Drivers of coral mortality in non-acute disturbance periods

Hillary A Smith[1,2*], Carla C.M. Chen[1,*], F. Joseph Pollock[3,4], Morgan Re[5], Bette L. Willis[1], David G Bourne[1,6,7,‡]

[1] College of Science and Engineering, James Cook University, Townsville QLD 4811 Australia

[2] Centre for Marine Science and Innovation, School of Biology, Earth and Environmental Sciences, University of New South Wales, Sydney NSW 2052, Australia

[3] Department of Biology, Pennsylvania State University, 208 Mueller Lab, University Park, PA 16802, USA

[4] The Nature Conservancy, Hawai'i and Palmyra Programs, 923 Nu'uanu Avenue, Honolulu, HI 96817, USA

[5] SeaWorld San Diego, San Diego, CA 92109, USA

[6] Australian Institute of Marine Science, PMB3, Townsville Australia

[7] AIMS@JCU, James Cook University, Townsville QLD 4811 Australia

[*] Co-first authorship

[‡] Corresponding author

Corresponding author email: David.bourne@jcu.edu.au

Running page head: Coral mortality during non-acute disturbance

ABSTRACT

Studies focused on understanding drivers of coral mortality often examine reef- or ecosystem-scale stressors and/or pulse events such as mass bleaching or disease outbreaks. While such work provides valuable information about large-scale changes to reef ecosystems, how stres- sors interact at the individual colony level across non-disturbance years is less understood. In this study, we tracked the fate of 400 plating Acropora coral colonies from 2 mid- and 2 outer-shelf reefs for 18 mo and examined (1) temporal changes in the prevalence of stressors, (2) how stres- sors affected the survival of individual colonies, and (3) survival rates of colonies after contracting disease. We found that 35.5% of all colonies died within the 18 mo observation period, a period free from acute disturbances (e.g. cyclones, mass bleaching, crown-of-thorns starfish [CoTS] out- breaks). Despite its low prevalence, predation (by Drupella spp. or CoTS) led to the greatest risk of complete mortality compared to corals that experienced no stressors (over 10-fold increased risk). Similarly, experiencing disease and physical injury (fragmentation, dislodgement) also increased the risk of complete mortality (~4-fold and ~2-fold, respectively). In contrast, while com- promised health (i.e. bleaching, algal overgrowth) was common, this did not significantly increase the risk of colony mortality. Survival analysis of colonies with white syndrome showed that colonies exposed to stressors prior to contracting disease were 3 times more likely to die compared to colonies with disease alone. Our results highlight the complex interactions that occur among multiple stressors on coral reefs, even in non-disturbance years, and quantify the increased risk of mortality for colonies experiencing accumulated stressors.

KEYWORDS

Coral mortality, Coral demographics, Survival analysis, Coral disease, White syndrome, Crown-of-thorns starfish, Coral bleaching, Coral predation

1. INTRODUCTION

Coral reefs worldwide are threatened by a wide range of global and local stressors that act synergistically, leading to unprecedented declines of these important ecosystems (Gardner et al. 2003, Pandolfi et al. 2003, Bruno & Selig 2007, De'ath et al. 2012, Hughes et al. 2017b). The major stressors driving the loss of coral reefs include rising sea temperatures as a result of climate change (Hughes et al. 2017a, 2018), ocean acidification (Hoegh-Guldberg et al. 2007, Doney et al. 2009, Kleypas & Yates 2009), water quality changes associated with coastal development (i.e. pollution, nutrient enrichment, and sedimentation from runoff and dredging; Fabricius et al. 2005, Connell 2007), and overfishing (Roberts 1995, Jack- son et al. 2001, Zaneveld et al. 2016). These anthropogenic stressors interact with other disturbances, such as tropical storms (De'ath et al. 2012), disease outbreaks (Harvell et al. 2007, Miller et al. 2009), and predation by corallivorous predators (i.e. *Acanthaster planci* and *Drupella* spp.; Rotjan & Lewis 2008, Baird et al. 2013), leading to increased risk of coral mortality and subsequent declines in live coral cover.

Management of the Great Barrier Reef (GBR), which spans over 2300 km along Australia's north- eastern coastline, is widely regarded as extensive and effective (McCook et al. 2010, Day & Dobbs 2013); however, this complex ecosystem is not immune to global and local stressors. A 50% reduction in coral cover over 27 yr (De'ath et al. 2012) has been attributed to cyclone damage, crown-of-thorns starfish (CoTS), and bleaching (i.e. the loss of endosymbiotic algae from coral tissue). In addition, acute stress associated with mass bleaching events resulted in a loss of 29% of shallow water corals in 2016 (Great Barrier Reef Marine Park Authority 2017), and a further 24 to 50% loss of coral cover fol- lowing the 2017 bleaching event (AIMS 2018). Although many local stressors are absent in remote regions of the GBR, even these most isolated reefs are affected by global climatic changes and acute disturbances (Bruno & Valdivia 2016, Harrison et al. 2019).

Due to the spatial extent of the GBR, which covers 14 degrees of latitude, most research to date has focused on factors contributing to broad scale mortality (i.e. transect- or reef-scale) (De'ath et al. 2012, Hughes et al. 2018), or the impacts of a single stressor (i.e. bleaching or disease). Such research has developed an understanding of how reefs respond to stress at large spatial scales, but knowledge of how multiple, accumulated threats impact individual colony survival in situ is less developed. Mortality, or selection, fundamentally acts at the scale of the individual colony. A colony will experience multiple, potentially interactive stressors in its lifetime, and thus it is critical to understand which stressors pose the greatest threats to survival at the colony level. Some studies have attempted to address this, with fate tracking conducted on individual corals, but the focus has generally been on the survival of corals affected by specific diseases, such as stony coral tissue loss dis- ease (Combs et al. 2021) and atramentous necrosis (Anthony et al. 2008), bleaching (Morais et al. 2021), or the combination of disease and bleaching (Brodnicke et al. 2019). Alternatively, individual colonies are often monitored for survival and to provide a metric of success for out-planted coral fragments in restoration studies (Goergen et al. 2020, McLeod et al. 2022). The method of fate tracking, however, can equally be applied to reef communities not undergoing acute disturbance events to better understand the pressures contributing to background mortality on coral reefs, as well as building understanding of how multiple stressors impact survival (Neely et al. 2021).

Several coral demographic studies have been conducted at Jiigurru (Lizard Island in the northern sector of the GBR), making it an ideal location to further examine the complex factors contributing to individual coral colony mortality. For example, fate tracking of individual Acropora colonies over 5 yr revealed boom-bust dynamics in response to bleaching (Morais et al. 2021). Other studies have quantified background- and disturbance-driven mortality rates at Jiigurru (Lizard Island), finding high rates of injury (~70%; Pisapia et al. 2016), low rates of partial mortality (~5%; Pisapia & Pratchett 2014), and variable rates of background (i.e. non-acute) complete mortality (~18 % per annum; Wakeford et al. 2008, ~2 % per annum; Pisapia et al. 2016). These previous studies provide a valuable platform against which to assess the factors contributing to individual colony survival.

To effectively manage coral reef health and to guide conservation efforts, it is critical to understand the hierarchy of risk factors for individual coral colony mortality. Therefore, the objectives of this study were 3-fold: (1) to provide prevalence rates for coral disease, compromised health, predation, and physical injury at a background level (i.e. during a non-acute disturbance phase); (2) to investigate the impact of accumulated, multiple stressors on mortality of individual coral colonies; and (3) to examine colony survival times after exposure to the most prevalent and lethal disease group identified in the study, white syndromes (WSs).

2. MATERIALS AND METHODS

2.1. Data collection

Coral health surveys were conducted at 2 mid-shelf (Vicki's Reef, 14.685°S, 145.444°E; Horseshoe Reef, 14.688° S, 145.444°E) and 2 outer-shelf reefs (No Name Reef, 14.648°S, 145.645°E; Yonge Reef, 14.583°S, 145.622°E) on the GBR, at 6 time points from July 2011 to January 2013 (July, October 2011; February, June, October 2012; January 2013). Three permanent 10 × 10 m quadrats were established at haphazardly chosen locations at approximately 5 m depth within the study site, and all plating Acropora corals (e.g. A. hyacinthus, A. cytherea, A. caroliniana, A. clathrata, A. subulata) within the quadrats were tagged and monitored. A total of 400 coral colonies from the 4 reefs were monitored over the course of 1.5 yr.

At each sampling point, divers with extensive training in recognising coral health indicators observed and photographed (with scale bar) individual tagged coral colonies and recorded the state of each colony (alive/dead) and the presence/absence of 19 health attributes (Table 1; defined and identified as per Beeden et al. 2008; Fig. 1), grouped into 4 main categories: disease, compromised health, predation, and physical injury. Differentiations between categories were made using close observations of colonies in situ with particular attention to the distinguishing characteristics described by Beeden et al. (2008). For example, CoTS scars often have scalloped borders, while *Drupella* spp. (hereafter referred to as *'Drupella'*) scars are more irregular, and WSs are characterised by diffuse patterns of tissue loss. Colony size was determined using ImageJ by tracing the 2-dimensional coral area (mm^2) in each colony photograph. Survey dates were categorised by sea- son, whereby February 2012 and January 2013 are defined as (austral) 'summer,' July 2011 and June 2012 are defined as 'winter,' and October 2011 and October 2012 are defined as 'spring.'

Table 1. List of 19 attributes recorded per coral colony at each observation.

Attribute	**Grouping**
White syndrome	Disease
Skeletal Eroding Band	
Growth Anomaly	
Brown Band	
Black Band (not observed in this study)	
Other diseases	
Bleaching	Compromised health
Overgrowth by red algae	
Overgrowth by green algae	
Overgrowth by sponge	
Pigmentation	
Sediment necrosis	
Other compromised health	
Predation by Crown-of-thorns starfish (CoTS)	Predation
Predation by *Drupella*	
Fragmentation	Physical injury
Flipped	
Broken	
Mucus	

The period of data collection coincided with the start of the 2010 CoTS outbreak (Babcock et al. 2020). Manta tow data collected by the Australian Institute of Marine Science's (AIMS) long-term monitoring program recorded an increase in CoTS density around Jiigurru (Lizard Island) between 2011 and 2013, from 0.16 to 0.74 CoTS per manta tow (AIMS 2011, 2012). While there were signs of an incipient regional-scale outbreak, only a small proportion of colonies in this study were observed to have scars from CoTS predation. Given that the impact of predation is likely to be localised, i.e. only affecting individual colonies upon which a CoTS predates, we assumed that colonies without CoTS scars were not affected by the CoTS outbreak. Where a colony was found dead in survey *t* but was alive and free from CoTS scars in survey *t – 1*, we assume that the colony did not die from CoTS predation. While we acknowledge that the temporal scale of sampling may have missed incidences of stressors (CoTS or otherwise), these assumptions were required, as we cannot unduly assign CoTS predation in the absence of observation. Aside from the CoTs outbreak, the 4 reefs were not subjected to a large-scale bleaching event nor to severe storm damage (i.e. cyclone impacts) over the course of the study period.

2.2. Data preparation and analyses

Demographic parameters of the 4 reefs were examined to provide context for the below objectives. The size frequency distributions for each reef at the start of the study were compared using Kolmogorov- Smirnov tests. Changes in the size of individual colonies (i.e. growth and partial mortality) were examined using a generalised linear mixed effects model (GLMM) with a Gamma distribution, modelling the percent change in size by the additive effect of reef identity and colony starting size.

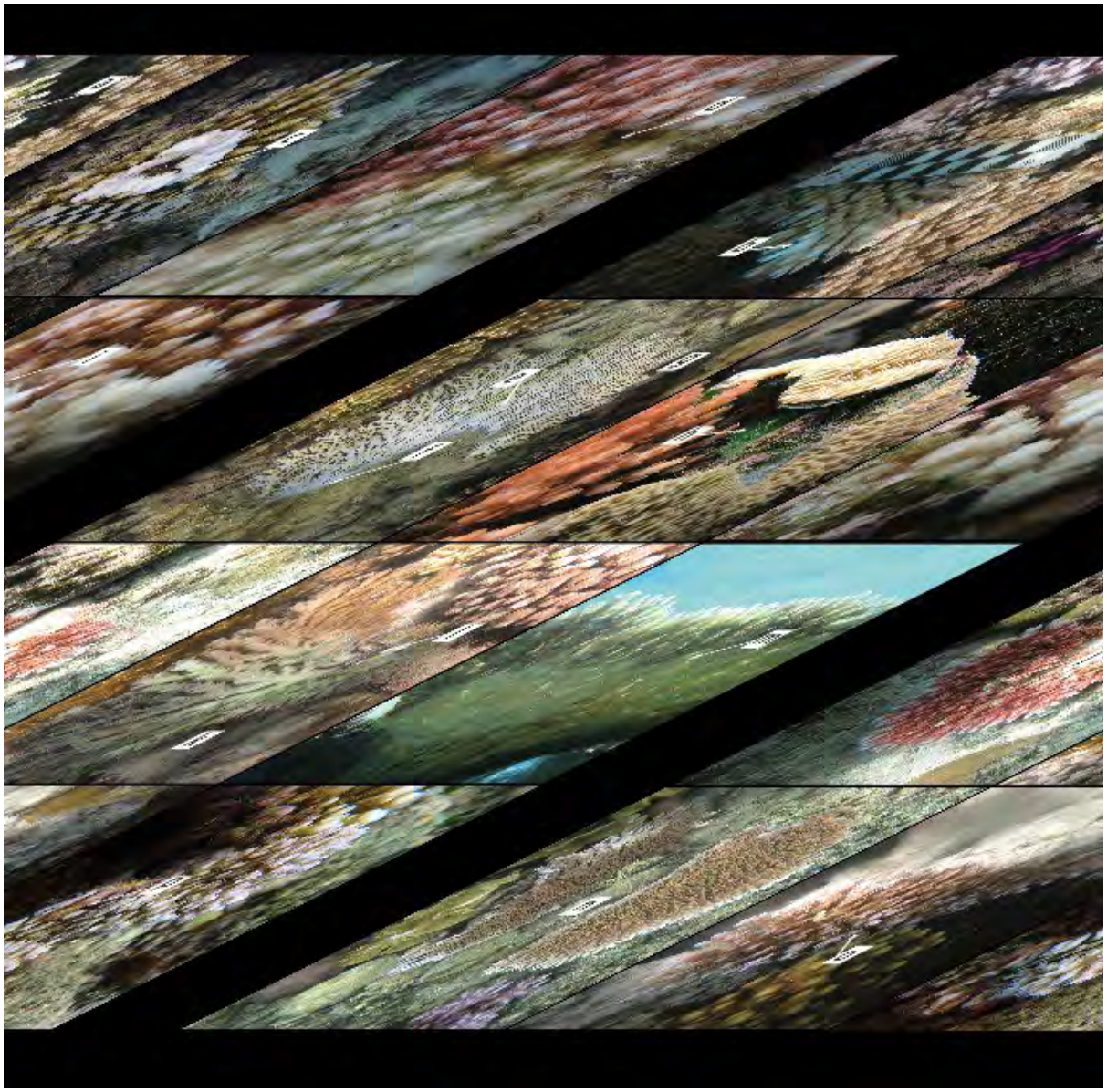

Fig. 1. Example images of 16 of the 19 health indicators (excluding black band, other diseases, other compromised health, see Table 1): (a) overgrowth by green algae; (b) white syndrome; (c) growth anomaly; (d) brown band; (e) bleaching; (f) pigmentation; (g) flipped; (h) overgrowth by sponge; (i) skeletal eroding band; (j) sediment necrosis; (k) broken; (l) *Drupella* predation; (m) CoTS predation; (n) fragmentation; (o) mucus; (p) overgrowth by red algae

2.2.1. Objective 1: Comparative prevalence of disease, compromised health, predation, and physical injury during the study period

First, we determined the prevalence of the 19 health attributes in coral populations at the 4 reefs across the 18 mo study period, to understand the relative occurrence of stressors in non-acute disturbance years. Prevalence is defined as the proportion of a population that has a specific disease or characteristic at a given time. Prevalence is used here to determine the relative occurrence of disease, com- promised health, predation, and physical injury in corals over a period of time and is measured as:

$$prevalence_{q,i,t} = n_{q,i,t}/N_{q,t}$$

where, in this study, $n_{q,i,t}$ is the number of colonies with condition i,i ⟩ [disease, compromised health, predation, physical injury] in quadrat q at time t, and $N_{q,t}$ is the number of live tagged colonies in quadrat q at time t. We examined temporal and spatial variation of the prevalence of each category using generalised linear models (GLM) with binomial distribution and logit link, with post-hoc pairwise tests based on estimated marginal means and significance adjusted using the Tukey method. Statistical significance was concluded at a level of ⟨ = 0.05.

2.2.2. Objective 2: Impact of stressors on the survival of coral colonies

Secondly, we determined the probability of mortality for individual coral colonies experiencing each stressor. To this end, we assumed the state of the coral (alive/dead) was directly related to the stressor(s) the colony experienced at the previous observation. Let $y_{i,t}$ denote the state of colony *i* at time *t*, and $x_{i,t-1}$, $d_{i,t-1}$, $c_{i,t-1}$, and $ph_{i,t-1}$ denote whether colony *i* experienced predation, disease, compromised health, and physical damage at *t–1*, respectively. The probability of mortality of colony *i* at time *t* is modelled using a Bernoulli distribution, where $p_{i,t}$ is modelled as:

$$y_{i,t} = \text{Bernoulli}(p_{i,t})$$

$$\text{Logit}(p_{i,t}) = \beta_0 + z_i + \beta_1 x_{i,t-1} + \beta_2 d_{i,t-1} + \beta_3 c_{i,t-1} + \beta_4 ph_{i,t-1} + \beta_5 shelf_i$$

where z_i is the random effect to account for individual colony variation, and β is the coefficient (i.e., quantifies the effect of) each predictor. Because surveys were timed approximately 3 mo apart, the probability of mortality calculated here is the probability that the colony is dead in approximately 3 mo time.

2.2.3. Objective 3: Expected survival time after displaying signs of white syndrome

Thirdly, we evaluated the dynamics of colony survival after experiencing a given stressor. Survival analysis is commonly used in clinical research to study how long patients live after experiencing an event. It is used here to examine the duration of survival after a colony contracted WSs, and to test if the survival time varies with exposure to other stressors (i.e. disease plus other stressor/s) prior to or post infection, as well as the location (reef) of the colony. We investigated survival of colonies displaying signs of WSs because (1) WSs were found to be a significant contributor to colony mortality in Objective 2, and (2) we had sufficient sample size. Only colonies displaying signs of WSs during the observation periods were included in the survival analysis. Survival time is approximated as the number of days that a colony was observed with the disease to the time it was observed to be dead.

To test whether exposure to other stressors before or after being observed with disease impacted survival time of a colony, a binary indicator variable was used to summarise the experience of a colony with other stressors. If a colony had a record of exposure to other stressors (i.e. compromised health, predation, or physical injury) in the observations prior to the infection, the variable was assigned a value of 1, otherwise zero. We used a similar approach to derive a variable for post-infection exposure.

A Cox-proportional hazard model was used to analyse the survival rate and impacts of covariates. Let *h(t)* denote the expected hazard rate (i.e. the probability of death) for a colony dying at time *t* :

179 $$h(t) = h_0(t)\exp(\beta_1 x_1 + \beta_2 x_2 + \beta_3 x_3)$$

180 where $h_0(t)$ is the baseline hazard, β_1,β_2 and β_3 are the expected log-scaled change in hazard ratio due to reefs (x_1),
181 prior (x_2) and post (x_3) exposure to other stressors. Hazard is an instantaneous mortality at time t. Survival rate is
182 given as a percent of colonies surviving at time t compared to the total number of colonies observed with WSs. The
183 probability of death is averaged across all time points.

184 All analyses were completed in the statistical software R, version 4.3.0 (R Core Team 2021).

185 **3. RESULTS**
186 **3.1. Population demographics**

187 A total of 400 individual colonies were tagged and monitored over the course of the 1.5 yr study period.
188 Approximately 38% of tagged colonies (i.e. 152 colonies) died during the study period. The highest mortality was
189 recorded at Vicki's Reef, followed by Horseshoe and No Name Reef, with Yonge Reef demonstrating the lowest
190 mortality during the study period (Table 2). Of the colonies that died, 30.1% (i.e. 46 colonies) did not experience any
191 observable stressor at the previous observation, and of these colonies, 61% (i.e. 28 colonies) were located on the
192 mid-shelf reefs.

193 Table 2. The number of coral colonies tagged at each reef, and the number and percent of corals that died during the study period.

Shelf position	Reef	Sample size (n)	No. Died	% Died
Mid-shelf	Horseshoe	101	40	39.6
	Vicki's	78	45	57.7
Outer	No Name	110	38	34.5
	Yonge	111	30	27.0
	Total	400	152	38.0

194 The median size of tagged colonies at the first observation was 205 mm^2; 50% of tagged corals were between 84 and
195 671 mm^2 (Fig. S1 in the Supplementary Materials). At the start of the study (t = 0), the size of tagged colonies was
196 similar among reefs, with the exception of Yonge Reef, which had a greater abundance of smaller colonies than other
197 reefs (<50 mm^2; Fig. S1).

198 Of the 400 tagged colonies, we were able to measure the starting and final 2-dimensional surface area (mm^2) of 208
199 colonies (from July 2011 to January 2013). More than half of these colonies (63.5%) increased in size over the study
200 period (523 d). The percentage annual increase varied significantly by reef (ANOVA; $F_{3,128} = 3.99$, $p = 0.009$) and by
201 the starting size of the colony ($F_{2,126} = 6.89$, $p = 0.001$). While there was no significant difference in the annual
202 percentage size increase between colonies from Vicki's (85.5 ± 21.5% increase; mean ± SE), No Name (57.0 ± 25.4%),
203 and Yonge Reefs (67.6 ± 26.0%), Horseshoe Reef had a significantly higher percentage annual growth (205 ± 55.4%)
204 during the study period compared to the other reefs (GLM; $t = -2.22$, $p = 0.028$, Fig. S2). For example, the predicted
205 percentage size increase for colonies with an initial size of <500 mm^2 on Horseshoe Reef was 490 ± 140% per annum,
206 which is 4 times higher than the expected growth for colonies of the same size on Vicki's Reef (120 ± 40% per annum;
207 $t = -2.2$, $p = 0.028$; Fig. S2). Furthermore, colonies with a starting size smaller than 500 mm^2 had significantly higher
208 annual percentage increase than colonies larger than 500 mm^2 ($t = -2.546$, $p = 0.01$). Corals that experienced a
209 decrease in size over the study period (n = 76) were reduced on average by 34% colony area per annum, and the
210 percentage annual size decrease did not differ by reef (ANOVA; $F_{3,72} = 0.61$, $p = 0.61$) or by the initial size of the
211 colony ($F_{2,70} = 1.75$, $p = 0.18$; Fig. S2).

212 **3.2. Objective 1: Comparative prevalence of disease, compromised health, predation, and physical injury**
213 **during the study period**

214 Physical injury and compromised health were the 2 most common stressors to all reefs during the 1.5 yr of
215 observation. The mean values for prevalence of com- promised health and physical injury (pooled across reefs and
216 timepoints) were 12.1 and 11.4%, respectively, followed by disease (4.2%) and predation (1.5%). There was
217 considerable variation in the prevalence of stressors between reefs (Table S1).

218 Prevalence of the compromised health state varied significantly between shelf position and pairwise among
219 timepoints. On average, corals on outer shelf reefs experienced higher prevalence of compromised health compared

to inner shelf reefs (GLMM; log-odds ratio of 0.42, z = 3.02, p = 0.003). The highest prevalence of compromised health was in spring and summer of 2011 (mean 19.4 and 18.5%, respectively, pooled across shelf positions; Fig. 2). There was no consistent pattern in the prevalence of com- promised health among seasons.

During the study period, the prevalence of physical injury varied significantly according to an interaction between shelf position and timepoints. On average, colonies located on the outer shelves sustained physical injury 1.89 (95% CI: 1.23, 2.93) times more frequently compared to the mid-shelf reefs (Fig. 2).

Four diseases were observed over the study period: WSs (11.25% prevalence across all reefs and time points), skeletal eroding band (0.45%), growth anomalies (2%), and brown band disease (3.75%). Disease prevalence varied significantly between shelf position and pairwise among survey timepoints, though lacked statistical evidence for seasonal patterns. Dis- eases were significantly less common on outer shelf reefs than mid-shelf reefs (GLMM; log-odds ratio of −0.64, z = −2.89, p = 0.004; Fig. 2). Although there were a limited number of observations in winter months, diseases were least prevalent in winter, increased in spring, and reached a peak in summer.

The reefs around Jiigurru (Lizard Island) were experiencing an active CoTS outbreak during the period of observation, though only a small number of the tagged colonies in our study showed signs of CoTS predation (13 of 400 colonies; 3.25%; Fig. 2). This is comparable to the number of tagged colonies which displayed signs of *Drupella* predation (19 of 400 colonies; 4.75%). None of the tagged colonies on the outer-shelf reefs had signs of CoTS predation and only 2 of the tagged colonies experienced *Drupella* predation. These small sample sizes precluded meaningful analyses of differences in predation among years, seasons, and reefs.

3.3. Objective 2: Impact of stressors on the mortality of coral colonies

The probability of mortality in an approximately 3 mo period (i.e. average duration between surveys) increased significantly if the colony was affected by predation, disease, or physical injury at the previous observation. Specifically, despite the small sample size, a colony was 10.49 (95% CI: 3.89, 30.23) times more likely to be found dead if it had signs of predation in the previous observation (GLMM; z = 4.5, p < 0.001). In the 17 cases where *Drupella* feeding scars were recorded on a colony, 10 (59%) of these colonies were found dead at the next survey. Similarly, in the 9 cases where CoTS predation scars were recorded, 5 (56%) of these colonies did not survive until the next observation. After a colony was re- corded in a diseased state, the probability of colony mortality in the next sampling increased 4.52-fold (95% CI: 2.43, 8.43; GLMM; z = 4.8, p < 0.001). Physical injury was also associated with colony mortality, with the probability of mortality within 3 mo increasing 2.05-fold (95% CI: 1.30, 3.20) following observations of colony injury (GLMM; z = 3.1, p = 0.002). Probability of colony mortality also increased 1.24- fold after a colony showed signs of compromised health, although the association was not statistically significant (GLMM; z = 0.892, p = 0.37).

The association between reef shelf and colony mortality remained statistically significant after accounting for the effects of different stressors, with colonies on the mid-shelf reefs experiencing 1.76-fold (95% CI: 1.18, 2.67) higher probability of mortality compared to colonies on the outer shelf reefs (GLMM; z = 2.7, p = 0.006). The sensitivity of the model (i.e. true positive; ability to predict death) was low at 0.17, while the specificity (i.e. true negative; ability to predict survival) was high, at 0.99. This is mostly due to the unbalanced design and small sample sizes, as most colonies were still alive at the end of study.

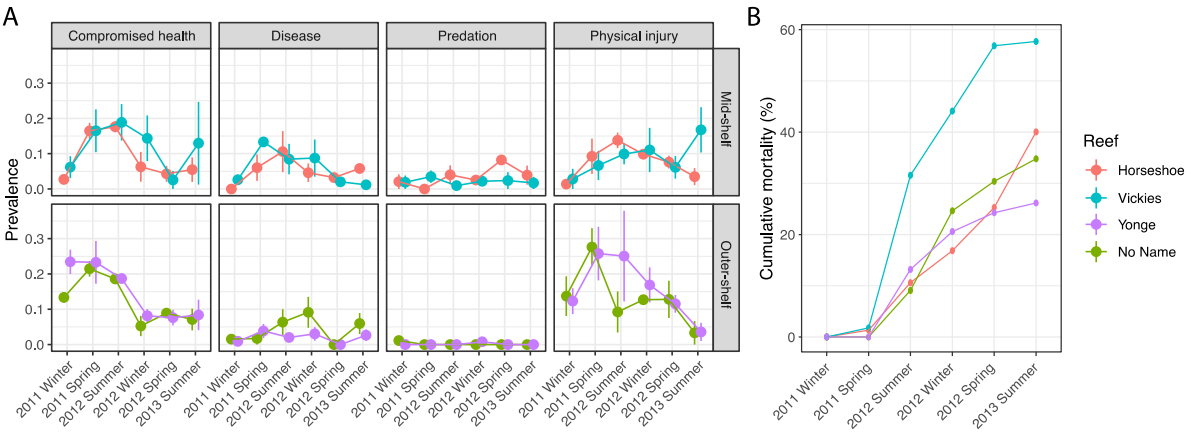

Fig. 2. (A) Mean ± SE comparative prevalence of compromised health, disease, predation, and physical injury across mid- and outer-shelf reefs. (B) Cumulative percent of colonies monitored experiencing complete mortality from each reef

3.4. Objective 3: Expected survival time of colonies with signs of WSs

Predation was associated with the greatest probability of mortality, though the small sample size of colonies experiencing predation did not allow for survival analysis. Instead, disease, and specifically WSs (the most prevalent disease observed in this study), represented the second greatest probability of mortality and was used to explore colony survival after expo- sure. Around 11% of tagged colonies (n = 400 tagged colonies) showed signs of WSs (i.e. 45 colonies) during the period of observation. Of the 45 colonies recorded with WSs, 9 were observed with WSs in the last survey, and were therefore excluded from further analyses.

Of the 36 colonies included in the survival analyses, 22 (61%) died after being observed with the disease. The median survival time for these colonies was 228 d (25% and 75% quantiles were 121.3 and 342 d, respectively).

Survival rate of individual colonies (% of colonies that survived out of all colonies with WSs) varied greatly among reefs and was dependent on colony exposure to other stressors prior to infection. Among all reefs, Vicki's Reef had the highest expected hazard (probability of mortality) for WS-infected colonies; probability of mortality was 3.49 times greater (95% CI: 1.25, 9.75) compared to Yonge or Horseshoe Reef (Fig. 3). After a colony was observed with WS signs, only 12.6% survived until the following survey at Vicki's Reef, while 55.2 to 89.4% of colonies survived at the other reefs in the same time frame (Fig. 3). At the last observation, no colonies survived at Vicki's Reef, while 11% survived at Horseshoe, 16% survived at Yonge, and the highest survival of 65.8% was observed at No Name Reef (Fig. 3).

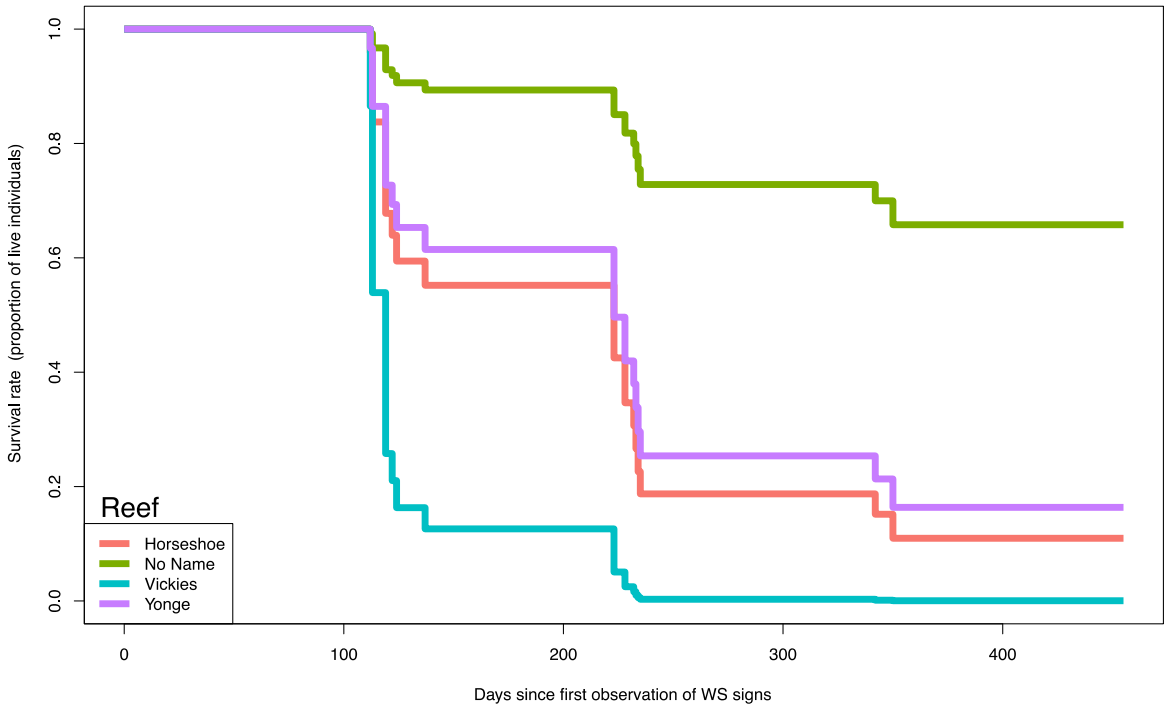

Fig. 3. Survival rate (proportion of live individuals) for coral colonies after being observed with signs of white syndrome at Horseshoe, Vicki's, Yonge, and No Name reefs

Exposure to other stressors prior to contracting WSs also significantly affected the probability of individual colony mortality. For colonies exposed to other stressors prior to showing disease signs, the probability of mortality was 3.07 times higher (95% CI: 1.14, 8.20) than colonies that were not exposed to other stressors prior to disease onset. For example, once a colony was observed with signs of WSs at Yonge Reef, the expected survival rate to the next survey was 61.5% for colonies exposed to other stressors prior to the infection, compared with a 95.3% survival rate

of colonies not exposed to prior stressors (Fig. 4). After 2 survey points following the first WS observation, the survival rate for colonies exposed to prior stressors was 25%, while colonies not exposed to prior stressors had a survival rate of 64% (Fig. 4). At the last observation, colonies that experienced a prior stressor had a survival rate of only 16%, compared to 55% for colonies without a prior stressor. Similar dynamics were observed at each reef, where colonies not experiencing prior stressors had higher survival than those that were exposed to stress before contracting disease (Fig. 4).

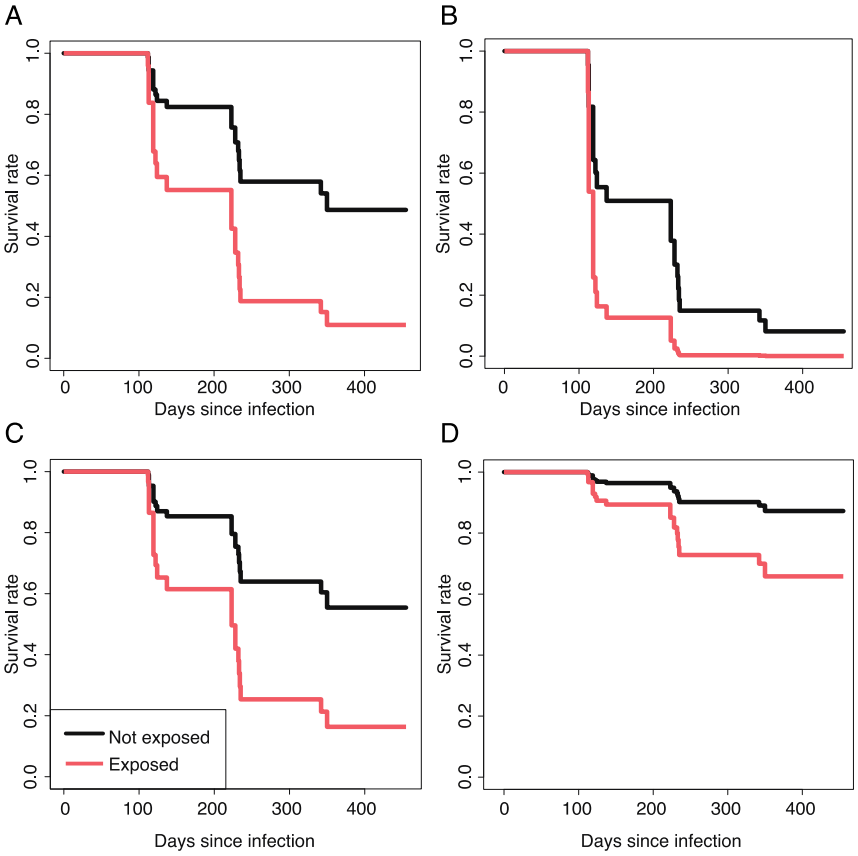

Fig. 4. Survival rate (proportion of live individuals) of diseased colonies exposed or not exposed to stressors prior to displaying signs of white syndromes at (A) Horseshoe Reef, (B) Vicki's Reef, (C) Yonge Reef, and (D) No Name Reef

4. DISCUSSION

This study tracked the fate of individual plating acroporid corals, with repeated surveys over 1.5 yr revealing high rates of mortality (25% per annum) during a period when they were not exposed to an acute disturbance, such as a mass bleaching event or a major storm. Our study attributes these high levels of mortality to non-acute stressors, particularly predation injuries and disease, although these stressors affected coral colony survival differentially across reef shelf position and time points. Overall, mortality for colonies that did not experience any detected stressor was approximately 7% per annum (i.e. background mortality rate). When colonies were subjected to a non-acute stressor, mortality per annum increased to 25%. The background mortality rate detected for corals experiencing no stressor at the sites studied was similar to rates found in previous studies. For example, one study documented a 2% per annum background mortality for Acropora hyacinthus over a similar timeframe (Pisapia et al. 2016). Similarly, up to approximately 20% annual mortality has been recorded for A. hyacinthus in inter-disturbance years between 1981 and 2003 in the Jiigurru (Lizard Island) region (Wakeford et al. 2008). Noting that the field aspect of the current study took place from 2011 to 2013, it is likely that rates of mortality have since increased in light of more severe chronic impacts affecting reef corals including recurrent mass bleaching events on the GBR (Hughes et al. 2017a, Pratchett et al. 2021).

The demographics of survival and mortality of individual coral colonies differed across the 4 reefs investigated. Specifically, almost 60% of colonies monitored at Vicki's Reef suffered complete mortality during the study period, in contrast to 27–40% mortality of colonies at the other reefs. Local environmental conditions are known to influence

315 survival, and hence it is possible that localised conditions at Vicki's Reef were challenging for resident corals. While
316 the model only assessed differences in complete mortality between reefs, the planar nature of the acroporids studied
317 here allowed for explicit size measurement, and hence negative change in colony size can serve as a proxy for partial
318 mortality. Despite higher whole colony mortality at Vicki's Reef, there was no difference in the percentage size
319 decrease among the 4 reefs. Furthermore, approximately 35% of colonies experienced a reduction in size over the
320 study period, which contrasts with the 71% of colonies experiencing partial mortality in other work examining
321 background mortality dynamics (Pisapia et al. 2016). Combining coral demographic processes with fine-scale
322 environmental data is challenging, but such efforts would be useful to further under- stand how environmental
323 factors affect the prevalence of biotic stressors and their interactions with colony growth and survival.

324 **4.1. Impact of compromised health, predation, and physical injury on coral mortality**

325 Compromised health signs, including signs of bleaching, pigmentation, overgrowth by algae and sponges, and
326 sediment necrosis, were most common in summer months, although these colonies were not significantly more at
327 risk of subsequent mortality than colonies without compromised health signs. High sea temperatures in summer are
328 positively correlated with coral bleaching (Brown 1997, Hughes et al. 2017a) and increased algal growth (Klumpp &
329 McKinnon 1989), though temperatures during the study period did not reach the sustained high levels necessary to
330 trigger extensive bleaching. Nevertheless, summer temperatures can result in paling or mild bleaching of the low
331 stress-tolerant acroporids that were the focus of this study (Darling et al. 2013, Smith et al. 2022). Corals are well
332 equipped to survive mild heat stress periods and, though a potential drain on energy budgets, the populations in this
333 study did not appear to be unduly impacted. Algal overgrowth can also occur more frequently in summer, as a result
334 of algae growing more rapidly (Klumpp & McKinnon 1989). At these sites, macroalgae abundance was low, so the
335 algal overgrowth category generally denoted overgrowth by turf and filamentous algae. Plating Acropora corals are
336 generally poor competitors in interactions with turf algae (Swierts & Vermeij 2016), bearing in mind that interactions
337 are likely coral- and algal- species specific (McCook et al. 2001). Although coral response to turf can be exacerbated
338 by sedimentation (Nugues & Roberts 2003), these reefs are located in mid- and outer-shelf positions and hence lie a
339 distance from sediment inputs. Pigmentation was uncommon (70 observations over the 18 mo) and generally
340 affected small portions of colonies (<10% of colony area). Increased melanisation is a recognised immune response
341 to a range of challenges (e.g. parasites, pests, diseases; Palmer et al. 2008), but can also be elicited by temperature
342 stress (van de Water et al. 2016), explaining its increased prevalence in summer surveys. Importantly, bleaching,
343 pigmentation, algal overgrowth, and sediment necrosis can often affect only a part of a coral colony. Our model
344 assessed only whole colony mortality, hence it is possible that corals experiencing compromised health did undergo
345 partial mortality. However, the low number of colonies experiencing a reduction in size, combined with the low
346 percent reduction in live tissue area of coral colonies throughout the study, suggest that partial mortality was
347 unlikely to have been a significant coral response to compromised health during the study period.

348 Signs of predation were not common during the study period despite signs of an incipient outbreak of CoTS in the
349 region (https://apps.aims.gov.au/reef- monitoring). The approximately 3 mo intervals between surveys, which were
350 conducted seasonally rather than at a finer temporal scale due to the remoteness of reefs, potentially precluded
351 detection of CoTS predation scars. Further, while every effort was made to differentiate CoTS predation from dis-
352 ease signs, particularly WSs, these 2 stressors manifest in similar physical outcomes (i.e. tissue loss), and records of
353 disease may have been inaccurately assigned. Nonetheless, if a colony was not recorded as showing signs of
354 predation in the previous survey, mortality was not attributed to predation. However, given that CoTS can move
355 across reefs rapidly, it is possible that the mortality of some colonies that died at time t was because of CoTS
356 predation in the time between t and $t - 1$. Similarly, there were few observations of active predation by *Drupella* and
357 hence it is likely that some of the background mortality found in this study may be attributed to CoTS and *Drupella*
358 predation. Nevertheless, despite the infrequent observations of predation, colonies that had predation injuries were
359 at the highest risk of mortality in the subsequent observation. Considering the 10-fold increased risk of mortality
360 found on mid-shelf reefs compared to outer-shelf reefs, and that CoTS outbreaks affect mid-shelf areas more
361 frequently (Moran et al. 1988, Vanhatalo et al. 2017), it is likely that CoTS had a larger role in causing mortality than
362 our study could detect.

363 The prevalence of physical injury (i.e. fragmentation, flipped, broken, or mucus production) was generally low across
364 all reefs and did not vary by season, though corals on outer shelf reefs did experience a 2- fold higher prevalence of
365 injury. This may be explained by wave action, which is generally stronger on outer shelf reefs (Bridge et al. 2019).
366 Tourist activity is an established factor that can increase the risk of coral mortality through physical injury (Hawkins
367 & Roberts 1992, Hawkins et al. 1999); however, the reefs included in this study are not frequented by tourists, and
368 hence damage from boat anchors and snorkeler fin damage are less likely to occur. While coral mucus can be
369 produced in response to a range of stressors, herein we recorded mucus as a separate category when it did not co-
370 occur with other stressors (e.g. disease, predation signs). As such, observations of mucus alone were rare, and may
371 indicate a longer-term systemic response to stressors undetected in our quarterly sampling regime.

372 **4.2. Role of disease in driving coral mortality**

Colonies that displayed disease signs (WSs, skeletal eroding band, growth anomalies, brown band) had a 4-fold increased risk of mortality in the next survey compared with colonies without disease signs. Disease signs were twice as prevalent on mid-shelf reefs, and, despite a lack of statistical significance, were generally more common in summer. Coral disease incidence, like bleaching, is correlated with high seawater temperatures (Selig et al. 2006, Ruiz- Moreno et al. 2012, Howells et al. 2020), likely explaining this higher disease prevalence in summer. Similar dynamics of disease incidence, with higher prevalence in summer, have previously been recorded at Heron Island (Haapkylä et al. 2010, Roff et al. 2011). Other factors that have been associated with increased WS prevalence include high coral population densities and warm winters (associated with increased prevalence of WSs in the subsequent summer periods) (Heron et al. 2010). Higher seawater temperatures were recorded in winter 2012 compared to winter 2011, but interestingly disease prevalence was lower in the spring 2012/summer 2013 surveys compared to the spring 2011/summer 2012 surveys, suggesting that warm winters are not always reliably linked to disease incidence. Longer-term monitoring of tagged individuals, paired with in situ temperature monitoring, would help to resolve the links between temperature, season, and disease prevalence.

Survival analysis of colonies that displayed WS signs revealed a strong effect of reef identity, with Vicki's Reef having the highest mortality of diseased corals. For example, none of the colonies with WSs survived on Vicki's Reef after 2 surveys, compared with 65% of colonies surviving on No Name Reef over the same period. Interestingly, only 2 colonies that showed signs of WSs at Vicki's Reef had experienced prior stressors, and hence the interaction with previous stress is unlikely to be the main determinant of high mortality at this reef. Disease is generally coupled with environmental conditions (Harvell et al. 2007), and based on the overall high mortality rate and high prevalence of all stressors at Vicki's Reef, it is possible that conditions on this reef were adverse for corals more generally. Detailed environmental metadata would be helpful in determining specifics, but it is possible that factors such as depth, sedimentation, or current patterns contributed to the high mortality rates on this reef. Disease is also coupled with coral cover, with high coral cover associated with increased spread of disease (Selig et al. 2006, Bruno et al. 2007). While we did not collect data explicitly on coral cover, the size distribution of coral colonies on Vicki's Reef was right-skewed, suggesting that large colonies are common on this reef. Indeed, colonies over 1500 mm^2 comprised 12% of the coral community at Vicki's Reef, while the same size class represented between 2 and 3% of the coral community on outer shelf reefs. Large colonies are more susceptible to WSs (Roff et al. 2011, Greene et al. 2020), and hence colony size could be an important component of disease dynamics on this reef. Furthermore, WSs encompass multiple distinct diseases with varying aetiologies (Bourne et al. 2015, 2022); some colonies may have slow-moving chronic lesions and others more rapidly progressing lesions that can quickly result in whole colony mortality. It is possible that different underlying aetiologies were present at each reef, and may have contributed to reef-scale variation in mortality.

Coral colonies experiencing WSs were at significantly higher probability of mortality if the colony demonstrated signs of other stressors before showing signs of WSs. Coral diseases are complex, and the causative factors and vectors for most are not known (Mera & Bourne 2018). However, it has been established that corals are more susceptible to displaying disease signs when stressed (Haapkylä et al. 2011, Vega Thurber et al. 2014, Brodnicke et al. 2019, Howells et al. 2020), and that disease severity is increased when corals are exposed to multiple stressors (Vega Thurber et al. 2014, 2020, Aeby et al. 2020). For example, the synergistic effects of bleaching and disease resulted in a 7-fold increase in mortality for corals on a mid-shelf reef (Brodnicke et al. 2019). Similarly, links between predation by *Drupella* and increased disease incidence have been established (Nicolet et al. 2013). Given these complex interactions, it is difficult to determine if disease alone is responsible for coral mortality in this study. However, the reduced survival rate for corals affected by stress before contracting disease compared to those with disease alone suggests that complex synergistic stressors result in cumulative mortality within coral populations. A coral colony may have the energetic resources to withstand 1 stressor (e.g. injury, elevated temperatures); however cumulative stressors are likely to overcome coral immune defences and result in whole colony mortality. The risk of mortality is likely to vary greatly among the variety of stressors that interact with diseases, and is worth further investigation. While a range of stressors were commonly observed in this study, we lacked the sample size to conduct survival analysis for each individual category.

It is critical to understand how individual coral colonies respond to multiple simultaneous and/or cumulative impacts to build an understanding of the interactions that drive coral demographic processes. This study provides important insight into the factors shaping coral population demographics in a period without acute stressors. While the models used detected significantly increased risk of mortality for a number of stressors, they had low sensitivity (i.e. ability to predict mortality) and hence other factors that were not measured or observed likely con- tributed to mortality. The 3 mo interval between surveys did not allow for more resolved tracking of coral colonies and it is probable that some stressors went undetected. Future research incorporating local-scale environmental conditions and greater temporal sampling would be useful to determine why some reefs (e.g. Vicki's Reef) experienced higher mortality than others. Despite the few colonies identified as experiencing multiple simultaneous stressors, the fate tracking of colonies that displayed WSs and were subjected to an additional stressor showed an increased probability of mortality. The approaches used in this study demonstrate the complexity of the impacts that interactions among biotic and environmental stressors have on the survival of individual coral colonies, as well as on processes governing selection.

Acknowledgements. The field aspect of this research was conducted on the Sea Country of the Dingaal people, and the desktop analyses were conducted on the lands of the Wulgurukaba and Bindal peoples. We acknowledge their ongoing connection to country, and pay our respects to their elders past, present, and emerging. Field work was conducted under permit G09/31013.1 issued to B.L.W. by the Great Barrier Reef Marine Park Authority. D.G.B. and H.A.S. are funded by a partnership between Mitsubishi Corporation and Earthwatch Institute.

Literature cited

Aeby GS, Howells E, Work T, Abrego D, Williams GJ, Wedding LM, Caldwell JM, Moritsch M, Burt JA (2020) Localized outbreaks of coral disease on Arabian reefs are linked to extreme temperatures and environmental stressors. Coral Reefs 39:829–846.

AIMS (2012) Survey Report 8-30 Oct 2012.

AIMS (2011) Survey Report 15 Oct 2011.

Anthony S, Page C, Bourne D, Willis B (2008) Newly characterized distinct phases of the coral disease 'atramentous necrosis' on the Great Barrier Reef. Dis Aquat Organ 81:255–259.

Australian Institute of Marine Science (2018) Great Barrier Reef suffers multiple, regional-scale impacts. Australian Institute of Marine Science.

Babcock RC, Plagányi ÉE, Condie SA, Westcott DA, Fletcher CS, Bonin MC, Cameron D (2020) Suppressing the next crown-of-thorns outbreak on the Great Barrier Reef. Coral Reefs 39:1233–1244.

Baird AH, Pratchett MS, Hoey AS, Herdiana Y, Campbell SJ (2013) *Acanthaster planci* is a major cause of coral mortality in Indonesia. Coral Reefs 32:803–812.

Beeden R, Willis BL, Raymundo LJ, Page CA, Weil E (2008) Underwater cards for assessing coral health on Indo-Pacific reefs. Coral Reef Targeted Research and Capacity Building for Management Program. Currie Communications, Melbourne.

Bourne DG, Ainsworth TD, Pollock FJ, Willis BL (2015) Towards a better understanding of white syndromes and their causes on Indo-Pacific coral reefs. Coral Reefs 34:233–242.

Bourne DG, Smith HA, Page CE (2022) Diseases of scleractinian corals. In: *Invertebrate Pathology*. Rowley AF, Coates CJ, Whitten MM (eds) Oxford University Press, p 656

Bridge TCL, Webster J, Sih TL, Bongaerts P (2019) The Great Barrier Reef outer-shelf. In: *The Great Barrier Reef: Biology, Environment and Management*. Hutchings P, Kingsford M, Hoegh-Guldberg O (eds) CSIRO Publishing

Brodnicke OB, Bourne DG, Heron SF, Pears RJ, Stella JS, Smith HA, Willis BL (2019) Unravelling the links between heat stress, bleaching and disease: fate of tabular corals following a combined disease and bleaching event. Coral Reefs 38:591–603.

Brown BE (1997) Coral bleaching: causes and consequences. Coral Reefs 16:S129–S138.

Bruno JF, Selig ER (2007) Regional decline of coral cover in the Indo-Pacific: timing, extent, and subregional comparisons. PLoS ONE 2:e711.

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 Biol 5:e124.

Bruno JF, Valdivia A (2016) Coral reef degradation is not correlated with local human population density. Sci Rep 6:29778.

Combs IR, Studivan MS, Eckert RJ, Voss JD (2021) Quantifying impacts of stony coral tissue loss disease on corals in Southeast Florida through surveys and 3D photogrammetry. PLOS ONE 16:e0252593.

Connell SD (2007) Water quality and the loss of coral reefs and kelp forests: alternative states and the influence of fishing. In: Connell SD, Gillanders BM (eds) Marine Ecology. Oxford University Press, Melbourne, pp 556-568. In: *Marine Ecology*. Connell SD, Gillanders BM (eds) Oxford University Press, Melbourne, p 556–568

Darling ES, McClanahan TR, Côté IM (2013) Life histories predict coral community disassembly under multiple stressors. Glob Change Biol 19:1930–1940.

Day JC, Dobbs K (2013) Effective governance of a large and complex cross-jurisdictional marine protected area: Australia's Great Barrier Reef. Mar Policy 41:14–24.

De'ath G, Fabricius KE, Sweatman H, Puotinen M (2012) The 27–year decline of coral cover on the Great Barrier Reef and its causes. Proc Natl Acad Sci 109:17995–17999.

Doney SC, Fabry VJ, Feely RA, Kleypas JA (2009) Ocean acidification: the other CO_2 problem. Annu Rev Mar Sci 1:169–192.

Fabricius K, De'ath G, McCook L, Turak E, Williams DMcB (2005) Changes in algal, coral and fish assemblages along water quality gradients on the inshore Great Barrier Reef. Mar Pollut Bull 51:384–398.

Gardner TA, Côté IM, Gill JA, Grant A, Watkinson AR (2003) Long-term region-wide declines in Caribbean corals. Science 301:958–960.

Goergen EA, Schopmeyer S, Moulding AL, Moura A, Kramer P, Viehman TS (2020) Coral reef restoration monitoring guide: Methods to evaluate restoration success from local to ecosystem scales. NOAA National Centers for Coastal Ocean Science, Silver Spring, MD.

Great Barrier Reef Marine Park Authority (2017) Significant coral decline and habitat loss on the Great Barrier Reef.

Greene A, Donahue MJ, Caldwell JM, Heron SF, Geiger E, Raymundo LJ (2020) Coral disease time series highlight size-dependent risk and other drivers of White Syndrome in a multi-species model. Front Mar Sci 7:601469.

Haapkylä J, Melbourne-Thomas J, Flavell M, Willis BL (2010) Spatiotemporal patterns of coral disease prevalence on Heron Island, Great Barrier Reef, Australia. Coral Reefs 29:1035–1045.

Haapkylä J, Unsworth RKF, Flavell M, Bourne DG, Schaffelke B, Willis BL (2011) Seasonal rainfall and runoff promote coral disease on an inshore reef. PLoS ONE 6:e16893.

Harrison HB, Álvarez-Noriega M, Baird AH, Heron SF, MacDonald C, Hughes TP (2019) Back-to-back coral bleaching events on isolated atolls in the Coral Sea. Coral Reefs 38:713–719.

Harvell D, Jordán-Dahlgren E, Merkel S, Rosenberg E, Raymundo L, Smith G, Weil E, Willis B (2007) Coral disease, environmental drivers, and the balance between coral and microbial associates. Oceanography 20:172–195.

Hawkins JP, Roberts CM (1992) Effects of recreational SCUBA diving on fore-reef slope communities of coral reefs. Biol Conserv 62:171–178.

Hawkins JP, Roberts CM, Van'T Hof T, De Meyer K, Tratalos J, Aldam C (1999) Effects of recreational scuba diving on Caribbean coral and fish communities. Conserv Biol 13:888–897.

Heron SF, Willis BL, Skirving WJ, Eakin CM, Page CA, Miller IR (2010) Summer hot snaps and winter conditions: modelling White Syndrome outbreaks on Great Barrier Reef corals. PLoS ONE 5:e12210.

Hoegh-Guldberg O, Mumby PJ, Hooten AJ, Steneck RS, Greenfield P, Gomez E, Harvell CD, Sale PF, Edwards AJ, Caldeira K, Knowlton N, Eakin CM, Iglesias-Prieto R, Muthiga N, Bradbury RH, Dubi A, Hatziolos ME (2007) Coral reefs under rapid climate change and ocean acidification. Science 318:1737–1742.

Howells EJ, Vaughan GO, Work TM, Burt JA, Abrego D (2020) Annual outbreaks of coral disease coincide with extreme seasonal warming. Coral Reefs 39:771–781.

Hughes T, Kerry J, Álvarez-Noriega M, Álvarez-Romero JG, Anderson K, Baird AH, Babcock RC, Beger M, Bellwood DR, Berkelmans R, Bridge TC, Butler IR, Byrne M, Cantin NE, Comeau S, Connolly SR, Cumming GS, Dalton SJ, Diaz-Pulido G, Eakin CM, Figueira WF, Gilmour JP, Harrison HB, Heron SF, Hoey AS, Hobbs J-PA, Hoogenboom MO, Kennedy EV, Kuo C, Lough JM, Lowe RJ, Liu G, McCulloch MT, Malcolm HA, McWilliam MJ, Pandolfi JM, Pears RJ, Pratchett MS, Schoepf V, Simpson T, Skirving WJ, Sommer B, Torda G, Wachenfeld DR, Willis BL, Wilson SK (2017a) Global warming and recurrent mass bleaching of corals. Nature 543:373–377.

Hughes TP, Barnes ML, Bellwood DR, Cinner JE, Cumming GS, Jackson JBC, Kleypas J, van de Leemput IA, Lough JM, Morrison TH, Palumbi SR, van Nes EH, Scheffer M (2017b) Coral reefs in the Anthropocene. Nature 546:82–90.

Hughes TP, Kerry JT, Baird AH, Connolly SR, Dietzel A, Eakin CM, Heron SF, Hoey AS, Hoogenboom MO, Liu G, McWilliam MJ, Pears RJ, Pratchett MS, Skirving WJ, Stella JS, Torda G (2018) Global warming transforms coral reef assemblages. Nature 556:492–496.

Jackson JBC, Kirby MX, Berger WH, Bjorndal KA, Botsford LW, Bourque BJ, Bradbury RH, Cooke R, Erlandson J, Estes JA, Hughes TP, Kidwell S, Lange CB, Lenihan HS, Pandolfi JM, Peterson CH, Steneck RS, Tegner MJ, Warner RR (2001) Historical overfishing and the recent collapse of coastal ecosystems. Science 293:629–637.

Kleypas JA, Yates KK (2009) Coral reefs and ocean acidification. Oceanography 22:108–117.

Klumpp DW, McKinnon AD (1989) Temporal and spatial patterns in primary production of a coral-reef epilithic algal community. J Exp Mar Biol Ecol 131:1–22.

McCook L, Jompa J, Diaz-Pulido G (2001) Competition between corals and algae on coral reefs: a review of evidence and mechanisms. Coral Reefs 19:400–417.

McCook LJ, Ayling T, Cappo M, Choat JH, Evans RD, Freitas DMD, Heupel M, Hughes TP, Jones GP, Mapstone B, Marsh H, Mills M, Molloy FJ, Pitcher CR, Pressey RL, Russ GR, Sutton S, Sweatman H, Tobin R, Wachenfeld DR, Williamson DH (2010) Adaptive management of the Great Barrier Reef: A globally significant demonstration of the benefits of networks of marine reserves. Proc Natl Acad Sci 107:18278–18285.

McLeod IM, Hein MY, Babcock R, Bay L, Bourne DG, Cook N, Doropoulos C, Gibbs M, Harrison P, Lockie S, van Oppen MJH, Mattocks N, Page CA, Randall CJ, Smith A, Smith HA, Suggett DJ, Taylor B, Vella KJ, Wachenfeld D, Boström-Einarsson L (2022) Coral restoration and adaptation in Australia: The first five years. PLOS ONE 17:e0273325.

Mera H, Bourne DG (2018) Disentangling causation: complex roles of coral-associated microorganisms in disease. Environ Microbiol 20:431–449.

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

Morais J, Morais RA, Tebbett SB, Pratchett MS, Bellwood DR (2021) Dangerous demographics in post-bleach corals reveal boom-bust versus protracted declines. Sci Rep 11:18787.

Moran PJ, Bradbury RH, Reichelt RE (1988) Distribution of recent outbreaks of the crown-of-thorns starfish (*Acanthaster planci*) along the Great Barrier Reef: 1985-1986. Coral Reefs 7:125–137.

Neely KL, Lewis CL, Lunz KS, Kabay L (2021) Rapid population decline of the pillar coral *Dendrogyra cylindrus* along the Florida Reef Tract. Front Mar Sci 8:656515.

Nicolet KJ, Hoogenboom MO, Gardiner NM, Pratchett MS, Willis BL (2013) The corallivorous invertebrate *Drupella* aids in transmission of brown band disease on the Great Barrier Reef. Coral Reefs 32:585–595.

Nugues MM, Roberts CM (2003) Coral mortality and interaction with algae in relation to sedimentation. Coral Reefs 22:507–516.

580 Palmer CV, Mydlarz LD, Willis BL (2008) Evidence of an inflammatory-like response in
581 non-normally pigmented tissues of two scleractinian corals. Proc R Soc B Biol Sci 275:2687–
582 2693.

583 Pandolfi JM, Bradbury RH, Sala E, Hughes TP, Bjorndal KA, Cooke RG, McArdle D,
584 McClenachan L, Newman MJH, Paredes G, Warner RR, Jackson JBC (2003) Global
585 trajectories of the long-term decline of coral reef ecosystems. Science 301:955–958.

586 Pisapia C, Anderson KD, Pratchett MS (2016) Temporal consistency in background mortality
587 of four dominant coral taxa along Australia's Great Barrier Reef. Coral Reefs 35:839–849.

588 Pisapia C, Pratchett MS (2014) Spatial variation in background mortality among dominant
589 coral taxa on Australia's Great Barrier Reef. PLoS ONE 9:e100969.

590 Pratchett MS, Heron SF, Mellin C, Cumming GS (2021) Recurrent mass-bleaching and the
591 potential for ecosystem collapse on Australia's Great Barrier Reef. In: *Ecosystem Collapse*
592 *and Climate Change*. Ecological Studies, Canadell JG, Jackson RB (eds) Springer
593 International Publishing, Cham, p 265–289

594 R Core Team (2021) R: A language and environment for statistical computing.

595 Roberts CM (1995) Effects of fishing on the ecosystem structure of coral reefs. Conserv Biol
596 9:988–995.

597 Roff G, Kvennefors ECE, Fine M, Ortiz J, Davy JE, Hoegh-Guldberg O (2011) The ecology
598 of 'Acroporid White Syndrome', a coral disease from the Southern Great Barrier Reef. PLoS
599 ONE 6:e26829.

600 Rotjan RD, Lewis SM (2008) Impact of coral predators on tropical reefs. Mar Ecol Prog Ser
601 367:73–91.

602 Ruiz-Moreno D, Willis B, Page A, Weil E, Cróquer A, Vargas-Angel B, Jordan-Garza A,
603 Jordán-Dahlgren E, Raymundo L, Harvell C (2012) Global coral disease prevalence
604 associated with sea temperature anomalies and local factors. Dis Aquat Organ 100:249–261.

605 Selig ER, Drew Harvell C, Bruno JF, Willis BL, Page CA, Casey KS, Sweatman H (2006)
606 Analyzing the relationship between ocean temperature anomalies and coral disease outbreaks
607 at broad spatial scales. In: *Coastal and Estuarine Studies*. Phinney JT, Hoegh-Guldberg O,
608 Kleypas J, Skirving W, Strong A (eds) American Geophysical Union, Washington, D. C., p
609 111–128

610 Smith HA, Prenzlau T, Whitman T, Fulton SE, Borghi S, Logan M, Heron SF, Bourne DG
611 (2022) Macroalgal canopies provide corals limited protection from bleaching and impede
612 post-bleaching recovery. J Exp Mar Biol Ecol 553:151762.

613 Swierts T, Vermeij MJ (2016) Competitive interactions between corals and turf algae depend
614 on coral colony form. PeerJ 4:e1984.

Van De Water JAJM, Lamb JB, Heron SF, Van Oppen MJH, Willis BL (2016) Temporal patterns in innate immunity parameters in reef-building corals and linkages with local climatic conditions. Ecosphere 7.

Vanhatalo J, Hosack GR, Sweatman H (2017) Spatiotemporal modelling of crown-of-thorns starfish outbreaks on the Great Barrier Reef to inform control strategies. J Appl Ecol 54:188–197.

Vega Thurber R, Mydlarz LD, Brandt M, Harvell D, Weil E, Raymundo L, Willis BL, Langevin S, Tracy AM, Littman R, Kemp KM, Dawkins P, Prager KC, Garren M, Lamb J (2020) Deciphering coral disease dynamics: integrating host, microbiome, and the changing environment. Front Ecol Evol 8:575927.

Vega Thurber RL, Burkepile DE, Fuchs C, Shantz AA, McMinds R, Zaneveld JR (2014) Chronic nutrient enrichment increases prevalence and severity of coral disease and bleaching. Glob Change Biol 20:544–554.

Wakeford M, Done TJ, Johnson CR (2008) Decadal trends in a coral community and evidence of changed disturbance regime. Coral Reefs 27:1–13.

Zaneveld JR, Burkepile DE, Shantz AA, Pritchard CE, McMinds R, Payet JP, Welsh R, Correa AMS, Lemoine NP, Rosales S, Fuchs C, Maynard JA, Thurber RV (2016) Overfishing and nutrient pollution interact with temperature to disrupt coral reefs down to microbial scales. Nat Commun 7:11833.