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## Analyzing the Relationship Between Ocean Temperature Anomalies and Coral Disease Outbreaks at Broad Spatial Scales

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### Abstract

Ocean warming due to climate change could increase the frequency and severity of infectious coral disease outbreaks by increasing pathogen virulence or host susceptibility. However, little is known about how temperature anomalies may affect disease severity over broad spatial scales. We hypothesized that the frequency of warm temperature anomalies increased the frequency of white syndrome, a common scleractinian disease in the Indo-Pacific. We created a novel 4 km satellite temperature anomaly dataset using data from NOAA's Pathfinder program and developed four different temperature anomaly metrics, which we correlated with white syndrome frequency at 47 reefs spread across 1500 km of the Great Barrier Reef. This cross-sectional epidemiological analysis used data from disease field surveys conducted by the Australian Institute of Marine Science six to twelve months after the summer of 2002, a year of extensive coral bleaching. We found a highly significant positive relationship between the frequency of warm temperature anomalies and the frequency of white syndrome. There was also a highly significant, nearly exponential relationship between total coral cover and the number of disease cases. Furthermore, coral cover modified the effect of temperature on disease frequency. Both high coral cover (>50%) and anomalously warm water appear to be necessary for white syndrome outbreaks to occur and these two risk factors explained nearly 75% of the variance in disease cases. These results suggest that rising ocean temperatures could exacerbate the effects of infectious diseases on coral reef ecosystems.

### 1. Introduction

Over the last four decades, coral cover has declined dramatically on reefs worldwide [Gardner et al., 2003; Bellwood et al., 2004]. Several factors are thought to be responsible for this decline including overfishing [Jackson, 1997; Pandolfi et al., 2003], terrestrial run-off [Fabricius, 2005], climate change [Hoegh-Guldberg, 1999; Hughes et al., 2003], and infectious disease [Aronson and Precht, 2001]. There is growing recognition that we

need to focus on possible synergisms among these and other stressors [Hughes and Connell, 1999; Lenihan et al., 1999]. In the last several years, the relationship between climate conditions and disease has received more attention as researchers have connected factors such as temperature and precipitation with increases in human and wildlife diseases [Pascual et al., 2000; Patz, 2002; Kutz et al., 2005; Pounds et al., 2006]. Yet, few studies have focused on the effects of climate change on diseases in the ocean, particularly at broad spatial scales.

Disease has already had significant effects on coral reef ecosystems. Several diseases have altered the landscape of Caribbean reefs, causing the near extirpation of the keystone herbivore *Diadema antillarum* [Lessios, 1988] and dramatic losses of *Acropora cervicornis* and *Acropora palmata* [Aronson and Precht, 2001; Aronson et al., 2002]. These disease outbreaks mediated a shift from coral- to algal-dominated communities in the Caribbean [Aronson and Precht, 2001]. The scale and severity of coral loss on many Caribbean reefs is unprecedented in the paleontological record [Aronson and Precht, 2001; Wapnick et al., 2004] of many reefs and indicative of the emergence of a novel stressor [Aronson and Precht, 2001]. Recent studies quantifying both disease reports [Ward and Lafferty, 2004] and the number of described coral diseases [Sutherland et al., 2004] suggest that the frequency of marine diseases is increasing and many of these diseases are the result of previously unknown pathogens. Reports from the Pacific indicate that diseases of reef-building corals may be far more widespread than previously believed [Sutherland et al., 2004; Willis et al., 2004; Aeby, 2005; Raymundo et al., 2005]. Although coral disease is likely underreported in the Pacific due to a lack of disease research, increases in coral disease cases have been detected since the late 1990s [Willis et al., 2004]. These lines of evidence provide strong support for the hypothesis that there has been a real increase in the number of coral disease reports over the last three decades [Harvell et al., 1999; Ward and Lafferty, 2004].

The causes underlying recent increases in coral disease outbreaks are complex and poorly understood, in part because of a paucity of knowledge about the identity and sources of most coral pathogens [Sutherland et al., 2004]. Pathogens have a variety of purported vectors and reservoirs including algae [Nugues et al., 2004], invertebrates [Rosenberg and Falkovitz, 2004; Williams and Miller, 2005], sediment transported from the Sahel region of northern Africa [Shinn et al., 2000], and sewage effluent [Patterson et al., 2002]. In addition, some diseases are hypothesized to be associated with changes in corals' microbial communities, rather than the result of an external infectious pathogen.

Abiotic factors like nutrients and temperature can exacerbate disease severity, but the scale of their effects may vary. For example, nutrients increased the severity of two coral diseases in experimental manipulations [Bruno et al., 2003]. However, inputs of terrestrial pollution, including nutrients, do not always result in elevated disease levels on affected reefs [Weil, 2004; Willis et al., 2004]. Although localized inputs of nutrients may play some role in disease outbreaks, the regional scale of most outbreaks [Aronson and Precht, 2001; Kim and Harvell, 2004] indicates that a climatic variable like temperature may be a critical driver of disease dynamics. For example, disease prevalence, or the proportion of the total population that is diseased, may be related to the frequency and magnitude of warm temperature anomalies. Extensive work clearly links these anomalies to coral bleaching events [Glynn et al., 1988; Glynn and D'croz, 1990; Hoegh-Guldberg, 1999; Bruno et al., 2001; Fitt et al., 2001; Liu et al., 2003; Strong et al., 2004]. Since bleaching is a sign of physiological stress in corals [Glynn, 1993; Brown, 1997], it is expected that bleached or thermally-stressed corals would be more susceptible to opportunistic and residential pathogens [Hayes et al., 2001; Rosenberg and Ben-Haim, 2002].

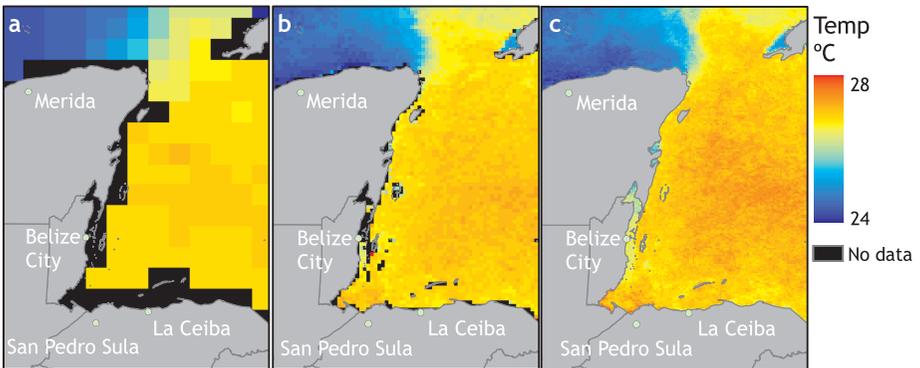


Plate 1. An example of the benefits of increasing resolution on the Yucatan peninsula and Caribbean Sea from; (a) 50 km HotSpot data to (b) 9 km Pathfinder data to (c) 4 km Pathfinder data. In the 4 km data, there is less missing data, allowing for greater coverage of coastal areas where many reefs occur. In addition, the 4 km data displays more spatial structure and precision in the temperature values. Data are from January climatological averages, monthly for the 50 km data and from the first week of January for the 9 km and 4 km data.

Forecast sea surface temperature (SST) models predict that the frequency and severity of warm temperature anomalies will increase with climate change [Hoegh-Guldberg, 1999; Sheppard, 2003; Sheppard and Rioja-Nieto, 2005]. Ocean temperature has already increased, on average, 0.4-0.8°C [Folland et al., 2001] from 1861 to 2000, with some regional variation [Casey and Cornillon, 2001]. These anomalies and the general warming of the ocean could have several effects on disease. One possible outcome of global warming is that the summer “disease season” may become more severe, as summer temperature maxima increase, and longer, as these elevated thermal regimes start earlier and persist later in the season. Seasonal variability in coral disease abundance has been found in multiple field studies in different regions. In the Caribbean, several coral diseases, including black band [Edmunds, 1991; Kuta and Richardson, 2002], white pox [Patterson et al., 2002], and dark spots disease [Gil-Agudelo and Garzon-Ferreira, 2001], are more prevalent or spread across colonies more rapidly during summertime than during cooler seasons. On the Great Barrier Reef (GBR), white syndrome frequency was greater in summer than winter on surveyed reefs [Willis et al., 2004]. Similarly, in the summer of 2002 on the GBR, Jones et al. [2004] documented a localized outbreak of atrematous necrosis. In addition, shorter or warmer winters may release some infectious diseases from the low-temperature control that provides hosts with a seasonal escape from disease [Harvell et al., 2002]. Climate warming has also been predicted to alter the geographic distribution of infectious disease by shifting host and pathogen latitudinal ranges pole-ward [Marcogliese, 2001; Harvell et al., 2002].

Testing the hypothesis that the frequency or intensity of temperature anomalies can influence coral disease dynamics requires high quality data on temperature and disease frequency. Here we discuss how analysis of these broad scale questions is now possible using a newly developed high-resolution satellite temperature anomaly dataset. We then present a case study where we examine the effects of the warm temperature anomalies on white syndrome, an emergent sign of disease on Great Barrier Reef corals. We also discuss future research directions for testing the relationship between temperature and disease.

## 2. Using Remote Sensing Data to Explore the Climate Warming Disease Outbreak Hypothesis

Most documented coral disease outbreaks have been at the scale of ocean basins [Lessios, 1988; Aronson and Precht, 2001; Willis et al., 2004]. Previously, correlating these outbreaks with ocean temperature was complicated by a scale mismatch. *In situ* temperature loggers can be highly effective at capturing small-scale variability (1-100 m) [Leichter and Miller, 1999; Castillo and Helmuth, 2005; Leichter et al., 2005], but are limited in their spatial extent. On the other hand, the 50 km HotSpot mapping (Plate 1a) [Strong et al., 2004], although effective for predicting bleaching at broad scales [Bruno et al., 2001; Berkelmans et al., 2004; Strong et al., 2004], is too coarse to accurately represent the temperature of waters surrounding many reefs and may not sufficiently capture local variability [Toscano et al., 2000]. Therefore, the development of new, higher resolution remote sensing products is required to detect correlations between temperature anomalies and disease outbreaks.

We developed a novel satellite temperature anomaly product to investigate the relationship between sea surface temperature and disease at broad spatial scales. Our dataset used the 4 km Advanced Very High Resolution Radiometer (AVHRR) Pathfinder Version 5.0 SST dataset

produced by NOAA's National Oceanographic Data Center and the University of Miami's Rosenstiel School of Marine and Atmospheric Science (<http://pathfinder.nodc.noaa.gov>). These data now provide the longest sea surface temperature record at the highest resolution of any global satellite dataset. The Pathfinder Version 4.2 dataset had a 9 km resolution [Kilpatrick et al., 2001], a marked improvement from the 50 km data. However, inaccuracies in the land mask, which defines the extent of the data, resulted in coverage of only 60% of reef areas (Plate 1b). Using a more refined land mask and other algorithm enhancements, the 4 km Pathfinder Version 5.0 dataset reprocessed the full AVHRR record from 1985-2005 to create an improved SST climate data record. With these improvements, temperature records are now available for more than 98% of reefs worldwide (Plate 1c). Appropriate use of satellite temperature data requires validation with *in situ* loggers [Reynolds et al., 2002]. In theory, an infrared observing satellite like AVHRR only measures an integrated temperature over approximately the top 10 micrometers of the ocean surface. Therefore, we validated the assumption that the Pathfinder 4 km Pathfinder Version 5.0 SST values reflect temperatures on shallow reefs, where the majority of reef-building corals are found, by comparing Pathfinder SST estimates to *in situ* temperature logger data collected by the Australian Institute of Marine Science ([www.reeffutures.org](http://www.reeffutures.org)) on nine shallow reefs on the GBR (5 m to 9 m depth). We fit a linear multi-level model and found a highly significant relationship ( $p < 0.0001$ ) with a common value for the slope of all the reefs of 0.96 (SE = 0.017). We found the difference between satellite-derived and benthic temperatures was generally less than the 0.2°C error of most *in situ* loggers. We also ran linear regression analyses for each reef to investigate the generality of this relationship at all nine reefs (Table 1) and found that the satellite temperature measurements were a very good predictor of benthic water temperature.

One key advantage of satellite data is their ability to provide a long-term temporal record of temperature values, which enables users to create accurate climatologies, or average long-term patterns (Plate 2a). These climatologies are then used as the basis for calculating deviations from typical weekly, monthly, seasonal, or annual temperatures. For disease analyses, relevant data include the frequency of deviations or temperature anomalies, the duration of anomaly events, occurrence of wintertime anomalies, and the geographic extent of the anomaly.

TABLE 1. Relationship between weekly averaged satellite and *in situ* temperatures at nine reefs on the GBR reefs at 5-9 m depth. Field data were collected by the Australian Institute of Marine Science in cooperation with CRC Reef Research Centre and the Great Barrier Reef Marine Park Authority.

Reef	Latitude	Longitude	Period	n	P	R <sup>2</sup>
Agincourt	-16.0384	145.8688	1996-2004	173	$p < .0001$	0.91
Chicken	-18.6521	147.7217	1996-2004	244	$p < .0001$	0.94
Davies	-18.8060	147.6686	1996-2004	200	$p < .0001$	0.96
Dip	-18.3999	147.4519	1997-2004	197	$p < .0001$	0.92
East Cay	-21.4698	152.5665	1995-2004	248	$p < .0001$	0.96
John Brewer	-18.6188	147.0815	1996-2004	239	$p < .0001$	0.94
Lizard Island	-14.6915	145.4692	1996-2004	134	$p < .0001$	0.90
Myrmidon	-18.2572	147.3813	1995-2004	205	$p < .0001$	0.93
Turner Cay	-21.7031	152.5601	1997-2004	204	$p < .0001$	0.96

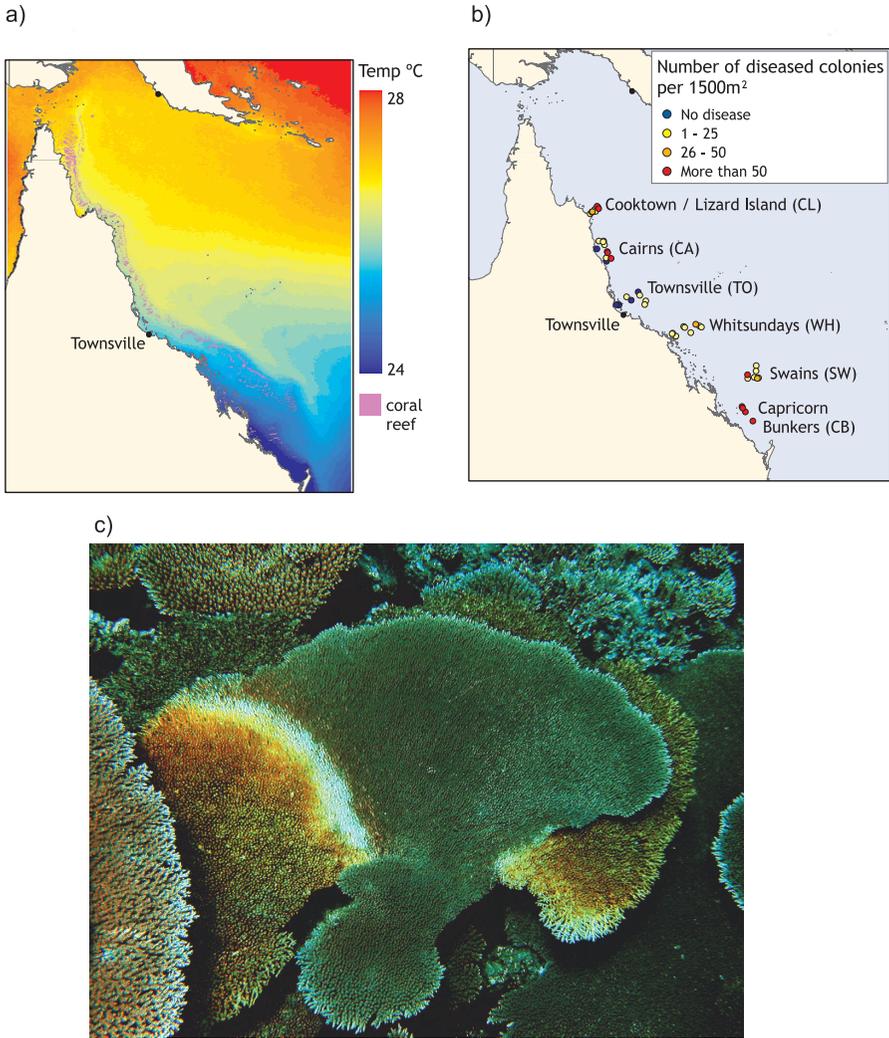


Plate 2. (a) Mean sea surface temperature (°C) from 1985-2003. Averages from this time period were used to create weekly climatologies for each surveyed reef. (b) Number of white syndrome cases in 2002-2003 based on data collected by the Australian Institute of Marine Science's Long-term Monitoring Program surveys. Each of the six latitudinal areas or sectors are labeled with their names and abbreviations. Cooktown/Lizard Island (CL) has nine surveyed reefs, Cairns (CA) has ten, Townsville (TO) has eight, Whitsundays (WH) has nine reefs, Swains (SW) has seven, and Capricorn Bunkers (CB) has four. For each reef, three different sites were surveyed. Disease cases were highest in the Cooktown/Lizard Island and Capricorn Bunkers sectors. (c) Example of white syndrome spreading across *Acropora cytherea*. White areas are coral skeleton that have been recently exposed following die-off behind the disease front.

### 3. Case Study on the Great Barrier Reef

The Great Barrier Reef (GBR) is the largest barrier reef system in the world and one of the most highly managed, with more than a third of reef area in marine reserves, or no-take areas [Fernandes et al., 2005]. Recent surveys suggest that disease is more prevalent than previously believed on the GBR [Willis et al., 2004]. Black band, white syndrome, brown band, and skeletal eroding band have all been reported on the GBR [Willis et al., 2004]. In spite of the well-documented relationship with increased temperatures and black band disease in the Caribbean [Edmunds, 1991; Kuta and Richardson, 2002], it did not vary in frequency over the course of our study [Willis et al., 2004]. We focused our analysis on white syndrome, which increased dramatically in 2002-2003 on some reefs. We tested specific hypotheses about how temperature might affect disease frequency by using an information-theoretic approach to evaluate different thermal stress metrics.

#### *Disease Surveys*

The disease data used for this analysis were collected by the Australian Institute of Marine Science's Long-term Monitoring Program (AIMS LTMP) during their 2002-2003 surveys. Surveys were performed at 47 reefs in a stratified design. Five permanent 50 m  $\times$  2 m belt transects were surveyed at three sites on each reef at approximately 6-9 m depth (full methods in *Sweatman et al.*, 2003). For each transect, the number of colonies infected with white syndrome was quantified. Reefs were grouped by latitudinal sectors, which together cover more than 1500 km and a variety of different oceanographic regimes (Plate 2b). White syndrome is characterized by a band of recently-exposed white skeleton, sometimes preceded by a band of bleached tissue at the tissue-skeleton interface. The white band moves across the colony as the front of tissue mortality progresses, potentially resulting in mortality of the whole colony (Plate 2c). White syndrome has been recorded on at least 17 species from 4 families, including Acroporidae, Pocilloporidae and Faviidae, families that constitute a significant percentage of overall coral cover on the GBR [Willis et al., 2004]. White syndrome is similar to Caribbean white diseases such as white band I, white band II, white plague I, and white plague II in its disease signs. Because the pathogen(s) that cause white disease in the Pacific are not known, we cannot state with certainty that the syndrome does not represent more than one distinct disease [Willis et al., 2004] or that the underlying etiology is infectious.

#### *Temperature Data*

Consistent with previous analyses of satellite data in coral studies, we used nighttime weekly temperature averages [Liu et al., 2003; Strong et al., 2004]. Nighttime daily averaged data had too many gaps and would have required extensive interpolation. Although they may not capture short duration events, weekly data provide substantially more continuity in the record and still represent a time scale short enough to capture most thermal stress events that negatively impact corals [Glynn, 1993; Podesta and Glynn, 2001]. To measure anomalies, we first calculated weekly climatologies, or the mean values at each calendar week from 1985-2003 for each 4  $\times$  4 km pixel (Plate 2a). Missing data in the climatologies were interpolated using a Piecewise Cubic Hermite Interpolating Polynomial (PCHIP) function in MATLAB [The Mathworks Inc., 2005]. The climatologies were then smoothed using a five-week running mean to minimize unusual fluctuations from periods of limited data availability. Gaps in weekly temperature observations were also interpolated using the PCHIP function without modifying the original data.

### *Thermal Stress Metrics*

To test the hypothesis that temperature affects disease frequency, we first designed temperature metrics relevant to disease. Infectious diseases are interactions between hosts and pathogens. Temperature can increase host susceptibility, but it can also increase pathogen growth rate, transmission rate, and over-wintering survival [Harvell et al., 2002]. Because of the potential complexity of this relationship and the paucity of data about most pathogens, no specific algorithm exists for disease prediction. However, data on how corals respond to stress and epidemiological theory provide a general guide to temperature thresholds that may be applicable to disease dynamics. We developed a series of temperature metrics for our case study based on metrics known to have a physiological effect on coral health. Three of our four thermal stress metrics are based on an anomaly threshold of 1°C, because this is widely assumed to estimate the point at which a warm temperature anomaly induces a measurable physiological stress in a coral host, and in general, increases of  $\geq 1^\circ\text{C}$  above normal summertime temperatures are thought to induce bleaching [Glynn, 1993; Glynn, 1996; Winter et al., 1998; Hoegh-Guldberg, 1999; Berkelmans, 2002]. All of the metrics measured deviations from the location-specific climatologies we created. Because disease surveys were conducted at different times of year (November-March), we standardized all of our metrics to include the number of anomalies during the 52 weeks prior to each disease survey. We used the latitude and longitude of each reef to match it with its corresponding pixel in the satellite data.

We developed each temperature metric to investigate a specific hypothesis related to the relationship between thermal stress and disease. The first three metrics are location-specific, which assumes that corals are acclimated to the thermal regime at their location. Work by Berkelmans and Willis [1999] suggests that corals exhibit some degree of local acclimation or adaptation. The fourth metric assumes that temperature is affecting disease rates at regional scales. If thermal anomalies are acting on the pathogen itself, by increasing growth or reproductive rates, they would likely be acting at a regional scale due to high pathogen mobility [McCallum et al., 2003].

### *Metric Descriptions*

- 1) *WSSTA = Weekly Sea Surface Temperature Anomalies* = deviations of 1°C or greater from the mean climatology during a particular week at a given location from 1985-2003. This metric is designed to be both location and season-specific by determining whether the temperature is unusual for that location at a particular week of the year.
- 2) *TSA = Thermal Stress Anomalies* = deviations of 1°C or greater from the mean maximum climatological weekly temperature from 1985-2003. The mean maximum climatological week is the warmest week of the 52 weekly climatologies. This metric is also site-specific but designed to detect deviations from typical summertime highs and is similar to metrics used by the Coral Reef Watch program [Liu et al., 2003; Strong et al., 2004].
- 3) *LSTA = Local Temperature Stress Anomalies* = deviations from the upper 2.5% of all weekly measurements taken at that location from 1985-2003. This metric is site-specific. It is designed to detect whether temperatures are unusual based on the distribution and extremes of all measured temperatures, regardless of calendar week.
- 4) *RTSA = Regional Thermal Stress Anomalies* = deviations from the upper 2.5% of all weekly measurements taken at all reefs. For this metric, local temperatures are compared to regional average values.

TABLE 2. For each thermal metric we calculated: the AIC, a measure of expected relative Kullback Leibler information; the  $AIC_C$ , AIC corrected for small sample size; the difference,  $\Delta_i$  between each model and the best model in the set; and the Akaike weight,  $w_i$ . The sum of all the Akaike weights is equal to 1.0 and each weight represents the approximate likelihood that a specific model is the best model of those compared, in a Kullback-Leibler information sense. For the models that involve a transformed response, likelihoods were adjusted with the Jacobian of the transformation to make the  $AIC_C$  values comparable.

Metric	AIC	$AIC_C$	$\Delta_i$	$w_i$
WSSTA	1340	1342	0	1
TSA	1464	1465	123	0
LTSA	1479	1481	138	0
RTSA	1476	1477	135	0

### *Metric Selection and Statistical Analysis*

We evaluated the different thermal stress metrics (Table 2) using Akaike Information Criterion (AIC) [Akaike, 1973; R 2.1.1, 2005]. Identifying which thermal stress metric best explains the relationship between temperature and disease can facilitate the development of an appropriate model for a specific coral-pathogen system. Different diseases may be affected by different temperature characteristics. For example, TSAs may be highly correlated with an increase of one disease while another disease may be more correlated with WSSTAs. AIC is an estimate of expected relative Kullback-Leibler information, an information-theoretic measure of the distance between models where smaller AIC values should be preferred. In practice, only the relative differences in AIC between models are meaningful. When these differences are normalized as Akaike weights, ( $w_i$ ), they can also be given a probabilistic interpretation. Each  $w_i$  is the weight of evidence favoring model  $i$  as the best model among the models under consideration [Anderson et al., 2000; Anderson and Burnham, 2002].

Coral cover is clearly related to the number of white syndrome cases on a reef (Figure 1A) and was also included in the analysis. We used multiple, nonlinear regression analysis [StataCorp LP, 2006] to analyze the relationship between the two independent variables, thermal stress and coral cover, and the dependent variable, the total number of white syndrome cases at each reef (i.e., the number/1500m<sup>2</sup>). We used a Poisson model and a quadratic function for the thermal stress metrics and a linear function for coral cover.

### *Results*

The austral summer in 2002 triggered the most extensive bleaching event ever documented on the GBR [Berkelmans et al., 2004]. Surveys conducted after this summer found a major increase in disease frequency with some sampled reef areas having more than 300 cases of disease [Willis et al., 2004].  $AIC_C$ , a small-sample (second order) bias-adjusted variant of AIC [Burnham and Anderson, 2002], indicated that the number of WSSTAs provided the best model fit (Table 2) and was used in the main analysis as the thermal

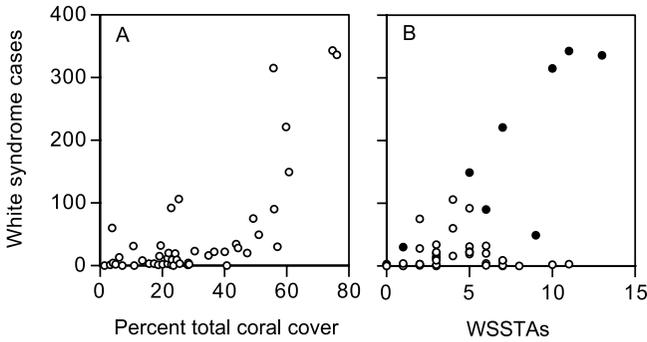


Figure 1. Relationships between total percent coral cover (A) and WSSTA frequency (B) and the number of white syndrome cases. Solid points in B represent reefs with >50% coral cover. Each point represents the values from a single reef (n = 47).

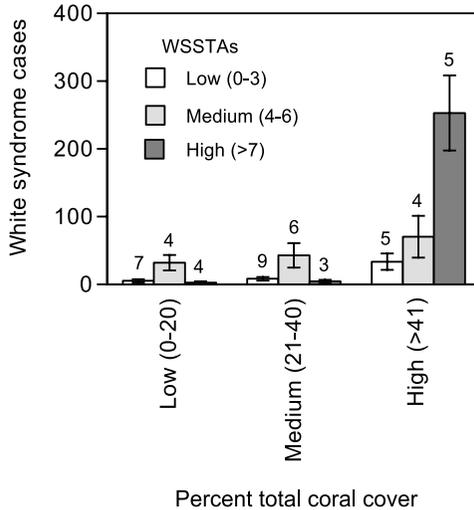


Figure 2. The effect of total coral cover and the number of WSSTAs on the number of white syndrome cases. Values are mean  $\pm$  1 SE. Values above error bars are the number of surveyed reefs in each of the nine categories.

stress metric. The whole Poisson regression model ( $R^2 = 0.73$ ) and both main effects (WSSTAs and coral cover) were all highly statistically significant at the reef scale (all  $P < 0.0001$ ,  $n = 47$ ). There was also a highly significant interaction between coral cover and WSSTAs ( $P < 0.0001$ ). At low and intermediate cover (0-40%), the number of white syndrome cases was greatest when annual WSSTAs were 4-6 and declined slightly with increasing WSSTA (Figure 2). However, when cover was high (>41%), white syndrome frequency was more than 3x greater when the frequency of WSSTAs was >7 than when it was 4-6 (Figure 2).

## Discussion

Temperature anomalies are known to be the underlying cause of mass coral bleaching [Podesta and Glynn, 2001; Liu et al., 2003; Berkelmans et al., 2004; Strong et al., 2004], but their relationship with infectious disease dynamics is not well understood. Our results suggest that warm temperature anomalies can significantly affect the frequency of white syndrome on the GBR, especially where coral cover is high. Total coral cover was clearly related to the number of white syndrome cases on a reef (Figure 1A) [Willis et al., 2004]. With few exceptions white syndrome frequency was relatively low (<30 cases/1500 m<sup>2</sup>, the area surveyed at each reef) when coral cover was <50% (Figure 1A) and was very high, 192 cases  $\pm$  46 (mean  $\pm$  1 SE) at the eight reefs where coral cover was >50%. The nearly exponential relationship between coral cover and disease cases (Figure 1A) suggests there is a threshold coral cover of approximately 50% that is generally required to for an outbreak of white syndrome to occur. This could be due to higher host densities on high coral cover reefs. Host density is widely known to influence disease dynamics [Anderson and May, 1986]. White syndrome has been documented in the major coral families on the GBR, including the abundant staghorn and tabular species of *Acropora* [Willis et al., 2004]. Therefore total coral cover is likely to be directly related to white syndrome host cover. However, the host density-prevalence relationship is not always clear for infectious coral diseases such as sea fan aspergillosis [Kim and Harvell, 2004] possibly because secondary transmission is rare among host colonies [Edmunds, 1991]. Additionally, several other aspects of total coral cover could also influence the dynamics of white syndrome and other infectious coral diseases. For example, coral cover could be positively related to animal vectors (i.e., coral predators and mutualists), which could increase rates secondary transmission, although Willis et al. [2004] found that the density of the corallivorous snail *Drupella* spp. was unrelated to white syndrome frequency on the GBR.

Temperature measured as WSSTA frequency also had a strong effect on the number of disease cases (Figure 1B). The selection of the WSSTA metric as the best model using an AIC approach suggests that the effect of thermal stress on white syndrome is both seasonal and location-specific. The WSSTA metric is the only metric we tested that incorporates temperature anomalies throughout the year. These findings are consistent with coral physiology studies, which have found that at the beginning of cooler months, zooxanthellae densities increase and coral tissue is built up [Brown et al., 1999]. With warmer or longer than usual summers or warmer winters these accumulations may not occur, increasing corals' vulnerability to future stress [Fitt et al., 2001]. Higher WSSTA frequencies may lead to chronic stress, which could increase host susceptibility and disease prevalence. However, WSSTAs could also have influenced disease frequency by increasing pathogen virulence. The relationship between WSSTA and white syndrome cases (Figure 1B) is also suggestive of a weak threshold response when the number of annual WSSTAs exceeds approximately seven. There were several reefs with >7 WSSTAs and a low number of cases (<10), however, all were low coral cover reefs (mean cover  $15.1 \pm 3.0$ ,  $n = 7$ ). In fact, the only reefs with >200 cases had >50% cover and a WSSTA frequency >7 (Figure 1B), suggesting that both conditions are necessary for white syndrome outbreaks to occur. Furthermore, these two risk factors explained nearly 75% of the variance in disease cases.

White syndrome frequency varied substantially among the six sectors, possibly due in part to regional temperature variation. Average WSSTA frequency within sectors was positively related to average disease frequency, largely because Capricorn Bunkers, the sector with by far the greatest number of cases also had the greatest number of WSSTAs (Figure 3) and the highest average coral cover [Willis et al., 2004]. Although other studies have found that higher nutrient levels are associated with increased disease severity [Kuta and Richardson,

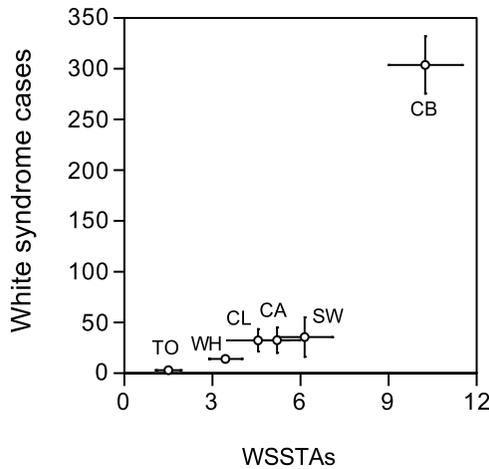


Figure 3. Relationship between mean thermal stress (WSSTAs) and the mean number of white syndrome cases at the sector scale. See Plate 2b for sector delineations and abbreviations. Values are mean  $\pm$  1 SE.

2002; Bruno et al., 2003], Willis et al. [2004] found that outer shelf reefs had higher levels of white syndrome than inner shelf reefs. Assuming that distance from shore is a proxy for nutrient availability, these findings suggest that nutrient and sediment input may not be primary contributors to increases in white syndrome [Willis et al., 2004]. In fact, several inner shelf reefs relatively close to shore in the Cairns sector had high WSSTA frequencies but very few disease cases in 2002, possibly because host density was also low or because other abiotic or biotic conditions inhibited white syndrome [Willis et al., 2004].

The importance of different abiotic and biotic factors driving white syndrome and other diseases is likely to vary with scale and the host-pathogen system [Bruno et al., 2003]. Ocean currents may facilitate spread or isolation of different pathogens [McCallum et al., 2003], but no empirical studies have yet quantified potential dispersal patterns. Biotic factors like host age or size structure are also likely to have significant effects on disease prevalence [Anderson and May, 1986; Dube et al., 2002; Lafferty and Gerber, 2002]. Older or larger hosts may be more vulnerable to disease [Dube et al., 2002; Borger and Steiner, 2005], which could be particularly devastating for coral populations where older or larger individuals are likely to have higher reproductive output [Hall and Hughes, 1996; Sakai, 1998; Dube et al., 2002].

Uncertainty about the identity and source of most pathogens represents a major challenge in understanding the factors that determine pathogen survival and development. Of the currently described coral diseases, only 5 of 18 [Sutherland et al., 2004] have been identified through fulfillment of Koch's postulates, which require a putative pathogen to be 1) isolated from a diseased individual, 2) grown in pure culture, and 3) transferred to a healthy organism where it induces the disease state [Koch, 1882]. An additional postulate, that the pathogen be reisolated from the infected organism, was not formulated by Koch, but is also typically recommended [Fredricks and Relman, 1996; Richardson, 1998; Sutherland et al., 2004]. Fulfilling Koch's postulates for coral pathogens has been challenging, in part due to the complex nature of the host-pathogen relationship, the possibility of multiple disease agents, and the difficulty of natural inoculation [Richardson, 1998;

Sutherland et al., 2004]. Identifying pathogens through Koch's postulates is not essential for epidemiological study [Fredricks and Relman, 1996], but without isolating them, it has been difficult to determine the mechanisms behind disease dynamics. For example, with a known, cultured pathogen, manipulative experiments on both the isolated pathogen and the host-pathogen system could yield insights into whether temperature is affecting disease by increasing expression of pathogen virulence factors, increasing pathogen growth or reproductive rates, or increasing host susceptibility [Harvell et al., 1999; Harvell et al., 2002].

Although the mechanisms are not known, this study found a strong correlative relationship between white syndrome and warm temperature anomalies. This relationship could have several implications for coral communities on the GBR. White syndrome affects key reef-building species on the GBR including the competitively dominant tabular acroporid corals that constitute a substantial percentage of total coral cover [Baird and Hughes, 2000; Connell et al., 2004]. Reductions in the abundance of these corals could cause shifts in species assemblages or abundances [Baird and Hughes, 2000]. In the Caribbean, loss of acroporid corals due to white band disease has led to a shift in dominance to *Agaricia* sp. on some reefs [Aronson et al., 2002] and precipitated a shift to macroalgal dominance on others. Predicted increases in the frequency and severity of thermal stress anomalies with global climate change [Hoegh-Guldberg, 1999] could exacerbate these kinds of disease effects. Our results and the balance of the published evidence from field studies comparing coral disease prevalence among seasons [Edmunds, 1991], years [Willis et al., 2004] and sites [Kuta and Richardson, 2002] suggest that water temperature plays a substantial role in coral disease dynamics. Because reef building corals are irreplaceable as marine foundation species [Bruno and Bertness, 2001], the synergism between temperature and disease could have cascading effects throughout reef ecosystems [Bruno et al., 2003].

#### 4. Future Research Directions

The development of 20 years of consistently processed satellite sea surface temperature and anomaly data for the GBR region represents a meaningful advancement in understanding the effects of temperature on several parameters of coral health including disease. Further validation of the satellite estimates will provide a better understanding of its limitations and enable more productive and effective use of the dataset. For example, in areas with persistent cloud cover, low data availability may decrease accuracy [Kilpatrick et al., 2001]. The relationship of the satellite-measured surface temperatures to temperatures at different depths will depend on bottom topography and the presence of oceanographic features like internal tidal bores [Leichter and Miller, 1999], which can alter the temperature regime experienced by corals. Assessing patterns of frequency, extent, and intensity of anomalies over the full time period of the dataset could also identify areas that are more or less vulnerable to thermal stress. The presence or absence of these correlations could help determine whether marine protected areas can be used to protect areas of greater resilience or resistance to thermal stress [West and Salm, 2003; Obura, 2005; Wooldridge et al., 2005].

To facilitate regional scale investigations of coral disease risk factors and dynamics, refinement of remote sensing tools must be complemented with rigorous disease monitoring protocols. Much of the current monitoring is idiosyncratic, often in response to a disease outbreak with little baseline or long-term monitoring data [but see Kim and Harvell, 2004; Willis et al., 2004; Santavy et al., 2005]. Long-term longitudinal and cross-sectional epidemiological studies are an essential component of elucidating density-dependence in

disease dynamics, susceptible age classes, possible pathogens or modes of transmission, and potential effects on reproductive output and population dynamics. Monitoring programs intended for use in conjunction with satellite temperature data should sample from different 4 km grid cells as defined by the Pathfinder dataset so that there is adequate replication for analysis. In addition, surveys should be conducted within a relatively close time frame so that the data do not covary with other temporal patterns. Finally, manipulative laboratory experiments are also an essential complement to these correlative studies to identify mechanisms driving correlations between temperature and rates of infection and spread.

## 5. Conclusions

Until very recently it was impossible to correlate satellite-derived temperature anomalies with *in situ* disease surveys. Temporally-consistent satellite temperature data at 4 km resolution enabled us to observe a positive relationship between temperature anomalies and outbreaks of white syndrome at broad spatial scales. A continuing challenge in assessing the importance of temperature anomalies as factors in disease dynamics will be to measure the role of other variables such as proximity to pathogen sources, local water quality, and physical oceanographic patterns. In some cases, these variables could be more important risk factors than temperature. A priority for coral reef scientists is the implementation of disease surveys on a global scale to adequately test the hypotheses that temperature anomalies and other factors drive coral disease. It is likely that there will be different thermal stress thresholds in different biogeographic regions for each disease. Work is underway to describe disease syndromes worldwide and develop a repeatable method for surveying coral disease [Willis et al., 2004] that can be implemented globally. In combination with satellite temperature data and other data sources, we can then begin to understand disease dynamics at regional scales.

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