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Relative role of disease and predators as drivers of decline in coral cover on the Great Barrier Reef.

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Abstract. The Australian Institute of Marine Science monitored causes of coral mortality annually on 46 reefs throughout the GBR from 1999 to 2005 and a further 46 reefs biannually from 2006. Sampling consisted of categorising corals lesions according to signs associated with coral disease, crown-of-thorns starfish and *Drupella* spp. feeding activity and lesions that could not be categorized. Of those categories recorded only increases in crown-of-thorns starfish lesions were associated with subsequent declines in coral cover. Between 1999 and 2005 there was no clear evidence to suggest that disease outbreaks had a significant impact on live coral cover on survey reefs. This result is in contrast to those recorded from the Caribbean where coral disease has lead to wholesale declines in coral cover on affected reefs. The relative proportions of diseases recorded show that lesions due to white syndrome and unknown causes make up the majority of those observed. The direct causes of a majority of coral lesions remain unexplained highlighting the difficulty of classifying coral lesions based on visual signs.

Key words: coral, disease, crown-of-thorns, lesion

Introduction

Little is known about the role disease plays in shaping the coral reef community on the Great Barrier Reef (GBR). This is despite the first disease of reef building corals being described some forty years ago (Squires 1965). Populations off hard corals in parts of the Caribbean were decimated by disease during the late 1970's and 1980's (Gladfelter 1982, McClanahan and Muthiga 1998, Aronson and Precht 1997, 2001). This has been followed by an apparent increase in disease numbers recorded on the GBR during the 1990's (McClanahan et al. 2004; Willis et al. 2004; Miller et al. 2006).

Reductions in hard coral cover on reefs have a serious impact on ecosystem function and can result in "phase shifts" where ecological services provided by the ecosystem are radically changed as the reef community is altered from one dominated by hard corals to one dominated by fleshy macro algae (Hughes 1994). Despite the events in the Caribbean and the growing recognition of the role disease plays in the ecology of Indo-Pacific hard coral species (Willis et al. 2004; Winkler et al. 2004; Raymundo et al. 2005; Dalton and Smith 2006; Page and Willis 2008) there have been few reports of disease outbreaks affecting coral cover on the GBR (e.g. Jones et al. 2004). An increase in reports of disease in other parts of the world has coincided with an increased publication rate that confounds interpretation of any patterns over time (Ward and Lafferty 2004). To help interpret the role disease plays in shaping coral communities it is important to understand background levels of disease induced mortality (i.e. the rate of unavoidable mortality that is independent of the organisms' state or behavior McNamara et al. 2004), what constitutes an increase in disease incidence above these levels and subsequently what are the impacts (Willis et al. 2004; Raymundo et al. 2005). This is increasingly important for the conservation of the GBR, already threatened by crown-of-thorns starfish outbreaks (Sweatman 2008) and other threats resulting from global warming such as coral bleaching, more frequent and intense cyclones (Hughes 2003) and increased disease (Harvell et al. 1999. The Australian Institute of Marine Science (AIMS) Long Term Monitoring Program (LTMP) has been collecting information on disease and coral cover on fixed survey sites since 1999. We examine whether observed increases in disease activity are linked to declines in coral cover on surveyed reefs between 1999 and 2005. We also compare changes in coral cover to abundance of disease and other sources of mortality on the GBR in order to determine the relative impact of identifiable diseases.

Material and Methods

Surveys of benthic organisms and SCUBA searches categorising the putative causes of coral lesions (white bare skeleton indicating recent mortality) were conducted on 46 reefs annually from 1999 to 2005 and a further 46 reefs biannually from 2006 by the

AIMS LTMP. Reefs targeted for survey were located in six latitudinal sectors over a distance of approximately 1500 km along the continental shelf. Three shelf positions were sampled (inner, mid, and outer shelf) in the four northernmost sectors, two shelf positions (mid and outer) were sampled in the Swains sector, and one (outer) was sampled in the Capricorn Bunker sector (Fig. 1).

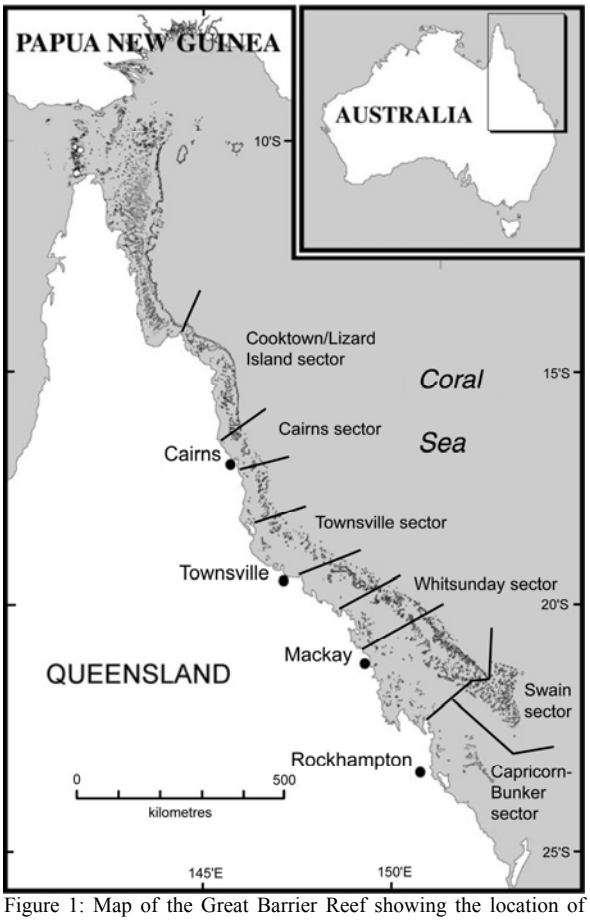

Figure 1: Map of the Great Barrier Reef showing the location of survey sectors targeted by the AIMS Long Term Monitoring Program.

At each reef, three sites positioned on the upper slope usually on the north-east flank were surveyed. Each site consisted of 5 x 50m transects along the 6-9m depth contour. Transects were marked with star pickets at the start and end, with steel rods at 10m intervals. Fibreglass measuring tapes attached to the pickets and rods allowed the same belt transect to be sampled in consecutive years.

A video recorder or still camera was used to record a 30cm wide strip along the edge of the measuring tape on each transect. Percent cover of hard corals was then estimated from the footage using a point-sampling technique. Images were captured from forty regularly-spaced frames of the video/still footage for each transect. Five systematically-distributed points were overlaid on each image and the identification of the benthic organism under each point was recorded. This provides a sample of 3000 points per reef. Hard and soft corals are identified to the highest taxonomic resolution possible, usually genus for hard coral and family for soft coral (see Abdo et al. 2004).

Causes of coral mortality were recorded using SCUBA searches conducted on a two-meter wide belt along each transect (search area $100m^2$). The observer recorded the number of individual lesions and ascribed their likely cause based on signs. These broadly fall into four major categories; disease including white syndrome (WS), black band disease (BBD), brown band disease (BrB), skeletal eroding band disease (SEB) and atramentous necrosis (AN); crown-of-thorns-starfish (COTS) ; *Drupella* spp. ; and unknown causes (see Miller 2004).

In 2006 a suite of 56 reefs (10 in common) were sampled using these methods as part of a separate research program (see Sweatman et al. 2008). The 46 LTMP reefs were again surveyed in 2007 but the time between surveys (2 years) meant that information collected in 2006 and 2007 could not be included in the statistical analysis as the model requires data collected in consecutive years. However, to determine the current relative frequency of disease types on the GBR a breakdown of the average number off disease lesions attributable to each of the categories recorded on all reefs over the period 2005 and 2007 was calculated.

To explore the relationship between changes in coral cover and coral lesion abundance a General-Linear-Model was used with log transformed coral cover as the response variable, log coral cover in the previous year as the main predictor and incidence of diseases as covariates. In log-space this kind of regression model is equivalent to the Gompertz growth model, a deterministic model frequently used to describe density-dependent population growth in discrete-time (Reddingius 1971; Dennis and Taper 1994). The intercept of the regression model estimates the log of the intrinsic growth rate, the coefficient of the population in the previous time period gives the strength of density dependence, and the coefficients of the covariates are modifiers of the growth rate and estimate the log of the proportional impact they have on population size.

Covariates (impacts) tested included the average number of lesions recorded between 1999 and 2005 for each of disease (pooled); *Drupella* spp.; COTS; and unknown causes. Covariates were square root transformed prior to analysis. All data were entered into an initial regression model with all covariates. Standardized residuals values were plotted against number of lesions due to COTS, unknown causes, disease and *Drupella* spp.. Outliers were identified

and removed for reefs where severe COTS outbreaks had decimated coral cover in the previous year, and where observed declines were due to mass coral bleaching in 1998 and cyclone damage on Low Isles reef also in 1998. A backward stepwise variable selection procedure was then used to test the importance of covariates. Covariates with the smallest partial correlation with the dependent variable were sequentially removed until all remaining covariates failed the removal criteria (probability of F-to-remove >= 0.10).

Results

Covariates for unknown causes, disease and *Drupella* spp. were all eliminated by the backward stepwise regression modeling procedure (Table 1) indicating that there was no significant association ($p > 0.10$) between coral cover in the subsequent year and lesions caused by these agents. There was, however a significant negative relationship between coral cover in subsequent years and increased COTS lesion abundance. COTS lesions were retained as a covariate in the final model (Table 2).

Covariate removed	Df1	Df2	Change in F	p (F)
Disease lesions	1	260	0.010	0.921
Drupella spp. lesions	1	261	1.256	0.263
Unclassified lesions	1	262	0.953	0.330

Table 1: Covariates eliminated during backward stepwise regression procedure and their corresponding changes in F-statistic

Covariate	Estimate	Std. error	t	P
Constant	0.224	0.073	3.09	0.002
Initial cover	0.939	0.021	44.1	<0.001
COTS lesions	-0.206	0.018	-11.6	<0.001

Table 2: Final regression model resulting from backwards stepwise regression procedure. The constant estimates the log of the intrinsic growth rate and the initial cover parameter estimates the strength of density dependence. Together they define the equilibrium coral cover [exp (0.224 / (1-0.939) = 39.3%]. The COTS lesions parameter estimates the proportional impact on next year's coral cover of COTS outbreaks that result in √x observed COTS lesions [exp (-0.206) = 0.81].

The majority of lesions recorded on fixed transects between 2005 and 2007 could not be assigned a cause (Fig. 2). White syndrome and *Drupella* spp. lesions were the most commonly assigned while lesions due to black band disease and atramentous necrosis were the least assigned. Only a small proportion (6.3 ± 1%) of the lesions observed could be attributed to COTS, which were on average less common than lesions due to *Drupella* spp., WS or SEB. There was some regional variation in the prevalence of lesion types. While unknown lesions dominate the counts in the majority of sectors WS was particularly common in the Capricorn Bunker sector. SEB was most common on northern reefs in the Cooktown Lizard Island sector while BrB was highest in the Pompey sector. BBD and AN were all observed at very low levels in the majority of sectors surveyed.

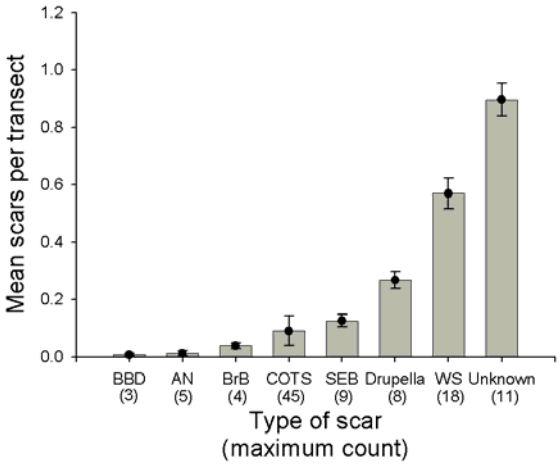

Figure 2: Abundance (mean ± SE) of different coral lesion types recorded per transect during AIMS scuba search surveys on fixed sites recorded from 2005-2007.

Discussion

The major finding of this study was the absence of a relationship between coral cover and the abundance of disease lesions on reefs of the GBR. In fact of all the factors tested only increases in lesion abundance due to COTS predation was positively associated with subsequent declines in coral cover. The latter result is not surprising as COTS have long been known to be a major source of coral mortality since outbreaks were first indentified in the 1960's (Birkeland and Lucas 1990). On the GBR three series of COTS outbreaks have caused declines in coral cover that have dwarfed losses from other disturbances such as storms or coral bleaching recorded over the same period (Sweatman et al. 2000). That there is no clear relationship between abundance of disease lesions and subsequent changes in coral cover on the GBR is in direct contrast to many other studies, particularly those in the Caribbean. In the Caribbean the most abundant reef building corals (*Acropora* spp.), were decimated by epizootics during the 1970's and early 1980's (Gladfelter 1982, McClanahan and Muthiga 1998, Aronson and Precht 1997). Since then coral cover on many reefs in the region has continued to decline with more epizootics recorded in recent times (Porter et al 2001, Patterson et al. 2002). While epizootics have been recorded from the GBR (Jones et al. 2004) they are rare. In fact reports of disease from the Indo-

Pacific, in general make up a much smaller proportion of reports when compared to those from the Caribbean (Willis et. al. 2004). This is despite the fact that the Indo-Pacific region is home to more than 80% of the reefs worldwide (Bryant et al. 1998). A possible explanation for the GBR is that coral diseases in this region are less virulent than in the Caribbean. Over the period 1977 to 2002 Gardner et al. (2003) reported a region-wide decline in hard coral cover in the Caribbean basin of 80% with coral disease playing a key role in this decline. Conversely declines in coral cover on the GBR over the last two decades have been generally driven by factors other than disease and where declines in some regions have been recorded they have been balanced by increases in others (Sweatman et al. 2008). In the Caribbean coral disease can have devastating affects on coral populations even when coral cover is low. For instance between 1996 and 1999 losses of *Acropora palmata* in the Florida Keys national Marine Sanctuary averaged 87% or greater from an already low (12% on average) cover (Sutherland 2004). In contrast on the GBR coral cover needs to be over 50% before outbreaks of white syndrome can occur (Bruno et. al 2007).

Another explanation for the observed result is that extrinsic factors may be differentially driving increases in the prevalence of coral disease in the Caribbean compared to those recorded from the GBR. However while many studies have pointed to rising disease prevalence (Ben-Haim and Rosenberg 2002, Sutherland et al. 2004, Weil et al. 2006), other studies have not always supported these conclusions (Voss and Richardson 2006) and a lack of baseline studies makes any interpretation of recent trends difficult (Ward and Lafferty 2004).

A third possible explanation is that lesions have been recorded at a level where they play a limited role driving observed changes in coral cover and that disease induced mortality on the GBR is simply outweighed by growth and renewal i.e. "background" levels of coral mortality (McNamara et al. 2004).

Previous studies have highlighted the increasing number of hard coral disease recognized on the GBR (Willis et al. 2004). Surveys by the AIMS LTMP in recent years have endeavored to include emerging diseases once signs for their identification have been confirmed. As a result the LTMP provides a clear picture of the ubiquity of coral disease on the GBR. Coral diseases appear widespread on the GBR with lesions caused by WS and SEB generally more frequent than those encountered for either *Drupella* spp. or COTS. This highlights that coral mortality due to disease, in particular SEB and WS, is a common occurrence on the GBR. All five disease types recorded by the LTMP have been generally found throughout the GBR system (AIMS data) though BBD, BrB and AN remain relatively rare. White syndrome is the most commonly encountered disease on the GBR. This is in contrast to other research that shows SEB to be generally more common than other diseases (Page and Willis 2008). The reason(s) for the apparently different results are unclear. Both WS and SEB have been well described with a known cause (Sussman et al. 2008, Antonius 1999). A possible explanation is that the divergent results are at least partially due to differences in sampling technique (i.e. observers, different locations on the reef surveyed, different survey methods including search area and time). Furthermore the most abundant lesion category recorded during this study was for those that remain unidentified. This highlights the difficulty of identifying signs of disease in the field. The problems of characterizing a disease (Richardson 1998) and then using the external appearance of corals for diagnosis is well recognized (Ainsworth et al. 2007a). The AIMS LTMP overcomes this problem by having a clear methodology (Miller 2004).

In terms of future scenarios for coral epizootics on the GBR indentifying disease and understanding disease dynamics, including trends in disease occurrence, is fundamental to conserving ecosystems faced with rising anthropogenic stresses (Ward and Lafferty 2004). This is particularly true given the relatively pristine nature of the GBR and that it is already under threat from COTS (Sweatman 2008), coral bleaching (Berkelmans et. al. 2004) and increased frequency and intensity of cyclones (Hughes 2003). The need to identify disease in the field and understand its role in the reef community can only become more important as increases in water temperatures (Jones et al. 2004, Bruno et al. 2007, Bourne 2008), nutrients (Bruno et al. 2003, Voss and Richardson 2006) bleaching (Muller et al. 2008) and in some cases, interactions with other organisms (Nugues et al. 2004, Aeby 2007, Nugues and Bak 2009) may be expected lead to increased prevalence of disease on the GBR in future years.

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