Coral Reefs (2011) 30:485–495
DOI 10.1007/s00338-010-0708-0

REPORT

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

J. A. Maynard · K. R. N. Anthony · C. D. Harvell · M. A. Burgman · R. Beeden · H. Sweatman · S. F. Heron · J. B. Lamb · B. L. Willis

Received: 23 September 2010 / Accepted: 3 December 2010 / Published online: 23 December 2010

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

Communicated by Biology Editor Dr. Andrew Baird

J. A. Maynard (✉) · M. A. Burgman
Australian Centre of Excellence for Risk Analysis, School of Botany, University of Melbourne, Parkville, VIC 3010, Australia
e-mail: maynardmarine@gmail.com

K. R. N. Anthony · B. L. Willis
ARC Centre of Excellence for Coral Reef Studies, James Cook University of North Queensland, Townsville, QLD 4811, Australia

K. R. N. Anthony
Global Change Institute, University of Queensland, St Lucia, QLD 4072, Australia

C. D. Harvell
Department of Ecology and Evolutionary Biology, Cornell University, Ithaca, NY 14853, USA

R. Beeden
Climate Change Group, Great Barrier Reef Marine Park Authority, Townsville, QLD 4810, Australia

H. Sweatman
Australian Institute of Marine Science, Cape Ferguson, QLD 4810, Australia

S. F. Heron
NOAA Coral Reef Watch, 675 Ross River Road, Townsville, QLD 4817, Australia

S. F. Heron
School of Engineering and Physical Sciences, James Cook University of North Queensland, Townsville, QLD 4811, Australia

J. B. Lamb · B. L. Willis
School of Marine and Tropical Biology, James Cook University of North Queensland, Townsville, QLD 4811, Australia

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

Introduction

Climate warming is projected to increase outbreaks of diseases in populations of humans, agricultural crops, terrestrial wildlife and marine organisms (Harvell et al. 2002, 2009). Thus, there is increasing urgency to develop tools that can predict where and when outbreaks will occur (e.g., crop disease forecasting programmes; Hijmans et al. 2000; Butterworth et al. 2010). On coral reefs, long-term monitoring programs indicate that outbreaks of infectious coral diseases, some of which are known to be temperature-facilitated (see Harvell et al. 2009; Sato et al. 2009), have been increasing worldwide over the past 40 years (Harvell et al. 2009; Mydlarz et al. 2009). In combination with human-induced and natural stressors, infectious diseases have radically altered coral reefs in the Caribbean, particularly in Florida (Hughes 1994; Patterson et al. 2002; Harvell et al. 2007). Although coral diseases have been assumed to be less abundant in the Indo-Pacific than in the Caribbean, recent studies have shown comparable mean prevalence between the two regions for some diseases (Willis et al. 2004; Myers and Raymundo 2009).

On Australia's Great Barrier Reef (GBR), three infectious diseases—White Syndromes, Black Band and Brown Band disease—have been identified as being of particular concern (Willis et al. 2004). These diseases are typically most prevalent on the GBR in the family Acroporidae, the coral family that is amongst the fastest growing on the GBR and is the most spatially dominant framework builder (Willis et al. 2004; Page and Willis 2008). Acroporidae are also the most susceptible to thermal bleaching (Marshall and Baird 2000), which has been shown to increase susceptibility to disease (Miller et al. 2009; Mydlarz et al. 2009). Both bleaching events and disease outbreaks are expected to become more frequent and severe under a changing climate and as anthropogenic regional and local-scale stressors exacerbate climate impacts (Mydlarz et al. 2009). Such predictions highlight the need to target research and monitoring efforts at sites where outbreaks are likely to occur (Bruckner 2002; Aeby et al. 2008). As a consequence, researchers will be best placed to determine the relative importance of outbreak risk factors. Concurrently, managers can reactively mitigate anthropogenic stressors at affected sites (e.g., through temporary closures or changed water quality targets, Maynard et al. 2009).

Unlike spatially extensive bleaching events, the causes of coral disease outbreaks are more complex and less understood. Coral diseases and disease outbreaks may be caused by abiotic and biotic factors working in combination. The best understood of these are the abiotic factors, temperature and water quality, and the biotic factors, host density and fish abundance and type (Bruno et al. 2007; Page et al. 2009; Raymundo et al. 2009; Diaz and Madin 2010; Williams et al. 2010). For example, the group of coral diseases known as White Syndromes (WS), the focus of this study, have been seen in greatest abundance following periods of anomalously high sea temperatures (Bruno et al. 2007). White Syndromes are an emerging group of diseases affecting Indo-Pacific, reef-building corals and, although little is known about their modes of transmission, at least some White Syndromes are caused by the coral pathogen *Vibrio coralliilyticus* (Sussman et al. 2008, 2009). White Syndromes are characterised by a white front that corresponds to the interface between recently killed tissue and exposed white skeleton (Willis et al. 2004). Tissue loss fronts move across coral colonies as the disease progresses causing either partial or whole colony mortality. The signs and impacts of WS are similar to those described for white band and white plague diseases, which have caused extensive mortality on Caribbean reefs (Aronson and Precht 2001; Miller et al. 2009).

Bruno et al. (2007) found that high temperatures caused WS outbreaks on the GBR only on reefs with >50% coral cover, suggesting that, like many other transmissible infectious diseases (Antolin 2008), high host density is required for outbreaks. Bruno et al. (2007) also showed, however, that variability in disease abundance was high, particularly during the only known outbreak year, 2002. Overall, WS abundance varied fourfold, even with consistently high temperature stress. Heron et al. (2010) advanced the work of Bruno et al. (2007), producing seasonal outlooks and near real-time monitoring of WS outbreak likelihood for the Great Barrier Reef and Coral Sea based on winter and summer sea temperature stress. Specific winter conditions were observed to pre-condition some corals against disease outbreak, while the level of outbreak for the remaining at-risk corals was related to subsequent summer stress. However, the resolution of the resulting images of outbreak likelihood (~50-km) may limit the utility of the tool for targeting research and monitoring efforts on the GBR (and hence facilitation of management responses), given that 50-km pixels often contain dozens of reefs. In addition, the absence of any measure of coral cover in the images of outbreak likelihood (though present in the algorithm development) could lead to misinterpretation; e.g., outbreak likelihood can be shown as high for

locations where the coral cover is below the empirically derived threshold for a WS outbreak to occur. This study is the next logical step in the evolution of the work first presented by Bruno et al. (2007) and builds on the research and tools produced by Heron et al. (2010).

To further develop and evaluate research and monitoring tools for coral diseases, our objectives were to (1) hindcast where WS outbreaks are likely to have occurred in years following the only known severe outbreaks on the GBR (i.e., in 2002) and (2) forecast where outbreak likelihood would be high in 2009 and subsequently survey these reefs to improve the knowledge of conditions triggering outbreaks. Despite increasing numbers of coral disease outbreaks globally and a critical need to increase our understanding of disease outbreak causation, this is the first effort to predict the likelihood of coral disease outbreaks at the management-relevant scale of an individual reef.

Methods

Surveys of WS abundance and coral cover

Data on coral cover and WS abundance were collected by the Long-Term Monitoring Program of the Australian Institute of Marine Science (AIMS, Sweatman et al. 2008). For coral cover, numerous GBR reefs spread over 13° of latitude (11–24S) have been surveyed on multiple occasions since 1986 using manta tows. The manta tow surveys involved a snorkeller with a "manta board" (hydrofoil) being towed slowly behind a small boat around the entire perimeter of each survey reef close to the reef crest so that the observer surveyed a 10-m-wide swathe of the shallow reef slope. The boat stopped every 2 min to allow the observer to record the mean coral cover for that "tow" ($\sim$200 m of reef edge) into one of 10 categories (Bass and Miller 1996). Observers were full-time monitoring staff whose cover estimates were cross-calibrated annually to minimise the variation amongst observers and between years. The towpath was also standardised; the boat handler followed a course marked on an aerial photograph of each reef so that approximately the same parts of each reef were surveyed at each visit. Initially, greater than 200 haphazardly selected reefs were surveyed each year, but this number declined as the program developed. Reefs that were surveyed only once during the 19 years have been excluded from the analysis used here, leaving 72–193 (median = 100) reefs surveyed in each year. The mean coral cover for each reef at each survey was calculated by taking the mid-point of the coral cover category for each 2-min tow and then averaging over all tows for that reef (Sokal and Rohlf 1995). The coral cover at each GBR reef was spatially modeled using generalised additive models (mgcv package R). The resulting contour maps denote ranges in coral cover and show areas of the GBR Marine Park for which these ranges apply, rather than designations for individual reefs. As a consequence, the maps are only interpreted with an overlay of reef boundaries since non-reef areas have very low (or zero) coral cover.

Surveys of WS abundance were conducted along the north-east flank of 48 reefs on six cross-shelf transects in the Great Barrier Reef Marine Park (see Fig. 1), along a depth contour of 6–9 m from 1998 to 2008. The abundance of WS was measured on SCUBA by counting infected colonies within 15 permanent belt transects (each 50 m by 2 m) per reef. Video was taken along a 25-cm-wide belt of the same permanent transects, and then hard coral cover estimated using a point sampling technique (see English et al. 2004). The year 2002 was identified as the only year in which severe (>100 cases/1,500 m^2) outbreaks occurred on the GBR, thus we used temperature conditions from the summer (Dec to

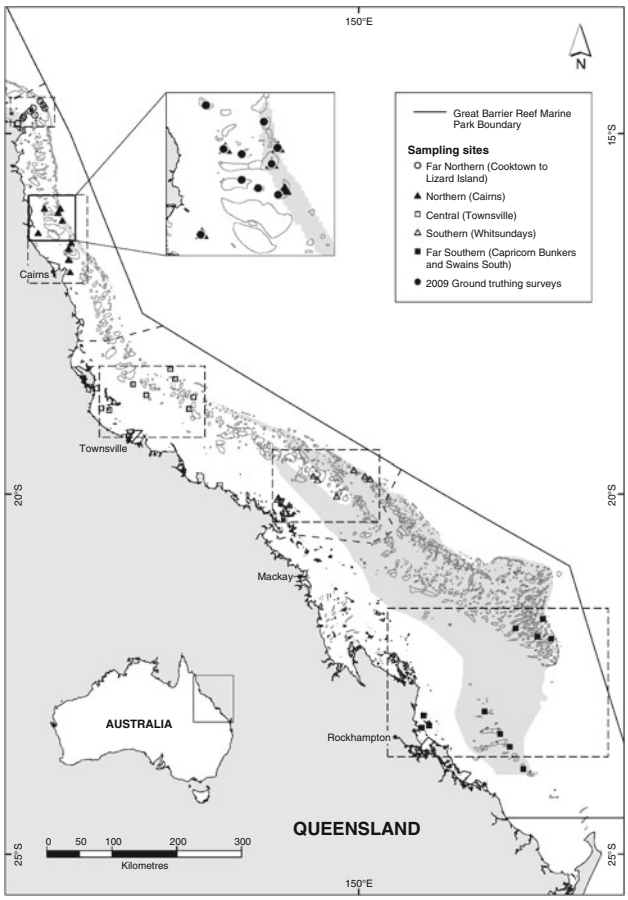

Fig. 1 Sites within the Great Barrier Reef Marine Park monitored by the Australian Institute of Marine Science's Long-Term Monitoring Program. The inset map for the Cairns area shows locations where ground-truthing surveys were conducted in 2009. The grey-shaded areas of the reef denote areas where surveys between 1986 and 2009 suggest long-term average coral cover to be above 26%

Feb) that preceded the first outbreaks in 2002 through to 2008 to constrain our model of outbreak likelihood. Earlier studies suggest that: (1) prevalence of diseases like WS increases in summer on the GBR (Willis et al. 2004), (2) increased disease susceptibility follows bleaching events (Harvell et al. 2009; Miller et al. 2009) and (3) density-dependent pathogen transmission results in a lag time between occurrences of high temperatures and the appearance of visible disease signs (Rudolf and Antonovics 2005). Therefore, variability in the seasonal timing of the AIMS surveys (annual surveys completed variably in summer–autumn and winter–spring months) is likely to contribute to variability seen in the disease frequency data, particularly during the 2002 outbreak. For this reason, only data from surveys made from August through November 2002, representing 5–9 months after the 2002 austral summer (December 2001 to February 2002), were used to develop the model. This time window covers a total of 28 surveys, all of which were made within 2 months either side of when maximum WS abundances were observed.

Remotely sensed temperature data and thermal stress

The daily sea surface temperature (SST) data were supplied by the Bureau of Meteorology, Australia, based on processing of the latest NOAA environmental satellite AVHRR thermal imagery. We used a 15-day composite SST data set (currently from NOAA AVHRR satellites 15, 17 and 18) at a pixel resolution of 0.017995° (~1.5 km, see also Maynard et al. 2008). SST values were backfilled with the most recent temperature for up to 14 days in some cases. For this study, more than 70% of the data were between 1 and 3 days old.

To calculate thermal stress, daily temperature data were compared to the average monthly temperatures calculated for the 10 years ('LMST') that preceded the surveys (e.g., mid-1993 to mid-2002 for surveys in mid- to late-2002). The climatology data set used is ~4-km resolution, and one standard deviation was added to the monthly average temperature to create a climatological baseline that identified temperatures outside the historically expected range during the summer months of 2002–2009. Quality control measures used in the development of the climatology data sets were comparable to those used in the processing of the daily data (see Griffin et al. 2004 for more detail on the climatology). The monthly climatology baselines were resampled to the ~1.5-km SST grid using a linear interpolation scheme that calculated a weighted average.

For the area where outbreaks were observed in 2002, the mean positive summer anomaly (MPSA) was found to be the best predictor of coral bleaching (referred to as the 'heating rate' in Maynard et al. 2008 and calculated from the mean rather than the mean + 1 SD baseline used here). Given links between susceptibility to bleaching and to disease (Palmer et al. 2010), MPSA is explored here as a predictor of disease risk. MPSA was calculated by comparing daily temperature data to the climatology data set for each of the three summer months. The number of 'degree heating days' (DHD, see Maynard et al. 2008) was calculated first and described the accumulation of heat stress at each site for each survey year through the summer (December–February). Specifically, DHD is the sum of daily average sea surface temperatures (T_{Heating}) that exceed the climatological baseline (LMST + SD), for the three summer months. The DHD metric therefore differs from the WSSTA metric used by Bruno et al. (2007) in integrating the amplitude of positive temperature anomalies through thermal stress events during the summer prior to disease surveys (rather than counting thermal stress events for the full year prior). DHD is calculated as:

$$\text{DHD} = \sum \left(T_{\text{Heating}} - (\text{LMST} + \text{SD}) \right) \quad (1)$$

However, degree heating days do not differentiate amongst a broad range of coral heat stresses. For this reason, the mean number of DHDs accumulated each day that temperatures (T_{Heating}) exceeded the long-term baseline temperature (LMST) is calculated—effectively the mean positive summer anomaly (MPSA, see 'heating rate' in Maynard et al. 2008), expressed as degrees Celsius. The MPSA is calculated as:

$$\text{MPSA} = \text{DHD} \Big/ \sum (\text{days heated}) \quad (2a)$$

Days heated are days at which:

$$T_{\text{Heating}} > \text{LMST} + \text{SD} \quad (2b)$$

Empirical regression model and mapping outbreak likelihood

To capture the observed interaction between MPSA, spatial cover of *Acropora* species (A_{Acr}), and the abundance of White Syndromes (A_{WS}), we used an empirical model of the form:

$$A_{\text{WS}} = c\,\text{MPSA}^{a} A_{\text{Acr}}^{b} \quad (3)$$

where c is a regression coefficient, and a and b are scaling parameters accounting for the relative contributions of each of the two factors to the interaction. This generic function approximates a suite of qualitatively different functional relationships between thermal regime, host abundance and WS abundance. The power function assumes that a WS outbreak can only occur under conditions of high host abundance and thermal stress (full interaction), which is consistent with observations (Bruno et al. 2007). Although this empirical approach does not provide detailed insight into the mechanisms of how these diseases spread, empirical models have proven successful in forecasting crop

diseases (Savary et al. 2006) and so have been explored here.

We produced images depicting WS outbreak likelihood for north-eastern Australia for each summer since and including 2002. Specifically, in each summer, pixels (~1.5-km resolution) were coloured red indicating high outbreak likelihood if both of the following conditions were met: (1) the pixel experienced MPSA values at least as great as those where outbreaks occurred in 2002 and (2) the pixel is in an area where long-term average coral cover data sets suggest coral cover on the reefs to be above an empirically derived threshold (see "Results" section). All other pixels were coloured white. Spatial overlays for the images were produced using ArcGIS™ software.

The regression model was conditioned on data from the documented outbreaks in the Great Barrier Reef area (i.e., the 2002 outbreaks), so maps of outbreak likelihood have been limited to this study area. Using the 2002 MPSA and coral cover thresholds to produce images of outbreak likelihood for the years 2003–2008 enabled an assessment of the predictive capacity of the model. Producing a similar image based on MPSA for the 2009 summer (December 2008–February 2009) enabled targeted research and monitoring at sites assessed as having high outbreak likelihood.

Ground-truthing surveys

Reef locations forecast to have both high and low outbreak likelihood in 2009 were identified to enable targeted coral disease surveys (see inset map, Fig. 1, for survey site locations). Surveys comprised 3 replicate manta tows covering an approximate area of 500 m^2, within which all cases of White Syndromes were recorded. To further verify manta tow observations, three 20 × 2 m belt transects were completed on SCUBA, within which all corals were classified as healthy or diseased with specific details of diseases recorded.

Results

Empirical regression model and threshold values

The empirical model (Eq. 3) provided an excellent fit to the data (Fig. 2), explaining over 90% of the variation in WS abundance recorded in the 2002 AIMS LTMP surveys. Initial analyses indicated that a more parsimonious model without the regression coefficient (parameter c) explained a similar amount of variation in WS (Table 1) and was hence used for further analyses. WS abundance scaled slightly more strongly with host abundance than with MPSA as indicated by differences in the scaling parameters ($b = 1.6 \pm 0.1$, $a = 1.1 \pm 0.4$, Table 1). The 2002 outbreaks in the southern GBR, where the highest abundances of WS were observed (maximum of 343 cases/1,500 m^2), had a high cover of *Acropora* hosts at that time (>45%) and experienced high (≥0.35°C) MPSA values (see topmost grouping of four points in Fig. 2).

No sites were surveyed during the 2002 outbreak year that had host cover between 26 and 45%, as well as MPSA values ≥0.35°C (see light grey shading, Fig. 2), indicating that the host density threshold for a WS outbreak on the

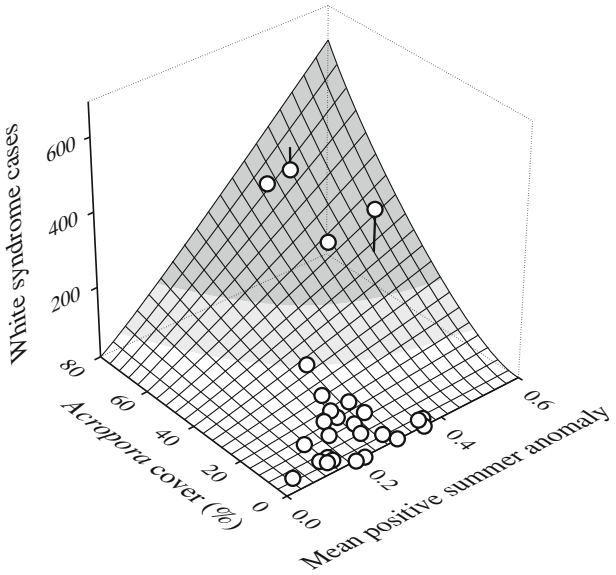

Fig. 2 Empirical regression model of host coral cover, mean positive summer anomaly (*MPSA*) and the number of WS cases observed at survey sites in 2002. The outbreak sites (Lady Musgrave Reef, One Tree Island Reef, Wreck Island Reef and Broomfield Reef, Fig. 3) experienced high MPSA values and had high coral cover. The dark grey-shaded area denotes conditions the model suggests are conducive to a severe outbreak (100+ cases/1,500 m^2) given conditions at outbreak sites in 2002. The paucity of data in the light grey area (referring to a minimum of 60 cases/1,500 m^2) highlights that the threshold cover density required for an outbreak is uncertain

Table 1 Summary results of non-linear regression of two versions of Eq. 3 (methods) to 2002 survey data ($N = 28$ sites) for the abundance of White Syndromes (A_{WS}), abundance of *Acropora* hosts (A_{Acr}) and thermal stress as estimated by MPSA. The regression coefficient, c, could only be estimated with high uncertainty and was not significantly different from unity (a). The more parsimonious (two-parameter) model without the inclusion of c explained a similar amount of variation in thermal stress and host density and was hence used to assess outbreak likelihood from 2003 to 2009

Model	R^2	Parameter	Estimate	SE	P-value
(a) $A_{WS} = c\ \mathrm{MPSA}^a A_{Acr}^b$	0.96	c	18.81	12.13	0.134
		a	1.57	0.27	<0.001
		b	0.98	0.14	<0.001
(b) $A_{WS} = \mathrm{MPSA}^a A_{Acr}^b$	0.93	a	1.07	0.37	0.008
		b	1.59	0.08	<0.001

GBR is unknown. Using the model parameter values (a, b) derived from the 2002 outbreak year, a threshold MPSA value of 0.35°C combined with a minimum of 26% cover yields 60 WS cases/1,500 m^2—a WS abundance level similar to the outbreak threshold of 50 WS cases/1,500 m^2 proposed by Heron et al. (2010). Therefore, using a gridded map of north-east Australia for each year between 2002 and 2009, all pixels (~1.5-km resolution) that experienced MPSA values at least as great as the MPSA threshold (0.35°C) and had at least 26% coral cover were designated as having 'high' outbreak (60 cases/1,500 m^2) likelihood (red in Fig. 3), with all other pixels being designated as having 'low' outbreak likelihood. Although the only long-term, GBR-wide records of coral cover are for overall average cover, the dominant coral genus on the GBR is *Acropora* (Sweatman et al. 2008), thus average cover is strongly indicative of the cover of *Acropora* hosts. Maintaining the cover threshold at the empirically derived 26% increases the conservativeness of the images of outbreak likelihood because the cover of *Acropora* hosts would be less than 26%. This conservative approach will lead to an increased understanding of host density thresholds following future outbreak monitoring.

Assessing outbreak likelihood and targeted surveys

For 2002, a map of outbreak likelihood shows that much of the southern GBR may have experienced an outbreak, particularly many of the outer-shelf sites in the Swain Reefs (the most south-eastern reefs of the GBR), where coral cover is highest (Fig. 3a). However, most of the southern GBR region was not surveyed in 2002, thus it is unknown whether WS outbreaks occurred (see Fig. 3), highlighting the value in using the model to target research and monitoring efforts.

For the 6 years between 2003 and 2008, outbreak likelihood was assessed as low almost everywhere in the GBR Marine Park, with the exception of a very few localised cases for which disease survey data are not available (Fig. 3). Concordantly, no outbreaks were documented during this period at the survey sites (see Fig. 1), indicating that there are no known false negatives (disease outbreaks when their presence was not predicted) or false positives (no disease outbreak when an outbreak was predicted) for the 6 years that followed the 2002 outbreaks.

The 2009 map identified areas of high outbreak likelihood for reefs in the north-central (see Fig. 3b) and southern GBR. In October 2009, targeted surveys were completed at 10 reefs in the Port Douglas region (north-central GBR, see inset map Fig. 1). This region was selected for surveys because: (1) reefs were comparatively more readily accessible and (2) almost all pixels assessed as having high outbreak likelihood in the southern GBR were non-reef areas. Based on the regression model, outbreak likelihood was high at four of the Port Douglas sites surveyed and low at the other six sites. WS abundance

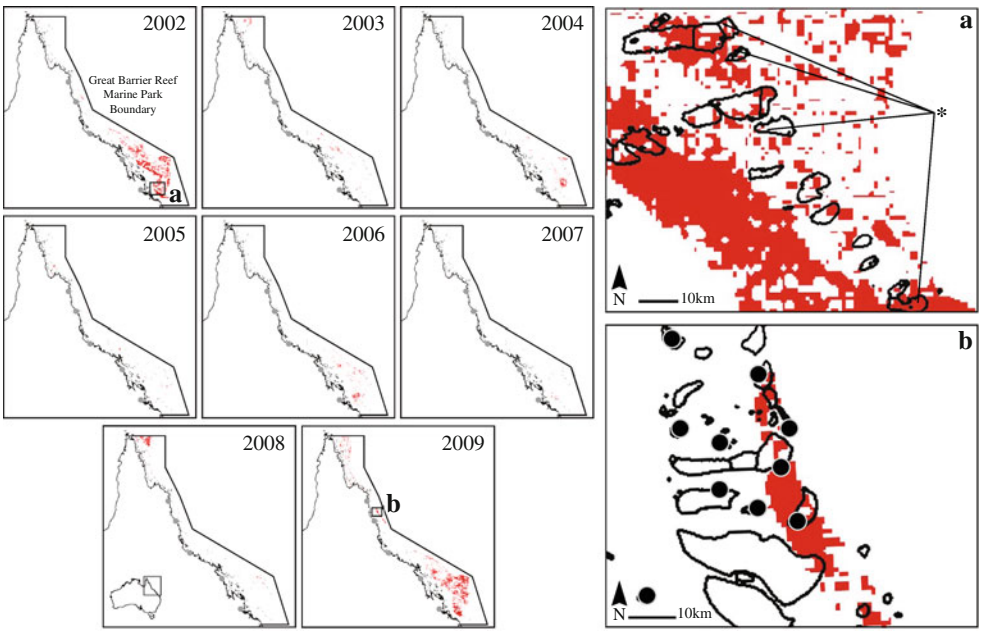

Fig. 3 WS outbreak likelihood from 2002 to 2009, where red pixels indicate high outbreak likelihood based on thermal stress and long-term average coral cover. The asterisk and arrows in (**a**) show sites where outbreaks were observed in the southern GBR in 2002 (from north to south: Broomfield Reef, Wreck Island Reef, One Tree Island Reef, and Lady Musgrave Reef). The grey circles in (**b**) show sites where ground-truthing surveys were conducted in 2009

approached outbreak levels at three of the four sites predicted to have high outbreak likelihood—Rudder Reef, Opal Reef and 16-017S (Table 2). WS abundance was more than three times greater at sites predicted to have a high outbreak likelihood (mean of 31 cases/1,500 m^2, Table 2) than at the low outbreak likelihood sites (mean of 9 cases/1,500 m^2, Table 2). Differences in WS abundance between the two-site groupings—high and low outbreak likelihood—were statistically significant (Student's *t*-test, $t(8) = 3.06$, $P = 0.01$, Table 2), whereas differences in coral cover between the two-site groupings were not significant ($t(8) = 0.416$, $P = 0.68$, Table 2), highlighting the importance of temperature stress as a WS outbreak trigger.

The minimum WS abundance at sites where severe outbreaks were observed in 2002 (221 cases/1,500 m^2, Fig. 2) was 12-fold greater than the highest average WS abundance calculated for the other survey years (18 cases/1,500 m^2 in 2009; Table 3). Considering the four severe outbreak sites in 2002 to be outliers, the mean ±1 SD WS abundance across all survey years (2002–2009) is 10.86 ± 17. Thus, we calculate that WS abundances exceeding 28 cases/1,500 m^2 (overall mean + 1 SD) are anomalously high and potentially of concern to reef managers. In 2009, WS abundances were anomalous (Table 2) at the three sites—Rudder Reef, Opal Reef and 16-017S—where abundance approached but did not exceed outbreak thresholds based on 2002 levels.

The maps of outbreak likelihood developed here work like an interactive decision-support tool in Google Earth™ facilitating monitoring and management responses by showing outbreak likelihood at the scale of an individual reef. Two versions of the tool were produced: (1) a publicly accessible tool based on temperature stress and a coral cover overlay from the AIMS LTMP long-term average coral cover data sets (as in Fig. 3), which works to limit the potential for the images to be misinterpreted by the public or the media and (2) a version developed for researchers and managers that complements version 1, but is based only on temperature stress. Version 2 requires users to interpret images based on the knowledge of coral cover in an area because, although state-of-art, the GBR-wide coral cover data sets use spatial modeling to estimate coral cover for sites not surveyed, resulting in potential inconsistencies between estimates and actual cover. As such, version 1 could incorrectly assess outbreak likelihood to be low at sites where coral cover is estimated to be lower than it

Table 3 Mean WS abundance for each survey year (), and the mean WS abundance across all sites and years. WS abundance values that exceed the mean across all sites and years plus one standard deviation are anomalous

Year	WS abundance (cases/1,500 m^2 ± 1 SD)
2002/03	20.11 ± 29.2[a]
2003/04	6.34 ± 8.58
2004/05	8.45 ± 12.23
2005/06	8.54 ± 13.91
2006/07	8.78 ± 13.37
2007/08	14.88 ± 14.18
2009	17.98 ± 15.3[b]
Across all sites and years[a]	10.86 ± 17
'Anomalous' WS abundance values	≥28

[a] Excludes four severe outbreaks, see Fig. 2

[b] Extrapolated from 500 m^2

Table 2 WS abundance at 10 sites in the north-central GBR (see Fig. 1) where ground-truthing surveys were conducted in 2009. Outbreak likelihood designations are derived from the empirical regression model (Eq. 3, "Methods")—the 'high' outbreak likelihood designation requires ≥26% coral cover and an MPSA value ≥0.35°C. Differences in WS abundance between the two-site groupings (high and low outbreak likelihood) are significant ($t(8) = 3.05$, $P = 0.01$), and abundance levels in bold italics are anomalously high (see Table 3)

Reef name	Coral cover (%)	MPSA > 0.35°C	Outbreak likelihood	WS abundance (cases/1,500 m^2)[a]
Opal Reef	48	Yes	High	***45.71***
Rudder Reef	30	Yes	High	***35.23***
Reef 16-017	60	Yes	High	***37.17***
Agincourt Reef #4	33	Yes	High	6.00
Chinaman Reef	20	No	Low	8.46
Agincourt Reef #1	10	No	Low	6.06
Mackay Reef	60	No	Low	11.53
St. Crispin Reef	29	No	Low	3.72
Low Island Reef	80	No	Low	13.11
Pickersgill Reef	20	No	Low	12.78

[a] Extrapolated from 500 m^2

actually is. When interpreted in combination with version 1, version 2 reduces the potential for such false negatives.

Discussion

The strong relationship found amongst thermal stress, *Acropora* abundance and WS abundance during the 2002 outbreak year enabled the development of an empirical model, which was then used to map outbreak likelihood for the GBR Marine Park from 2003 to 2009. From 2003 to 2008, outbreak likelihood was low throughout the GBR, highlighting the uniqueness of the conditions that caused the 2002 outbreaks. When outbreak likelihood was high at four of ten north-central GBR sites surveyed in 2009, WS abundances were anomalous at three of the four sites and approached but did not exceed outbreak levels. This result highlights that the regression model developed here successfully targeted surveys at sites that can increase our understanding of WS outbreak causation and/or serve as trial sites for management actions to mitigate disease impacts.

That WS abundances were anomalous but approached rather than exceeded outbreak levels at the high outbreak likelihood sites in 2009 could be explained by the lack of bleaching in 2009 and the conservative nature of the coral cover threshold used to develop the images of outbreak likelihood. In bleached corals, loss of autotrophic capacity as a consequence of loss of endosymbiotic algae can compromise nutritional economy (Anthony et al. 2009; Wooldridge 2009). As a result, maintenance of the surface mucus layer (SML) and the associated microbial communities that are integral to coral health (Ritchie 2006; Bourne et al. 2009) can be compromised, potentially allowing growth of pathogens (Bourne et al. 2009). Exemplifying this, following severe thermal stress and widespread bleaching of corals in the north-eastern Caribbean during 2005 (Wilkinson and Souter 2008), disease observations increased 13- to 51-fold compared with pre-bleaching levels of disease (Miller et al. 2009). On the GBR in 1998, a spatially extensive and severe bleaching event (Berkelmans and Oliver 1999) preceded the first observations of WS (reviewed in Willis et al. 2004), though WS abundance in 1998 was far lower than observed in 2002. Similarly, the first reports of severe WS outbreaks in 2002 were preceded by the most severe bleaching event recorded on the GBR (Berkelmans et al. 2004). Although bleaching tended to be minor in 2002 for reef areas that included the WS outbreak sites (Berkelmans et al. 2004), it is likely that sub-lethal bleaching effects would have increased the susceptibility of corals to disease at these sites (Bourne et al. 2009; Wooldridge 2009). In contrast, almost no bleaching was observed in the north-central GBR in 2009 (GBRMPA 2009), thus it is less likely that host resistance mechanisms were impaired, potentially explaining that abundances were close but not beyond outbreak levels in this year.

Coral cover at sites assessed as having high outbreak likelihood in 2009 (mean of 31%) was less than half that at any of the 2002 severe outbreak sites (min of 65%), highlighting the need for further records of thermal stress, host abundance and WS abundance to refine host density thresholds for WS outbreaks. The 2009 survey results suggest the host density threshold for a WS outbreak lies between 35 and 65%. Importantly though, it is also likely that the host density threshold for an outbreak depends in part on the severity of temperature stress, which would affect both the condition of corals and their susceptibility to pathogens (Lafferty and Holt 2003; Work et al. 2009). Thus, when thermal stress is high, WS infections may develop on corals with increased disease susceptibility at multiple locations concurrently, reducing the relative importance of high host density to facilitate disease spread. Conversely, at lower thermal stress, rapid spread of WS may require higher host density (see review in Harvell et al. 2002). The regression model used here lends strong support to these suggestions, as WS abundance during the 2002 outbreak year increased as both temperature stress and host density increased but scaled more strongly with coral cover than thermal stress (Table 1, Fig. 2).

Although WS abundances in 2009 did not reach the 2002 severe outbreak threshold, they were anomalously high (>28 cases/1,500 m^2, Table 3) at three sites where outbreak likelihood was predicted to be high, raising questions as to what abundance level should define a WS outbreak on the GBR. In wildlife disease ecology, it is commonly accepted that an outbreak is defined as the occurrence of disease at levels greater than expected for a given time and place (e.g., Wobeser 2006). Hence, WS abundance at Rudder Reef, Opal Reef and 16-017S would be consistent with an outbreak according to such definitions, meaning that these sites do not, as we label them above, constitute 'false positive' predictions by the model. Heron et al. (2010) use a slightly different process to calculate an outbreak threshold of 50 WS cases/1,500 m^2. These authors isolated unusually high disease events and then took the mean and standard deviation of the maximum of the remaining observed disease abundance each year, following an iterative process that removed outliers at each iteration (later designated outbreaks). To calculate our threshold of 28 WS cases/1,500 m^2, we consider the severe outbreaks of 2002 as outliers and calculate the average of the remaining abundance values. Future work in this area is needed to further refine current understanding of what constitutes a WS outbreak. We suggest using the lower outbreak threshold level calculated here as a 'threshold of concern to managers' and that it could be included as part

of a precautionary approach for targeting management responses.

The retrospective analysis of survey data from 2003 to 2008 suggested the model did not predict outbreak likelihood to be low at a location where an outbreak occurred (false negative). More supporting evidence was provided for this in 2009, when outbreak likelihood was assessed as high for the first time in the GBR since 2002, and ground-truthing surveys showed no false negatives. From the perspective of reef managers tasked with responding to threats, avoiding false negative predictions is even more important than avoiding false positives. False negative predictions could lead to a reef visitor encountering an outbreak of which managers had no knowledge, creating awkward issues for managers tasked with interacting with the public and potentially the media. Worse, false negative predictions could result in a delayed management response or, if the outbreaks are never observed, no management response at all. Heron et al. (2010) found the number of false positive predictions of disease outbreaks could be minimised by including winter temperatures in a predictive model for White Syndromes on the Great Barrier Reef, since cold temperatures could knock back the pathogen load. Winter temperatures have been excluded from the model presented here. Targeted research and monitoring requires resources but surveys revealing a model prediction to be a false positive can increase our understanding of outbreak causation. In future years, the targeted research facilitated by the model presented here may increase our understanding of the relative importance of winter versus summer temperatures, at which point the case for combining the model presented here with that of Heron et al. (2010) may be better substantiated.

The means by which managers or conservationists might contain outbreaks once they have started are certainly limited and all, like phage therapy (Matsuzaki et al. 2005; Rosenberg et al. 2007) and stimulating coral immune systems (see examples for other invertebrates in Little and Kraaijeveld 2004), are strictly experimental and likely to be prohibitively expensive over all but small spatial scales. Nevertheless, there is a need to know where disease outbreaks are occurring, given the responsibility managers have to communicate about the condition of reefs. Aside from communication-based awareness-raising efforts, managers can work towards identifying Reef-wide management strategies for mitigating disease impacts in two ways: (1) by testing the effectiveness of reactively mitigating anthropogenic stress that could otherwise lengthen recovery timeframes at outbreak sites (e.g., water quality and physical stressors, Maynard et al. 2009) and (2) work with researchers to increase our understanding of the role of process level actions like herbivory and predation by butterflyfish on disease transmission (Aeby and Santavy 2006).

A disease response plan has been developed collaboratively with marine managers in Australia (Beeden et al. in review), and our model-based tools (versions 1 and 2), forms part of the early warning system within the plan. In combination with: (1) a volunteer monitoring network used to ground-truth predictions and (2) a seasonal outlook of disease risk based on preceding winter conditions produced by the model presented in Heron et al. (2010), our tool will enable more surveys at sites predicted to have high outbreak likelihood. Such surveys can, when combined with experimental research on disease causality, lead to further increases in our understanding of outbreak causation and further refinements of the model presented here. Insight could also be gained by integrating the disease model presented here with physiological stress models (e.g., Anthony et al. 2009), which would represent an important step towards a more mechanistic framework for predicting disease outbreak likelihood.

Climate change projections (e.g., Meehl et al. 2007) suggest that the conditions that caused severe outbreaks in 2002 and anomalous WS abundance levels in 2009 will occur more frequently in the future. Above, we highlight that an increased frequency and severity of bleaching events in the future could increase disease susceptibility and lower host threshold densities required for outbreaks. This would significantly increase the chance of outbreaks even on moderate cover (25–40%) reefs, which are far more common than high cover (40+%) reefs on the GBR and throughout most of the Indo-Pacific. Accordingly, developing new tools and refining existing ones like that presented here will continue to be critical to increasing our understanding of outbreak causation and could lead to the capacity to produce more defensible projections of reef state in an era of climate change.

In the iterative approach presented here, a predictive model was constrained around conditions at sites where disease outbreaks are known to have occurred in the past. The model was then used to predict outbreaks and validated and refined when stressful conditions predicted outbreaks would occur. This approach is already leading to an improved understanding of disease causation, can facilitate management responses and can be applied to other coral diseases and/or other regions.

Acknowledgments This work has been made possible by grants from the Marine and Tropical Sciences Research Facility through the Reef and Rainforest Research Centre and the Great Barrier Reef Marine Park Authority (GBRMPA) and benefited from the support of: the Coral Disease Working Group of the GEF Coral Reef Targeted Research project, the Australian Greenhouse Office, Australian Bureau of Meteorology, CSIRO Marine and Atmospheric Research

and the Ocean Biology Processing Group of NASA. Field staff of the AIMS Long-Term Monitoring Program provided the disease survey data for 2002–2008, and G. Torda and Y. Sato assisted with surveys conducted in 2009. The AEDA CERF Hub and the University of Melbourne provided support to the first author and versions of the manuscript improved following insightful comments from several anonymous reviewers. This is a contribution by the ARC Centre of Excellence for Coral Reef Studies. The manuscript contents are solely the opinions of the authors and do not constitute a statement of policy, decision, or position on behalf of NOAA or the U. S. Government.

References

Aeby GS, Santavy DL (2006) Factors affecting susceptibility of the coral *Montastrea faveolata* to black-band disease. Mar Ecol Prog Ser 318:103–110

Aeby GS, Hutchinson M, MacGowan P (2008) Hawaii's rapid response contingency plan for events of coral bleaching, disease or crown-of-thorns starfish outbreaks. The Division of Aquatic Resources, Department of Land and Natural Resources, Honolulu, HI

Anthony KRN, Hoogenboom M, Maynard JA, Middlebrook R, Grottoli A (2009) Energetics approach to predicting coral mortality risk from bleaching. Funct Ecol 23:539–550

Antolin MF (2008) Unpacking β: Within-host dynamics and evolutionary ecology of pathogen transmission. Annu Rev Ecol Evol Syst 39:415–437

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

Bass DK, Miller IR (1996) Crown-of-thorns starfish and coral surveys using the manta tow and scuba search techniques. Long-term Monitoring of the Great Barrier Reef Standard Operational Procedure Number 1. Australian Institute of Marine Science, Townsville

Berkelmans R, Oliver JK (1999) Large-scale bleaching of corals on the Great Barrier Reef. Coral Reefs 18:55–60

Berkelmans R, De'ath G, Kininmonth S, Skirving WJ (2004) A comparison of the 1998 and 2002 coral bleaching events on the Great Barrier Reef: Spatial correlation, patterns, and predictions. Coral Reefs 23:74–83

Bourne DG, Garren M, Work TM, Rosenberg E, Smith GW, Harvell CD (2009) Microbial disease and the coral holobiont. Trends Microbiol 17:254–262

Bruckner AW (2002) Priorities for effective management of coral diseases. NOAA, Washington, DC

Bruno JF, Selig ER, Casey KS, Page CA, Willis BL, Harvell CD, Sweatman H, Melendy AM (2007) Thermal stress and coral cover as drivers of coral disease outbreaks. PLOS Biology 5:1220–1227

Butterworth MH, Semenov MA, Barnes A, Moran D, West JS, Fitt BDL (2010) North-South divide: contrasting impacts of climate change on crop yields in Scotland and England. J R Soc Interface 7:123–130

Diaz M, Madin J (2010) Macroecological relationships between coral species' traits and disease potential. Coral Reefs. doi:10.1007/s00338-010-0668-4

English S, Wilkinson CR, Baker V (eds) (2004) Survey manual for tropical marine resources, 2nd edn. Townsville, Australian Institute of Marine Science

Griffin DA, Rathbone CE, Smith GP, Suber KD, Turner PJ (2004) A decade of SST satellite data. On-line report: http://www.cmar.csiro.au/remotesensing/oceancurrents/ten_years_of_SST.doc Hobart, Australia

Harvell CD, Altizer S, Cattadori Im, Harrington L, Weil E (2009) Climate change and wildlife diseases: When does the host matter the most? Ecology 90:912–920

Harvell CD, Mitchell CE, Ward JR, Altizer S, Dobson AP, Ostfeld RS, Samuel MD (2002) Climate warning and disease risks for terrestrial and marine biota. Science 296:2158–2162

Harvell CD, Jordan-Dahlgren E, Merkel S, Rosenberg E, Raymundo L, Garriet S, Ernesto W, Willis BL (2007) Coral disease, environmental drivers, and the balance between coral and microbial associates. Oceanography 20:172–195

Heron SF, Willis BL, Skirving WJ, Eakin MC, Page CA, Miller IR (2010) Summer hot snaps and winter conditions: modelling white syndrome outbreaks on Great Barrier Reef Corals. PloS One. doi:10.1371/journal.pone.0012210

Hijmans RJ, Forbes GA, Walker TS (2000) Estimating the global severity of potato late blight with GIS-linked disease forecast models. Plant Pathol (Oxf) 49:697–705

Hughes TP (1994) Catastrophes, phase shifts, and large-scale degradation of a Caribbean coral reef. Science 265: 547–1551

Lafferty KD, Holt RD (2003) How should environmental stress affect the population dynamics of disease? Ecol Lett 6:454–664

Little TJ, Kraaijeveld AR (2004) Ecological and evolutionary implications of immunological priming in invertebrates. Trends Ecol Evol 19:58–60

Marshall PA, Baird AH (2000) Bleaching of corals on the Great Barrier Reef: differential susceptibilities among taxa. Coral Reefs 19:155–163

Matsuzaki S, Rashel M, Uchiyama J, Sakurai S, Ujihara T, Kuroda M, Ikeuchi M, Tani T, Fujieda M, Wakiguchi H, Imai S (2005) Bacteriophage therapy: a revitalized therapy against bacterial infectious diseases. J Infect Chemother 11:211–219

Maynard JA, Johnson JE, Marshall PA, Goby G, Spillman C (2009) A strategic framework for responding to coral bleaching events in a changing climate. Environ Manage 44:1–11

Maynard JA, Turner PJ, Anthony KRN, Baird AH, Berkelmans R, Eakin CM, Johnson JE, Marshall PA, Packer GR, Rea A, Willis BL (2008) ReefTemp: an interactive monitoring system for coral bleaching using high-resolution SST and improved stress predictors. Geophys Res Lett. doi:10.1029/2007GL032175

Meehl GA, Covery C, Delworth T, Latif M, McAvaney B, Mitchell JFB, Stouffer RJ, Taylor KE (2007) The WCRP CMIP3 multimodel dataset: a new era in climate change research. Bull Am Meteorol Soc 88:1383–1394

Miller J, Muller E, Rogers C, Waara R, Atkinson A, Whelan KRT, Patterson M, Witcher B (2009) Coral disease following a massive bleaching in 2005 causes a 60% decline in coral cover on reefs in the US Virgin Islands. Coral Reefs 28:925–937

Mydlarz LD, Couch CS, Weil E, Smith G, Harvell CD (2009) Immune defenses of healthy, bleached and diseases *Montastrea faveolata* during a natural bleaching event. Dis Aquat Org 87:67–78

Myers RL, Raymundo LJ (2009) Coral disease in Micronesian reefs: a link between disease prevalence and host abundance. Dis Aquat Org 87:97–104

Page CA, Baker DM, Harvell CD, Golbuu Y, Raymundo L, Neale SJ, Rosell KB, Rypien KL, Andras JP, Willis BL (2009) Influence of marine reserves on coral disease prevalence. Dis Aquat Org 87:135–150

Page CA, Willis BL (2008) Epidemiology of skeletal eroding band on the Great Barrier Reef and the role of injury in the initiation of this widespread coral disease. Coral Reefs 27:257–272

Palmer CV, Bythell JC, Willis BL (2010) Levels of immunity parameters underpin bleaching and disease susceptibility of reef corals. FASEB J. doi:10.1096/fj.09-152447

Patterson KL, Porter JW, Ritchie KB, Polson SW, Mueller E, Peters EC, Santavy DL, Smith GW (2002) The etiology of white pox, a lethal disease of the Caribbean elkhorn coral, *Acropora palmata*. Proc Natl Acad Sci USA 99:8725–8730

Raymundo LJ, Halford AR, Maypa AP, Kerr AM (2009) Functionally diverse reef-fish communities ameliorate coral disease. Proc Natl Acad Sci USA 106:17067–17070

Ritchie KB (2006) Regulation of microbial populations by coral surface mucus and mucus-associated bacteria. Mar Ecol Prog Ser 322:1–14

Rosenberg E, Koren O, Reshef L, Efrony R, Zilber-Rosenberg I (2007) The role of microorganisms in coral health, disease and evolution. Nat Rev Microbiol 5:356–362

Rudolf VHW, Antonovics J (2005) Species coexistence and pathogens with frequency-dependent transmission. Am Nat 166: 112–118

Savary S, Teng PS, Willocquet L, Nutter FW (2006) Quantification and modeling of crop losses: A review of purposes. Annu Rev Phytopathol 44:89–112

Sato Y, Bourne DG, Willis BL (2009) Dynamics of seasonal outbreaks of black band disease in an assemblage of *Montipora* species at Pelorus Island (Great Barrier Reef, Australia). Proc R Soc Lond B Biol Sci 276:2795–2803

Sokal RR, Rohlf FJ (1995) Biometry: the principles and practice of statistics in biological research, 3rd edn. WH Freeman and Co, New York

Sussman M, Willis BL, Victor S, Bourne DG (2008) Coral pathogens identified for white syndrome (WS) epizootics in the Indo-Pacific. PLoS One. doi:10.1371/journal.pone.0002393

Sussman M, Mieog JC, Doyle J, Victor S, Willis BL, Bourne DG (2009) *Vibrio* zinc-metalloprotease causes photoinactivation of coral endoysmbionts and coral tissue lesions. PLoS One. doi: 10.1371/journal.pone.0004511

Sweatman H, Cheal A, Coleman G, Emslie M, Johns K, Jonker M, Miller I, Osborne K (2008) Long-term monitoring of the Great Barrier Reef, Status Report No. 8. Australian Institute of Marine Science

Williams GJ, Aeby GS, Cowie ROM, Davy SK (2010) Predictive modelling of coral disease distribution within a reef system. PLoS One. doi:10.1371/journal.pone.0009264

Willis BL, Page CA, Dinsdale EA (2004) Coral disease on the Great Barrier Reef. In: Rosenberg E, Loya Y (eds) Coral health and disease. Springer-Verlag, Berlin, pp 69–104

Wilkinson C, Souter D (eds) (2008) Status of Caribbean coral reefs after hurricanes and bleaching in 2005. Global Coral Reef Monitoring Network, Townsville

Wobeser GA (2006) Essentials of disease in wild animals. Blackwell Publishing, Oxford, UK

Wooldridge SA (2009) A new conceptual model for the enhanced release of mucus in symbiotic reef corals during 'bleaching' conditions. Mar Ecol Prog Ser 396:145–152

Work TM, Richardson LL, Reynolds TL, Willis BL (2009) Biomedical and veterinary science can increase our understanding of coral disease. J Exp Mar Biol Ecol 362:63–70