Pathologies and mortality rates caused by organic carbon and nutrient stressors in three Caribbean coral species

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ABSTRACT: Anthropogenic inputs, including organic carbon and nutrient loading, are increasingly changing the water quality on coral reefs. Herein we show that treating *Montastraea annularis*, *Agaricia tenuifolia* and *Porites furcata* with various organic carbon sources (starch, lactose, arabinose and mannose) results in different species-specific and carbon-specific pathologies and rates of mortality. The variation in the pathological characteristics caused by stressors showed that visual cues for determining coral health and disease may be misleading. The probability of mortality increased significantly over time with continual exposure to several of the stressors, suggesting that chronic stressors may be more harmful than acute stressors. In contrast to the organic carbon sources, high concentrations of nutrients (phosphate, ammonium and nitrate) did not directly kill corals. The variation in coral responses to anthropogenic stressors means that changes on disturbed coral reefs will depend on the type of and duration of exposure to the stressor, as well as on the species of coral.

KEY WORDS: Coral · Disease · Symptoms · Chronic stress · Ecotoxicology

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INTRODUCTION

Coral reefs are being severely changed by natural and anthropogenic stressors, resulting in a substantial decline of these ecosystems globally (Jackson et al. 2001, Gardner et al. 2003, Pandolfi et al. 2003). In the Caribbean, hard coral cover has been significantly degraded over the last 3 decades (Gardner et al. 2003). Growing human populations and projected climatic changes will result in more severe and variable stressors on coral reefs (Hughes et al. 2003). These changes will continue to test the survival of different coral species (Harvell et al. 1999, Green & Bruckner 2000).

Diseases (Dustan & Halas 1987, Aronson et al. 1998, Harvell et al. 1999) and bleaching events (Glynn 1993, Brown 1997, Hughes & Connell 1999) have caused major phase shifts in coral reef communities (Knowlton et al. 1990, Hughes 1994, Harvell et al. 1999, Aronson et al. 2002). Determining species-specific differences in coral vulnerability to disease is critical for explaining and predicting changes on disturbed community structure. Previous studies have shown that closely related coral species demonstrate large differences in their response to disturbances (Marcus & Thorhaug 1981, Fitt & Warner 1995, Yap & Molina 2003). Several coral species thrive in 'marginal' environments and proliferate on impacted reefs while others suffer high mortality. *Porites furcata*, for example, is abundant in disturbed environments, which experience extreme fluctuations in temperature, sedimentation and salinity that exclude most other coral species (e.g. Biscayne Bay, Florida; Lirman et al. 2003).

In this study, *Montastraea annularis*, *Agaricia tenuifolia* and *Porites furcata* were exposed to varying

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organic carbon sources (mono- and polysaccharides) and nutrients (combinations of phosphate, ammonium and nitrate). Our results show that the 3 coral species displayed different pathologies and mortality rates in response to these treatments.

MATERIALS AND METHODS

Coral collection and culturing system. Experiments were conducted at the Smithsonian Tropical Research Institute's Caribbean marine station in Bocas del Toro, Panama. The coral species were chosen because *Montastraea annularis* is the most common Caribbean reef-building species, while *Agaricia tenuifolia* and *Porites furcata* have become dominant shallow water corals since the decline of *Acropora cervicornis* due to white band disease (WBD; Aronson et al. 1998, 2002). Corals were collected from nearby waters using a hammer and chisel for *M. annularis* nubbins (~2 to 3 cm diameter) and steel bone clippers for *A. tenuifolia* and *P. furcata* (both ~4 cm length). In all cases, only the bare skeleton below the coral tissue was cut. Corals fragments were transported back to the field station in 19 l buckets fitted with plastic separators to minimize damage. The fragments were allowed to recover for at least 1 wk before beginning the experiments.

We conducted 2 experimental runs using a flow-through treatment system called AADACS (Aquatic Automated Dosing and Culturing Systems; D. I. Kline & F. Rohwer unpubl. data). Individual coral fragments were placed in separate 100 ml polypropylene cups within 8 larger water baths to maintain ambient reef water temperature. The AADACS system was set up in a custom-made culturing room with a ceiling made of clear poly vinyl chloride (PVC) sheets and walls of window screening to increase the overall light levels in the room. Extra screening was used to equalize light levels among the baths. A light meter (Licor; Model LI-193SA) with a 4π sensor was used to measure light levels just above each of the 8 water baths. Light levels at noon were 153.6 ± 39.6 µE on overcast days and 263.7 ± 53.2 µE on clear days.

The AADACS uses Venturi valves to automatically add treatments to incoming reef seawater just prior to entering the cup containing the coral. For these experiments, the AADACS was programmed to turn on for 6 s of each minute, completely replenishing each coral's water every 7.6 ± 2.3 min. Experimental runs were conducted during October and November 2003. Both runs lasted for 30 d, with each coral nubbin's condition noted and photographed approximately every 3 d. For each experimental run, 10 nubbins of each coral species were exposed to the treatments and at least 10 nubbin controls were exposed to seawater only. Each of the 10 nubbins per treatment came from a separate coral colony.

Nutrient and organic carbon treatments. In the first 30 d experiment, *Montrastraea annularis*, *Agaricia tenuifolia* and *Porites furcata* fragments received 2 treatments: 5 and 25 mg l^{-1} of lactose. In separate treatments, all 3 corals species were exposed to combinations of nutrients. The 'high' nutrient doses were treatments of 2.5 µM potassium phosphate, 25 µM ammonium chloride and 7.5 µM calcium nitrate; the 'low' nutrient doses were treatments of 0.5 µM potassium phosphate, 5 µM ammonium chloride and 2.5 µM calcium nitrate. In the second 30 d experiment, *M. annularis* and *P. furcata* were separately exposed to 25 mg l^{-1} of starch, lactose, arabinose and mannose. All treatment concentrations reflect levels previously reported for impacted reefs (March 1977, Webber & Roff 1995, Costa et al. 2000, Van Duyl & Gast 2001).

Statistical analyses. The cumulative effects of the treatments on coral survivorship throughout the 30 d of each experiment were compared for each coral species. Coral mortality of each colony was recorded as a continuous variable representing the fraction of the colony with dead polyps. A polyp was considered dead when all tissue was lost from the skeleton. Each *Montastraea annularis* nubbin had approximately 30 to 70 polyps. Mortality was more frequently complete (i.e. 1 whole nubbin) than partial (i.e. a fraction of the nubbin), resulting in a severely non-normal distribution of mortality within each treatment. Therefore, a non-parametric rank test (Kruskal-Wallis test with corrections for ties and unequal sample sizes; Zar 1984) was used to compare differences in relative mortality across treatments. Post-hoc multiple comparisons using rank sums (again with corrections for ties and unequal sample sizes; Dunn 1964) were computed for species in experiments with significant differences among treatments to determine the outlying groups. Because the differences of coral mortality with and without altered seawater were being tested, post-hoc tests on the comparisons of treatment against control were used. To be conservative, results were tested against 2-tailed probability distributions, although using a 1-tailed test (assuming that treatments will only have increased mortality relative to controls) would not have changed the results.

To test for qualitative differences in the pathology of each treatment, the patterns of mortality were explored throughout the experiment. Given counts of coral survivorship over time, the per capita daily rate of mortality for each treatment, m_s, for the days preceding Sample s were computed as $m_s = 1 - (n_s/n_{s-1})^{1/t_s - t_{s-1}}$, where n_s is the number of corals alive and t_s is the time (d) at Sample s. Note that for these analyses partial morality was ignored and only the entire colony lost was considered, thus constraining all n_s to integers.

The data were fit to 2 models of mortality rate over time: (1) constant mortality over time, or $m_t = m$ for all Time t, and (2) mortality as a function of time. To allow maximum flexibility of this second mortality rate function while constraining the value between 0 and 1, the logistic equation $\mu_t = [1 + (\beta^{-1} + 1)\exp(-\alpha t)]^{-1}$ was used, where α and β were estimated parameters. Maximum likelihood techniques were used to identify the best-fit parameters for each model for each treatment. Given estimated parameters (m for Model 1 and both α and β for Model 2), a per capita probability of mortality, m_t, for each of the 30 d of the experiment was calculated. The probability of there being n_s corals alive in Sample s was a function of the number of corals alive in the previous sample, n_{s-1}, and of the daily per capita mortality probabilities, m_t, for days $[t_{s-1}, (t_s - 1)]$. The probability of the number of surviving corals changing from n_{s-1} to n_s was computed as

$$\Pr(n_s | n_{s-1}) = \qquad (1)$$
$$\sum_{i=n_s}^{n_{s-1}} \sum_{j=n_s}^{i} \dots \sum_{y=n_s}^{x} \mathrm{bin}(i, n_{s-1}, 1 - \mu_{s-1}) \mathrm{bin}(j, i, 1 - \mu_{t_{s-1}+1}) \dots \mathrm{bin}(n_{s-1}, y, 1 - \mu_{t_{s-1}})$$

where $\mathrm{bin}(a, b, 1 - m)$ was the binomial probability of having a individuals alive in the next time step after starting with b individuals with a per capita probability of survivorship, $1 - m$. The number of summations in Eq. (1) was the number of days separating Samples $s - 1$ and s. Summing the probability from Eq. (1) across all samples of coral survivorship samples during the 30 d run produced the total probability of the data given mortality function m_t. The log-likelihood value is the logarithm of this total probability. For each of the 2 models, parameter space was explored to identify the best-fitting parameter (or the combination of parameters, for the logistic model) that resulted in the maximum log-likelihood value.

To test whether the mortality probability changed with time, the 2 best-fit models for each treatment were compared to determine relative fit of each model. Because the time-independent mortality model was nested within the logistic model (as by setting α to zero in the logistic model), a simple statistical test for the presence of time-dependent mortality could be used (i.e. the likelihood ratio test; Hilborn & Mangel 1997). Specifically, the estimate of α was significantly different from zero at the $p < 0.05$ level if the log-likelihood of the best-fit logistic mortality model was larger by >1.92 than the log-likelihood of the best-fit nested time-independent mortality model ($1.92 = 0.5 \cdot X^2_{0.05\,(1)}$, because the time-dependent model had 1 more parameter than the nested time-independent model). Thus, a significant value of α means that the rate of mortality for that treatment changes significantly over time.

RESULTS

Mortality and pathology caused by different levels of lactose

Lactose treatments caused significant mortality of *Montastraea annularis* and *Agaricia tenuifolia* (Kruskal-Wallis H_c = 27.3 and 13.7, respectively; df = 2, $p < 0.05$ for both), but not of *Porites furcata* (Kruskal-Wallis H_c = 2.42, df = 2, $p > 0.25$). The high lactose treatment (25 mg l^{-1}) caused significant mortality of *M. annularis* relative to the control (90 vs. 10%; $p < 0.001$). Both the 5 and 25 mg l^{-1} treatments of lactose caused significant mortality of *A. tenuifolia* relative to control (77 vs. 73%, respectively, vs. 10% for control; $p < 0.02$ for both). In contrast, the 5 and 25 mg l^{-1} lactose treatments caused no significant mortality of *P. furcata* at either concentration after 30 d (27 and 23%, respectively, vs. 10% for control; $p > 0.1$, Fig. 1). With the high
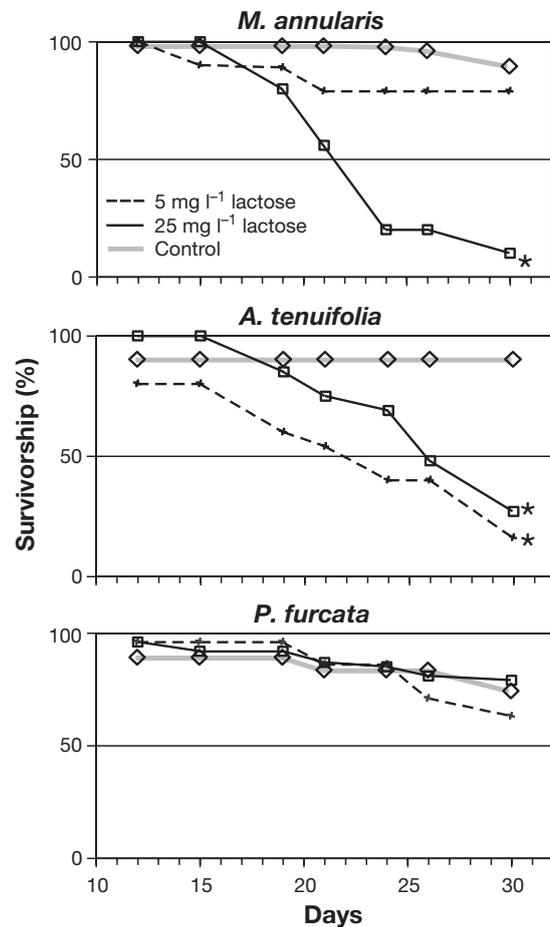
Fig. 1. *Montastraea annularis*, *Agaricia tenuifolia* and *Porites furcata*. Variation in coral survivorship during exposure to 5 and 25 mg l^{-1} lactose over a 30 d period. ⋆: Significant mortality of *M. annularis* and *A. tenuifolia*; control sample size = 49 nubbins for *M. annularis* and 10 each for *A. tenuifolia* and *P. furcata*; 10 nubbins were used in all treatments

Table 1. *Montastraea annularis*, *Agaricia tenuifolia* and *Porites furcata*. Pathologies observed during treatment with different nutrient and lactose concentrations. 'Low' nutrients: 0.5 µM potassium phosphate, 5 µM ammonium chloride, 2.5 µM calcium nitrate; high nutrients: 2.5 µM potassium phosphate, 25 µM ammonium chloride, 7.5 µM calcium nitrate.

Species	Lactose concentration		Nutrient concentration	
	5 mg l^{-1}	25 mg l^{-1}	Low	High
M. annularis	Progressive mortality beginning along coral edge	Sloughing of tissue in 1 event	Bleaching	Bleaching; partial mortality along edges
A. tenuifolia	Progressive mortality beginning along coral edge	Bleaching of conosteum or entire coral bleaching; progressive mortality beginning along coral edge	Bleaching; partial mortality, beginning along edges	Bleaching; partial mortality along edges
P. furcata	Retraction of polyps; progressive mortality beginning at coral edge	Retraction of polyps; increased mucus secretion; progressive mortality beginning at coral edge	Nothing observed	Retraction of polyps

lactose treatment, the rate of mortality (lethal time until 50% mortality, LT_{50}) was 21 and 26 d for *M. annularis* and *A. tenuifolia*, respectively. At the lower lactose dosage, *A. tenuifolia* had an LT_{50} of 24 d.

Sublethal effects of the lower lactose treatment (5 mg l^{-1}) were similar in all 3 coral species, including partial tissue loss at the edges (Table 1). Pathologies associated with the higher lactose treatment (25 mg l^{-1}) varied among the 3 coral species. *Agaricia tenuifolia* displayed bleaching of the coenosteum of the entire coral followed by progressive mortality, which began along the coral's edge until it destroyed the fragment. In contrast, *Montastraea annularis* usually appeared healthy until all the tissue sloughed off the coral.

Species-specific mortality caused by different carbon sources

Montastraea annularis and *Porites furcata* demonstrated distinct trends in response to different carbon sources (Fig. 2). For example, mannose caused significant mortality of *P. furcata* relative to the con-
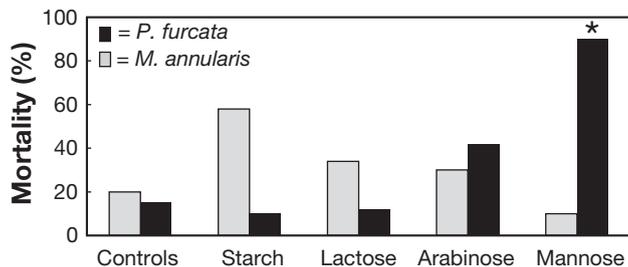
Fig. 2. *Montastraea annularis* and *Porites furcata*. Percent mortality after exposure to various mono- and polysaccharides (25 mg l^{-1}) for 30 d. *: Significant mortality; sample size = 10 for both treatments and controls

trol (90 vs. 15%, p < 0.001), but did not affect *M. annularis*.

Some of the *Porites furcata* corals were bleached from the arabinose exposure, while the mannose treatment produced progressive mortality that began at the coral tip. Pathologies associated with *Montastraea annularis* exposed to the different sugars were similar to those for lactose. The progressive mortality due to increased carbon levels resulted in a clear demarcation, in the form of a banding pattern between the degrading tissue and dead coral in 23% of the lethal cases (Fig. 3). The progression of mortality was symptomatically reminiscent of several coral diseases (review by Richardson 1998).

Mortality and pathologies caused by elevated nutrients

Combinations of potassium phosphate (2.5 µM), ammonium chloride (25 µM) and calcium nitrate (7.5 µM) had caused no significant coral mortality after 30 d (Fig. 4): *Agaricia tenuifolia* (H_c = 4.2, df = 2, p > 0.1), *Montastraea annularis* (H_c = 2.4, df = 2, p > 0.25), and *Porites furcata* (H_c = 0.6, df = 2, p > 0.75). Some of the *M. annularis* nubbins bleached at both nutrient levels, as did some of the *A. tenuifolia* fragments at the 'high' nutrient concentrations (Table 1).

Time-dependent mortality

Exposure to chronic stress caused a significant increase in the likelihood of coral mortality in some treatments (Fig. 5). In the first 30 d experiment, mortality increased significantly for the high lactose treatments with *Montastraea annularis* and both lactose level treatments with *Agaricia tenuifolia* (Fig. 5A). The time-dependent mortality in the second experiment was
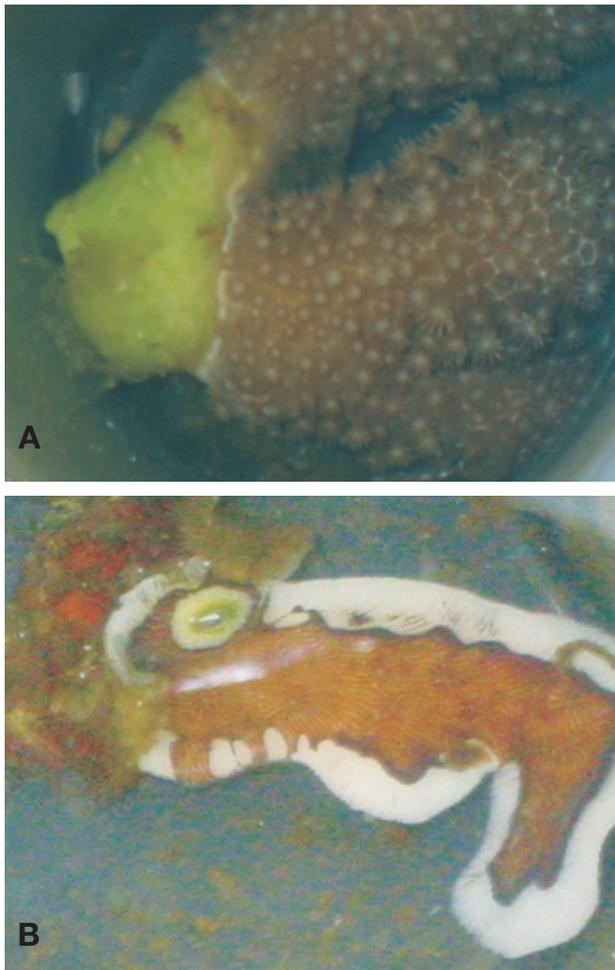
Fig. 3. *Agaricia tenuifolia* and *Porites furcata*. Examples of progressive mortality arising from increased lactose levels in (A) *A. tenuifolia* (25 mg l^{-1} lactose at 30 d) and (B) *P. furcata* (25 mg l^{-1} lactose at 21 d)

significant with starch and cellulose treatments of *M. annularis* and mannose exposure for *Porites furcata* (Fig. 5B).
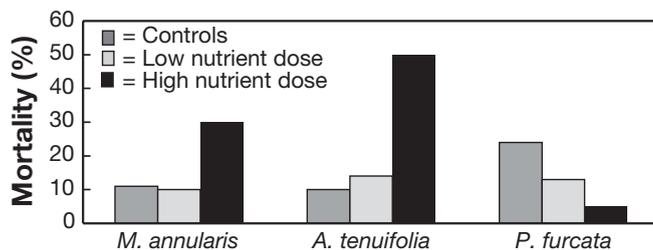

Fig. 4. *Montastraea annularis*, *Agaricia tenuifolia* and *Porites furcata*. Mortality after exposure to different levels of nutrients containing mixtures of ammonium, phosphate and nitrate (see 'Nutrient and organic treatments' for details of low and high nutrient doses). Control sample size = 49 for *M. annularis* and 10 each for *A. tenuifolia* and *P. furcata*

DISCUSSION

Variation in coral pathologies

Coral diseases have, by necessity, been described based on pathology (review by Richardson 1998). No ubiquitous pathological characteristics, however, were associated with increased nutrients or organic carbon levels. This means that a specific stressor can elicit multiple pathologies that vary between coral species (Table 1). The discrepancy in visual pathologies illustrates the limitation of traditional methods for assessing coral health. Similarly, the sudden 'sloughing' of tissue in what had previously appeared to be a healthy *Montastraea annularis* highlights the need for better markers for the assessment of coral health. Fitt & Warner (1995) also noted a rapid tissue sloughing from 'healthy' colonies when *M. annularis* was subjected to increased seawater temperature up to 34°C. Microscopic analyses have shown that there is a subdermal layer of microbes in stressed corals just prior to such rapid mortality events (O. Pantos pers. comm.).

Pantos et al. (2003) showed that the bacterial community on apparent 'healthy tissue' of white plague-diseased *Montastraea annularis* differed significantly from that on healthy tissue of an uninfected and unstressed colony. Together these observations suggest that a coral's response to stress frequently involves changes in the microbial community, and the response of the holobiont may be an effective tool in identifying stress in coral colonies before visible symptoms appear (Pantos et al. 2003). Molecular measurements have also been proposed as bioassays for coral health (Burns 1993, Harriot 1993, Downs et al. 2000). Logistical limitations, however, may make molecular techniques too difficult for rapid coral health assessments. A tool that is deployable and responsive *in situ* and combines data from several of a coral's symbionts is greatly needed. Potential targets could be *in situ* ATP measurements to determine microbial activity or fluorometry with DNA stains to monitor microbial numbers.

Increased probability of mortality with chronic stressor

In these experiments, the probability of mortality increased significantly with continual exposure to several of the stressors. This effect was noted for all of the stressors found to cause significant mortality after a 30 d period (Fig. 5). These results show that the impact of a chronic stress on susceptible species is not constant, but instead has an increasingly potent effect over time. Chronic stress has been shown to have a devas-
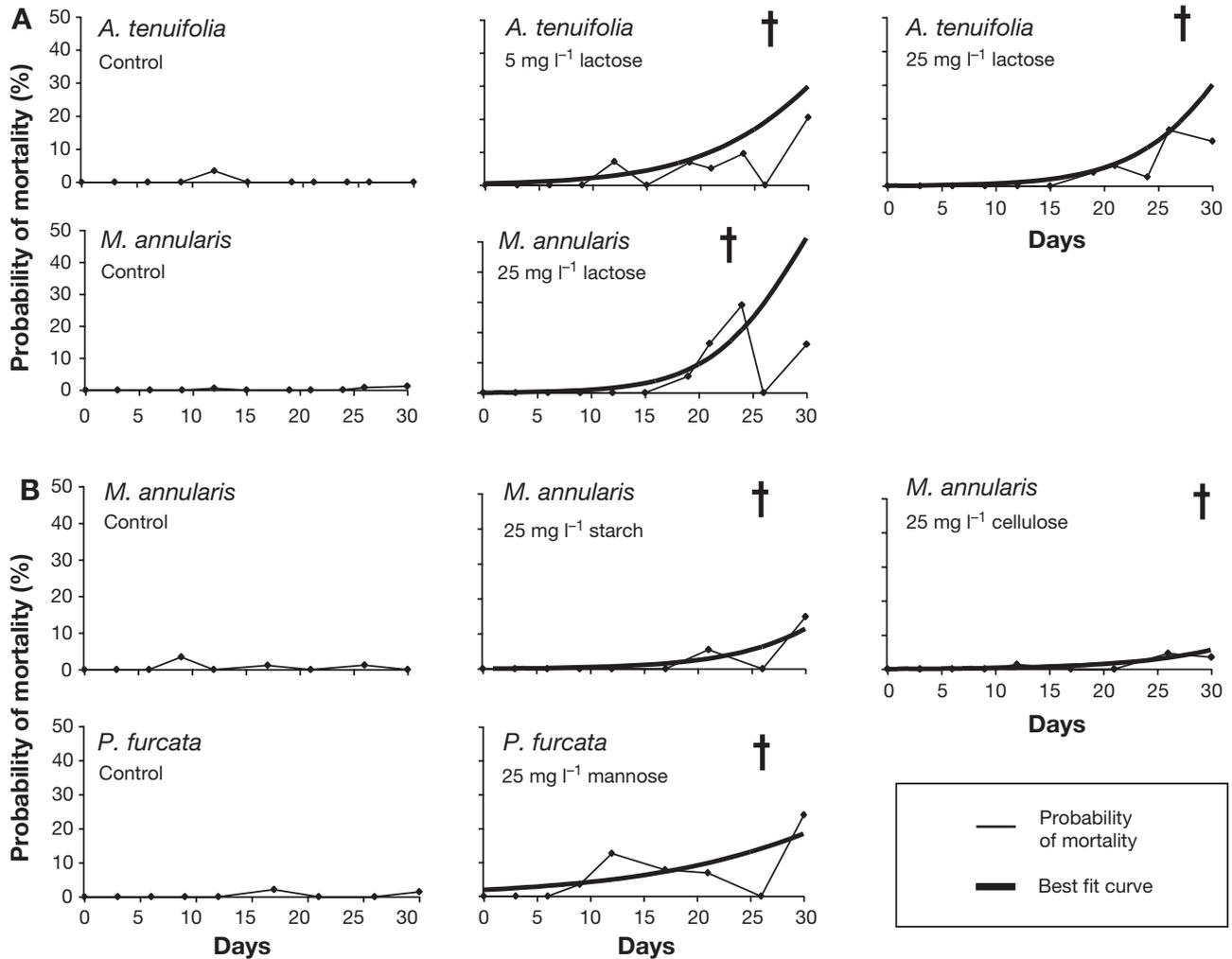
Fig. 5. *Montastraea annularis*, *Agaricia tenuifolia* and *Porites furcata*. Time-dependent mortality during exposure to chronic stressor over a 30 d period. Exposure was to (A) lactose and (B) various mono- and polysaccharides. Probability of mortality was computed as per capita daily rate of mortality for each treatment. (♦) Actual daily probability of mortality; †: significant rate of mortality changes over time as indicated by time-dependent model

tating effect on coral reef systems (Walker & Ormond 1982, Scott 1990). Sewage runoff into tropical marine systems is considered one of the principal causes of coral reef degradation (Risk & Erdmann 2000). Continual sewage input (a major source of organic carbon) has been shown to severely depress and eliminate the coral coverage in an immediate discharge area of Honolulu, Hawaii (Mamala Bay; Grigg 1995). This is especially worrisome because most coral reefs are in less economically developed regions of the world, where wastewater treatment is limited or non-existent. Distinguishing the effects of chronic and acute stress remains an outstanding problem in ecotoxicological studies on coral reefs. Responses of corals and their symbionts to anthropogenic stressors will depend on the period of exposure to those stressors. These results highlight the fact that coral mortality patterns may depend on each type of stressor, the species of coral, and the duration of exposure time.

Differential effects of stressors on corals and coral holobiont

Corals naturally harbor Bacteria and Archaea (Di-Salvo 1971, DiSalvo & Gundersen 1971, Ducklow & Mitchell 1979, Ritchie & Smith 1995, 1997, Rohwer et al. 2001, 2002, Kellogg 2004, Wegley et al. 2004), as well as zooxanthellae, endolithic algae (Bentis et al. 2000) and endolithic fungi (Odum & Odum 1955, Shashar et al. 1997). Disrupting the coral–microbe relationship by organic carbon loading (e.g. glucose) can directly cause

coral mortality by over-stimulating growth of coral mucus-associated microbes (Mitchell & Chet 1975, D. I. Kline & F. Rohwer unpubl. data). We propose that variation in coral responses to stressors may be mediated by species-specific dynamics of the symbiont community (Rohwer & Kelley 2004), similar to the variation observed in bleaching based on zooxanthellae type (Rowan et al. 1997, Toller et al. 2001). Additional experiments are needed to determine how nutrient and organic carbon loading changes the composition and/or dynamics of the coral-associated microbial communities. Resolving this distinction will advance greatly our understanding of the complex and dynamic changes occurring in coral communities today.

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