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Drivers of coral mortality in non-acute disturbance periods

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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 stressors 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 stressors 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] outbreaks). 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 compromised 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.

KEY WORDS: 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 sedimenta-

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tion from runoff and dredging; Fabricius et al. 2005, Connell 2007), and overfishing (Roberts 1995, Jackson 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 northeastern 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 following 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 disease (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 haphaz-

ardly 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 season, 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.

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

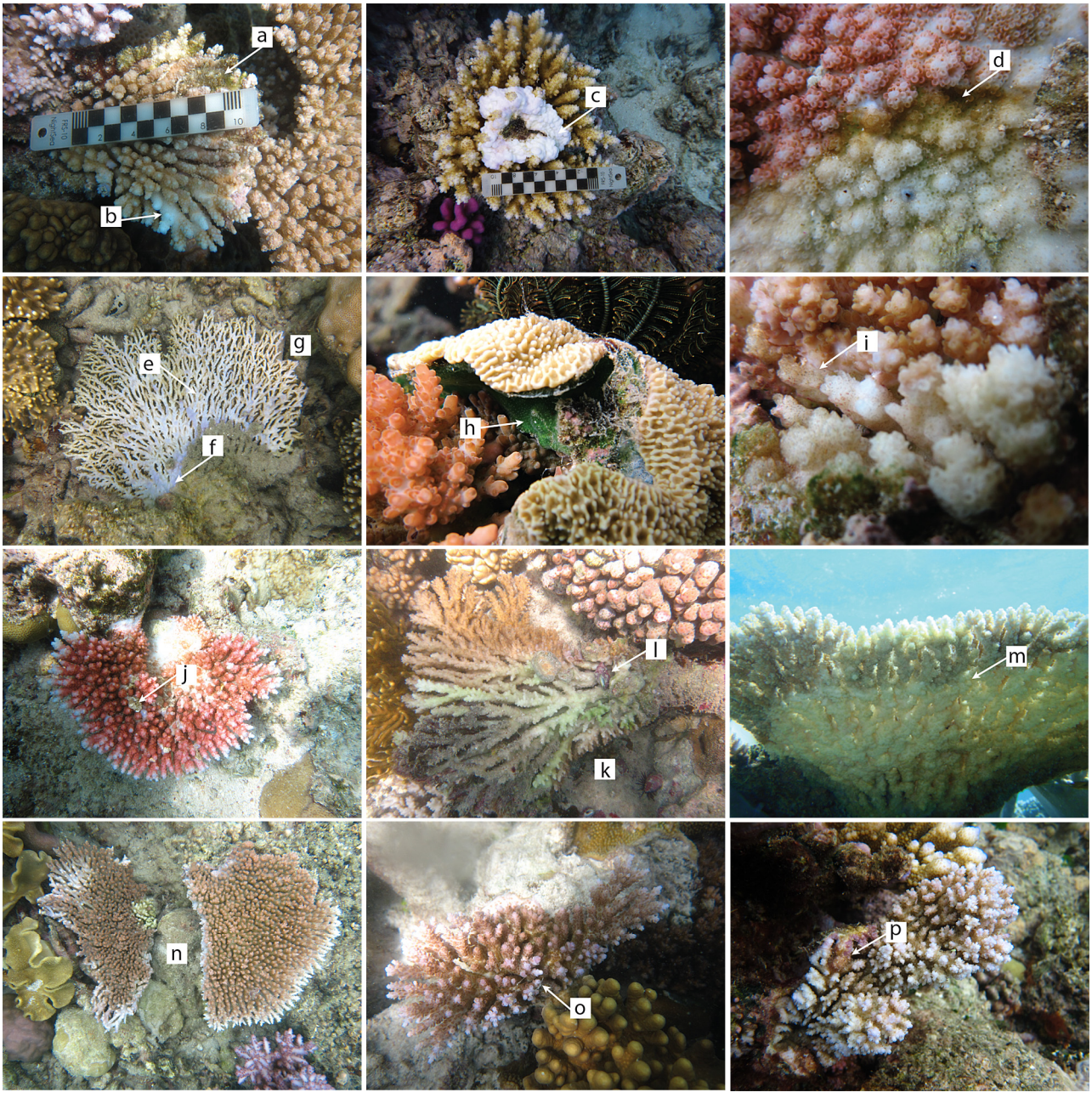

Fig. 1. Example images of 16 of the 19 health indicators (excluding black band, other diseases, other compromised health, see Table 1): (a) overgrowth by green algae; (b) white syndrome; (c) growth anomaly; (d) brown band; (e) bleaching; (f) pigmentation; (g) flipped; (h) overgrowth by sponge; (i) skeletal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

determine the relative occurrence of disease, compromised health, predation, and physical injury in corals over a period of time and is measured as:

$$\text{prevalence}_{q,i,t} = n_{q,i,t}/N_{q,t} \quad (1)$$

where, in this study, $n_{q,i,t}$ is the number of colonies with condition i,i α [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 $\alpha = 0.05$.

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:

$$\begin{gathered} y_{i,t} \sim Bernoulli(p_{i,t}) \\ \text{Logit}(p_{i,t}) = \beta_0 + z_i + \beta_1 x_{i,t-1} + \beta_2 d_{i,t-1} \\ + \beta_3 c_{i,t-1} + \beta_4 ph_{i,t-1} + \beta_5 shelf_i \end{gathered} \quad (2)$$

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:

$$h(t) = h_0(t)\exp(\beta_1 x_1 + \beta_2 x_2 + \beta_3 x_3) \quad (3)$$

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), prior (x_2) and post (x_3) exposure to other stressors. Hazard is an instantaneous mortality at time t. Survival rate is given as a percent of colonies surviving at time t compared to the total number of colonies observed with WSs. The probability of death is averaged across all time points.

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

3. RESULTS

3.1. Population demographics

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

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)	Died (n)	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

The median size of tagged colonies at the first observation was 205 mm^2; 50% of tagged corals were between 84 and 671 mm^2 (Fig. S1 in the Supplement at www.int-res.com/articles/suppl/m717p037_supp.pdf). At the start of the study ($t = 0$), the size of tagged colonies was similar among reefs, with the exception of Yonge Reef, which had a greater abundance of smaller colonies than other reefs (<50 mm^2; Fig. S1).

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

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

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

Prevalence of the compromised health state varied significantly between shelf position and pairwise among 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 compromised 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. Diseases 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

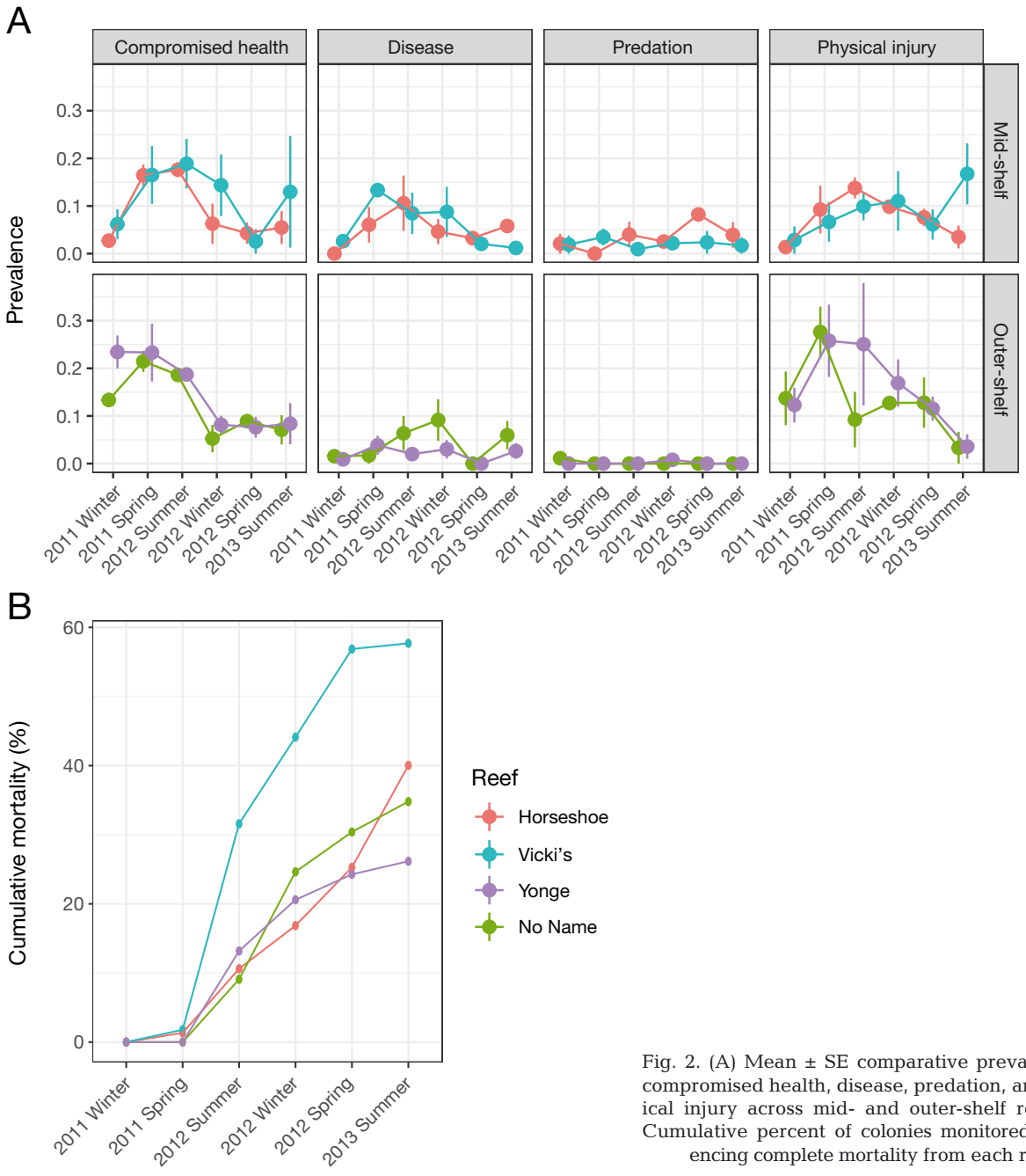

Fig. 2. (A) Mean ± SE comparative prevalence of compromised health, disease, predation, and physical injury across mid- and outer-shelf reefs. (B) Cumulative percent of colonies monitored experiencing complete mortality from each reef

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 recorded 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 account-

ing 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.

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 exposure. 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).

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 stres-

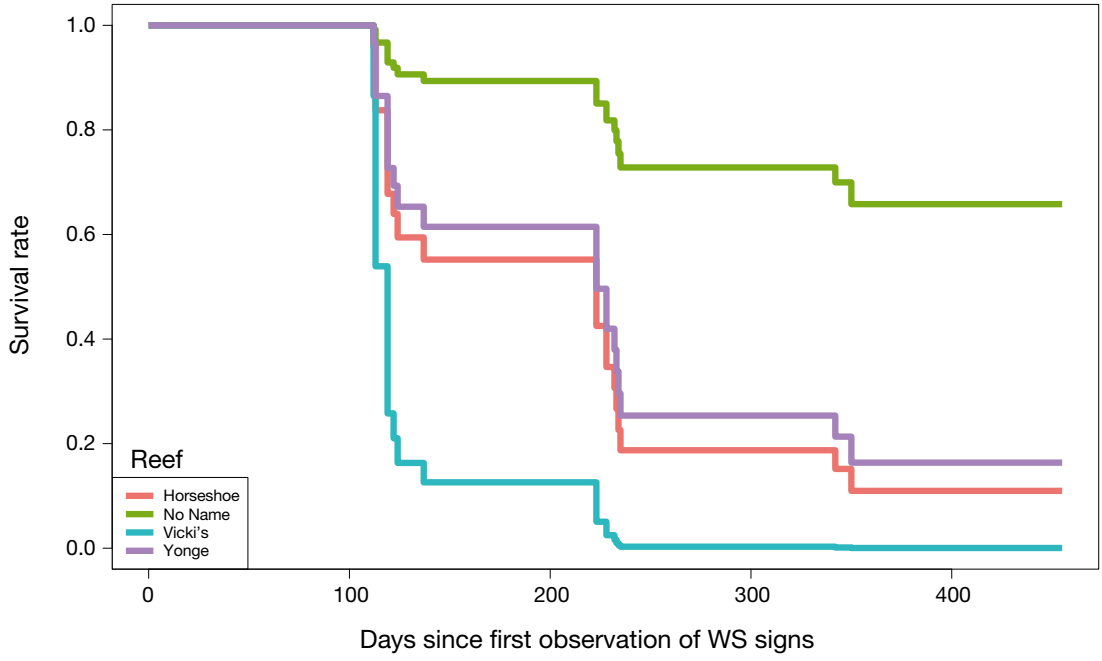

Fig. 3. Survival rate (proportion of live individuals) for coral colonies after being observed with signs of white syndrome at Horseshoe, Vicki's, Yonge, and No Name reefs

sors 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).

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

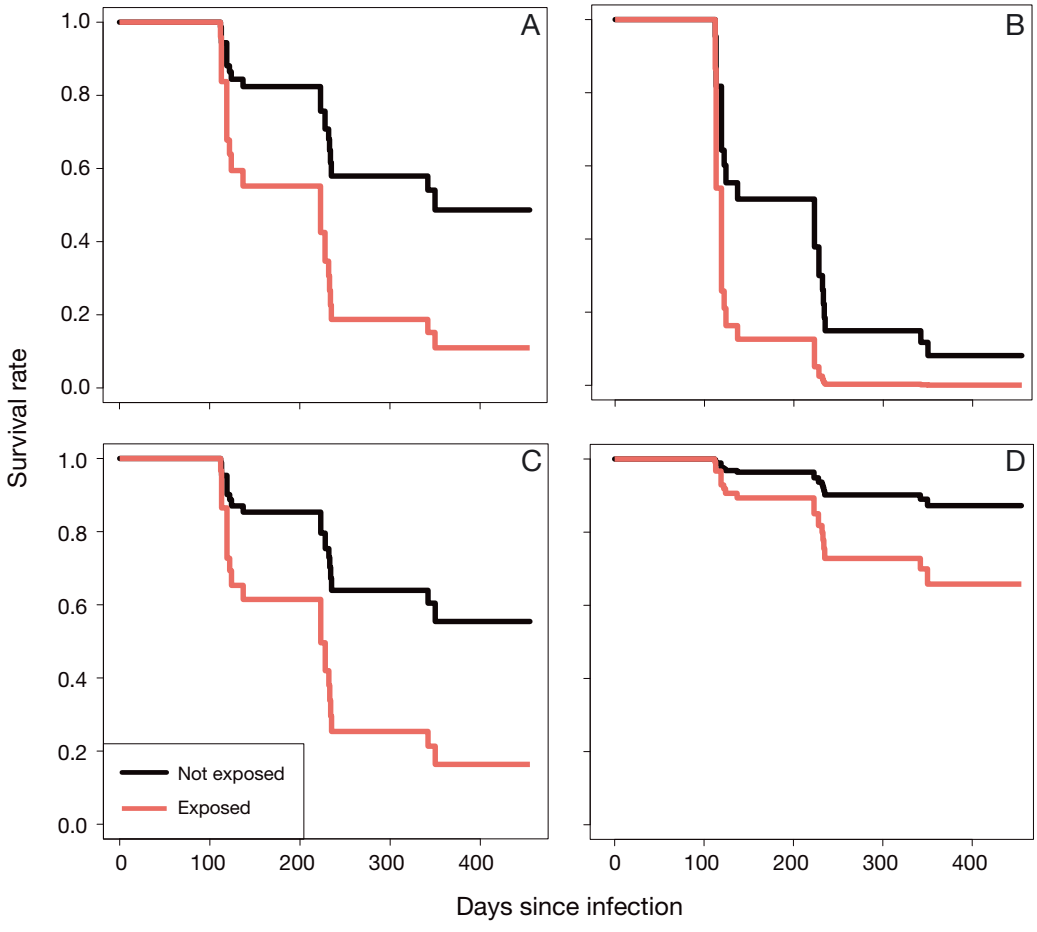

Fig. 4. Survival rate (proportion of live individuals) of diseased colonies exposed or not exposed to stressors prior to displaying signs of white syndromes at (A) Horseshoe Reef, (B) Vicki's Reef, (C) Yonge Reef, and (D) No Name Reef

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

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

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

Signs of predation were not common during the study period despite signs of an incipient outbreak of CoTS in the region (https://apps.aims.gov.au/reef-monitoring). The approximately 3 mo intervals between surveys, which were conducted seasonally rather than at a finer temporal scale due to the remoteness of reefs, potentially precluded detection of CoTS predation scars. Further, while every effort was made to differentiate CoTS predation from disease signs, particularly WSs, these 2 stressors manifest in similar physical outcomes (i.e. tissue loss), and records of disease may have been inaccurately assigned. Nonetheless, if a colony was not recorded as showing signs of predation in the previous survey, mortality was not attributed to predation. However, given that CoTS can move across reefs rapidly, it is possible that the mortality of some colonies that died

at time t was because of CoTS predation in the time between t and $t-1$. Similarly, there were few observations of active predation by *Drupella* and hence it is likely that some of the background mortality found in this study may be attributed to CoTS and *Drupella* predation. Nevertheless, despite the infrequent observations of predation, colonies that had predation injuries were at the highest risk of mortality in the subsequent observation. Considering the 10-fold increased risk of mortality found on mid-shelf reefs compared to outer-shelf reefs, and that CoTS outbreaks affect mid-shelf areas more frequently (Moran et al. 1988, Vanhatalo et al. 2017), it is likely that CoTS had a larger role in causing mortality than our study could detect.

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

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 contributed 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.

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