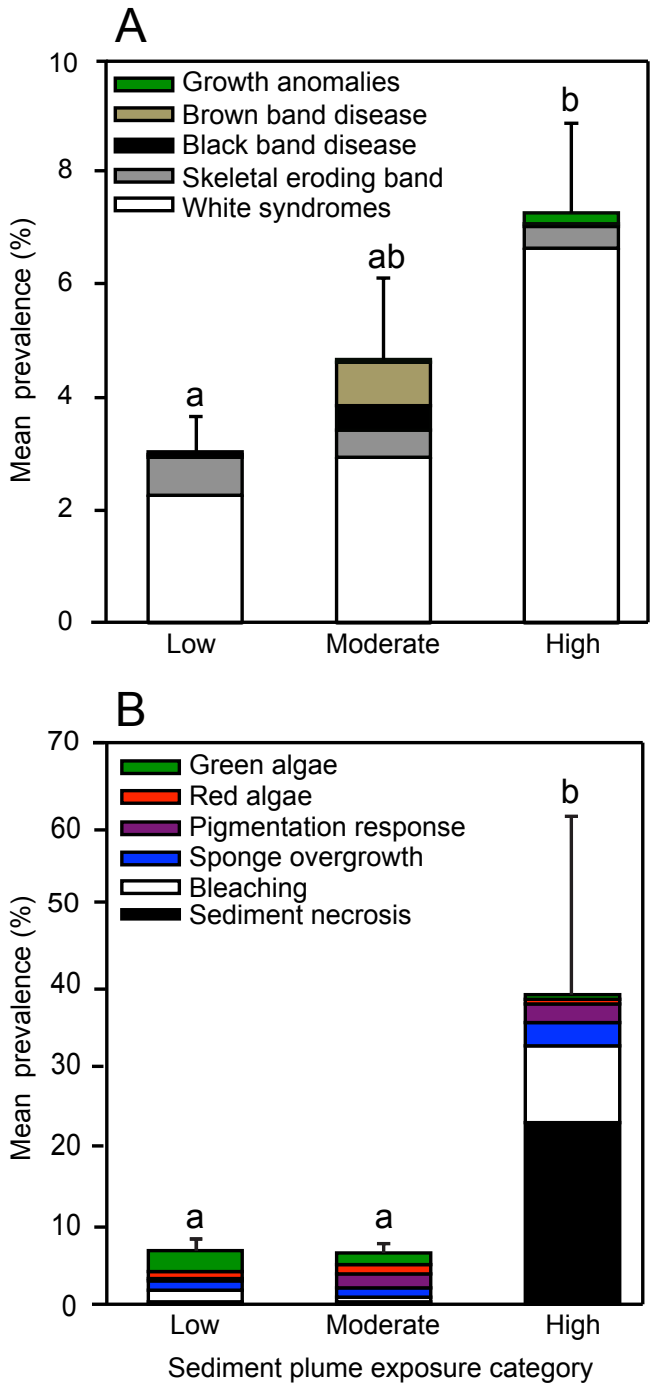


Figure 4.2 Mean prevalence of (a) coral disease and (b) compromised coral health indicators at sites within three sediment plume exposure categories: low (0 to 9 plume exposure days; n = 18 transects, 5351 corals surveyed), moderate (40 to 78 plume exposure days; n = 9 transects, 4292 corals surveyed), and high (296 to 347 plume exposure days; n = 6 transects, 1013 corals surveyed). Stacked bars indicate disease or compromised health prevalence by category and error bars indicate standard error among transects for total disease or compromised coral health prevalence. Letters indicate homogenous post-hoc groups among sediment plume exposure categories.

4.3.3 Influence of coral community composition and morphology on disease and other signs of compromised health

There was no significant difference in coral community composition between sediment plume exposure categories (*Pseudo-F = 1.38, P > 0.1*; Figure 4.3). However, coral community composition did vary significantly among sites within exposure categories (*Pseudo-F = 7.54, P < 0.001*; Figure 4.3).

Disease levels did not differ significantly among growth forms (i.e., massive, plating and branching colonies) at high or low exposure sites ($P > 0.05$; Figure 4.4a). However, massive corals at moderate exposure sites sustained significantly less disease than branching and plating colonies ($P > 0.05$; Figure 4.4a). The prevalence of other compromised health indicators did not differ between growth forms within any sediment plume exposure category ($P > 0.05$; Figure 4.4b).

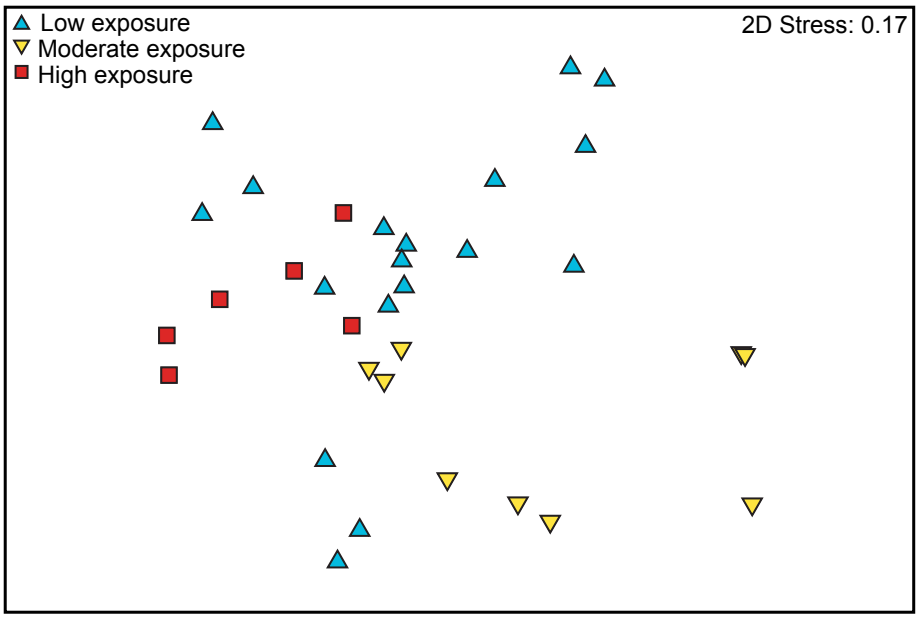


Figure 4.3 Non-metric multidimensional scaling (nMDS) plot visualising variation in the taxonomic composition (genus-level) of coral assemblages at transects within each sediment plume exposure category: low (0 to 9 plume exposure days; n = 18 transects; light blue triangles), moderate (40 to 78 plume exposure days; n = 9 transects; yellow triangles), and high (296 to 347 plume exposure days; n = 6 transects; red squares).

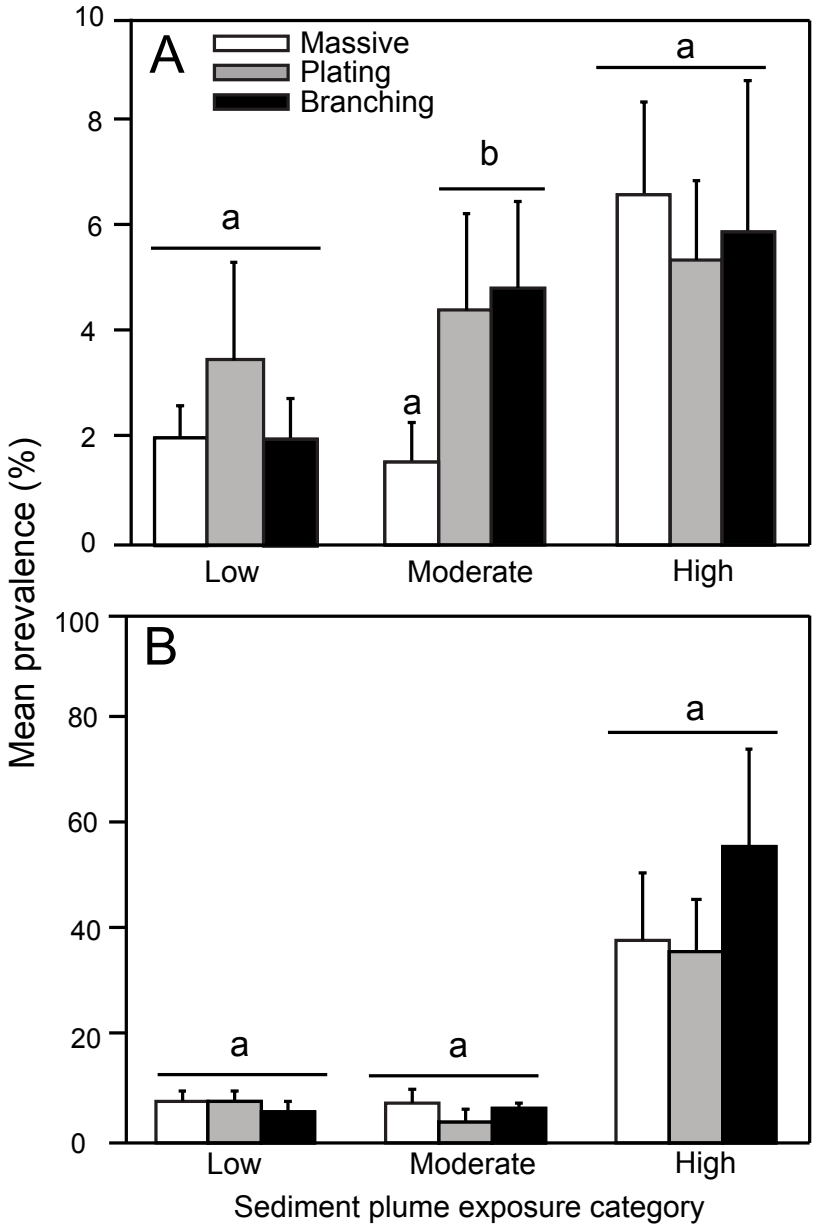


Figure 4.4 Mean prevalence of (a) coral disease and (b) compromised coral health indicators at sites within three sediment plume exposure categories, low (0 to 9 plume exposure days; n = 15 transects; white bars), moderate (40 to 78 plume exposure days; n = 9 transects; grey bars) and high (296 to 347 plume exposure days; n = 6 transects; black bars), for three coral growth morphologies, massive (n = 1725 colonies), plating (n = 1768 colonies) and branching (n = 3351 colonies). Lettered bars indicate homogenous post-hoc groups among morphologies within each exposure category.

4.3.3 Environmental drivers of disease and compromised health

ANOVA and DISTLM (visualised through dbRDA) both identified Sediment plume exposure level as the most significant environmental driver of elevated levels of disease and other indicators of compromised health. Among all environmental parameters assessed (see Table 4.1), sediment plume exposure days was the only metric that differed significantly among exposure categories ($P < 0.001$; Table 4.1). Furthermore, the abundance of disease predicted by satellite-derived temperature-based stress metrics did not differ significantly among sediment plume exposure groups ($P > 0.05$; Table 4.1).

The dbRDA diagram depicting coral disease composition (based on the simplest best fit DISTLM, $AICc = 216.65$, $R^2 = 0.33$), showed a clear separation of high exposure sites from moderate and low exposure sites (Figure 4.5a). The greatest amount of variation in the disease prevalence data was explained by sediment plume exposure days (*Pseudo-F = 3.97, P < 0.05*) and total hard coral cover (*Pseudo-F = 4.01, P < 0.05*), while the accumulation of thermal anomalies greater than the long-term summer mean temperature plus one standard deviation (Hot Snap) explained a lesser, albeit still significant amount of variation in the data (*Pseudo-F = 3.31, P < 0.05*; Figure 4.5a). The overlay vector for sediment plume exposure days corresponded to the axis separating high exposure sites from low and moderate exposure sites, while the vectors for hard coral cover and Hot Snap largely corresponded to the axis separating sites within low and moderate exposure categories (Figure 4.5a).

The dbRDA diagram depicting the composition of other compromised health indicators (based on the simplest best fit DISTLM, $AICc = 215.57$, $R^2 = 0.28$) also showed a clear separation between tightly clustered high exposure transects and the more dispersed low and moderate exposure transects (Figure 4.5b). Sediment plume exposure days (*Pseudo-F = 5.99, P < 0.005*) and predation by *Drupella* spp. (*Pseudo-F*

$= 3.65, P < 0.05$) explained the greatest amount of variation in the data. The overlay vector for sediment plume exposure days corresponded to the axis separating high exposure sites from low and moderate exposure sites, while the vector for *Drupella* largely corresponded to the axis separating sites within low and moderate exposure categories (Figure 4.5b).

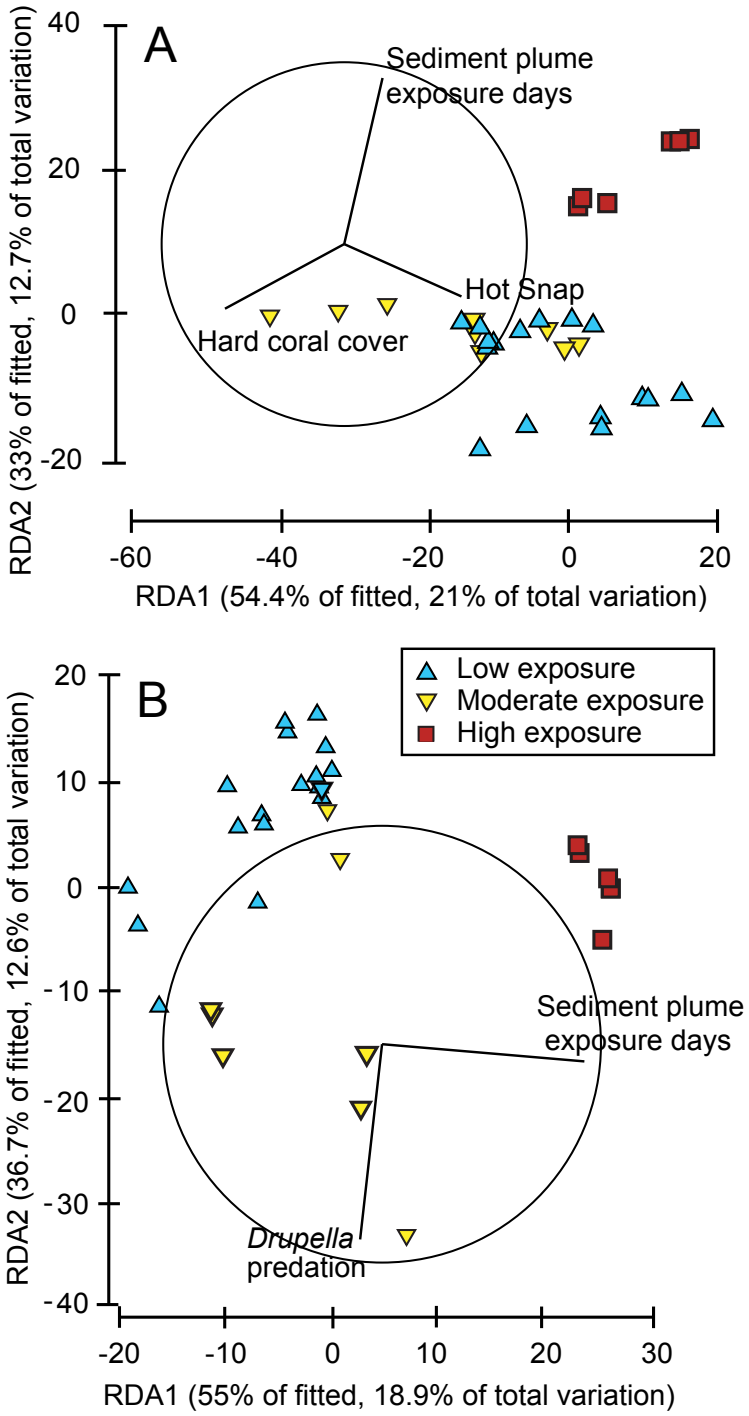


Figure 4.5 Distance-based redundancy analysis (dbRDA) ordination plots illustrating the relationship between environmental predictors that best explain the variation of (a) coral disease and (b) compromised coral health indicators among sites. The dbRDA was constrained by the best-fit explanatory variables from a multivariate multiple regression analysis (DISTLM) and vectors overlays are shown for predictor variables explaining a significant proportion of the variation in the prevalence of (a) coral disease, and (b) other signs of compromised coral health. Hot Snap = accumulation of thermal anomalies greater than the long-term summer mean temperature plus one standard deviation (°C-weeks).

4.4 Discussion

This study provides the first empirical evidence linking turbidity and sedimentation with elevated levels of coral disease and other indicators of compromised coral health *in situ*. I found two-fold higher disease prevalence, largely driven by increases in white syndromes (WS), and six-fold higher levels of other compromised health indicators at high sediment plume exposure sites. While previous studies have suggested a myriad of environmental stressors as potential drivers of coral disease (Hodgson 1990; Kline et al. 2006; Haapkyla et al. 2011), the current study highlights a direct link to sedimentation and turbidity. The initial, relatively pristine condition of the study sites, in combination with their location distant to any major land-based influences, provided the ideal opportunity to identify dredging-related sedimentation and turbidity as the key parameters driving elevated disease prevalence. On Australia's east coast, the UNESCO World Heritage Center recently cited increasing coastal development and catchment runoff as serious threats to the "outstanding universal value" of Australia's Great Barrier Reef World Heritage Area (Douvere & Badman 2012), and on Australia's west coast, an estimated 200 million cubic meters of sediment will be dredged and dumped in current projects passing through Western Australia's approvals and/or regulatory system alone (Masini et al. 2011). Clearly, these findings will have direct implications for coastal managers charged with balancing economic development with the imperative to maintain healthy coral reefs. As the rate of coastal development near coral reef ecosystems continues to escalate in many parts of the world, insights into the types of activities that promote disease, like those revealed in this study, are becoming increasingly important.

Elevated disease levels at high sediment plume exposure sites were primarily the result of the more than 2.5-fold higher prevalence of WS, an important group of diseases that have affected reefs throughout the Indo-Pacific and which are

characterised by a distinct band of sloughing coral tissue revealing underlying coral skeleton (Willis et al. 2004; Aeby 2005; Sussman et al. 2008; Long & Holmes 2009; Hobbs & Frisch 2010). WS prevalence is often associated with environmental stress, and strong correlations between warm temperature anomalies and elevated WS levels have shown that thermal stress is an important driver of this disease on Indo-Pacific reefs (Heron et al. 2010; Maynard et al. 2011). However, I found no differences in multiple thermal stress metrics or model-derived predictions of disease among the three sediment plume exposure groups, indicating that temperature was not the factor driving WS prevalence in this study and providing further evidence that multiple stressors cause tissue loss characteristic of white syndromes. While Hot Snap and total hard coral cover helped to explain the distribution of disease among sites, this metric largely correlated with differences among low and moderate exposure sites. Haapkylä et al. (2011) noted a relationship between seasonal coastal runoff and disease prevalence, and suggested that increased availability of nutrients and organic matter could reduce host fitness or increase pathogen virulence, while other seasonal environmental conditions, including decreased salinity and elevated water temperature could place further synergistic stress on corals. Sediment plume exposure was the main driver of elevated WS levels in the current study, providing further evidence for the role of environmental stress, specifically increased sedimentation and turbidity, in WS pathogenesis.

Greater prevalence of brown band disease (BrB) at moderate exposure sites compared to high and low exposure sites may reflect differences in turbidity and hence light levels among sites, with optimal levels for ciliate proliferation most closely approximating those at moderate exposure sites. BrB is characterised by a dense, brown mat of ciliates packed with *Symbiodinium* derived from consumed coral tissue (Willis et al. 2004; Bourne et al. 2008). Since *Symbiodinium* cells residing within ciliates are

photosynthetically competent during BrB progression, it has been proposed that BrB ciliates could derive nutrition from photosynthates produced by ingested *Symbiodinium*, while also benefiting from an additional oxygen source within the densely populated and presumably oxygen-limited BrB mat (Willis et al. 2004; Bourne et al. 2008). At highly turbid, high sediment plume exposure sites, *Symbiodinium* photosynthesis would be hindered, potentially removing the advantage that BrB ciliates might have over their presumably immune-compromised coral hosts. However, moderate-exposure sites could provide the right balance of compromised host immunity and sufficient light availability to facilitate infection and proliferation of BrB ciliates, although further studies specifically investigating the influence of reduced light levels on the partitioning of photosynthates between *Symbiodinium* and ciliates are required to test this hypothesis.

Total mean disease levels at low exposure sites (3.1%) were similar to levels reported from nearby Ningaloo Marine Park (Onton et al. 2011), indicating that these sites provide a good approximation of background disease levels in the region. Prevalence levels of black band disease, skeletal eroding band disease and growth anomalies did not differ significantly among sediment plume exposure groups and were consistent with levels reported from Ningaloo Reef (Onton et al. 2011).

The greater prevalence of other indicators of compromised coral health at high sediment plume exposure sites was largely the result of elevated levels of sediment-associated necrosis and bleaching, which were 57-fold and 9-fold higher, respectively. Increased turbidity reduces the amount of light available for photosynthesis, while sediment deposition further shades corals and taxes energy budgets through the need to allocate energy to sediment removal. Although corals are able to actively remove sediment particles through ciliary and tentacular movement, combined with polyp distension and mucus production (Hubbard & Pocock 1972; Rogers 1990; Philipp &

Fabricius 2003), these mechanisms can become overwhelmed during periods of intense and/or chronic sediment deposition. When sediment stress is chronic, even low levels can dramatically alter coral energy budgets by reducing *Symbiodinium* densities (i.e., bleaching) and by decreasing the photochemical efficiencies (F_v/F_m) of the *Symbiodinium* that remain (Hubbard & Pocock 1972; Rogers 1990; Philipp & Fabricius 2003). If resulting energy deficits are not relieved through either metabolic depression or heterotrophic feeding, bleaching can lead to mortality of the affected coral tissue (i.e., sediment necrosis).

While bleaching and sediment necrosis observed in this study were mostly confined to depressions on the coral surface, these patches of partial mortality could expose the coral to further mortality or subsequent infection by disease, even after the completion of dredging operations. In laboratory sedimentation experiments, Flores et al. (2012) reported that corals with only 10% partial mortality at the end of a period of sediment exposure subsequently suffered complete mortality during a 4-week "recovery" period. Although bleaching and sediment necrosis are known sources of coral mortality during periods of prolonged sediment exposure (Glynn 1996; Brown 1997), future studies should investigate the potential of sediment-induced lesions to develop into coral disease, which could progress even after the sediment stress is removed.

Elevated prevalence of pigmentation responses (PR) at high exposure sites provides further evidence of sediment plume-induced coral stress. PR has been observed in corals subjected to a suite of stressors and has been proposed to be a general immune response to a variety of physical and biological challenges, including to competitors and pathogens (Willis et al. 2004; Bongiorni & Rinkevich 2005; Ravindran & Raghukumar 2006; Palmer et al. 2008). Tissues associated with PR possess high

levels of melanin, an important component of invertebrate innate immunity that can act as a defensive barrier against foreign bodies (Palmer et al. 2008). Elevated PR levels at high exposure sites may represent an inflammatory-like response by the coral to either sediment particles clogging tentacles and polyp surfaces or to invading pathogens.

Competition on space-limited coral reefs is intense, and even small perturbations can alter competitive hierarchies by reducing the competitive abilities of some species relative to others (Connell 1978). In this study, sponge overgrowth was greater at high exposure sites, while levels of red and green algae overgrowth did not differ significantly among sediment plume exposure levels. Increased sediments and nutrients derived from dredging activities may enhance primary production and biomass in the water column, benefiting heterotrophic sponges while simultaneously limiting the potential of corals for autotrophic nutrition (Pastorok & Bilyard 1985). However, in competitive interactions between coral and algae, both sides rely, at least partially, upon autotrophic photosynthesis for the energy necessary to defend themselves or to mount an attack against the other (Barott & Rohwer 2012). Unlike coral-sponge competition, elevated sedimentation and turbidity did not appear to provide a clear benefit to either side of the coral-algal competitive hierarchy.

The prevalence of disease and other compromised coral health indicators did not vary between coral growth forms (i.e., massive, plating and branching) at high sediment plume exposure sites. The influence of coral morphology (i.e., growth from) on sediment clearing rates has been well investigated (Hubbard & Pocock 1972; Bak & Elgershuizen 1976; Rogers 1983; Stafford-Smith 1993; Sanders & Baron-Szabo 2005), with branching corals generally considered to be more effective at passive sediment removal, while massive and plating forms retain more sediment due to their shapes, which inhibit passive rejection and removal (Brown & Howard 1985). However,

sediment rejection rates and sediment tolerance are not directly related (Stafford-Smith 1993). For example, Stafford-Smith (Stafford-Smith 1993) reported some plating species (e.g. *Montipora aequituberculata*) with high sediment tolerance despite poor sediment rejection capacity, whereas some massive species (e.g. *Favia stelligera* and *Leptoria phrygia*) are efficient sediment rejecters but exhibit low sediment tolerance. While previous investigations have focused on only a few coral species in relatively artificial conditions, this is the first field study to investigate the relationship between growth form, sedimentation, turbidity and coral health among all hard coral species present on a natural reef. Although this study indicates that growth form is not a strong predictor of corals' susceptibility to disease and other compromised health indicators associated with increased sedimentation and turbidity, some caution is required in this interpretation, as the most susceptible corals may have experienced complete mortality prior to surveys being undertaken.

This study provides empirical, field-based evidence linking dredging-associated sedimentation and turbidity to elevated levels of coral disease and other compromised coral health indicators on reefs. To date, disease has been largely overlooked as a contributor to dredging-induced reef degradation, with most impact monitoring programs focusing almost exclusively upon tissue necrosis and bleaching as indicators of coral health. Additionally, while poor water quality has been suggested as a driver of coral disease, little ecological evidence exists linking specific water quality parameters and coral disease data. WS responded strongly to elevated sedimentation and turbidity demonstrating a clear link between water quality and coral disease, though the mechanisms underlying this response remain unclear. Future studies that investigate the response of the coral host (e.g., immune function and energy reserves) and potential pathogens (e.g., shifts in bacterial and viral communities on corals and in surrounding

seawater) to elevated sedimentation and turbidity are required. As coastal development intensifies in many parts of the world, the health of coral reefs will depend upon a thorough understanding of the impacts of dredging and water quality changes on coral reef health. To accurately quantify the impact of dredging on coral health, comprehensive monitoring of coral diseases and other indicators of compromised coral health must be included in environmental impact assessments.

CHAPTER 5.

Marine reserves mitigate coral disease on inshore reefs in the Great Barrier Reef Marine Park[†]

[†]Submitted for review

5.1 Introduction

Coral reefs are under increasing pressure to provide food, employment and recreation for millions of people within coastal populations, but there is widespread concern that increasing and often conflicting usage is leading to progressive degradation of coral reef health (Worm et al. 2006; Halpern et al. 2008). Management efforts are primarily focused towards strengthening reef resilience by ameliorating local and regional anthropogenic pressures, such as those caused by terrestrial runoff and dredging, destructive fishing, overfishing, and coastal development (Bellwood et al. 2005; Hoegh-Guldberg et al. 2007; De'ath et al. 2012). Because it is often difficult to modify human actions directly, physical solutions to resource management problems, like restricting site access, are typically adopted (Boersma & Parrish 1999). Within the past few decades, marine parks and reserves have been strongly advocated as a strategy to protect environments from anthropogenic pressures. Marine protected areas (MPAs) have been shown to be a relatively simple and effective strategy for managing multi-species reef fisheries (Russ et al. 2008), and are widely perceived as a means to protect marine habitats and communities (Done 2001; Selig & Bruno 2010), separate conflicting use of marine resources (Gell & Roberts 2003), and enhance tourism opportunities (Roberts & Hawkins 2000). However, despite the increasing threat that coral disease outbreaks pose to food security and ecosystem services in coastal reef areas, the potential of MPAs to ameliorate coral health has received comparatively limited attention.

Few options are available to manage diseases in marine environments (Harvell et al. 2004; McCallum et al. 2005; Beeden et al. 2012), and the few studies that have evaluated the utility of MPAs to mitigate epizootics in coral populations have produced conflicting results. In the Philippines, effectively managed MPAs significantly reduced coral disease, with the mechanism purportedly related to the maintenance of taxonomically diverse fish assemblages (Raymundo et al. 2009). In contrast, Page et al. (2010) found no evidence that MPAs enhanced coral health on Palauan reefs, potentially because of poor compliance with restrictions on activities within reserves, although an inverse correlation found between fish diversity and coral disease prevalence when MPA status was ignored suggested that diverse fish assemblages are important for coral health. Comparisons of the prevalence of growth anomalies on *Porites* colonies in Kenya (McClanahan et al. 2009) and of several diseases at Little Cayman in the Caribbean (Coelho & Manfrino 2007) provided further evidence that MPAs are not effective in reducing disease in these regions, although such conclusions are potentially confounded by poor compliance with site restrictions or by influences that permeate boundaries, such as sediment runoff, nutrient or sewage pollution, and thermal anomalies (McClanahan et al. 2009). There is a clear need for further studies of well-managed MPAs to separate the impact of reef protection status from other potentially confounding factors affecting coral health and to evaluate the efficacy of MPAs in mitigating coral disease.

Several lines of reasoning suggest that MPAs are likely to influence levels of disease in coral populations, although it could be argued that influences could be either beneficial or detrimental to coral health. Areas that exclude activities that damage corals may reduce disease prevalence by limiting injury, which provides an entry point for coral pathogens (Page & Willis 2008; Nicolet et al. 2012; Chapter 3). Higher levels of

coral disease at sites associated with high intensity scuba diving and tourism in Thailand and the Great Barrier Reef (Lamb & Willis 2011; Chapter 3) support this hypothesis. In addition, many of the fishing methods and gear types used to catch coral reef fishes cause direct physical damage to corals (Bavestrello et al. 1997; Schleyer & Tomalin 2000; Yoshikawa & Asoh 2004; Mangi & Roberts 2006; Asoh et al. 2006). In Kenya, spear fishers had the highest number of contacts with live corals per unit catch, while beach seines, spears and gill nets cause the most direct physical damage (Mangi & Roberts 2006). In Hawaii, the mean proportion of dead or damaged colonies was higher in fished than in adjacent unfished zones, and there was a positive linear relationship between the proportion of colonies entangled with fishing lines and the proportion of dead or damaged colonies (Asoh et al. 2006). More recently, corals entangled in derelict fishing line were more likely to have disease than nearby colonies without fishing line (Chapter 3). On the other hand, increases in host cover or density as a consequence of fishing exclusion might facilitate the spread of pathogens through populations of susceptible hosts (Anderson & May 1979; Bruno et al. 2007). Further tests of the relative veracity of these opposing predictions of the impacts of MPAs on coral health are needed.

In addition to the direct impacts discussed above, fishing has the potential to affect coral health through indirect shifts in reef fish community structure. Loss of functional diversity in reef fish communities (Bohnsack 1982; Russ & Alcala 1989) as a consequence of reduced reef habitat complexity associated with deployment of fishing gear (Roberts 1995), particularly loss of a wide range of feeding guilds, could disrupt the balance between corals, competitors and organisms that act as potential vectors or reservoirs of pathogens. For example, high densities of herbivorous fish within MPAs could reduce negative algal-coral interactions (Jompa & McCook 2003) by limiting

growth of algae (Bellwood et al. 2003), which have been implicated as reservoirs of pathogens in Caribbean corals (Nugues et al. 2004; Smith et al. 2006). In addition to reducing habitat complexity, line fishing has been shown to target piscivores, important in structuring coral reef fish assemblages (Mumby et al. 2006) and indirectly, coral reef communities (McClanahan & Muthiga 1988; Roberts 1995; Graham et al. 2003). Moreover, MPAs could increase disease prevalence if they result in increasing densities of fishes that (1) act as vectors for coral pathogens, (2) injure coral tissues during feeding (Aeby & Santavy 2006), or (3) deposit faeces on live coral surfaces leading to changes in microbial community composition towards more pathogenic taxa (Weil 2004; Pantos & Bythell 2006).

The Great Barrier Reef Marine Park (GBRMP) represents a particularly relevant case study to test the utility of MPAs as a management tool because coral reefs within the Marine Park have been classified as the world's least threatened (Burke et al. 2011) and compliance with MPA restrictions is typically high (Davis et al. 2004). The minor impacts that local anthropogenic disturbances, such as anchor damage, destructive fishing practices, pollution, vessel groundings, and oil spills have had on the GBR to date (De'ath et al. 2012), allow examination into the role of visitor activities and reef fish assemblages in mitigating disease in the absence of other disturbances. Furthermore, spatial zoning for multiple-use provides nominally strong protection to certain areas, whilst allowing varying levels of extractive activities in others (Day 2002). Consequently, inshore reefs of the GBRMP are exposed to a range in intensity of recreational use, from no-take zones to areas exposed to significant levels of fishing effort near urban centres (Higgs & McInnes 2003), enabling meaningful comparisons between coral populations which are protected versus exposed to recreational fishing activities.

An increase in no-take zones within the GBRMP from 5% to 33% in 2004, the largest spatial closure to fishing to date (Fernandes et al. 2005), caused intense and divisive public debate, highlighting the need for reef managers to continuously assess the effectiveness of established no-take areas (Day 2008). There has been ongoing assessment of the utility of MPAs to maintain densities of coral trout (*Plectropomus* spp.), the primary target group of the recreational and commercial hook and line fishery in the GBRMP (Mapstone et al. 2004), with densities shown to increase in rapid timeframes when reef status is changed from fished to no-take reserves (Mapstone et al. 2004; Russ et al. 2008). No-take reserves in the GBRMP also appear to benefit overall ecosystem health and resilience by reducing the frequency of outbreaks of coral-eating, crown-of-thorns starfish (Sweatman 2008) and enhancing coral cover (Williamson et al. 2004). However, there has been no assessment of the impact of no-take reserves on overall coral health within the GBRMP.

In this study, I examine the utility of marine reserves to mitigate coral disease in the most frequented recreational inshore island region in the GBRMP. I use multiple factors, including MPA status, habitat and environmental characteristics, fish assemblages, and differences in gear restrictions to evaluate variation in coral disease assemblages and individual disease types in the presence versus absence of fishing. In order to obtain fine-scale spatial resolution of factors driving coral disease, I also examine associations between disease and recreational activities within management zones.

5.2 Methods

5.2.1 Multiple-use management zones in the Great Barrier Reef Marine Park

Three management zones were used to assess the efficacy of marine reserves as tools for mitigating coral disease (Table 5.1). Marine National Parks (MNP) make up

about 33% of the GBRMP and are 'no-take' areas, thus extractive activities like fishing or collecting are not allowed without a permit. Habitat Protection (HP) zones, which comprise 28% of the marine park, are open to moderate levels of fishing and extractive activites. Conservation Park (CP) zones are also open to fishing and extractive activities, however fishing gear is restricted to one rod, line and hook per person. Anchoring is also allowed in all zones, however in high use and sensitive areas, buoys define no anchoring areas and the use of moorings may be necessary.

Table 5.1 Permitted and restricted activities in three management zones surveyed in this study.

Activity	Marine National Park MNP	Habitat Protection HP	Conservation Park CP
Line fishing	No	Yes	Limited [a]
Boating, diving, photography	Yes	Yes	Yes
Crabbing (trapping)	No	Yes	Limited [b]
Harvest fishing for aquarium fish, coral and beachworm	No	Permit	Permit
Harvest fishing for sea cucumber, trochus, tropical rock lobster	No	Permit	No
Limited collecting	No	Yes	Yes
Limited impact research (non-extractive)	Yes	Yes	Yes
Limited spearfishing (snorkel only)	No	Yes	Yes
Bait netting	No	Yes	Yes
Netting (other than bait netting)	No	Yes	No
Research (other than non-extractive)	Permit	Permit	Permit
Shipping (other than a designated shipping area)	Permit	Permit	Permit
Tourism program	Permit	Permit	Permit
Traditional use of marine resources	Permit	Permit	Permit
Trawling	No	No	No
Trolling	No	Yes [c]	Yes [c]

[a] One hand-held rod or one hand-held line per person, with no more than one hook attached

[b] Four crab pots or collapsible traps

[c] No more than three lines per person and up to six hooks combined total per person

5.2.2 Study locations and characteristics

This study was conducted on fringing inshore coral reefs in the Whitsunday Islands (20º08'S, 148º56'E), a group of 53 islands approximately 25 km from the mainland in the central sector of the GBRMP (Figure 5.1). The Whitsunday Island region is a high-use recreational area and a popular destination for boating, fishing, and diving. The islands are destinations for approximately half of the 1.4 million tourists that visit the Great Barrier Reef each year (Tourism Queensland 2013), and there is a significant level of recreational fishing pressure (hook and line, spear) on the fringing reef communities (Blamey & Hundloe 1991; Higgs & McInnes 2003). In comparison to more remote areas of the GBRMP, there is a relatively high level of formal surveillance of fishing activities in the island group. Daily management, surveillance and enforcement of the GBRMP is undertaken by government agencies, such as the Queensland Parks and Wildlife Service, Queensland Boating and Fisheries Patrol, the Queensland Water Police, the Australian Federal Police and the Australian Customs Service vessels and aircraft (Davis et al. 2004). Furthermore, general compliance with Marine Park zoning and fishery regulations is generally high (Davis et al. 2004).

I surveyed 21 sites (n = 62 transects) within Marine National Park reserves and 20 sites open to fishing (n = 60 transects). Of the sites open to fishing, 11 were within fished Habitat Protection zones (n = 33 transects), and 9 sites (n = 27 transects) were within limited fishing Conservation Park zones (Figure 5.1). The protected reefs around Border and Hook Island had been zoned as MNP's for 25 years when this study was conducted. All other islands were zoned in 2004, as either MNP or limited fishing CP zones, providing 8 years of management zonation. In addition, the islands host a resort on Hayman Island and 42 boat moorings were located near survey sites in reserves (see Figure 5.1).

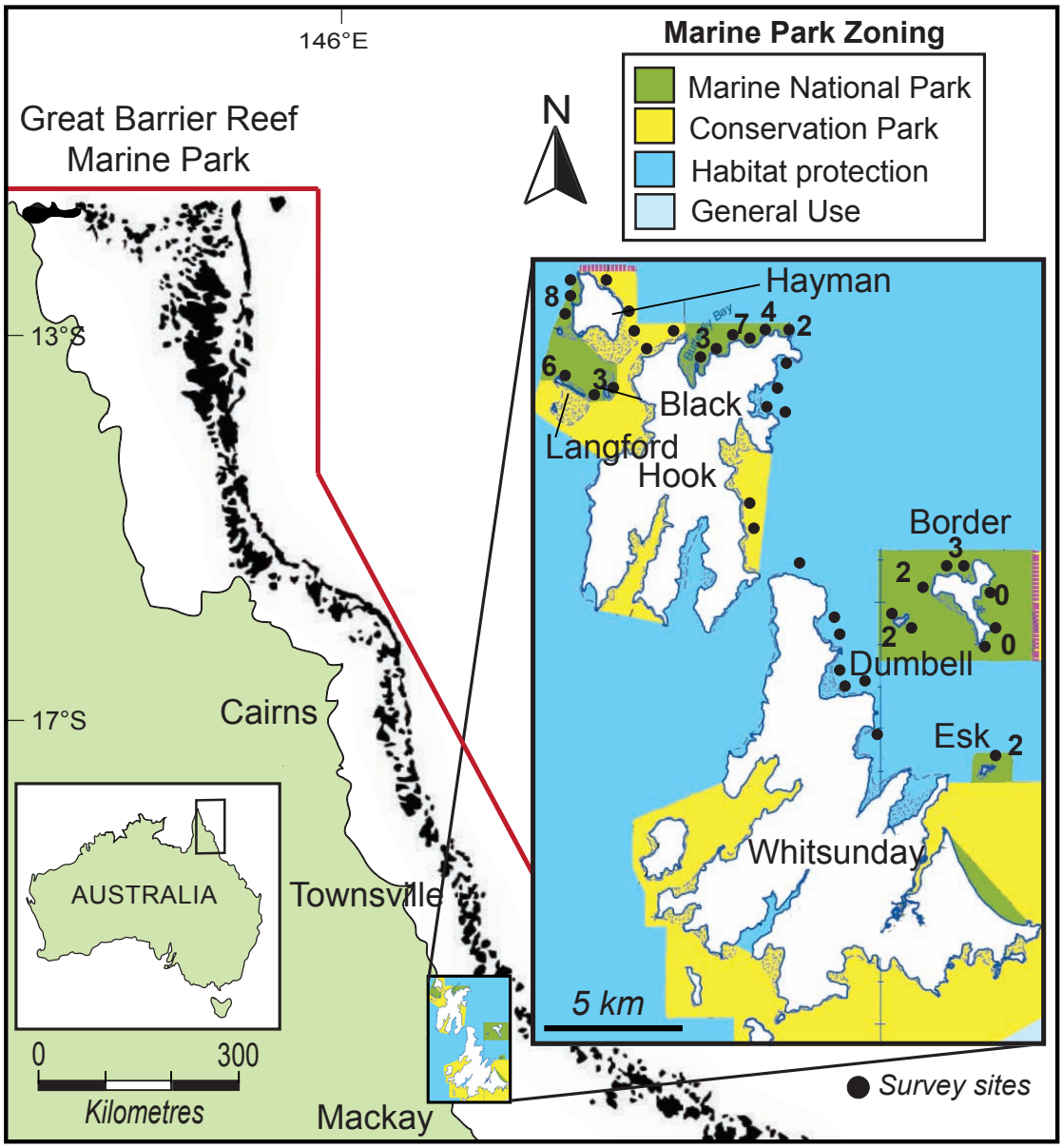


Figure 5.1 Regional map showing the location of the Whitsunday Island group and sites surveyed within each management zone in the Great Barrier Reef Marine Park. Areas shaded in green are protected Marine National Park zones or 'no-take reserves' (n = 21 sites); areas shaded in dark blue are Habitat Protection zones (n = 11 sites) and open to fishing; areas shaded in yellow are Conservation Park zones (n = 9 sites) and are open to fishing but with gear limitations (see Table 5.1). Black circles indicate survey sites. Numbers in bold indicate the number of boat moorings located near the study sites.

5.2.3 Data collection

Surveys were conducted at the beginning of the austral summer in late October and November 2012.

5.2.3.1 Coral health surveys At each site, three 15 x 2 m belt transects were surveyed, corresponding to the first 15m of each concurrent UVC fish transect (see section 5.2.3.1). Transects were located at depths ranging between 6 and 12 m and were approximately 35 m apart on mid reef slopes. Within each 30 m^2 belt transect, each coral colony over 5 cm in diameter was identified to genus and further classified as either healthy (no disease observed) or affected by one or more of six diseases: black band disease (and other cyanobacterial bands), brown band disease, white syndromes, growth anomalies, skeletal eroding band and atramentous necrosis (Willis et al. 2004). As an estimate of the intensity of site use (see Chapter 3), other indicators of compromised coral health were also recorded, such as physically damaged colonies (recently exposed skeleton from breakage), the abundance and health status of corals entangled in derelict monofilament fishing line, tissue necrosis due to sediment accumulation, bleaching, non-normal pigmentation of tissue, and cuts and scars from predation by crown-of-thorns starfish and corallivorous marine snails (*Drupella* spp.) (Willis et al. 2004; Lamb & Willis 2011). Benthic coral and macroalgae cover were determined using standard line-intercept surveys along each 15 m transect by recording the length under the transect tape covered by each organism present to the nearest centimetre.

5.2.3.2 Visual census of reef fishes In order to minimize disturbance to the fish community, a modified version of the underwater visual census (UVC) technique was

used to record the abundance of fish to the species level (Williamson et al. 2004). A single observer slowly swam a 50 m transect line identifying and recording fish within 4m either side of the transect tape (a 200 m^2 total survey area per transect), while a second diver ran the transect tape behind the observer for subsequent coral health surveys, as described above. Since reef topography and habitat complexity affect the abundance of reef fish (Beukers & Jones 1998), a site complexity index (SCI) was calculated using visual estimates of rugosity and slope at each site (see Williamson et al. 2004)

5.2.3.3 Environmental data At each site, five replicate cores of the top 3 cm of bulk sediment were sampled along survey transects at intervals of approximately 10m. Each sediment core was sampled by driving a 60 ml plastic syringe with the end removed perpendicularly into the sediment in order to not disturb the layers. Replicate cores for each site were placed in a sterile 50 ml polypropylene tube and immediately placed on ice and stored at -20ºC until further use. Sediment cores were visually classified into an incremental categorical scale ranging between 1 (very coarse) and 10 (very fine): (1) 1410-2000µm; (2) 1000-1410µm; (3) 710 - 1000µm; (4) 500-710µm; (5) 250- 500µm; (6) 250-350µm; (7) 177-250µm; (8) 125-177µm; (9) 88-125µm; and (10) 62-88µm by taking the mode of 5 measurements for each sample. Working depth and water temperature at working depth were measured at each site by averaging values recorded every 5 minutes using a Sensus Ultra temperature and depth recorder (ReefNet Inc., Ontario, Canada).

5.2.4 Data analyses

All prevalence values were calculated within each 30 m^2 belt transect by dividing the number of colonies with disease or other signs of compromised health by

the total number of colonies present.

5.2.4.1 Univariate analyses Differences in pooled and individual disease types were investigated using a univariate 3-level nested analysis of variance (ANOVA), where wave exposure (sheltered versus exposed) and protection status (reserves versus fished zones) were fixed factors and site was nested into exposure and protection status as a random factor. Differences among management zones were investigated using a univariate 2-level nested ANOVA, nesting site (random factor) into management zone (MNP, HP, CP), which was treated as a fixed factor. Analyses of variance were followed by a posteriori Tukey's HSD tests when comparisons were found to be significant ($P < 0.05$). Prior to all univariate analyses, assumptions of normality (Shapiro-Wilks) and homogeneity of variance (Levene's test of homogeneity) were tested. Data were transformed to meet assumptions of normality where necessary. Univariate analyses were performed using Statistica 12 (StatSoft, Tulsa, Oklahoma, USA).

5.2.4.2 Modeling drivers of disease assemblages A variety of measures of coral and fish community structure, in combination with a number of environmental variables (Table 5.2), were modeled to evaluate their roles in ameliorating coral health. Biodiversity indices (Shannon-Weiner diversity and Pielou evenness) were calculated according to the lowest taxonomic group using the total number of individuals surveyed per transect area (coral genera per 30 m^2 and fish species per 200 m^2). Prior to inclusion in the model, each fish species was grouped into one of 12 broad functional roles on coral reef habitats: algal croppers, corallivores, territorial pomecentrids, planktivorous pomecentrids, omnivorous pomecentrids, dentrivores, benthic carnivores, excavating grazers and scraping grazers (see Appendix 1 for specific functional groupings of

species). Large and intermediate-sized predators were grouped according to their fishing pressure on the GBR: primary target predators, secondary target predators, and non-target predators (Appendix 1). In addition, the taxonomic diversity of fish assemblages (Δ) was calculated for each transect to assess the average relatedness or the degree to which species in a sample were related taxonomically, by measuring the average path length between every pair of species through a taxonomic tree (Pienkowski et al. 1998). This measurement is independent of sample size, which circumvents problems with small sample sizes confounding other measures of diversity. It has been shown to be more sensitive to disturbance effects than traditional indices such as Shannon-Wiener (Pienkowski et al. 1998).

A multivariate distance-based linear regression model (DISTLM) was used to measure the strength and significance of the relationships between coral disease assemblages and 31 predictor variables (Table 5.2; McArdle & Anderson 2001). DISTLM is robust to zero-inflated data sets and makes no assumptions about the distribution of the response variable. Regression-based models can be sensitive to variables that are correlated, therefore variables with correlations of > 0.80 were identified using draftsman's plots and excluded from the final analysis (Leathwick et al. 2006). Fish abundances were down-weighted using a fourth root transformation to account for clumped distributions of abundant schooling species (Anderson et al. 2008). Individual predictors were transformed on a case-by-case basis to meet assumptions of normality and were fitted conditionally in a step-wise manner, with tests based on 9999 permutations of the residuals under the reduced model (McArdle & Anderson 2001; Anderson et al. 2008). Because of the large number of predictor variables, model selection (to obtain the best-fit model while maintaining model parsimony) was based on Bayesian Information Criterion (BIC, Schwarz 1978).

To visualise each best-fit model, distance-based redundancy plots (dbRDA) (McArdle & Anderson 2001) were created based on the prevalence patterns between independent observations. The optimal predictor variable vector(s) (model base variables) was then overlaid as a bi-plot (Anderson et al. 2008). In addition, variables that might be responsible for any differences detected in the dbrda plots were investigated by calculating Pearson correlations with rda axes (Anderson & Willis 2003). All prevalence modeling analyses were based on zero-adjusted Bray-Curtis similarity matrices (Clark et al. 2006) and conducted using PRIMER v6 (Clark & Gorley 2006) and PERMANOVA+ (Anderson et al. 2008).

Table 5.2 Predictor variables, codes and units included in the distance-based multiple regression model (DISTLM).

Variable	Code	Description and units	Min	Max
Spatial management predictors				
Protection status	Protection status	Categorical	Fished, Reserve	
Environmental predictors				
Exposure	Exposure	Categorical	Exposed, Sheltered	
Temperature	Temp	ºC	25.9	30.4
Depth	Depth	m	4	12
Sediment grain size	Grainsize	Categorical $1 \geq$ size ≥ 10	3	10
Biological predictors				
Fish abundance	FishAbund	number per 200 m^2	45	749
Fish species diversity	FishDiv	H' index	1.2	3.4
Fish taxonomic diversity	FishTaxDiv	Δ index	66.7	98.1
Detritivores	Dent	number per 200 m^2	0	24
Algal croppers	AlgCrop	number per 200 m^2	0	112
Corallivores	Corallivores	number per 200 m^2	0	27
Benthic carnivores	BenthCarn	number per 200 m^2	1	44
Primary target predators	PrimTarg	number per 200 m^2	0	22
Secondary target predators	SecTarg	number per 200 m^2	0	176
Non-target predators	NonTarg	number per 200 m^2	1	32
Omnivorous pomacentrids	OmPom	number per 200 m^2	1	319
Planktivorous pomacentrids	PlankPom	number per 200 m^2	0	104
Territorial pomacentrids	TerrPom	number per 200 m^2	0	359
Excavating grazers	ExScarids	number per 200 m^2	0	23
Scraping grazers	ScScarids	number per 200 m^2	0	193
Benthic predictors				
Scleractinian genera diversity	CoralDiv	H' index		
Scleractinian genera richness	CoralRich	d index		
Scleractinian cover	CoralCov	%	0	96.7
Scleractinian density	CoralDens	number per 30m^2	11	6560
Acropora cover	AcroCov	%	0	31.3
Coral bleaching	Bleaching	% colonies affected	0	16.3
Coral physical damage	Damage	% colonies affected	0	10.3
Coral sediment necrosis	SedNec	% colonies affected	0	9.5
Coral *Drupella* scars	Pred	% colonies affected	0	3.3
Fishing line	Fishing line	number per 30 m^2	0	10
Macroalgae cover	MacroAlg	%	0	60

5.3 Results

5.3.1 Influence of protection from fishing on coral disease prevalence

Surveys of 80,866 scleractinian coral colonies at sites covering 3,660 m^2 of reef around the most frequently visited recreational inshore island group in the Great Barrier Reef Marine Park revealed that protection from fishing in no-take reserves has a significant impact on coral health. Overall, pooled coral disease prevalence was approximately 4 times lower in reserves (mean ± SE = 1.0% ± 0.2, range = 0% to 2.9%, 272 colonies with disease) than at sites open to fishing (4.1% ± 0.4; range = 0.7% to 8.1%, 848 colonies with disease; $F = 43.4$, $P < 0.001$; Figure 5.2, Table 5.3). Three diseases dominated disease assemblages at all sites, with skeletal eroding band (SEB) accounting for approximately 60% of all disease cases, followed by white syndromes (WS, 16% of disease cases) and brown band disease (BrB, 15% of disease cases). In each case, disease prevalence was significantly decreased within reserves where reefs are protected from fishing activities (Table 5.4). In contrast, protection from fishing did not result in significant differences in the mean prevalence of black band disease (BBD), growth anomalies (GA) or atramentous necrosis (AtN) (Table 5.3). Of these latter three diseases, black band disease (BBD) and growth anomalies (GA) were recorded at all sites, accounting for the remaining 9% of disease cases overall and AtN was recorded at very low levels (prevalence = 0.05%, 8 total cases). Disease prevalence did not differ significantly among sites differing in exposure to wave energy, either when all diseases were pooled or when they were considered individually (Table 5.3). Two measures of coral health, the prevalence of skeletal eroding band (SEB) and of all coral diseases pooled, varied significantly among sites (SEB: $F = 1.6$, $P = 0.04$; Pooled disease: $F = 1.8$, $P = 0.01$; All other diseases: $P > 0.05$).

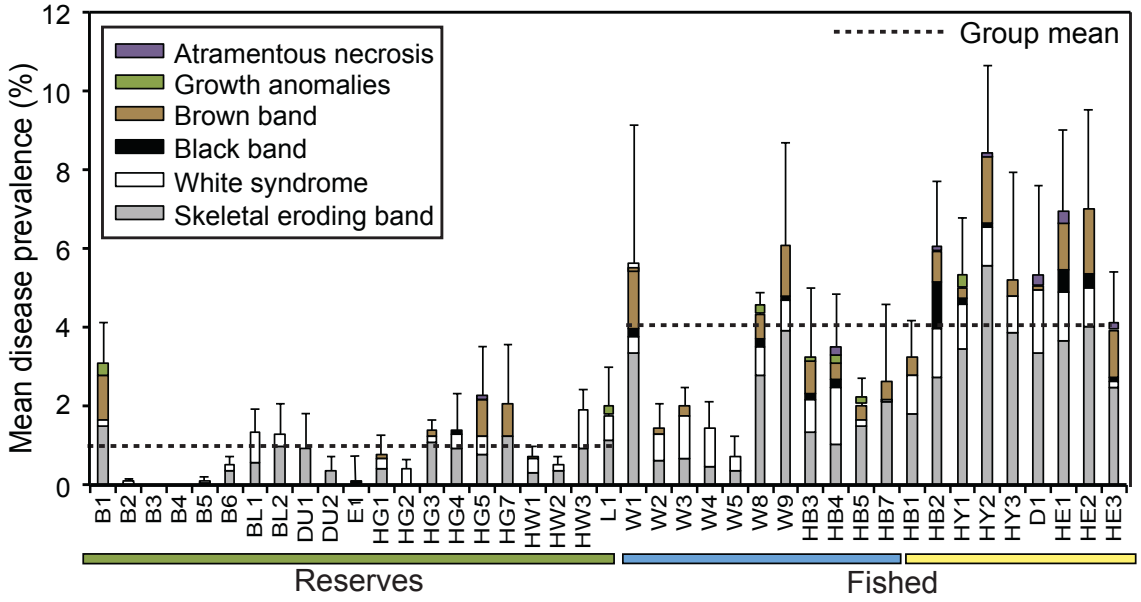


Figure 5.2 Mean prevalence (± SE) of six coral diseases at 41 sites surveyed in the Whitsunday Islands. Dashed line represents the group mean for sites protected (reserves) or exposed to fishing. Coloured axes: Green = Marine National Park Zones (reserves), blue = Habitat Protection Zones (fished), yellow = Conservation Park Zone (limited fishing). Abbreviations preceding site number: B – Border Island, BL – Black Island, DU – Dumbell Island, E – Esk Island, HG – Hook Island, HW – Hayman Island (west), L – Langford Island, W – Whitsunday Island, HB – Hook Island (southeast), HY – Hook Island (northeast), D – Deloraine Island, HE – Hayman Island (east). n = 3 replicate transects per site.

Table 5.3 Mean prevalence[a] of coral disease at sites within reserves and sites open to fishing, and results (F-statistics) of univariate 3-level nested analysis of variance (ANOVA) comparing disease prevalence between sites differing in protection status (fished vs protected), wave exposure (sheltered versus exposed) and among replicate sites nested within these two factors (all replicate site comparisons were significant; results not shown). n: denotes number of transects. Colonies surveyed = 80,866

	Reserve (n = 62)	Fished (n = 60)	ANOVA		
Variable	Mean ± SE prevalence	Mean ± SE prevalence	Exposure	Protection status	Exposure x Protection Status
Total disease	0.98 ± 0.15	4.05 ± 0.36	3.6 (ns)	43.4 (***)	< 0.01 (ns)
Skeletal eroding band	0.55 ± 0.09	2.34 ± 0.25	2.7 (ns)	35.0 (***)	0.2 (ns)
Brown band	0.14 ± 0.06	0.65 ± 0.09	0.4 (ns)	11.7 (**)	0.3 (ns)
Black band	0.01 ± 0.007	0.16 ± 0.05	1.5 (ns)	2.3 (ns)	0.2 (ns)
White syndrome	0.24 ± 0.05	0.81 ± 0.08	4.0 (ns)	30.2 (***)	0.1 (ns)
Growth anomalies	0.02 ± 0.01	0.06 ± 0.02	1.3 (ns)	2.1 (ns)	0.03 (ns)
Atramentous necrosis	-	0.05 ± 0.02	0.03 (ns)	3.6 (ns)	0.2 (ns)

[a] Mean prevalence calculated as the percentage of colonies with disease for each disease type or as a percentage of the total number of diseased corals per transect. Analyses performed on data transformed to the square root. ***: $p < 0.001$; **: $p < 0.01$, *: $p < 0.05$; ns: not significant

5.3.2 Influence of no-take reserves on fish and benthos

Three indicators of disturbance, i.e. bleaching, coral damage, and the abundance of derelict fishing line, were significantly higher in fished locations compared to reserves (Table 5.4). However, protection from fishing did not significantly influence mean coral density, richness or cover (Table 5.4). Taxonomic diversity of reef fish species (totals recorded across all sites: 238 species, 17 families, 12 functional groups), as well as the densities of coral trout and plankivorous pomecentrids were all significantly higher inside reserves (Table 5.4). All other benthic characteristics, additional indicators of coral compromised health, and fish functional groups and assemblages did not differ significantly between reserves and fished zones (Table 5.4).

Table 5.4 Comparisons of included model variables between reserves (n = 62 transects) and fished locations (n = 60 transects). Results (F-statistics) of a 2-level nested ANOVA comparing abundance or disease prevalence between reserve status (fished vs protected) and replicate sites (nested within reserve status).

Variable	Reserve Status		ANOVA		**Difference**
	Reserve mean ± SE	Fished mean ± SE	Status	Site (Status)	
(A) Coral Assemblages and cover					
Coral density	740.3 ± 154.6	582.7 ± 145.8	0.1(ns)	9.6(***)	No
Coral genera diversity	2.4 ± 0.2	2.6 ± 0.2	1.3(ns)	1.9(**)	No
Coral genera richness	0.6 ± 0.3	0.7 ± 0.2	0.6(ns)	1.6(*)	No
Total coral cover	26.3 ± 1.9	26.5 ± 2.4	0.4(ns)	5.7(***)	No
Acroporidae	5.3 ± 0.9	7.1 ± 0.9	1.1(ns)	3.4(***)	No
Agaricidae	1.3 ± 0.3	1.6 ± 0.7	0.02(ns)	1.7(*)	No
Faviidae	4.5 ± 0.8	3.7 ± 0.6	1.5(ns)	5.6(***)	No
Fungidae	0.1 ± 0.05	0.1 ± 0.04	0.03(ns)	1.0(ns)	No
Merulinidae	0.4 ± 0.1	0.9 ± 0.3	0.8(ns)	4.7(***)	No
Mussidae	0.7 ± 0.2	0.5 ± 0.1	1.3(ns)	1.5(ns)	No
Oculiniidae	1.0 ± 0.7	0.3 ± 0.07	0.5(ns)	2.6(***)	No
Pectiniidae	1.0 ± 0.3	1.1 ± 0.3	0.5(ns)	5.4(***)	No
Pocilloporidae	0.9 ± 0.2	0.9 ± 0.3	0.009(ns)	1.3(ns)	No
Poritidae	10.4 ± 2.0	9.7 ± 2.5	0.002(ns)	8.2(***)	No
Siderastreidae	0.2 ± 0.09	0.2 ± 0.1	0.3(ns)	1.1(ns)	No
Other hard coral	0.4 ± 0.1	0.2 ± 0.1	2.4(ns)	3.1(***)	No
(B) Benthic characteristics and coral health					
Sediment grain size	8.1 ± 0.5	6.9 ± 0.4	1.4(ns)	1.7(*)	No
Coral physical damage	0.9 ± 0.1	3.5 ± 0.9	8.2(**)	1.6(*)	Fished > Reserve
Coral sediment necrosis	2.2 ± 0.3	2.4 ± 0.3	0.8(ns)	3.6(***)	No
Derelict fishing line	2.1 ± 1.5	6.5 ± 1.8	6.8(**)	3.0(***)	Fished > Reserve
Site complexity	13.3 ± 0.6	12.6 ± 0.7	0.2(ns)	7.6(***)	No
Macroalgae cover	2.5 ± 0.7	4.3 ± 1.3	1.9(ns)	5.4(***)	No
Coral *Drupella* scars	0.5 ± 0.2	0.9 ± 0.2	3.5(ns)	5.8(***)	No
Coral bleaching	0.6 ± 0.08	1.0 ± 0.2	5.0(*)	1.7(ns)	Fished > Reserve
(C) Fish assemblages and functional groups					
Density of coral trout	2.7 ± 0.4	1.0 ± 0.2	13.2(***)	1.1(ns)	Reserve > Fished
Taxonomic diversity	71.9 ± 1.1	76.0 ± 0.9	5.1(*)	2.9(***)	Reserve > Fished
Total species	37.5 ± 0.9	37.1 ± 0.5	0.004(ns)	3.2(***)	No
Total individuals	281.8 ± 13.5	271.6 ± 16.2	0.2(ns)	4.2(***)	No
Algal croppers	6.8 ± 1.6	4.5 ± 0.5	1.0(ns)	2.1(ns)	No
Scraping grazers	17.9 ± 1.9	18.1 ± 2.8	0.05(ns)	3.7(ns)	No
Benthic carnivores	16.1 ± 1.0	19.1 ± 1.2	1.4(ns)	4.2(***)	No
Excavating grazers	1.5 ± 0.5	2.6 ± 0.6	3.4(ns)	1.1(ns)	No
Om. Pomacentrids	135.1 ± 8.9	144.1 ± 10.6	0.4(ns)	8.3(***)	No
Terr. Pomacentrids	11.3 ± 1.7	18.7 ± 1.8	3.6(ns)	6.4(***)	No
Plank. Pomacentrids	63.4 ± 7.1	31.4 ± 6.6	5.0(*)	3.4(***)	Reserve > Fished
Corallivores	7.1 ± 0.6	6.7 ± 0.5	0.05(ns)	3.6(***)	No
Dentrivores	2.0 ± 0.3	2.0 ± 0.3	0.03(ns)	1.7(*)	No
Other primary target pred.	2.4 ± 0.2	1.6 ± 0.3	3.7(ns)	1.3(ns)	No
Secondary target pred.	7.3 ± 2.2	11.4 ± 1.1	2.9(ns)	0.8(ns)	No
Non-target pred.	9.7 ± 0.5	11.5 ± 0.7	3.8(ns)	1.9(**)	No

Data square root transformed to meet assumptions of variance where necessary.
***: $p < 0.001$; **: $p < 0.01$; *: $p < 0.05$; ns: not significant.

5.3.3 Modeling drivers of coral disease assemblages

Three measures of reserve status and compliance, in combination with two environmental variables, were found to explain a high proportion (>40%) of the variability in coral disease assemblages on inshore reefs of the GBRMP (Table 5.5). Protection from fishing explained the greatest percentage of the variance identified in the distance-based multivariate linear model (DISTLM), accounting for 39.6% of variability in coral disease assemblages among sites (*BIC = 784.7, pseudo-F = 44.8, df = 120, P < 0.001*). Sediment accumulation causing tissue necrosis was the most important benthic variable, explaining 3.5% of the variability in disease assemblages (*BIC = 776.1, pseudo-F = 13.9, df = 119, P < 0.001*). Although significant (*P < 0.005*), the remaining variables combined, i.e. sediment grain size (Grainsize), physical damage (Damage) and the abundance of derelict fishing line, only represented a further 1.1% of variability in disease assemblages fitted by the best solution model (best solution DISTLM: *BIC = 770.5,* R^2 *= 0.45*; Table 5.5).

Table 5.5 Results of distance-based multivariate linear model (DISTLM) for coral disease prevalence showing the proportion of variation explained by significant zoning, environmental and biological variables (p < 0.005).

Axis	Percentage variation explained by individual axes			
	% explained variation out of fitted model		% explained variation out of total variation	
	Individual	Cumulative	Individual	Cumulative
Protection Status	89.70	89.70	39.65	39.65
SedNec	7.81	97.52	3.45	43.10
Grainsize	1.66	99.17	0.73	43.83
Damage	0.60	99.77	0.26	44.10
Fishing line	0.23	100.00	0.10	44.23

A distance-based redundancy analysis (dbRDA) used to visualise results of the model revealed that disease assemblages recorded on the 122 transects clearly separated along the RDA1 vector that described protection from fishing (89.7% of fitted variation; Table 5.5 & Figure 5.3). Interestingly, coral disease assemblages at fished sites with gear restrictions (Conservation Protection zones) were more distant from reserve assemblages along the RDA1 axis than assemblages at fished sites without gear restrictions (Habitat Protection zones) (Figure 5.3). Although sediment accumulation causing tissue necrosis did not differ significantly between reserves and fished groups (*F = 2.4, P = 0.07;* Table 5.4), the DISTLM analysis revealed that variation in sediment necrosis influenced coral disease assemblages across all sites, regardless of protection status (RDA2, 7.8% of fitted variation; Table 5.5 & Figure 5.3a). To determine which variables were best represented by protection from fishing and sediment necrosis, raw Pearson correlations of each significant variable identified by the model were examined for correlations with RDA1 and RDA2, respectively. Coral damage ($\rho = 0.527$) was strongly associated with coral disease assemblages in fished zones, followed by the abundance of derelict fishing line ($\rho = 0.346$) (Figure 5.3a & Table 5.6). On the second axis, smaller sediment grain sizes ($\rho = 0.487$) were more representative of disease assemblages at sites with increased sediment necrosis (Figure 5.3a & Table 5.6).

To establish which individual diseases were driving changes in the overall coral disease assemblages, raw Pearson correlations between each disease and the original RDA axes were calculated and overlaid on the dbRDA (Figure 5.3b). Because correlations for each disease were calculated without considering all other diseases contributing to the RDA axes, these results can only be used as a guide. All six coral diseases were more associated with fished sites (positive correlations with RDA1), particularly SEB ($\rho = 0.710$), BrB ($\rho = 0.553$), and WS ($\rho = 0.451$), whereas BBD, AtN

and GA were less influenced by protection from fishing (ρ = *0.273*, ρ = *0.180*, ρ = *0.117*, respectively; Figure 5.6b). Within zones open to fishing, sites with higher levels of WS had the highest levels of sediment necrosis (ρ = *0.202*). In contrast, sites with higher levels of SEB and BrB within fished zones had the lowest levels of sediment necrosis (ρ = *-0.166 and* ρ = *-0.128*, respectively; Figure 5.3b).

Table 5.6 Pearson correlations between reserve status or environmental variables and each of the two redundancy axes (RDA).

	Protection status	SedNec	Grainsize	Damage	Fishing line
RDA1	-0.902	-0.007	-0.201	0.527	0.346
RDA2	0.034	0.856	0.487	-0.258	0.293

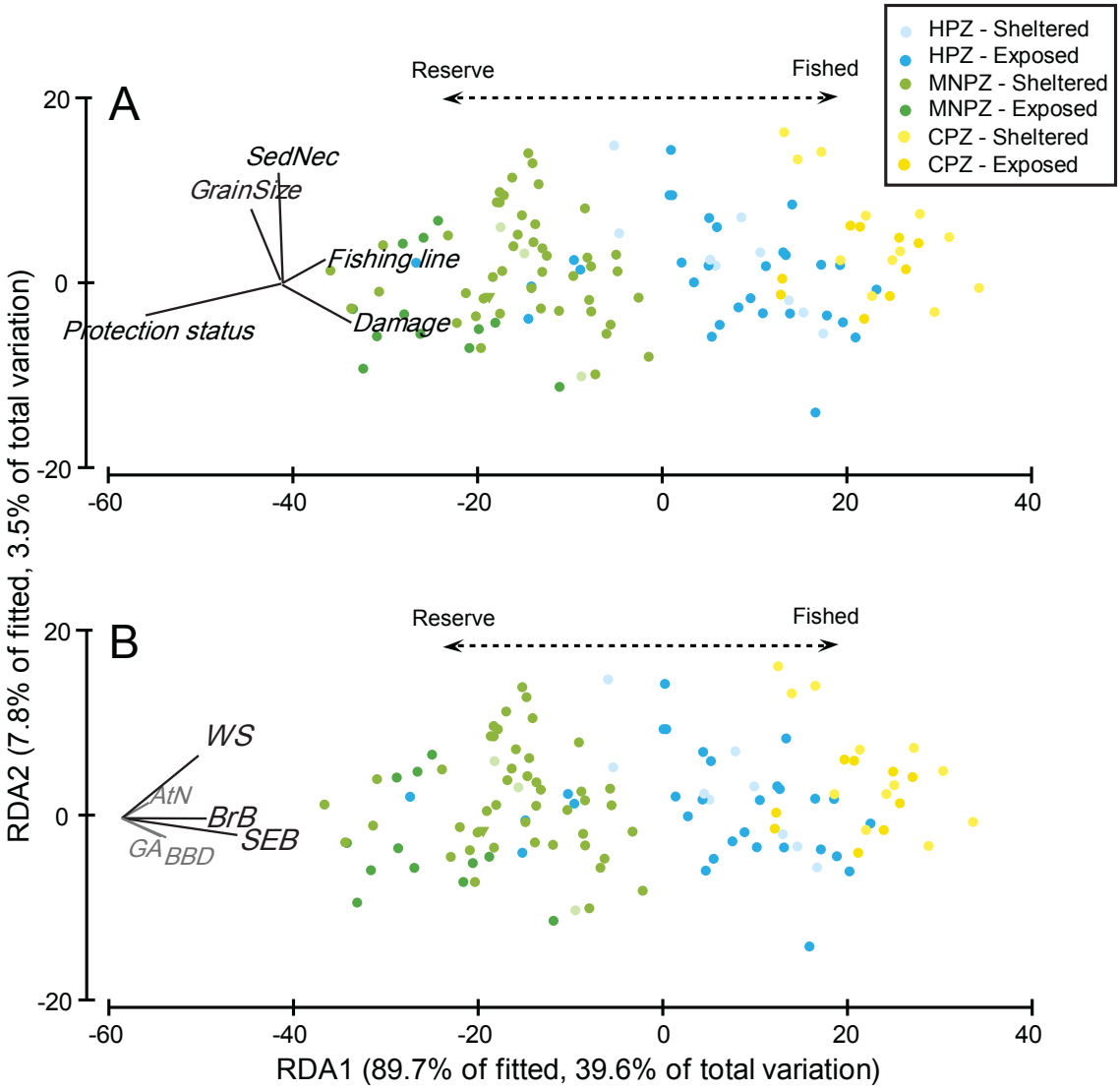


Figure 5.3 Distance-based redundancy analysis (dbRDA) visualizing the similarity in coral disease assemblages among transects in 3 management zones. Green symbols denote 'no-take' reserves (Marine National Park Zones, n = 103 transects); blue symbols denote fished Habitat Protection Zones (n = 65 transects); and yellow symbols denote limited fishing Conservation Park Zones (n = 27 transects). Vectors in (a) depict significant zoning management, environmental and biological variables (Table 5.2) forming the best-fit DISTLM identified using Bayesian Information Criterion (BIC). Vectors in (b) represent coral diseases super-imposed on the ordination as vectors (raw Pearson correlations). The length and direction of the vectors represent the strength and direction of the relationship. The separation of the survey transects indicates a strong zoning protection status gradient increasing along RDA1. Model performed on a Bray-Curtis similarity matrix. SEB = skeletal eroding band, WS = white syndromes, BBD = black band disease, BrB = brown band disease, GA = growth anomalies, and AtN = atramentous necrosis.

5.3.4 Impacts of derelict fishing line

A total of 252 scleractinian coral colonies were entangled in derelict fishing line, 92% of which were recently dead (i.e. 232 dead colonies). The majority of colonies entangled in fishing line were located in areas open to fishing (78%), with corals in these zones more likely to be affected by fishing line than corals in reserves ($\chi^2_1 = 6.6$, $P = 0.01$). Of the 8% of living colonies entangled in fishing line (n = 20), 40% also had the ciliate disease, skeletal eroding band (SEB), while the remaining 60% were considered healthy (Table 5.7). However, colonies entangled in fishing line were no more likely to have SEB than remain healthy ($\chi^2_1 = 1.6$, $P = 0.2$). Coral disease was observed on 9 genera in 6 families of reef-building corals entangled in derelict fishing line, primarily on branching, bushy and corymbose morphologies (Table 5.7).

Table 5.7 Number of live colonies entangled in derelict fishing line (FL) grouped by genera or coral morphology and resulting health status. SEB = Skeletal eroding band.

Coral genus and morphology	FL + SEB	FL + Healthy
Acropora - corymbose		1
Acropora - branching	2	2
Echinopora - branching		1
Galaxea - plating	1	
Leptastrea - massive	1	
Montipora - plating		1
Pavona - branching		1
Porites - branching	2	2
Seriatopora - branching	1	2
Stylophora - branching	1	2
Total (%)	**8** (40%)	**12** (60%)

5.3.5 Influence of fishing gear limitations on coral disease prevalence

When the presence or absence of gear restrictions was included in analyses, thereby dividing management zones open to fishing into Conservation Protection zones (with gear restrictions) and Habitat Protection zones (without gear restrictions), pooled coral disease prevalence differed significantly among the three management zones surveyed ($F = 42.3$, $P < 0.001$). Unexpectedly, fished zones with gear restrictions (CP Zones) had an approximately two-fold increase in pooled coral disease prevalence (mean ± SE = 5.4% ± 0.5) compared to fished zones without gear restrictions (HP Zones, 2.9% ± 0.2; Tukey's HSD: $P < 0.05$). Predictably, disease prevalence was five-fold greater in fished (CP) zones with gear restrictions compared to reserves (1.0% ± 0.2; Tukey's HSD: $P < 0.05$). The prevalence of skeletal eroding band (SEB) and white syndromes (WS) differed significantly in all comparisons between each pairing of the three management zones, with prevalence significantly greater in fished sites (in both CP and HP zones) compared to reserves (Tukey's HSD: all $P < 0.05$; Figure 5.4 & Table 5.8). The prevalence of brown band (BrB) also differed among management zones in the overall ANOVA and was significantly greater in fished sites, but BrB prevalence did not differ between fished sites with gear restrictions (CP zones) versus without restrictions (HP zones) (Tukey's HSD: all $P > 0.05$). The prevalence of black band disease (BBD), growth anomalies (GA) and atramentous necrosis (AtN) did not differ significantly in any comparison, either among management zones or between fished sites with versus without gear restrictions (Tukey's HSD: all $P > 0.05$; Figure 5.4 & Table 5.8).

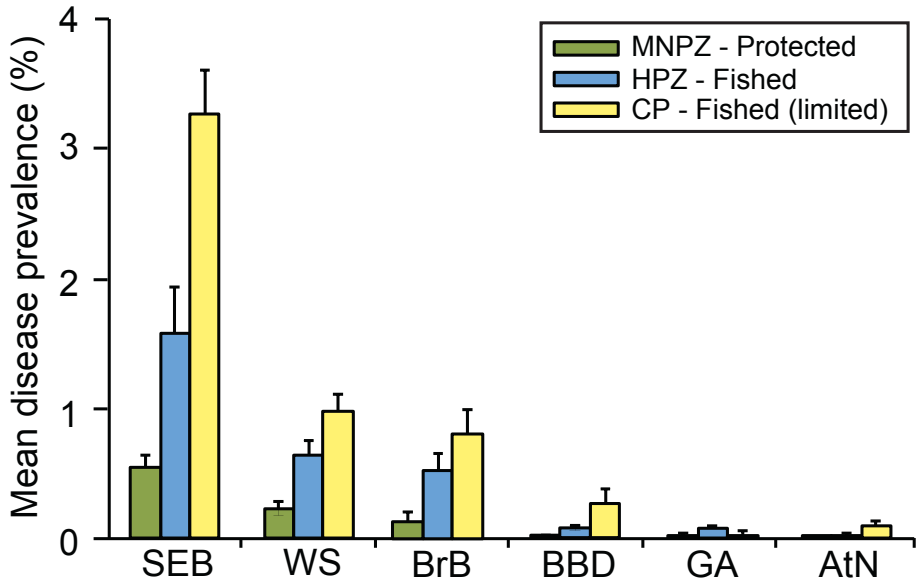


Figure 5.4 Mean prevalence of six coral diseases among three management zones in the Whitsunday Island region. SEB = skeletal eroding band, WS = white syndromes, BBD = black band disease, BrB = brown band disease, GA = growth anomalies, and AtN = atramentous necrosis. MNPZ – Marine National Park Zone (n = 62 transects), HPZ – Habitat Protection Zone (n = 33 transects), CP – Conservation Park Zone (n = 27 transects). Permitted activities in each zone are listed in Table 5.1). Error bars represent standard error of the mean.

Table 5.8 Mean prevalence of six coral diseases in three management zones in the Whitsunday Island region (see Table 5.1). n = number of transects per management zone. SE = standard error of the mean, SEB = skeletal eroding band, WS = white syndromes, BBD = black band disease, BrB = brown band disease, GA = growth anomalies, and AtN = atramentous necrosis. Number of colonies surveyed = 80,866.

	Reserves (60 transects)	Fished (33 transects)	Limited fishing (27 transects)	ANOVA	
	Prev ± SE	Prev ± SE	Prev ± SE	Zone	Site (Zone)
SEB	0.5 ± 0.1	1.5 ± 0.3	3.3 ± 0.3	35.2 (***)	32.1 (ns)
WS	0.2 ± 0.05	0.6 ± 0.1	1.0 ± 0.1	18.7 (***)	0.2 (ns)
BrB	0.1 ± 0.06	0.5 ± 0.2	0.9 ± 0.2	8.5 (***)	1.2 (ns)
BBD	0.01 ± 0.007	0.08 ± 0.03	0.3 ± 0.1	2.3 (ns)	1.3 (ns)
GA	0.02 ± 0.02	0.07 ± 0.03	0.03 ± 0.02	1.4 (ns)	1.2 (ns)
AtN	-	0.02 ± 0.02	0.1 ± 0.1	1.9 (ns)	0.8 (ns)

[a] Mean prevalence calculated as the percentage of colonies with disease for each management zone as a percentage of the total number of corals per transect. Analyses performed on data transformed to the square root. ***: $p < 0.001$; **: $p < 0.01$; *: $p < 0.05$; ns: not significant.

5.3.6 Measures of the intensity of site use among management zones

Derelict monofilament fishing line was recorded in all three management zones, but its mean abundance differed significantly among zones ($F_{zone} = 5.8, P = 0.006$; Figure 5.5a), and also among sites within management zones ($F_{site\ (zone)} = 2.7, P < 0.001$). Mean abundance of fishing line tripled between reserves and fished HP zones (2.1 ± 1.5 and 4.2 ± 1.8, respectively), however the difference was not significant (Tukey's HSD: $P > 0.05$; Figure 5.5a). The mean abundance of derelict fishing line was four-fold higher at sites with gear restrictions (8.0 ± 1.7) compared to reserves, but contrary to expectations, two-fold higher at fished sites with gear restrictions than at sites without gear restrictions (HP zones) (Tukey's HSD: both $P < 0.05$). Four out of nine sites located in CP zones had the highest abundance of derelict fishing line recorded throughout the study ($n \geq 8$ total pieces per site).

Mean prevalence of coral damage (recently exposed white skeleton) differed significantly among the three management zones ($F_{zone} = 13.4, P < 0.001$; Figure 5.5b), but not among sites within management zones ($F_{site\ (zone)} = 1.2, P = 0.26$). Similar to patterns observed for the abundance of derelict fishing line, the mean prevalence of coral damage did not differ between reserves and fished HP zones (0.9 ± 0.1 and 2.1 ± 0.7, respectively; Tukey HSD: $P > 0.05$; Figure 5.5b), but again unexpectedly, mean prevalence of colony injury doubled at sites with gear restrictions (CP zones, 4.6 ± 0.6) compared to fished HP zones and reserves (Tukey HSD: both $P < 0.05$). Mean prevalence of physical injury at 67% of sites in CP zones was higher than the maximum prevalence of injury in the other two zones (maximum: reserves = 2.3%, HPZ = 3.9%).

Mean densities of coral trout differed significantly among the three management zones ($F_{zone} = 9.0, P < 0.001$; Figure 5.5c), but not among sites within management zones ($F_{site\ (zone)} = 1.1, P = 0.41$). Mean density of coral trout in reserves (2.7 ± 0.4) was

twice that of fished HP zones (1.4 ± 0.2), however this difference was not statistically significant (Tukey's HSD: *P > 0.05;* Figure 5.5c). Compared to fished HP zones and reserves, mean density of coral trout at sites with gear restrictions (CP zones, 0.6 ± 0.2) was 1.5- and 3-fold lower, respectively (Tukey's HSD: both *P < 0.05)*.

Sediment necrosis did not differ among management zones (*F = 0.6, P = 0.55*), however it did differ significantly among sites within management zones (*F = 3.3, P < 0.001*).

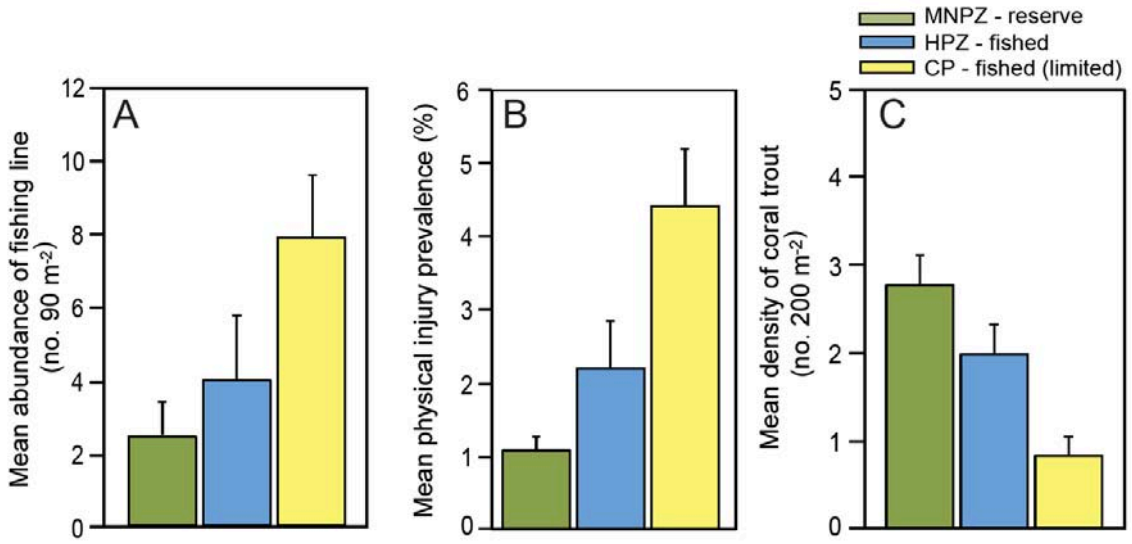


Figure 5.5 Indicators of use-levels in the Whitsundays Island region. Mean (a) abundance of derelict fishing line, and (b) prevalence of coral physical injury (recently exposed white skeleton), and (c) density of coral trout (*plectopomus* spp.). Marine National Park Zone (MNPZ, n = 62 transects), Habitat Protection Zone (HPZ, n = 33 transects), Conservation Protection Zone (CPZ, n = 27 transects). Error bars represent standard error of the mean.

5.3.7 Spatial patterns of use-levels and coral disease

Within marine reserves, there was a significant positive association between the number of permanent boat moorings located near the sites surveyed (within a 200 m radius) and both the prevalence of tissue necrosis from sediment accumulation and white syndromes (*r = 0.63, P = 0.002* and *r = 0.66, P < 0.001*, respectively; Figure 5.6a-b). There were

no additional significant associations between the number of boat moorings and other use-level indicators or the prevalence of individual or pooled diseases found within reserves (*r < 0.35, P > 0 .05*).

Within Conservation Park Zones (i.e. gear restricted), there was an unexpected negative association between the distance (km) from the nearest marine reserve boundary and the mean prevalence of three indicators of coral health: skeletal eroding band disease (SEB), coral injury, and the abundance of derelict fishing line (SEB: *r = -0.68, P = 0.04*; injury: *r = -0.77, P = 0.02*; fishing line: *r = -0.65, P = 0.05*; Figure 5.7a-c). Conversely, the density of coral trout was significantly and positively associated with distance (km) from the nearest marine reserve boundary (*r = 0.67, P = 0.007*; Figure 5.7d). Within HP zones, there were no significant associations between distance to the nearest marine reserve boundary and indicators of use-level or of the prevalence of either individual or pooled diseases (*r < 0.4, P > 0 .05*).

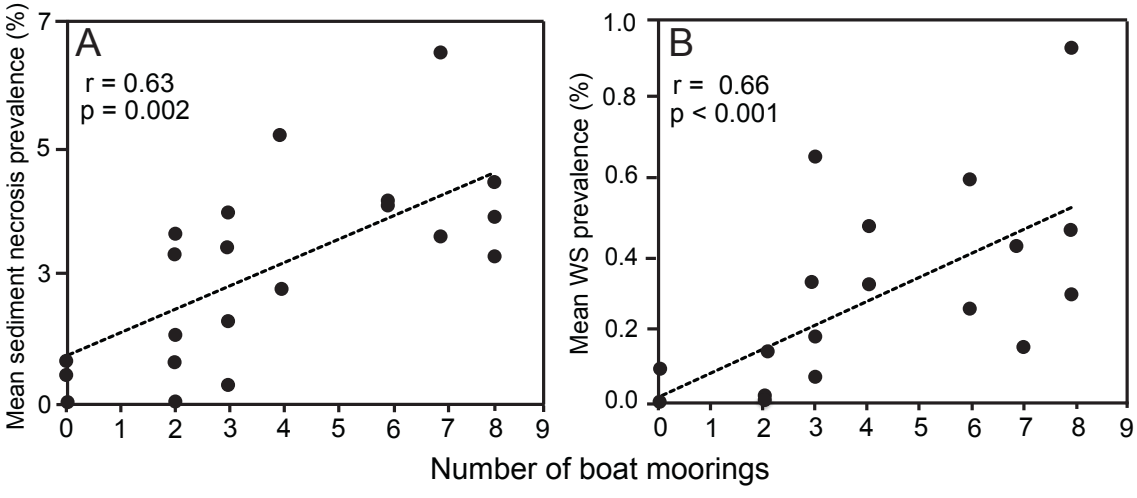


Figure 5.6 Associations between the number of boat moorings within a 200 m radius of the site and the mean (a) prevalence of white syndromes (WS), and (b) prevalence of coral tissue necrosis due to sediment in 'no-take' Marine National Park Zones (Table 5.1) in the Whitsundays Island region of the GBRMP (n = 21 sites). Surveyed colonies = 45,894.

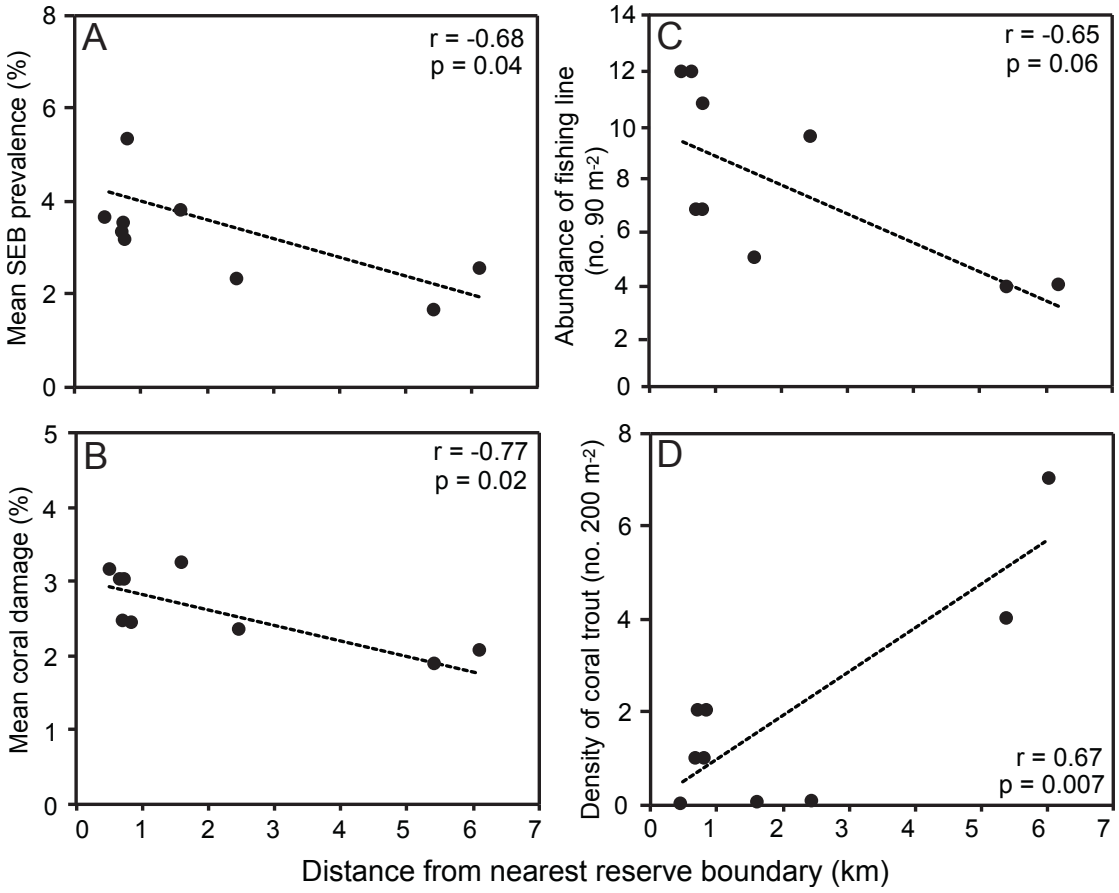


Figure 5.7 Associations between the distance (km) from the nearest marine reserve boundary and the mean (a) prevalence of skeletal eroding band disease (SEB), (b) prevalence of recent coral injury (recently exposed white skeleton), (c) abundance of derelict fishing line (d) density of coral trout (*Plectropomus* spp.) in gear restricted Conservation Park Zones (CP, Table 5.1) in the Whitsundays Island region of the GBRMP (n = 9 sites with 3 replicate transects each). Surveyed colonies = 7,602.

5.4 Discussion

Marine reserves ameliorate coral disease

The four-fold lower levels of coral disease within zones protected from fishing than in zones with fishing permitted provide clear evidence that marine reserves are a promising approach for managing coral disease within the Great Barrier Reef Marine Park (GBRMP). Additionally, significant reductions in levels of the three most abundant diseases, i.e. skeletal eroding band, white syndromes and brown band disease, offer insights into the major mechanisms by which marine reserves mitigate coral disease. Specifically, increasing abundance of these three diseases with either breakage (skeletal eroding band and brown band disease) or sediment necrosis (white syndromes) strongly suggests that reduction in activities associated with fishing underlies the capacity of marine reserves to ameliorate coral health. This conclusion is further supported by analyses showing that, of the 31 factors analysed, protection from fishing is the single dominant factor predicting coral disease assemblages at the 41 sites surveyed on the inshore reefs of the GBRMP. Given that colony damage and the abundance of derelict fishing line were the major factors driving dissimilarities between reserves and sites open to fishing, I conclude that it is the activity of fishing itself, rather than changes in fish communities, that accounts for the striking differences in disease levels between reserves and zones open to fishing.

The nature of reef fish assemblages has limited influence on coral health

The lack of a significant relationship between coral disease and either the abundance or diversity of fish assemblages suggests that characteristics of reef fish assemblages do not exert a major influence on coral health. My results are contrary to the conclusion of a recent study on coral health within MPAs in the Philippines, which suggested that taxonomic diversity of reef fish assemblages may be the principal driver

of differences in coral disease prevalence between reserves and fished areas (Raymundo et al. 2009). The different conclusion reached in this study may reflect regional disparities in the primary reef fish species targeted by fishers and their role in ecosystem functioning. For example, the removal of major groups of herbivores, such as those targeted in the Philippines (Abesamis et al. 2006), increases the vulnerability of coral communities to phase shifts towards reefs dominated by algae (Bellwood et al. 2006), which are known to enhance dissolved organic carbon, thereby stimulating microbial growth (Haas et al. 2011). However herbivorous fishes face insignificant fishing pressure on the GBR (Bruno et al. 2009). Increases in the abundance of coral trout within GBR reserves, combined with similarity in the abundance of other predatory piscivorous reef fish in fished and protected zones, highlight the selective targeting of fishers on the GBR, a pattern also found in earlier studies (Williamson et al. 2004). Since piscivorous fishes are perhaps the most significant consumers of fish biomass on coral reefs (Grigg et al. 1984), coral trout may play a large role in the top-down control of fish assemblages in this region (Graham et al. 2003), potentially causing the reduced taxonomic diversity of reef fish on unprotected reefs recorded in this study. Although ecological similarity of taxa is often used as an indicator of reef degradation (Graham et al. 2006), fish community alterations and differences in the densities of coral trout did not emerge as significant influences driving dissimilarities in coral disease assemblages between protected and fished zones in my study.

Mechanisms by which fishing activities impact coral health

The veracity of my conclusion that increases in the abundance of both derelict fishing line and injured corals in fished zones compared to adjacent unfished zones is a primary mechanism underlying enhanced coral disease prevalence is supported by the

results of studies in a number of other reef regions that have linked fishing line with increased coral breakage (Basvestrello et al. 1997; Scheyler & Tomalin 2000; Yoshikawa & Asoh 2004; Asoh et al. 2006; Chapter 3). Injuries are generally assumed to enhance coral disease transmission because they provide entry wounds for pathogens, disrupt the antibacterial mucus layer on the surface of corals (Ritchie 2006), and redirect energy towards healing processes (Mydlarz et al. 2006). For example, injury increased the susceptibility of corals to colonization by the ciliate *Halofolliculina corallasia*, the pathogen that causes skeletal eroding band disease (Page & Willis 2008; Chapter 3), and similarly, ciliate infections causing brown band disease have been associated with coral tissue injury and predation by both the coral-feeding gastropod *Drupella* spp. (Nicolet et al. 2013) and the crown-of-thorns starfish, *Acanthaster planci* (Nugues & Bak 2009). Histologically, ciliates are typically associated with necrotic and fragmented tissues, indicating an active invasive process that allows little opportunity for the host to regenerate tissues (Work et al. 2012). Wound repair in corals takes days to several weeks depending on the size of the wound (Lester & Bak 1985; Work & Aeby 2010; Palmer et al. 2011), thus wounds provide an extended period of time for the establishment of ciliate infections. In this study, the two most prevalent diseases elevated as a result of fishing, skeletal eroding band and brown band disease, are associated with ciliate-mediated tissue loss. The dominance of ciliate diseases in fished zones provides further corroborative evidence that fishing activities which cause wounding and breakages have a major impact on coral health.

Mean levels of damaged colonies in this study were markedly lower than levels reported on more heavily fished reefs in Hawaii (up to 44% of *Pocillopora meandrina*; Asoh et al. 2006), South Africa (up to 60% of reef organisms; Scheyler & Tomalin 2000) and the Mediterranean reefs of northeastern Italy (up to 40% of gorgonian

colonies, Basvestrello et al. 1997), highlighting the extent of damage that unregulated fishing can have on coral assemblages and the risk of disease outbreaks if fishing activities are not managed. Since derelict fishing line can drift or become dislodged, management programs such as reef cleaning by divers have been suggested as a way to reduce impacts on coral health (Asoh et al. 2006). However, removal of fishing line directly from entangled corals could increase tissue damage, hindering recovery from injury (Bak et al. 1977). Given evidence that larger injuries (< 30 mm^2) were noted to increase the likelihood of infection by brown band ciliates from 25% to 40% in experimental trials (Nicolet et al. 2013), enlarging the initial coral injuries should be avoided. Interestingly, corals entangled in fishing line did not have signs of brown band disease in this study, instead 40% of entangled colonies suffered from skeletal eroding band disease. It is possible that lesions caused by fishing line entanglement may not be severe enough for colonization of ciliates associated with brown band disease, or that ciliates associated with skeletal eroding band are better competitors under the environmental conditions corals experienced during my surveys. Another possibility is that the more rapid progression rates of brown band disease (approximately 2 cm day^{-1}; Boyett 2006, Nicolet et al. 2013) compared with skeletal eroding band disease (up to 0.3 cm day^{-1}; Page & Willis 2008) resulted in complete colony mortality prior to surveys, an interpretation consistent with the 92% level of whole colony mortality found for entangled corals.

Necrosis from sediment sitting on the surface of coral tissues and the fineness of sediment grain sizes were significant factors driving variation in disease assemblages, particularly white syndromes in fished zones. Because the prevalence of sediment necrosis did not differ between reserves and fished sites, it is likely that exposure to finer sediment grain sizes coupled with mechanical damage as a result of fishing

activities caused the increased prevalence of white syndromes outside of reserves. Outbreaks of white diseases have been associated with mechanical sources of damage and stress in the past. For example, high levels of a white plague-like disease occurred one month after high damage associated with the passage of two hurricanes on reefs surrounding the Caribbean island of Navassa (Miller & Williams 2007) and within weeks of the passage of a hurricane in Puerto Rico (Bruckner & Bruckner 1997). More recently, a multispecies rapid tissue loss disease was associated with colony fragmentation and physical contact with sediment in the Virgin Islands (Brandt et al. 2013), implying a direct link with injury and sediment. Not only are fine sediment fractions the most difficult for corals to expel and remove (Weber et al. 2006), fine sediments are often positively correlated with total organic carbon content (De Falco et al. 2004). In experimental studies, elevated organic carbon contributed to disease development and mortality of corals, signifying that coral pathogens are carbon-limited (Kuntz et al. 2005; Kline et al. 2006). Taken together, the multiple lines of evidence discussed above suggest that physical disruption of coral tissue as a result of fishing activities, in addition to reductions in energy resources as a consequence of allocating a major proportion of resources to sediment removal and wound-healing processes, increase the probability of infection.

Spatial dynamics of site-use and its influence on coral disease prevalence

The use of moorings within reserves is a doubled-edged sword. There is clear evidence that coral reefs with high intensities of boating activities generally have higher levels of broken corals as a result of anchor damage (Dinsdale 2004), thus damage from anchoring has been identified as a management problem on the GBR. Accordingly, the use of permanent moorings is encouraged within reserves in the Whitsundays, ensuring

that visitors have access to bays while concurrently protecting corals from breakage associated with anchoring. Since coral injury, on average, was less than 1% inside reserves (compared to an average of 3.5% in fished zones), the use of moorings appears to be a successful management strategy to prevent damage and diseases associated with coral injury. However, increases in both coral tissue necrosis from sediment and white syndromes associated with the number of nearby boat moorings inside reserves suggest that concentrating recreational activities around promoted infrastructure negatively affects the health of corals. Analogous to many terrestrial parks, areas with limited infrastructure and facilities often incur higher levels of continuous visitor pressure and road traffic near easily accessible and well-advertised sites (Leung & Marion 2000), resulting in higher levels of impacts. These results suggest that areas with fewer than 3 moorings nearby had very low levels of white syndromes compared to areas with 3 or more moorings. Similarly, corals with sediment-associated tissue necrosis were also more likely to suffer from white syndromes in Thailand (Chapter 3). Therefore, limiting levels of turbidity and sediment accumulation on coral tissues by decreasing the concentration of boat moorings appears to be a viable option to reduce disease associated with boat traffic.

Gear restrictions, such as limitations to the number of fishing poles allowed, have been suggested as a practical and effective management strategy for reducing coral damage and entanglement associated with line fishing (Asoh et al. 2006). However, my study unexpectedly revealed that coral disease prevalence, coral damage and derelict fishing line all increase significantly in areas open to fishing with gear restrictions compared to those without gear restrictions. It is plausible that fishers perceive stocks in zones without gear restrictions to be more depleted and therefore consciously avoid them. Ease of accessibility is another factor that can affect the amount of fishing

pressure an area will experience (Wilcox & Pomeroy 2003). For instance, Smallwood et al. (2012) noted that vessels generally disperse up to a median radius of 5 km from a boat recreation site, with a rapid decline in the number of vessels that travel greater than 10 km. The presence of a high number of boat moorings within reserves that are immediately adjacent to zones with gear limitations may explain unexpected increases in coral disease within these zones compared to other fished zones without gear limitations. This hypothesis is further supported by the observed decrease in coral disease, damage and derelict fishing line as the distance from the nearest marine reserve boundary increased.

In addition to ease of access to fishing sites, if stocks are perceived to be depleted in areas outside reserves relative to those inside reserves, fishers may be attracted to the perimeters of reserves in anticipation of fish spilling over the boundaries. Concentration of fishing activities at the edge of a reserve has been observed in other reef regions (McClanahan & Kaunda-Arara 1996; Johnson et al. 1999). This behaviour by fishers is of concern from the perspective of its impacts on both coral health and reserve functioning. Increases in damage or loss of coral habitat due to disease can lead to habitat fragmentation, which has important implications for coral recruitment and movement patterns of reef fish, potentially influencing the efficacy and management objectives of a marine reserve. For example, many fishes are habitat-specific and are reluctant to disperse across 'foreign' habitats, such as sand or rubble (Chapman & Kramer 2000). For this reason, the number of fish successfully emigrating into areas surrounding a reserve is greatly influenced by the health of the bordering habitat. If fishers perceive reserves as ineffective for producing spillover, compliance and support of additional closures that benefit coral health will also be affected. Nevertheless, further analyses of within-destination movement patterns of recreational users are

needed to aid assessment of infrastructure placement, evaluation of zoning effectiveness, and zone accessibility modeling, to maximise benefits to coral health and reduce the likelihood of coral disease.

Marine reserves play a significant role in mitigating coral disease on extensively fished inshore reefs in the Great Barrier Reef Marine Park, providing an additional conservation tool to ameliorate disease in marine environments. Because humans are easier to exclude than pollutants, the results of this study suggest that marine reserves are most useful as tools for managing diseases associated with the direct effects of resource extraction and recreational use. While reserves have been proposed and implemented in many ecosystems throughout the world, this is the first study to link coral disease prevalence with spatial adaptations by fishers and recreational users to the imposition and placement of site closures and infrastructure. Assessing user adaptations is critically important to the success and efficacy of marine reserves as tools to ameliorate coral health and reduce disease prevalence.

CHAPTER 6. General Discussion

Preventing outbreaks of coral disease associated with anthropogenic activities is highly dependent on effective strategies to identify and moderate potential impacts. This thesis combines field-based surveys, remotely sensed environmental metrics and multivariate modelling to consistently demonstrate that activities associated with marine-based industries adjacent to coral reefs compromise overall coral health and elevate disease prevalence compared to reefs without these impacts (Objective A, Section 1.4; results summarised in Table 6.1). Importantly, the results presented here highlight the management value of minimising stressors associated with rapidly growing industries adjacent to coral reefs and support the utilisation of existing approaches to manage marine environments, notably marine reserves, as tools to mitigate disease impacts on reef corals.

Table 6.1 Summary of changes in the prevalence of coral disease in response to activities associated with four marine industries studied in this thesis, in comparison to reefs not exposed to these activities. ↑ : denotes increased disease prevalence

Chapter	Assessed Impact	SEB	WS	BrB	BBD	GA	AtN
2	Sites with adjacent tourism platforms	↑*	↑*	↑*	↑*	↑*	–
3	High use reef-based tourist sites	↑*	↑*	–	ns	ns	–
4	High sediment exposure days from dredging [a]	ns	↑*	ns	ns	ns	–
5	Sites open to fishing	↑*	↑*	↑*	ns	ns	ns

* = statistically significant
ns = not significant
– = not observed

[a] High sediment exposure (< 296 days) compared to sites with low exposure (> 9 days). SEB = skeletal eroding band disease, WS = white syndromes, BrB = brown band disease, BBD = black band disease, GA = growth anomalies, AtN = atramentous necrosis.

6.1 Elucidating drivers of disease associated with industries near coral reefs

By exploring the impacts of marine-based industries on coral health and disease, the present study contributes to an emerging framework that links specific indicators of stress with potential causes (Objective B, Section 1.4). Specifically, research presented in this thesis repeatedly demonstrates that excluding or reducing tourism and fishing can moderate disease prevalence by limiting damage or injury to corals (Chapters 2, 3, and 5), a finding supported by several studies which have suggested that wounds provide entry points for coral pathogens (e.g. Bak and Cirens 1981; Antonius and Riegl 1998; Page & Willis 2008; Nugues and Bak 2009; Nicolet et al. 2012). Mean levels of damaged colonies at high use sites in Thailand (4.7% ± 1.1, Chapter 3) and fished sites in Australia (3.5% ± 0.9, Chapter 5) were more than two-fold lower than levels reported on more heavily dived reefs in Egypt and Israel (approximately 10%, Riegl & Velimirov 1991; Hawkins & Roberts 1992) and ten-fold lower than levels reported on fished reefs in the Mediterranean, South Africa and Hawaii (upper levels ranging from 40 – 60%, Basvestrello et al. 1997; Scheyler & Tomalin 2000; Asoh et al. 2006). This highlights the extent of damage that unregulated tourism and fishing can have on coral assemblages and the risk of disease outbreaks if these activities are not addressed and managed in the future.

One of the most unexpected results from my research was the discovery that derelict fishing line entangled around corals increased their susceptibility to skeletal eroding band disease at high use sites in Koh Tao (Chapter 3). Although several studies have shown that fishing line can cause considerable coral mortality on reefs (Bavestrello et al. 1997; Yoshikawa & Asoh 2004), the specific mechanism was previously unclear. Since wound repair in corals takes days to several weeks depending on the size of the wound (Lester & Bak 1985; Work & Aeby 2010; Palmer et al. 2011),

fishing line entangled on corals may continue to act as a substrate from which opportunistic pathogens directly invade wounded tissue or immunocompromised corals (Mydlarz et al. 2006), providing an extended period of time for the establishment of infection. However, on fished reefs in the Great Barrier Reef, colonies entangled in fishing line were equally as likely to have skeletal eroding band as remain healthy (Chapter 5), suggesting that additional factors are needed to facilitate the development of pathogenic infections from fishing line entanglement. Like other wildlife diseases, disease in corals is likely to involve a web of causation, including nutritional and environmental stressors (Wobeser 2006), therefore further studies are required to assess environmental and/or host-related factors that might affect the disease susceptibility of corals entangled in fishing line. Nevertheless, these novel findings provide additional support for the importance of no-take marine reserves and spatially-explicit zones for tourism and fishing as tools to moderate coral disease in marine environments.

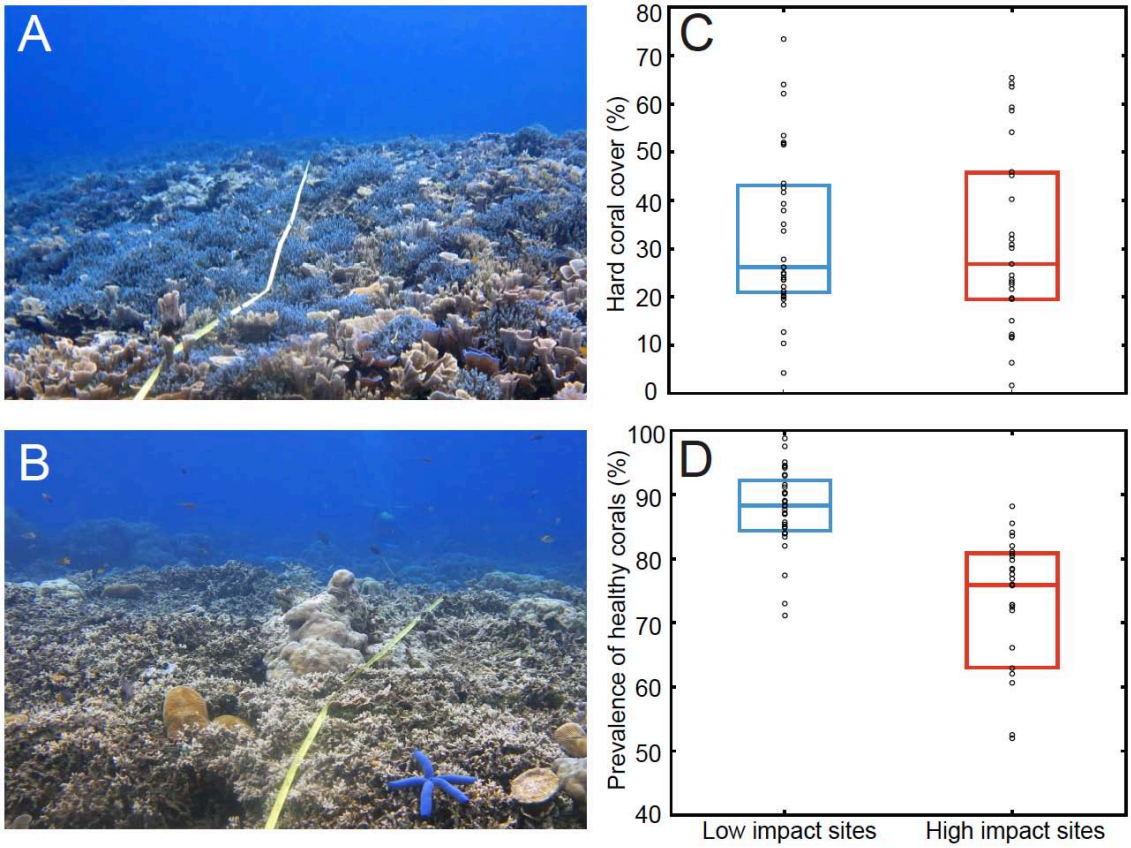
Coral susceptibility to disease as a consequence of sediment exposure was another consistent pattern identified in this thesis (Chapters 3, 4, & 5). While poor water quality has been suggested as a driver of coral disease (e.g. Bruno et al. 2003; Kline et al. 2006; Haapkyla et al. 2011; Vega Thurber 2013), little field-based evidence exists to link specific water quality parameters with different coral diseases. By comparing disease prevalence at high impact sites with levels at relatively pristine control sites distant to land-based sources of pollution, I found that exposure to sediment was the key parameter driving elevated levels of white syndromes following an 18-month dredging project in Western Australia (Chapter 4). The strong correlation I found between the prevalence of sediment-associated tissue necrosis and the prevalence of white syndromes in Koh Tao, irrespective of site use intensity (Chapter 3), further corroborates my conclusion that sediment is an important environmental driver for this

disease, potentially through promotion of increased microbial growth rates (e.g. Hodgson 1990; Kline et al. 2006). Moreover, on inshore reefs of the Great Barrier Reef, necrosis from sediment sitting on the surface of coral tissues and the fineness of sediment grain sizes were significant factors driving variation in disease assemblages, particularly white syndromes in fished zones (Chapter 5). Because the prevalence of sediment necrosis did not differ between reserves and fished sites, it is likely that exposure to finer sediment grain sizes coupled with mechanical damage as a consequence of fishing activities (see Chapter 3) caused the increased prevalence of white syndromes outside of reserves. Although strong associations between anomalously warm sea surface temperatures and elevated levels of white syndromes have shown that thermal stress is an important driver of this disease (Heron et al. 2010; Maynard et al. 2011 in Appendix 2), results from studies presented in this thesis indicate that tissue loss characteristic of white diseases may have multiple pathogeneses. Whether sediment causes coral disease by introducing pathogens or is a result of a compromised host from other stressors warrants further study. Clearly, these findings will have direct implications for managers charged with balancing economic development of tourism and coastal infrastructure with the imperative to maintain healthy coral reefs. As industrial activities near coral reef ecosystems continue to intensify in many parts of the world, insight into the types of activities that promote disease, like those revealed in this thesis, are becoming increasingly critical.

6.2 New perspectives for reef coral assessments

A major conclusion from my research, reinforced by results from all four data chapters, is that inclusion of disease monitoring with more commonly-used metrics of coral health, like percent cover, increases the likelihood of selecting appropriate

management strategies on reefs facing intensifying levels of human disturbance. Similarities in coral cover, density and community composition among sites that clearly differed in levels of coral disease and other compromised health indicators (Chapters 2, 3, 4, & 5) suggest that quantifying the prevalence of healthy colonies provides a pragmatic and general approach for targeting locations of anthropogenic disturbance and for prompting management intervention so that actions can be initiated prior to the occurrence of disease outbreaks (see Box 6.1). Coral health status reveals valuable information about reef health that is not detected by less discerning metrics, such as total coral cover (e.g. Hughes et al. 2010). Thus, the results of this thesis contribute to the emerging consensus that percent cover of live coral is of limited value for gauging ecosystem health in areas with anthropogenic disturbances (Muthiga & McClanahan 1997; Santavy & Peters 1997; Hawkins et al. 1999; Dinsdale & Harriott 2003), and further explains why total coral cover is not always a good predictor of community dynamics or ecosystem state (e.g. Tanner et al. 2009; McClanahan et al. 2011; Darling et al. 2013). Moreover, while multiple metrics of coral health may increase one's capacity to differentiate between human impacts and other drivers of disease in this study (see Sections 3.4 & 6.1), monitoring the prevalence of healthy corals can be readily implemented into existing coral survey programs with little to no additional training, thereby providing more comprehensive and meaningful reef health assessments. Inclusion of disease metrics in monitoring programs would also contribute to much-needed baseline data (e.g. Willis et al. 2004; Ward et al. 2006) to enable future detection of changes in the health of reef corals.



Box 6.1 Photographic examples of two reefs located (**a**) approximately 1 km from a village off the Sulawesi coast in Indonesia, and (**b**) adjacent to the village (< 100 m). Both sites have similar ($P > 0.05$) coral cover, density and scleractinian family composition, however the prevalence of healthy corals (no visual signs of disease or other indicators of compromised health) recorded at site (**a**) is six times greater than at site (**b**).

Summary comparison of (**c**) total hard coral cover, and (**d**) prevalence of healthy corals recorded at all sites presented in this thesis. **Low impact sites** (n = 36) include control sites without tourist platforms (Chapter 2), low use tourism sites (Chapter 3), low sediment exposure sites (Chapter 4), and no-take reserves (Chapter 5). **High impact sites** (n = 31) include tourist sites with platforms (Chapter 2), high use tourism sites (Chapter 3), high sediment exposure sites (Chapter 4), and sites open to fishing (Chapter 5). Boxes represent the interquartile range (25 to 75th percentiles), the horizontal line is the median, and black circles represent raw means for each site (n = 3 transects per site).

Photo credits: J. Lamb and W. Mangile

6.3 Are spatial management strategies a solution to help mitigate coral disease?

My research presents new and encouraging evidence that no-take marine reserves are effective tools for moderating coral disease in marine environments (Chapter 5, thesis objective C in section 1.4), and underlines the need to further evaluate the role of spatial management strategies for ameliorating coral health (Chapters 2, 3, & 5). Since activities directly associated with human use are easier to exclude than pollutants, the recurring message of my research is that spatial closures represent the most useful tool available for managing diseases associated with the direct effects of resource extraction and recreational use. Unfortunately, the global coverage of marine protected areas is strongly biased away from areas of greatest human threat (Burke et al. 2011), limiting the potential to demonstrate their effectiveness for moderating disease in areas of heavy impacts. As a result, the effectiveness of spatial closures in sustaining coral health will be largely dependent on the level of user compliance, community awareness and support that exists for their use (e.g. Alcala & Russ 2006). It is essential that users be made aware of the potential long-term benefits of sustained marine reserve protection (see Selig & Bruno 2010).

The capacity of spatial closures and marine reserves to ameliorate coral disease will also depend upon the mechanism of disease pathogenesis. Evidence that climate warming has caused profound and often complex changes in the prevalence or severity of some infectious diseases in corals (Altizer et al. 2013; Burge et al. 2014) suggests that, in some cases, environmental factors may be of greater importance in governing disease prevalence than mechanical damage. Minor differences in the prevalence of coral growth anomalies, black band disease and atramentous necrosis between reserves and fished zones (Chapter 5) suggest that environmental factors which enhance pathogen virulence are more likely to govern the abundance of these diseases than factors associated with fishing activities, which function more by compromising host

resistance. Similarly, the abundance of coral growth anomalies on reefs in Kenya was not influenced by reserve status; rather, anomalous warm water and environmental factors associated with bleaching were implicated (McClanahan et al. 2011). On protected inshore reefs in the Great Barrier Reef Marine Park, increases in the prevalence of atramentous necrosis were linked to seasonal sediment runoff and reduced salinity following monsoonal rain events (Haapkyla et al. 2011), while seasonal fluctuations of seawater temperatures and light are associated with recurrent outbreaks of black band disease (Sato et al. 2009). Thus evidence so far suggests that environmental factors are likely to override benefits provided by spatial closures for the mitigation of certain diseases.

On local scales, site closures and marine reserves have a role to play in mitigating disease caused by point sources of pollution. Coral diseases have been linked to anthropogenic sources of pollution associated with increased human population centers (Aeby et al. 2011), developed and altered coastal land (Guilherme Becker et al. 2012), sewage (Patterson et al. 2002; Voss & Richardson 2006; Sutherland et al. 2010; Redding et al. 2013), tourism (Chapters 2 & 3) and dredging (Chapter 4). Site closures could potentially alleviate disease impacts by regulating point sources of pollution, particularly discharge from terrestrial or vessel-based sources.

Since many coral reefs are located in poor, developing countries (Donner & Portere 2007), use restrictions can undermine local livelihoods and can be difficult to justify and enforce (McClanahan et al. 2005). Total prohibition on use, while perhaps ideal from a coral health perspective, may pose an unrealistically difficult burden on local communities (Cinner et al. 2009). Users are generally more likely to support restrictions on specific types of use rather than outright closures (McClanahan et al. 2005). For these reasons, spatial strategies that confine the aggregate extent of impacts of site use to a specified area are often adopted (Leung & Marion 1999). For the first

time, the impact of a spatial containment strategy on coral reefs was examined in relation to coral disease (Chapter 2: permanent tourist platforms; Chapter 3: high use tourist sites; and Chapter 5: boat mooring concentrations and reserve boundaries). Recurring patterns in elevated levels of disease in these studies suggest that spatial containment of recreational activities significantly reduces the overall health of reef corals. Therefore, dispersing visitors over extensive areas and reducing the intensity of use may be a preferred management strategy for preventing disease outbreaks. In Chapter 2, mean coral disease prevalence was generally less than 1% at several popular dive sites ('controls') that were visited irregularly or frequently rotated by operators, although their offshore locations and status as no-take marine reserves (see Chapter 5) undoubtedly also contributed to low disease prevalence. For the tourism industry, rotational dive site use or mooring exclusivity to a single operator is a conceivable solution for areas with intensifying levels of coral reef tourists. Further evaluations of the applicability of existing spatial management strategies for coral reefs are necessary to alleviate disease impacts and enhance coral health.

6.4 Future scenarios and their implications for reef coral health and disease

Global trends show that the proportion of intense tropical storms reaching categories 4 and 5 has increased significantly within the past three decades (Walsh et al. 2004; Webster et al. 2005; Klotzbach 2006), and is best explained by increasing sea-surface temperatures (Hoyos et al. 2006). Although the relationship between cyclone intensities and warming ocean temperatures is still subject to research and debate (Kossin et al. 2007), the consequences of intensifying storms on the development of coral disease outbreaks are likely to be severe, since the energy dissipated by a storm above water increases as the cube of the storm's maximum wind speed, where the diameter and transition time of the storm contribute additionally to its likely damage

(Emanuel 2005). Although the disease susceptibility of corals is increased as a result of mechanical damage (see Chapters 3 & 5; Page et al. 2008; Nicolet et al. 2012), there are no studies which directly link intensive storm damage with disease. A handful of studies have associated the passage of intense tropical storms with subsequently elevated levels of coral disease (i.e. Knowlton et al. 1981; Bruckner & Bruckner 1997; Miller & Williams 2007; Brandt et al. 2013; Haapkyla et al. 2013), but recent estimates of coral mortality associated with storm damage (e.g. Osborne et al. 2010; De'ath et al. 2012) may overlook or underrepresent disease as a driving factor for long-term declines in coral cover.

In addition to climate change, which is expected to cause heavier and more frequent precipitation in many areas (Jones et al. 2007), expanding human populations in coastal regions are expected to exacerbate pollution and sediment runoff to reefs as a result of losing natural ecological buffers on shorelines though land reclamation, port development, and natural resource exploitation (Burke et al. 2011). Research presented in this thesis stresses the importance of linkages between land and ocean for managing coral health and disease in coastal areas (see Chapter 4), however more often than not, marine and terrestrial environments are regarded as two separate ecosystems, and managed as independent entities. The impacts of coastal development on outbreaks of coral disease could be greatly reduced through effective planning and regulations. For example, methods to reduce impacts from increased sediment in waters adjacent to shoreline development include the use of silt fences, settling ponds, and vegetated buffer strips to trap sediments before they enter waterways (Rogers 1990). In addition, conserving marine littoral zones may reduce levels of disease-causing pollutants that enter coastal reefs. For instance, mangroves and constructed wetlands are often used as bio-filters for natural sewage control and have also been shown to be effective filtration systems to remove sedimentation, nutrients and organic matter (Yang et al. 2008). In

addition, bivalves could have huge potential for reducing transmission of disease to coral reefs from terrestrial sources by filtering out human pathogenic microorganisms from the water column (Shuval 2003; Faust et al. 2009). Ecosystem filtration of toxins, nutrients and pathogenic microorganisms provided by coastal mangroves, seagrasses, and bivalves has not yet been examined as a tool to alleviate coral disease. An important area for future research would be to assess the level to which these habitats sequester pollutants and ameliorate coral health along coastlines, which will provide additional support for addressing the need to protect coastal ecosystems from emerging industries near coral reefs.

6.5 Applications for early-warning systems and forecasting disease outbreaks

The implications of anthropogenic and climate-driven outbreaks of disease for society will require preemptive solutions and mitigation. Early-warning systems form an important component of any such potential solutions. For example, a forecasting system linking global ocean and atmospheric climate models to malaria risk in Botswana enabled the prediction of anomalously high probability areas so that strategies for mitigation could be initiated (Thomson et al. 2006). Forecasting is well-established in crop disease management and leads to improved timing of pesticide application and deployment of planting strategies to lower disease risk (Schaafsma & Hooker 2007). Moreover, modeling to better predict disturbance events has been shown to reduce crop losses (Garrett et al. 2013). On coral reefs, accurate forecasting programs for coral bleaching have become core to marine resilience programs (Eakin et al. 2010) and are leading to the development of climate-driven, coral disease–forecasting algorithms (Maynard et al. 2011, see Appendix 2). In Chapter 4, the combination of *in situ* coral disease assessments with satellite-derived measures of sediment exposure using freely accessible ocean colour imagery from Moderate Resolution Imaging Spectroradiometer

(MODIS) sensors, represents the first example of a direct application of such tools to understand the interface between water quality, coastal development or land-use, and coral disease. Incorporating additional remotely-derived products which can be linked with coral disease, such as wind and wave intensity from severe tropical storms, sediment or nutrients from dredging, coastal development or land-use, and/or reserve protection status, will further improve early-warning programs and facilitate the rapid-response of management actions at practical scales (e.g. < 1 km; MODIS sensor resolution). Often, the time-intensive and costly nature of disease prevalence surveys limits the geographic scale and range of field-based data collection to three or fewer reefs (Loya et al. 1984; Willis et al. 2004). Knowledge resulting from studies presented here will enable predictions of the regions where coral reefs are most vulnerable to degradation from infectious disease, which is especially critical in locations where reefs are remote and difficult to access.

6.6 Concluding remarks

Understanding the roles that human activities play in facilitating outbreaks of coral diseases is the critical first step in developing mitigation strategies to ensure the ongoing conservation of coral reefs and persistence of dependent reef-associated species and industries. As a result of research presented in this thesis, the influence of marine-based industries on coral health and disease is now more clearly demarcated. The identification of new management strategies to improve coral health will provide additional practical tools for increasing the resilience of vulnerable reef ecosystems in a rapidly developing world and changing climate.

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APPENDIX 1.

Table A.1 Fish species categorised into functional and taxonomic groups according to targeted fishery status in the Great Barrier Reef Marine Park.

Family	Species	Functional Group	Fishery status
Acanthuridae	*Acanthurus blochii*	Detritivore	Non-target
	Acanthurus dussumieri	Detritivore	Non-target
	Acanthurus grammoptilus	Detritivore	Non-target
	Acanthurus lineatus	Algal cropper	Non-target
	Acanthurus nigricauda	Detritivore	Non-target
	Acanthurus nigrofuscus	Algal cropper	Non-target
	Acanthurus xanthopterus	Detritivore	Non-target
	Ctenochaetus binotatus	Detritivore	Non-target
	Ctenochaetus striatus	Detritivore	Non-target
	Naso annulatus	Algal cropper	Non-target
	Naso brevirostris	Algal cropper	Non-target
	Naso lituratus	Algal cropper	Non-target
	Naso tuberosus	Algal cropper	Non-target
	Naso unicornis	Algal cropper	Non-target
	Prionurus microlepidotus	Algal cropper	Non-target
	Zebrasoma scopas	Algal cropper	Non-target
	Zebrasoma veliferum	Algal cropper	Non-target
Chaetodontidae	*Chaetodon aureofasciatus*	Corallivore	Non-target
	Chaetodon auriga	Corallivore	Non-target
	Chaetodon baronessa	Corallivore	Non-target
	Chaetodon citrinellus	Corallivore	Non-target
	Chaetodon flavirostris	Corallivore	Non-target
	Chaetodon lineolatus	Corallivore	Non-target
	Chaetodon lunula	Corallivore	Non-target
	Chaetodon lunulatus	Benthic carnivore	Non-target
	Chaetodon melannotus	Benthic carnivore	Non-target
	Chaetodon ornatissimus	Corallivore	Non-target
	Chaetodon plebeius	Corallivore	Non-target
	Chaetodon rafflesi	Corallivore	Non-target
	Chaetodon rainfordi	Corallivore	Non-target
	Chaetodon speculum	Corallivore	Non-target
	Chaetodon trifascialis	Corallivore	Non-target
	Chaetodon ulietensis	Benthic carnivore	Non-target
	Chaetodon vagabundus	Corallivore	Non-target
	Chelmon rostratus	Benthic carnivore	Non-target
	Coradion altivelis	Benthic carnivore	Non-target
	Coradion chrysostomus	Benthic carnivore	Non-target

	Heniochus acuminatus	Benthic carnivore	Non-target
	Heniochus monoceros	Benthic carnivore	Non-target
	Heniochus varius	Benthic carnivore	Non-target
	Parachaetodon ocellatus	Benthic carnivore	Non-target
Ephippidae	*Platax orbicularis*	Benthic carnivore	Non-target
	Platax teira	Benthic carnivore	Non-target
	Platax pinnatus	Benthic carnivore	Non-target
	Platax teira	Benthic carnivore	Non-target
Haemulidae	*Diagramma pictum*	Large predator	Secondary Target
	Plectorhinchus	Large predator	Secondary Target
	Plectorhinchus flavomaculatus	Large predator	Secondary Target
	Plectorhinchus gibbosus	Large predator	Secondary Target
	Plectorhinchus lessonii	Large predator	Secondary Target
	Plectorhinchus unicolor	Large predator	Secondary Target
Kyphosidae	*Kyphosus spp.*	Algal cropper	Non-target
	Microcanthus strigatus	Benthic carnivore	Non-target
Labridae	*Anampses geographicus*	Benthic carnivore	Non-target
	Anampses neoguinaicus	Benthic carnivore	Non-target
	Bodianus axillaris	Benthic carnivore	Non-target
	Bodianus loxozonus	Benthic carnivore	Non-target
	Bodianus mesothorax	Benthic carnivore	Non-target
	Cheilinus chlorurus	Benthic carnivore	Non-target
	Cheilinus fasciatus	Benthic carnivore	Non-target
	Cheilinus trilobatus	Benthic carnivore	Non-target
	Cheilinus undulatus	Benthic carnivore	Non-target
	Choerodon anchorago	Benthic carnivore	Secondary target
	Choerodon cyanodus	Benthic carnivore	Secondary target
	Choerodon fasciatus	Benthic carnivore	Non-target
	Choerodon graphicus	Benthic carnivore	Secondary target
	Choerodon monostigma	Benthic carnivore	Secondary target
	Choerodon schoenleinii	Benthic carnivore	Secondary target
	Choerodon vitta	Benthic carnivore	Non-target
	Epibulus insidiator	Benthic carnivore	Non-target
	Gomphosus varius	Benthic carnivore	Non-target
	Halichoeres melanurus	Benthic carnivore	Non-target
	Hemigymnus fasciatus	Benthic carnivore	Non-target
	Hemigymnus melapterus	Benthic carnivore	Non-target
	Labrichthys unilineatus	Benthic carnivore	Non-target
	Labroides bicolor	Benthic carnivore	Non-target
	Labroides dimidiatus	Benthic carnivore	Non-target
	Labropsis australis	Benthic carnivore	Non-target
	Oxycheilinus diagramma	Benthic carnivore	Non-target
	Psuedolabrus guentheri	Benthic carnivore	Non-target
	Stethojulis bandanensis	Benthic carnivore	Non-target
	Stethojulis strigiventer	Benthic carnivore	Non-target

	Thalassoma hardwicke	Benthic carnivore	Non-target
	Thalassoma jansenii	Benthic carnivore	Non-target
	Thalassoma lunare	Benthic carnivore	Non-target
	Thalassoma lutescens	Benthic carnivore	Non-target
Lethrinidae	*Gymnocranius spp.*	Intermediate predator	Secondary target
	Lethrinus atkinsoni	Intermediate predator	Secondary target
	Lethrinus laticaudis	Intermediate predator	Secondary target
	Lethrinus lentjan	Intermediate predator	Secondary target
	Lethrinus miniatus	Intermediate predator	Primary target
	Lethrinus nebulosus	Intermediate predator	Secondary target
	Lethrinus obsoletus	Intermediate predator	Secondary target
	Lethrinus ornatus	Intermediate predator	Secondary target
	Monotaxis grandoculis.	Intermediate predator	Secondary target
Lutjanidae	*Lutjanus argentimaculatus*	Large predator	Secondary target
	Lutjanus carponotatus	Intermediate predator	Secondary target
	Lutjanus fulviflamma	Intermediate predator	Secondary target
	Lutjanus fulvus	Intermediate predator	Secondary target
	Lutjanus lemniscatus	Intermediate predator	Secondary target
	Lutjanus lutjanus	Intermediate predator	Secondary target
	Lutjanus monostigma	Intermediate predator	Secondary target
	Lutjanus quinquelineatus	Intermediate predator	Secondary target
	Lutjanus russelli	Intermediate predator	Secondary target
	Lutjanus sebae	Intermediate predator	Primary target
	Lutjanus vitta	Intermediate predator	Secondary target
	Symphorus nematophorus	Large predator	Non-target
Mullidae	*Parupeneus barberinus*	Benthic carnivore	Non-target
	Parupeneus bifasciatus	Benthic carnivore	Non-target
	Parupeneus ciliatus	Benthic carnivore	Non-target
	Parupeneus indicus	Benthic carnivore	Non-target
Muraenidae	*Echidna nebulosa*	Intermediate predator	Non-target
	Gymnothorax favagineus	Intermediate predator	Non-target
	Gymnothorax javanicus	Intermediate predator	Non-target
	Gymnothorax meleagris	Intermediate predator	Non-target
	Gymnothorax undulatus	Intermediate predator	Non-target
	Gymnothorax javanicus	Intermediate predator	Non-target
	Gymnothorax meleagris	Intermediate predator	Non-target
Nemipteridae	*Scolopsis bilineatus*	Intermediate predator	Non-target
	Scolopsis margaritifer	Intermediate predator	Non-target
	Scolopsis monogramma	Intermediate predator	Non-target
Pomacanthidae	*Centropyge bicolor*	Benthic carnivore	Non-target
	Centropyge bispinosus	Benthic carnivore	Non-target
	Centropyge nox	Benthic carnivore	Non-target
	Centropyge tibicen	Benthic carnivore	Non-target

	Centropyge vrolikii	Benthic carnivore	Non-target
	Chaetodontoplus douboulayi	Benthic carnivore	Non-target
	Chaetodontoplus meredithi	Benthic carnivore	Non-target
	Pomacanthus imperator	Benthic carnivore	Non-target
	Pomacanthus semicirculatus	Benthic carnivore	Non-target
	Pomacanthus sexstriatus	Benthic carnivore	Non-target
	Pomacanthus xanthometapon	Benthic carnivore	Non-target
	Pygoplites diacanthus	Benthic carnivore	Non-target
Pomacentridae	*Abudefduf bengalensis*	Omnivorous pomacentrid	Non-target
	Abudefduf sexfasciatus	Omnivorous pomacentrid	Non-target
	Abudefduf vaigiensis	Omnivorous pomacentrid	Non-target
	Abudefduf whitleyi	Omnivorous pomacentrid	Non-target
	Abudefduf sexfasciatus	Omnivorous pomacentrid	Non-target
	Abudefduf vaigiensis	Omnivorous pomacentrid	Non-target
	Abudefduf whitleyi	Omnivorous pomacentrid	Non-target
	Acanthochromis polyacanthus	Omnivorous pomacentrid	Non-target
	Amblyglyphidodon aureus	Omnivorous pomacentrid	Non-target
	Amblyglyphidodon curacao	Omnivorous pomacentrid	Non-target
	Amblyglyphidodon	Omnivorous pomacentrid	Non-target
	Amphiprion akindynos	Omnivorous pomacentrid	Non-target
	Amphiprion chrysopterus	Omnivorous pomacentrid	Non-target
	Amphiprion clarkia	Omnivorous pomacentrid	Non-target
	Amphiprion melanopus	Omnivorous pomacentrid	Non-target
	Amphiprion perideraion	Omnivorous pomacentrid	Non-target
	Chromis amboinensis	Planktivorous pomacentrid	Non-target
	Chromis atripectoralis	Planktivorous pomacentrid	Non-target
	Chromis atripes	Planktivorous pomacentrid	Non-target
	Chromis nitida	Planktivorous pomacentrid	Non-target
	Chromis retrofasciatus	Planktivorous pomacentrid	Non-target
	Chromis ternatensis	Planktivorous pomacentrid	Non-target
	Chromis weberi	Planktivorous pomacentrid	Non-target
	Chrysiptera rex	Omnivorous pomacentrid	Non-target
	Chrysiptera rollandi	Omnivorous pomacentrid	Non-target
	Chrysiptera talboti	Omnivorous pomacentrid	Non-target
	Dascyllus aruanus	Omnivorous pomacentrid	Non-target
	Dascyllus melanurus	Omnivorous pomacentrid	Non-target
	Dascyllus trimaculatus	Omnivorous pomacentrid	Non-target
	Dascyllus reticulatus	Omnivorous pomacentrid	Non-target
	Dischistodus melanotus	Territorial pomacentrid	Non-target
	Dischistodus perspicillatus	Territorial pomacentrid	Non-target
	Dischistodus prosopotaenia	Territorial pomacentrid	Non-target
	Dischistodus	Territorial pomacentrid	Non-target
	Hemiglyphidodon	Territorial pomacentrid	Non-target
	Neoglyphidodon melas	Territorial pomacentrid	Non-target
	Neoglyphidodon nigroris	Territorial pomacentrid	Non-target
	Plectroglyphidodon dickii	Territorial pomacentrid	Non-target
	Plectroglyphidodon	Territorial pomacentrid	Non-target
	Pomacentrus adelus	Territorial pomacentrid	Non-target

	Pomacentrus amboinensis	Omnivorous pomacentrid	Non-target
	Pomacentrus australis	Omnivorous pomacentrid	Non-target
	Pomacentrus bankanensis	Territorial pomacentrid	Non-target
	Pomacentrus brachialis	Omnivorous pomacentrid	Non-target
	Pomacentrus chrysurus	Territorial pomacentrid	Non-target
	Pomacentrus coelestis	Omnivorous pomacentrid	Non-target
	Pomacentrus lepidogenis	Planktivorous pomacentrid	Non-target
	Pomacentrus moluccensis	Omnivorous pomacentrid	Non-target
	Pomacentrus nagasakiensis	Omnivorous pomacentrid	Non-target
	Pomacentrus vaiuli	Territorial pomacentrid	Non-target
	Pomacentrus wardi	Territorial pomacentrid	Non-target
	Stegastes apicalis	Territorial pomacentrid	Non-target
	Stegastes fasciolatus	Territorial pomacentrid	Non-target
Scaridae	*Bolbometapon muricatum*	Excavating grazer	Non-target
	Cetoscarus bicolor	Excavating grazer	Non-target
	Chlorurus bleekeri	Excavating grazer	Non-target
	Chlorurus microrhinus	Excavating grazer	Non-target
	Chlorurus sordidus	Excavating grazer	Non-target
	Hipposcarus longiceps	Excavating grazer	Non-target
	Scarus altipinnis	Scraping grazer	Non-target
	Scarus chamaeleon	Scraping grazer	Non-target
	Scarus dimidiatus	Scraping grazer	Non-target
	Scarus flavipectoralis	Scraping grazer	Non-target
	Scarus frenatus	Scraping grazer	Non-target
	Scarus ghobban	Scraping grazer	Non-target
	Scarus globiceps	Scraping grazer	Non-target
	Scarus niger	Scraping grazer	Non-target
	Scarus psittacus	Scraping grazer	Non-target
	Scarus rivulatus	Scraping grazer	Non-target
	Scarus rubroviolaceus	Scraping grazer	Non-target
	Scarus schlegeli	Scraping scarid	Non-target
	Scarus spinus	Scraping grazer	Non-target
	Scarus tricolor	Scraping grazer	Non-target
Serranidae	*Aethaloperca rogga*	Intermediate predator	Secondary target
	Anyperodon leucogrammicus	Large predator	Secondary target
	Cephalopholis boenak	Intermediate predator	Non-target
	Cephalopholis cyanostigma	Intermediate predator	Secondary target
	Cephalopholis microprion	Intermediate predator	Non-target
	Cromileptes altivelis	Large predator	Primary target
	Diploprion bifasciatus	Intermediate predator	Non-target
	Epinephelus caerulopunctatus	Large predator	Secondary target
	Epinephelus fasciatus	Intermediate predator	Secondary target
	Epinephelus fuscoguttatus	Large predator	Secondary target
	Epinephelus lanceolatus	Large predator	Non-target
	Epinephelus merra	Intermediate predator	Secondary target
	Epinephelus ongus	Intermediate predator	Secondary target
	Epinephelus quoyanus	Intermediate predator	Secondary target

	Plectropomus laevis	Large predator	Primary target
	Plectropomus leopardus	Large predator	Primary target
	Plectropomus maculatus	Large predator	Primary target
Siganidae	*Siganus argenteus*	Algal cropper	Non-target
	Siganus corallinus	Algal cropper	Non-target
	Siganus doliatus	Algal cropper	Non-target
	Siganus fuscescens	Algal cropper	Non-target
	Siganus javus	Algal cropper	Non-target
	Siganus lineatus	Algal cropper	Non-target
	Siganus puellus	Algal cropper	Non-target
	Siganus punctatus	Algal cropper	Non-target
	Siganus spinus	Algal cropper	Non-target
	Siganus vulpinus	Algal cropper	Non-target
Zanclidae	*Zanclus cornutus*	Benthic carnivore	Non-target

Conservation Biology

Contributed Paper

Using Coral Disease Prevalence to Assess the Effects of Concentrating Tourism Activities on Offshore Reefs in a Tropical Marine Park

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Abstract: *Concentrating tourism activities can be an effective way to closely manage high-use parks and minimize the extent of the effects of visitors on plants and animals, although considerable investment in permanent tourism facilities may be required. On coral reefs, a variety of human-related disturbances have been associated with elevated levels of coral disease, but the effects of reef-based tourist facilities (e.g., permanent offshore visitor platforms) on coral health have not been assessed. In partnership with reef managers and the tourism industry, we tested the effectiveness of concentrating tourism activities as a strategy for managing tourism on coral reefs. We compared prevalence of brown band disease, white syndromes, black band disease, skeletal eroding band, and growth anomalies among reefs with and without permanent tourism platforms within the Great Barrier Reef Marine Park. Coral diseases were 15 times more prevalent at reefs with offshore tourism platforms than at nearby reefs without platforms. The maximum prevalence and maximum number of cases of each disease type were recorded at reefs with permanently moored tourism platforms. Diseases affected 10 coral genera from 7 families at reefs with platforms and 4 coral genera from 3 families at reefs without platforms. The greatest number of disease cases occurred within the spatially dominant acroporid corals, which exhibited 18-fold greater disease prevalence at reefs with platforms than at reefs without platforms. Neither the percent cover of acroporids nor overall coral cover differed significantly between reefs with and without platforms, which suggests that neither factor was responsible for the elevated levels of disease. Identifying how tourism activities and platforms facilitate coral disease in marine parks will help ensure ongoing conservation of coral assemblages and tourism.*

Keywords: Acroporidae, anthropogenic impacts, coral disease, Great Barrier Reef, marine park, reef tourism, visitor concentration

Utilización de la Prevalencia de Enfermedades del Coral para Evaluar los Efectos de la Concentración de Actividades Turísticas en Arrecifes en un Parque Marino Tropical

Coral Reefs
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REPORT

Predicting outbreaks of a climate-driven coral disease in the Great Barrier Reef

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Abstract Links between anomalously high sea temperatures and outbreaks of coral diseases known as White Syndromes (WS) represent a threat to Indo-Pacific reefs that is expected to escalate in a changing climate. Further advances in understanding disease aetiologies, determining the relative importance of potential risk factors for outbreaks and in trialing management actions are hampered by not knowing where or when outbreaks will occur. Here, we develop a tool to target research and monitoring of WS outbreaks in the Great Barrier Reef (GBR). The tool is based on an empirical regression model and takes the form of user-friendly interactive ~1.5-km resolution maps. The maps denote locations where long-term monitoring suggests that coral cover exceeds 26% and summer temperature stress (measured by a temperature metric termed the mean positive summer anomaly) is equal to or exceeds that experienced at sites in 2002 where the only severe WS outbreaks documented on the GBR to date were observed. No WS outbreaks were subsequently documented at 45 routinely surveyed sites from 2003 to 2008, and model hindcasts for this period indicate that outbreak likelihood was never high. In 2009, the model indicated that outbreak likelihood was high at north-central GBR sites. The results of the regression model and targeted surveys in 2009 revealed that the threshold host density for an outbreak decreases as thermal stress increases, suggesting that bleaching could be a more important precursor to WS outbreaks than previously anticipated, given that bleaching was severe at outbreak sites in 2002 but not at any of the surveyed sites in 2009. The iterative approach

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used here has led to an improved understanding of disease causation, will facilitate management responses and can be applied to other coral diseases and/or other regions.

Keywords Climate change · Coral disease · Great Barrier Reef · Environmental management · Outbreaks · White Syndromes