Macroecological relationships between coral species' traits and disease potential

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Abstract

Coral disease is a growing problem for reef corals and a primary driver of reef degradation. Incidences of coral disease on the Great Barrier Reef (GBR) are rapidly increasing; however, our understanding of differences among species in their potential for contracting disease is poor. Here, we integrate observations of coral disease on the GBR from the primary literature in order to identify the morphological, ecological, and biogeographical traits of coral species that influence a species' "disease potential." Most of the examined traits influenced species' disease potential when considered alone; however, a master analysis finds that the diversity of predators, geographical range size, and characteristic local abundance are the primary predictors. Observational biases associated with species local abundance and phylogeny are tested but do not overpower relationships. This large-scale macroecological evaluation of coral disease provides important insights into species-level traits that drive disease susceptibility globally.

Keywords: Reef corals, disease potential, species traits, macroecology, Great Barrier Reef, predation

Introduction

Coral reefs are a productive and economically important, yet highly threatened, global ecosystem (Lesser et al. 2007; Mumby and Steneck 2008). Studies reveal declines of 20% and 5% in coral cover per decade in the Caribbean (Gardner et al. 2003) and the Indo-Pacific (Bruno and Selig 2007), respectively. Projections suggest that up to 30% of reefs worldwide will be severely damaged by 2030 (Hughes et al. 2003). The ubiquity of reef decline over the last millennium has led to the conclusion that no pristine reefs remain (Pandolfi et al. 2003). Causes of coral decline are numerous, including climate change (predominantly increasing sea water temperature), overfishing and destructive fishing techniques, coral bleaching, coral disease, trophic level dysfunction (phase shifts), predation, and pollution (Lesser 2004; Mumby and Steneck 2008; Fabricius 2005). Of these threats, coral disease is now considered a key driver of decline for corals and the reefs they build (Harvell et al. 1999); a problem that is aggravated by environmental stressors caused by human-induced change (Jones et al. 2004; Selig et al. 2006; Bruno et al. 2007; Francini-Filho et al. 2008).

A recent review found 19 described coral diseases, four of which have a worldwide distribution, nine of which are found only in the Caribbean, and six of which are endemic to the Indo-Pacific region (Sutherland et al. 2004). Although the Caribbean is considered the coral disease hotspot, there have been increasing reports of disease on the Great Barrier Reef (GBR) (Antonius and Lipscomb 2001; Willis et al. 2004).

However, individual reports are disease based and typically focus on few coral species and at specific geographic locations, and therefore our understanding of disease susceptibility at broader scales and across all species is limited. One exception is a study by Willis et al. (2004) in which disease observations were conducted up and down the GBR providing an assessment of reef condition through time. However, studies to date have stopped short of assessing the ecological, morphological, and environmental characteristics of coral species that influence their potential for infection. Therefore, in this study we integrate the current state of knowledge about coral disease on the GBR and paint a macroecological picture of the species-level traits that are likely to influence a species' potential to contract disease. Given the heterogeneous data collected in coral disease studies (explained further in the methods) and subsequent lack of sampling standardized disease prevalence data, we define "disease potential" simply as whether or not a scleractinian coral species has been observed in the primary literature with a disease. We select a broad range of species-level traits based on expected relationships with disease, and include ecological (local abundance, number of predators, range size), morphological (corallite size, growth form complexity), and environmental (wave exposure, water clarity, depth range) information.

Similar to human populations, coral species living at higher local abundances have been shown to be more susceptible to disease (Willis et al. 2004; Page and Willis 2008). For example, there has been an increase in the prevalence of the coral disease white syndrome in areas where coral cover is greater (Willis et al. 2004). The underlying mechanism is that disease can spread more readily within crowded populations, suggesting that locally common species have a greater disease potential

than rare species (Aeby and Santavy 2006). A potential vector for disease transmission that is exacerbated in dense populations is coral predation. Predators act as vectors by oral or fecal transmission of pathogens (Aeby and Santavy 2006; Rotjan and Lewis 2008). Some diseases, such as black band disease, flourish in the presence of corallivorous fishes that are suspected to increase the rate at which the disease is spread from infected to non-infected colonies (Aeby and Santavy 2006; Page and Willis 2006). Predators also increase disease potential by making scars in corals that allow pathogens to penetrate and infect tissues (Page and Willis 2008). Certain diseases (such as skeletal eroding band) require a tissue lesion in order to infect corals (Page and Willis 2008). For example, lesions may be driving the positive association between coral polyp-eating chaetodontids and disease prevalence (Raymundo et al. 2009).

Coral polyp size is a morphological characteristic that is related to coral predation and may also be related to disease potential. For instance, most corallivorous fishes belong to the families Chaetodontidae (butterflyfishes) and Labridae (wrasses) that have smaller mouths often specialized for the removal of tentacles from individual polyps (Aeby and Santavy 2006). Colony growth form is another important characteristic that is related to energy allocation among physiological processes such as growth and colony defence. For instance, branching corals invest more energy into growth and therefore allocate less energy to maintenance and disease resistance (Jackson 1979; Palmer et al. 2008). Mass transfer also tends to be reduced in more complex branching morphologies (Chamberlain 1978; Nakamura and van Woesik 2001) where lower internal water velocities slow or stop mucus and sediment shedding and increase the chance of pathogen infection. Complex colony morphology

is also related with a higher degree of physiological integration between the polyps (Soong and Lang 1992) that is likely to increase the probability of disease spread within colonies.

Hydrodynamic exposure levels associated with coral species' preferred habitats are also expected to influence the potential for corals to contract disease for a similar reason to mass transfer within colonies (i.e., the flushing of accumulated materials potentially containing pathogens). Indeed, disease prevalence in the genus *Acropora* is found to be greater in areas of the reef that are sheltered from wave action, and lesser in exposed habitats such as the reef crest (Willis et al. 2004). Coral disease prevalence also increases with levels of nutrients and sedimentation generated by terrestrial run-off and exacerbated by human activities such as coastal development, deforestation and agriculture (Bruno et al. 2003; Francini-Filho et al. 2008). Some coral species have adaptations to cope with these conditions, including polyp retraction, lowered photosynthetic (and thus metabolic) rates, and increased mucus production (Sofonia and Anthony 2008; Lirman and Manzello 2009), each of which might decrease disease infection rates in turbid water species. Therefore, as terrestrial influences spread further from land, corals without such adaptations (e.g., clear water dwelling outer reef colonies) are expected to have a greater disease potential.

Several other species traits are hypothesised to be important for coral disease potential, but have not yet been widely tested. First, a species with a broader geographical distribution is more likely to intercept disease and subsequently spread it to other parts of its distribution via vectors such as predators (especially if a disease and/or disease vector is strictly associated with one or few coral species). Second,

several studies have shown that high temperatures increase the prevalence of disease (Jones et al. 2004; Selig et al. 2006; Bruno et al. 2007). The upper depth limit of coral species is directly associated with water temperature (Kuta and Richardson 1996), and therefore species with higher upper depth limits (closer to the ocean surface) are expected to have a greater potential for disease contraction than deeper dwelling species.

This study tests for associations between disease observations in the literature and morphological, ecological, and geographical characteristics of coral species by compiling a dataset of species-level traits for all known species in the GBR region. The outcome is the first ever macroecological trait-based analysis of disease potential, which provides important insights about coral traits that drive disease susceptibility globally.

Methods

Species-level traits

Coral species-level trait information was collected for all known (406) scleractinian coral species found on the GBR. Species and family data were assigned according to Veron and Stafford-Smith (2002), Wallace (1999), and Carpenter et al. (2008). For each species, nine different traits were recorded. Characteristic local abundance was assigned as rare, uncommon, or common (following Veron and Stafford-Smith 2002; Carpenter et al. 2008), which is a general metric of local abundance developed by Veron based on comprehensive ecological and taxonomic information throughout the GBR region. Given the low number of rare species, rare and uncommon categories

were grouped resulting in two local abundance classes: common and uncommon. Geographical range size was calculated as the global area occupied by a species found on the GBR measured as number of map pixels (Veron and Stafford-Smith 2002). Colony growth form was assigned following descriptions and pictures from Wallace (1999) and Veron and Stafford-Smith (2002) as either solitary, encrusting, massive, columnar, foliaceous, digitate, branching, tabulate, or corymbose. Corallite size was obtained from a variety of taxonomic monographs (Veron and Pichon 1976, 1980; Veron et al. 1977) or measured directly from scaled pictures (Veron and Stafford-Smith 2002). For the genus *Acropora*, the corallite size information was obtained from Wallace (1999). Wave exposure was assigned as protected, exposed, or broad (protected and exposed) (Veron and Stafford-Smith 2002). Preferred water clarity was assigned as either turbid, clear, or both (turbid and clear) (Veron and Stafford-Smith 2002). The upper (shallowest) depth at which the coral is found was obtained from Carpenter et al. (2008). The number of predatory species was determined by an extensive review of the primary literature for corallivorous animals on the GBR and the coral species they have been observed to prey upon (361 species-level observations).

Coral disease observations

A total of 210 species-specific disease observations (for 95 species) were found in the primary literature that included nine disease types (white syndrome, white patch, pigmentation response, tumours, black band disease, skeletal eroding band, brown band, atramentous necrosis, or unidentified pathologies). Studies spanned the GBR, but were undertaken with a diverse range of objectives and tended to focus on diseases rather than details about their coral hosts (see Supplemental Material, Table).

For example, twice as many disease observations were found in the literature (>400), but coral hosts were not described sufficiently for species-level analyses (i.e., they were recorded at higher taxonomic levels or by growth form). Furthermore, coral disease studies almost exclusively reported presence-only information, thereby preventing the extraction of more robust sampling standardized measures such as disease prevalence. The several studies that did report disease prevalence information focused on single coral diseases, which limited the generality for all disease types. There were too few studies about GBR coral disease to standardise disease observations based on the frequency at which a species is reported in the literature. Finally, observation methodologies differed dramatically among studies, ranging from sampling replicate belt transects of known area through to targeted a certain number of colonies with a given disease type.

Given the data at hand, it was not possible to look at species' disease susceptibility using sampling standardized measures of prevalence. Therefore, to improve analytical power and use all the available information disease, data were grouped so that a species observed to have any disease anywhere on the GBR was categorized as having the "potential" to contract disease (i.e., a binomial response variable). This definition of disease potential assumes that the literature reflects species' vulnerability to disease in the field, and that species not observed with disease have a lower potential for infection (but not necessarily zero potential). This assumption results in the chance of underestimating the actual potential for species that are poorly studied, are locally rare, or have restricted geographic distributions (type II errors), which we test for by contrasting disease patterns for locally common and uncommon species separately (below).

Data analysis

Relationships between coral species characteristics and disease potential were analysed: (1) separately, to gain a better understanding of patterns of disease potential within traits, (2) altogether using a generalized linear mixed-effects model (GLMM), to determine which traits are the best predictors of disease potential, and (3) in two separate GLMM models for common and uncommon species, and once again with species from the common and well-studied family Acroporidae removed to assess for observational biases in the literature.

Separate trait analyses assessed if a given categorical grouping of species (e.g., all species with a "branching" growth form) had on average a greater potential to contract disease than all other species groupings. Exact binomial tests are used to determine if average disease susceptibility for a given grouping was significantly different to that of all other groups. Results are presented as the proportional difference between focal groupings relative to all other groupings (i.e., bars above zero indicate that a group is more susceptible on average, and vice versa). 95% confidence intervals illustrate any overlap with zero ("no difference"). Statistical differences are indicated for each focal group using stars (* = p-value < 0.05; ** = p-value < 0.01; *** = p-value < 0.001; ns = not significant). Logistic regression is used for analysis of the continuous predictor variables (e.g., geographical range size). Best-fit logistic estimates are presented, because overlapping points conceal underlying trends.

GLMMs allow for (1) both continuous and categorical predictor variables, (2) a non-linear response variable (Venables and Ripley 2002), and (3) the removal of group effects by making family a random variable (Zuur et al. 2009). This latter feature is accomplished by removing variation due to differences among families from the error term and allowing families to vary randomly around the overall mean (Pinheiro and Bates 2000; Krackow and Tkadlec 2001). Other independent variables (fixed effects) can then be examined, and any significant results can then be generalized for all species (Pinheiro and Bates 2000; Jovani and Serrano 2001). Disease potential was considered to have binomial distributions of errors (observed in the literature with disease = 1, not observed with disease = 0; these data were examined using a logit link function). The GLMM was run with the function glmmML from the R package glmmML (Broström 2009; R Development Core Team). To determine the best predictive model for disease potential, we compared the full model with models in which one of the predictor variables are dropped (using the "drop1" function in the R base statistics distribution). If an analysis of variance found a dropped variable to have no significant effect on the model, then the variable was left out. Interactions (up to two-way) were examined and dropped in the same fashion.

The analyses suggest that coral species that are characteristically locally common are significantly more susceptible to disease than are uncommon species (Fig. 1a). Despite possible biological explanations for this result (see Discussion), it raises questions about observational biases. For example, there is a higher probability of observing a common coral species on the reef, and subsequently a higher probability of observing disease in common species. Also, researchers tend to focus on locally abundant species, because they are easier to locate in the field. Furthermore, despite

the use of a GLMM to reduce phylogenetic biases, there were concerns that the large number of species in the family Acroporidae might overwhelm trait / disease relationships. Therefore, to test that either "species commonness" or "belonging to the family Acroporidae" are not driving disease patterns, the separate trait and GLMM analyses were repeated for either common or uncommon species (alone), as well as with Acroporidae removed. Any substantial divergences in disease susceptibility / trait analysis patterns between full and partial analyses are reported in the results.

Results

Families

Family groupings exhibited a large spread of disease susceptibilities, ranging from ~20% less than to ~20% greater on average than the expected disease potential based on the total species mean (Fig. 2). However, the majority of differences were not significant. Acroporidae is the only family with a greater potential to contract disease. Conversely, Fungiidae and Agariciidae are the only families significantly less susceptible. Locally common families such as Pocilloporidae and Faviidae appear to have a greater disease potential on average, while the common family Poritidae appears to be less susceptible. However, these differences cannot be statistically distinguished from the total species pool. Test for observation biases show no differences in pattern nor in statistical significance; except when the Acroporidae are removed, and the Faviidae exhibit a marginally significant disease potential.

Coral predators

Corals have a wide range of predators noted in the literature, including 15 families of corallivorous fishes and invertebrates (46 species), which prey on 11 coral families, comprising of more than 66 coral species. The dominant fish predators, Chaetodontidae and Scaridae, prey on 65 and 4 coral species, respectively. The dominant invertebrate predators, the crown-of-thorns starfish (*Acanthaster planci*) and the gastropod *Drupella sp.,* targeted 8 and 4 coral genera, respectively. There was a marked relationship between the number of predator species that eat coral species and disease potential ($p < 0.001$; Fig. 3a). All coral species observed with more than ~3 predators have been observed with a disease regardless of their characteristic local abundance or taxonomic affinities. However, having fewer or no predators does not necessarily imply that a coral species does not have the potential to contract disease.

Geographical range size and habitat preference

Logistic regression of disease potential as a function of geographical range size illustrates that coral species with larger ranges tend to be much more prone to disease than those with more restricted distributions (Fig. 3b). Species that prefer protected reef areas are found to be ~40% less susceptible to disease than species that prefer exposed environments or are found in a wide range of habitats (protected and exposed). Protected habitat reef species have a significantly lower disease potential, whereas the other two groups have a significantly greater potential (Fig. 1c). Clear water dwelling species are ~ 30% (and significantly) more likely to contract disease than turbid water dwelling species and than species found in both turbid and clear environments (Fig. 1d). Finally, species with shallower upper depth limits appear to have a greater disease potential than species preferring deeper waters (Fig. 3d). The

pattern for upper depth became highly significant when Acroporidae was removed, as well as insignificant when analysing uncommon species alone, suggesting a strong observational bias (e.g., shallower species are intercepted more by researchers).

Growth form and polyp diameter

There was a general increase in disease potential as a function of the morphological complexity (Fig. 1b). Robust and simple forms such as massive and encrusting have an up to ~17% reduced potential to contract disease. Whereas, intermediate morphologies such as columnar, and foliaceous almost show no difference or are not significantly different from the proportion expected for all species. Branching and corymbose forms, which are delicate and complex, have significantly higher disease potentials than the average of all the other growth forms. Finally, solitary corals that are generally one large polyp tend to have a very low potential for disease infection. When analysing uncommon species alone, corymbose species were the only morphological group with a significantly higher potential to contract disease. When Acroporidae was removed only solitary species were found to have a significantly low disease potential and branching species are marginally insignificant, suggesting that the morphologically complex species that dominate *Acropora* are driving the pattern for all species. A negative relationship was found between polyp diameter and disease potential (Fig. 3c), where colonies with larger polyps have a significantly lower potential for disease contraction.

Generalised linear mixed-effects model

The GLMM illustrates that the most important predictors of coral species disease potential are the number of geographical range size and corallivorous predator species

(Table 1). Slightly less important is characteristic local abundance. All other species' traits (and any interactions among traits) did not significantly alter the predictive power of the model when they were removed. That is, species growth form, polyp size, upper depth limit, water clarity, and hydrodynamic exposure are not important predictors of disease potential when compared with the predictive power of geographical range size and number of coral predator species. Separate analyses for common and uncommon species uncovered the same pattern as for the full model; although, marginal changes occurred in the significance levels for range size and predation. Finally, the analysis following the removal the family Acroporidae also showed a similar pattern as the full model; however, local abundance becomes a non-significant predictor, and thus strengthening the key finding that geographical range size and predation are driving disease potential.

Discussion

The coral species traits collected and analysed in this study each displayed clear and significant trends with respect to disease potential when analysed separately (Figs. 1-3). In general, species observed in the literature as having fewer predators, restricted geographical ranges, lower abundances, simple growth forms, larger corallites, and a preference for protected, turbid and deeper reef habitats tend to be observed with disease less frequently than species that have more predators, broad geographical ranges, high abundances, complex growth forms, smaller corallites, and a preference for exposed, clear and shallower reef habitats. However, when all traits are analysed together, the geographical range size, the number of predatory species, and characteristic local abundance were consistently the most significant predictors of a coral species' potential to contract disease (Fig. 4).

Locally common species have a higher potential to contract disease than uncommon species (Fig. 1a). This conspicuous result prompted the investigation of possible observational biases associated with sampling scientific literature (i.e., a higher probability of observing common species within a sampling area). Distinguishing between observation biases and a genuine biological signal is not possible without a standardised sampling approach from the literature (which is not possible given limitations of the disease literature; see Methods). Nonetheless, the GLMM and separate trait analyses show no substantial changes in either pattern or statistical significance when characteristically common or uncommon were analysed separately. This result suggests that species' abundance does not interact substantially with most traits, and subsequent trait / disease potential patterns are presumably genuine macroecological signals. Furthermore, several studies have found that population density is an important driver of disease susceptibility (Willis et al. 2004; Bruno et al. 2007), which is likely facilitated by increased rates of disease transmission as a result of diminished distances between individual colonies of the same species. Host density can also be related to vector abundance (e.g., coral predators; Bruno et al. 2007; Raymundo et al. 2009). The allocation of resources to competition in more densely populated reefs can also render species more vulnerable to pathogen transmission and infection (Bruno et al. 2007). For example, there is a distinct relationship between coral cover and disease incidence for white syndrome (Bruno et al. 2007); whereas, coral cover has no effect on disease prevalence for black band disease (Page and Willis 2006). Despite these purported differences in disease transmission within coral assemblages, the results presented here suggest that, as a

whole, local abundance is a significant predictor of a species' potential to contract disease.

Our analyses of coral families corroborate reports that members of the family Acroporidae are more likely to contract disease on the GBR (Willis et al. 2004; Ulstrup et al. 2007; Page and Willis 2008). Despite this higher susceptibility and the shear number of species in Acroporidae, trait /disease potential patterns did not change appreciably when species from Acroporidae were removed from analyses; indicating that this family is not driving the study's broader results. Other families were no different or less susceptible to disease than expected (Fig. 2). The susceptibility of Acroporidae species may explain the observed declines in coral communities since acroporids dominate GBR reefs and are found in almost every reef habitat, making them one of the most important reef builder (Wallace 1999; Willis et al. 2004; Page and Willis 2008). Furthermore, the two species of *Acropora* found in the Caribbean (*A. palmata* and *A. cervicornis*) were almost driven to extinction by disease in the 1990s (Richardson 1998). Page and Willis (2008), however, found pocilloporids to be more susceptible to disease than acroporids, concluding that pathogenic microorganisms disproportionately target faster growing corals and not necessarily the most spatially dominant species. While our regional-scale analysis finds that pocilloporids have a higher disease potential on average, this proportion is not significantly different to expectation due to this family's small number of species. Previous work has also found that species from the families Poritidae and Faviidae have a reduced disease potential on average (Willis et al. 2004; Page and Willis 2008); however, our analyses suggest that their potential to contract disease is not significantly different from expectation. Interestingly, the family Fungiidae has a

significantly reduced potential to contract disease compared with other families. Fungiids are typically solitary, single-polyp forms, and therefore are likely to invest in disease resistance strategies to defend this single polyp. Additionally, this family does not have any reports of predators, which we found to be the primary trait driver of disease potential.

Geographical range size emerges as a highly significant factor in determining disease potential, where more widespread species are expected to be more susceptible to disease in general (Fig. 4). Most species range more widely than the study's coverage (the GBR, approximately 200 map pixels) and study locations in the literature (typically the same research stations and surrounding reefs), indicating that this result is not a geographical sampling bias. One explanation is that greater ranging species have a higher probability of intercepting pathogens and passing them to con-specifics. However, because most coral diseases are not host-specific, they can be passed readily from broader to narrower ranging species. A more plausible explanation is that predators tend to target broader ranging coral species in order to increase the probability of having local resources following dispersal. Indeed, there is a positive triangular relationship between the number of coral predator species and geographical range size (Fig. 5), suggesting that coral predation is likely to be the primary causal driver because it tends to operate increasingly on broader ranging coral species.

The separate analysis of species growth form here confirms the current hypothesis that massive and encrusting corals have a reduced potential to contract disease. Simple growth forms have more effective mass transfer, by which toxic metabolites produced by the pathogenic microorganism can be easily expelled from the polyp and

water flux subsequently removes pathogens and metabolites lingering on the surface (Nakamura and van Woesik 2001). It has been reported that the pathogens responsible for black band disease are usually found in sediments within the crevices and folds of coral colonies (Richardson 1998) making more complex morphologies more prone to sediment accumulation, and therefore more prone to infection (e.g., branching and intricate colony patterns). Complex morphologies also tend to be associated with faster growing species that spend more energy on growth and less on defence mechanisms (e.g., branching acroporids; Palmer et al. 2008). Meanwhile, slow growing, simpler forms tend to invest more energy in the implementation of stronger defence (e.g., production of melanin, phenoloxidase and special proteins; Palmer et al. 2008) and regeneration systems since they cannot recover as quickly from partial colony mortality (Jackson 1979). Finally, corals with these complex growth forms (especially acroporids) tend to have higher colony integration than massive corals, where polyps are physiologically independent (Soong and Lang 1992). This is hypothesised to affect bleaching susceptibility (Baird and Marshall 2002) and could also act as a mechanism for pathogen transmission within the colony. Despite the plausible mechanisms for greater disease potential in more complex growth forms, the GLMM presented here suggests that growth form is not a dominant predictor when compared with coral predation and geographic range size.

When all traits were analysed together, polyp diameter was found not to be an important driver relative to other species-level traits. Nonetheless, when analysed separately, a species' polyp diameter also shows a clear association with disease susceptibility, where smaller corallite species tend to be more susceptible to disease (Fig. 3c), and might explain why acroporids and pocilloporids are particularly prone

to infection. Smaller corallites infer higher polyp densities for a given colony surface area, and therefore each polyp is likely to have relatively less protection from disease. Meanwhile, colonies with larger polyps and lower densities per unit surface area might be more resistant, because each polyp contributes in greater proportion to the colony's energy budget. Larger corallites also appear to be less targeted by coral predators, which are hypothesized to be a primary transmission vector of coral diseases (Aeby and Santavy 2006; Raymundo et al. 2009); and there is evidence that the scars left by *A. planci* on the coral tissue might promote the transmission of some coral disease (Nugues and Bak 2009). The most common predators (from reef fish families Chaetodontidae, Labridae, and Monacanthidae) have small mouths specifically designed for removing tissue from individual polyps, and therefore specifically target colonies with small corallites (Rotjan and Lewis 2008). A recent study found that chaetodontids are the only fish family to be significantly and positively related with coral disease prevalence, supporting the hypothesis that they act as vector of disease (Raymundo et al. 2009). Moreover, chaetodontids have been found to specifically target diseased tissue; decreasing the subsequent disease spread within colonies, but illustrating the potential for the fishes to interact with disease and act as vectors (Cole et al. 2009).

Although the GLMM found that habitat preferences are not significant, relative to other traits, coral species preferring turbid, protected reef habitats, and/or greater depths tend to be less likely to be observed with a coral disease when analysed individually (Fig. 3a and b). This result might be because these species are already adapted for living in environments where pathogen levels are higher and sediment-shedding water motion is slower. It is hypothesized that corals living in turbid water

are less sensitive to sedimentation and have mechanisms for dealing with excess sediment loads that might contain disease pathogens, decreasing infection rates (Lirman and Manzello 2009). On the other hand the increased disease potential in species that prefer clear water and/or high wave exposure might be explained by the fact that they do not have the necessary adaptations to cope with increasing levels of pathogens in their environment. It is suggested that the majority of disease-causing pathogens are of terrestrial origin, either from terrestrial sediments or are coliforms coming from fecal contaminated waters that are dumped into the oceans without proper treatment (Frias-Lopez et al. 2003; Lesser et al. 2007, Francini-Filho et al. 2008). Depth showed no significant effects on disease potential in the GLMM, but when analysed individually it had a significant effect. Studies have suggested that depth could potentially be an important variable because shallower waters tend to be warmer, which increases the spread and degree of virulence of the disease/pathogens (Jones et al. 2004; Selig et al. 2006; Bruno et al. 2007). Some diseases such as black band disease are more prevalent in shallower environments (<6m; Page and Willis 2006), and so the grouping of disease types in our analyses would have obscured such patterns. Overall, habitat preference was not found to be an important driver of general disease susceptibility in corals on the GBR.

A lack of understanding about the causes of coral disease has hampered its management and prevented the detection of mechanistic links between disease occurrence and environmental perturbation (Work et al. 2008). Our species-specific trait-based analysis is an important step towards better understanding disease potential, and could be used to support conservation initiatives by highlighting species-level characteristics that render corals more prone to disease infection, as well

as providing insights into modelling community vulnerability. Given the general lack of coral taxonomic resolution and disease prevalence metrics reported in the disease literature, and our subsequent grouping of diseases to increase statistical power, our analyses were not able to tease apart disease-specific patterns. Improved taxonomic resolution and the integration of disease etiologies and ecologies and coral defence mechanisms (immune response) into our macroecological framework would further improve our understanding of coral disease ecology. It would also be important to take a closer look at the phylogenetic component of the data, to see if there is a major effect or if species are indeed independent units of study. Preliminary Phylogentically Independent Contrasts (PIC) analyses show that the patterns found are true macroecological signals that are not been driven by phylogeny.

Reference list

Aeby G, Santavy D (2006) Factors affecting susceptibility of the coral *Montastraea faveolata* to black-band disease. Mar Ecol Prog Ser 318: 103-110

Antonius A, Lipscomb D (2001) First protozoan coral-killer identified in the Indo-Pacific. Atoll Res Bull 481: 1-21

Baird AH, Marshall PA (2002) Mortality, growth and reproduction in scleractinian corals following bleaching on the Great Barrier Reef. Mar Ecol Prog Ser 237: 133-141

Broström G (2009) glmmML: Generalized linear models with clustering: a maximum likelihood and bootstrap approach to mixed models. R Development Core Team

Bruno J, Petes L, Harvell D, Hettinger A (2003) Nutrient enrichment can increase the severity of coral diseases. Ecol Lett 6: 1056-1061

Bruno J, Selig E (2007) Regional decline of coral cover in the Indo-Pacific: timing, extent, and subregional comparisons. PLoS ONE, 8: e711, 1-8

Bruno J, Selig E, Casey K, Page C, Willis B, Harvell D, Sweatman H, Melendy A (2007) Thermal stress and coral cover as drivers of coral disease outbreaks. PLoS Biology 5: e124, 1220-1227

Carpenter E, Abrar M, Aeby G, Aronson R, Banks S, Bruckner A, Chiriboga A, Cortés J, Delbeek C, DeVantier L, Edgar G, Edwards A, Fenner D, Guzmán H, Hoeksema B, Hodgson G, Johan O, Licuanan W, Livingstone S, Lovell E, Moore J, Obura D, Ochavillo D, Polidoro B, Precht W, Quibilan M, Reboton C, Richards Z, Rogers A, Sanciangco J, Sheppard A, Sheppard C, Smith J, Stuart S, Turak E, Veron J, Wallace C, Weil E, Wood E (2008) One-third of reef -building corals face elevate extinction risk from climate change and local impacts. Science 321: 560-563

Chamberlain JA (1978) Mechanical properties of coral skeleton: compressive strength and its adaptive significance. Paleobiology 4: 419-435

Cole AJ, Chong Seng KM, Pratchett MS, Jones GP (2009) Coral-feeding fishes slow progression of black band disease. Coral Reefs 28: 965

Fabricius KE (2005) Effects of terrestrial runoff on the ecology of corals and coral reefs: reviews and synthesis. Mar Pollut Bull 50: 125-146

Francini-Filho R, Moura R, Thompson F, Reis R, Kaufman L, Kikuchi R, Leão Z (2008) Diseases leading to accelerated decline of reef corals in the largest South Atlantic reef complex (Albrolhos Bank, eastern Brazil). Mar Pollut Bull 56: 1008-1014

Frias-Lopez J, Bonheyo G, Fouke B (2003) Cyanobacteria associated with coral black band disease in Caribbean and Indo-Pacific reefs. Appl Environ Microbiol 69: 2409-2413

Gardner T, Coté I, Gill J, Grant A, Watkinson A (2003) Long-term region-wide declines in Caribbean corals. Science 301: 958-960

Harvell CD, Kim K, Burkholder JM, Colwell R, Epstein P, Grimes DJ, Hofman EE, Lipp EK, Osterhaus DME, Overstreet RM, Porter JW, Smith GW, Vasta GR (1999) Emerging marine diseases – climate links and anthropogenic factors. Science 285: 1505-1510

Hughes T, Baird A, Bellwood D, Card M, Connolly S, Folke C, Grosberg R, Hoegh-Guldberg O, Jackson BC, Kleypas J, Lough JM, Marshall P, Nyström M, Palumbi SR, Pandolfi JM, Rosen B, Roughgarden J (2003) Climate change, human impacts, and the resilience of coral reefs. Science 301: 929-933

Jackson JBC (1979) Morphological strategies of sessile animals. In: Larwood G, Rosen BR (eds) Biology and systematics of colonial organisms. Academic Press, New York, pp499-555
Jones R, Bowyer J, Hoegh-Guldberg O, Blackall L (2004) Dynamics of a temperature-related coral disease outbreak. Mar Ecol Prog Ser 281: 63-77
Jovani R, Serrano D (2001) Feather mites (*Astigmata*) avoid moulting wing feathers of passerine birds. Anim Behav 62: 723-727
Krackow S, Tkadlec E (2001) Analysis of brood sex ratios: implications of offspring clustering. Behav Ecol Sociobiol 50: 293-301
Kuta KG, Richardson L (1996) Abundance and distribution of black band disease on coral reefs in the northern Florida Keys. Coral Reefs 15: 219-223
Lesser M (2004) Experimental biology of coral reef ecosystems. J Exp Mar Biol Ecol 300: 217-252
Lesser M, Bythell J, Gates J, Johnstone R, Hoegh-Guldberg O (2007) Are infectious diseases really killing corals? Alternative interpretations of the experimental and ecological data. J Exp Mar Biol Ecol 346: 36-44
Lirman D, Manzello D (2009) Patterns of resistance and resilience of the stress-tolerant coral *Siderastrea radians* (Pallas) to sub-optimal salinity and sediment burial. J Exp Mar Biol Ecol 369: 72-77
Mumby P, Steneck R (2008) Coral reef management and conservation in light of rapidly evolving ecological paradigms. Trends Ecol Evol 23: 555-563
Nakamura T, van Woesik R (2001) Water-flow rates and passive diffusion partially explain differential survival of corals during the 1998 bleaching event. Mar Ecol Prog Ser 212: 301-304
Nugues MM, Bak RP (2009) Brown-band syndrome on feeding scars of the crown-of-thorn starfish *Acanthaster planci*. Coral Reefs 28: 507-510
Page C, Willis B (2006) Distribution, host range and large-scale spatial variability in black band disease prevalence on the Great Barrier Reef, Australia. Dis Aquat Org 69: 41-51
Page C, Willis B (2008) Epidemiology of skeletal eroding band on the Great Barrier Reef and the role of injury in the initiation if this widespread coral disease. Coral Reefs 27: 257-272
Palmer CV, Mydlarz LD, Willis B (2008) Evidence of an inflammatory-like response in non-normally pigmented tissues of two scleractinian corals. Proc R Soc Lond B Biol Sci 275: 2687-2693
Pandolfi J, Bradbury R, Sala E, Hughes T, Bjorndal K, Cooke R, McArdle D, McClenachan L, Newman M, Paredes G, Warner R, Jackson J (2003) Global trajectories of the long-term decline of coral reef ecosystems. Science 301: 955-958
Pinheiro J, Bates D (2000) Mixed-Effects Models in S and S-P*LUS*. Springer, New York
Raymundo LJ, Halford AR, Maypa AP, Kerr AM (2009) Functionally diverse reef fish communities ameliorate coral disease. Proc Natl Acad Sci USA 106: 17067-17070
Richardson L (1998) Coral diseases: what is really known? Trends Ecol Evol 13: 438-443
Rotjan R, Lewis S (2008) Impact of coral predators on tropical reefs. Mar Ecol Prog Ser 367: 73-91
Selig E, Harvell D, Bruno J, Willis B, Page C, Casey K, Sweatman H (2006) Analyzing the relationships between ocean temperature anomalies and coral

disease outbreaks at broad spatial scales. In: Phinney J, Hoegh-Guldberg O, Kleypas J, Skirving W, Strong A (eds) Coral reefs and climate change: science and management. American Geophysical Union, Washington, DC. pp 111-128

Sofonia J, Anthony K (2008) High- sediment tolerance in reef coral *Turbinaria mesenterina* from the inner Great Barrier Reef lagoon (Australia). Estuar Coast Shelf Sci 78: 748-752

Soong K, Lang JC (1992) Reproductive integration in reef corals. Biol Bull 183: 418-431

Sutherland K, Porter J, Torres C (2004) Disease and immunity in Caribbean and Indo-Pacific zooxanthellate corals. Mar Ecol Prog Ser 266: 273-302

Ulstrup K, Kühl M, Bourne D (2007) Zooxanthellae harvested by ciliates associated with brown band syndrome of corals remain photosynthetically competent. Appl Environ Microbiol 73: 1968-1975

Venables WN, Ripley BD (2002) Modern applied statistics with S. Fourth Edition. Springer, New York

Veron JEN, Pichon M (1976) Scleractinia of eastern Australia. Part I. Families Thamnasteriidae, Astrocoeniidae, Pocilloporidae. Australian Government Publishing Service, Canberra

Veron JEN, Pichon M (1980) Scleractinia of eastern Australia. Part III. Families Agariciidae,Siderastreidae, Fungiidae, Oculinidae, Merulinidae, Mussidae, Pectiniidae, Caryophyllidae, Dendrophyliidae. Australian Institute of Marine Science in association with Australian National University Press, Canberra

Veron JEN, Stafford-Smith M (2002) Coral ID. Australian Institute of Marine Science and CCR Qld Pty Ltd

Veron JEN, Pichon M, Wijsman-Best M (1977) Scleractinia of eastern Australia. Part II. Families Faviidae, Trachyphylliidae. Australian Government Publishing Service, Canberra

Wallace C (1999) Staghorn corals of the world: a revision of the genus *Acropora*. CSIRO Publishing, Collingwood

Willis B, Page C, Dinsdale E (2004) Coral disease on the Great Barrier Reef. In: Rosenberg E, Loya Y (eds) Coral health and disease. Springer-Verlag, Berlin, pp. 69-104

Work T, Richardson L, Reynolds T, Willis B (2008) Biomedical and veterinary science can increase our understanding of coral disease. J Exp Mar Biol Ecol 362: 63-70

Zuur AF, Ieno EN, Walker NJ, Saveliev AA, Smith GM (2009) Mixed Effects Models and Extensions in Ecology with R. Springer, New York

Table 1. Generalized linear mixed-effects model (GLMM) for disease potential (family as random variable; n = 406). See text for details.

	Master analysis			**Common only**			**Uncommon only**			**Acroporidae removed**		
Parameter	*Est*	*SE*	*p-val*	*Est*	*SE*	*p-val*	*Est*	*SE*	*p-val*	*Est*	*SE*	*p-val*
(Intercept)	-5.198	0.969	0.000 ***	-5.258	1.412	0.000 ***	-5.770	1.166	0.000 ***	-7.050	1.664	0.000 ***
Geographical range size	0.000	0.000	0.000 ***	0.000	0.000	0.002 **	0.000	0.000	0.002 **	0.000	0.000	0.000 ***
Local abundance uncommon	-1.082	0.342	0.001 **	NA	NA	NA	NA	NA	NA	-0.801	0.476	0.092 ns
Predation	2.122	0.411	0.000 ***	2.162	0.490	0.000 ***	2.00	0.781	0.010 *	2.131	0.507	0.000 ***

Figures

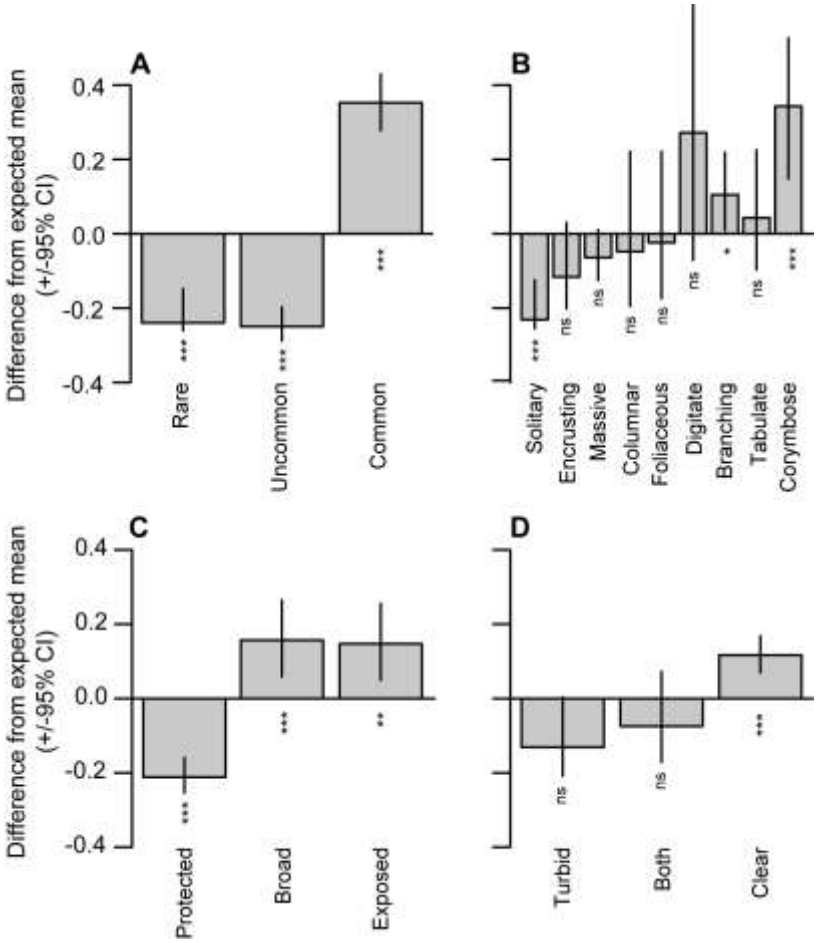

Figure 1. Categorical proportions of disease observation relative to expectation for species' (a) local abundance, (b) growth form, (c) wave exposure, and (d) water clarity. 95% binomial confidence intervals and significance levels show if proportions can be distinguished from expectation. See Methods for further details.

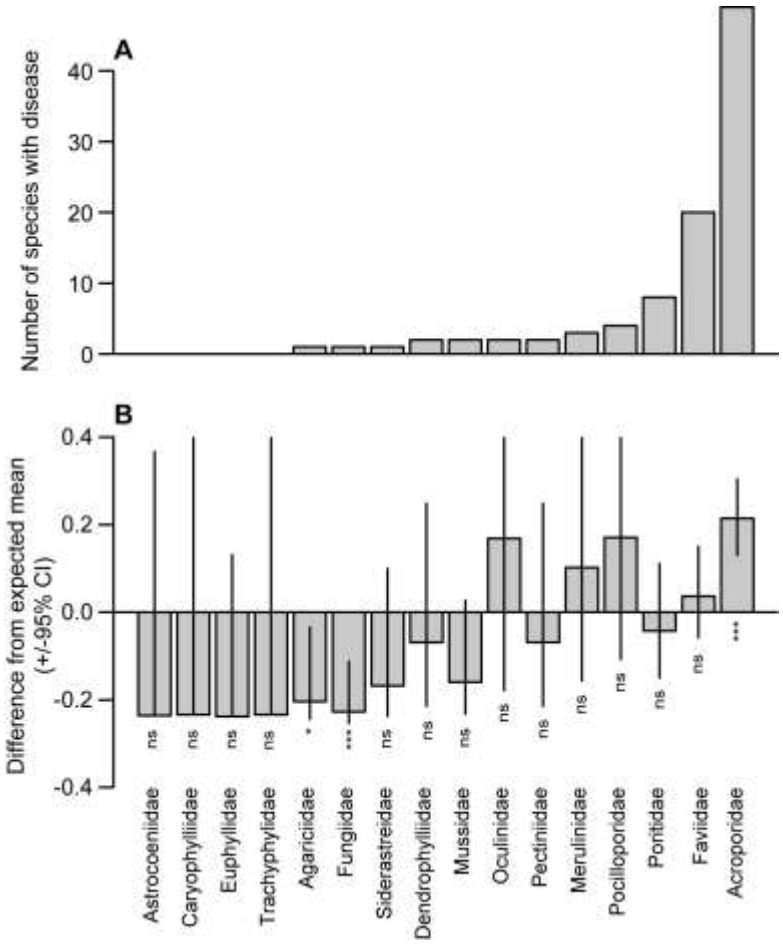

Figure 2. (a) The number of species observed with a coral disease in the GBR grouped by family. (b) Differences in family proportions of disease observation relative to the expected proportion for all families. 95% binomial confidence intervals and significance levels show if proportions can be distinguished from expectation. See Methods for further details.

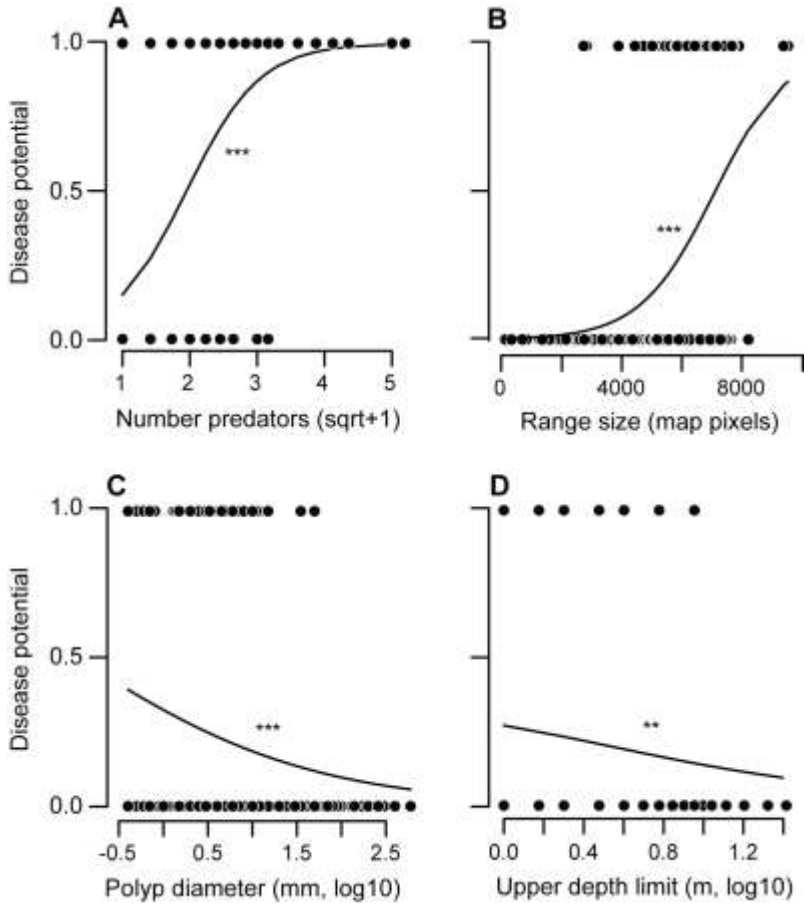

Figure 3. Disease potential as a function of (a) the number of predator species, (b) geographical range occupied by a species (measured as number of pixels), (c) polyp diameter, and (d) upper depth limit. Significance levels for logistic regressions are given in adjacent to fitted curves. See Methods for further details.

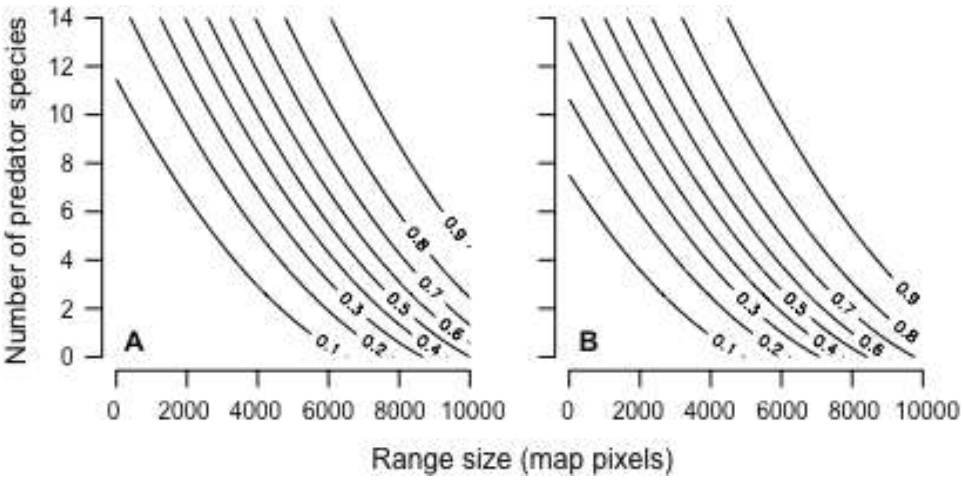

Figure 4. A contour plot of predicted disease potential as a function of both species' geographic range sizes and number of predatory species, illustrated separately for characteristically (a) common and (b) uncommon species.

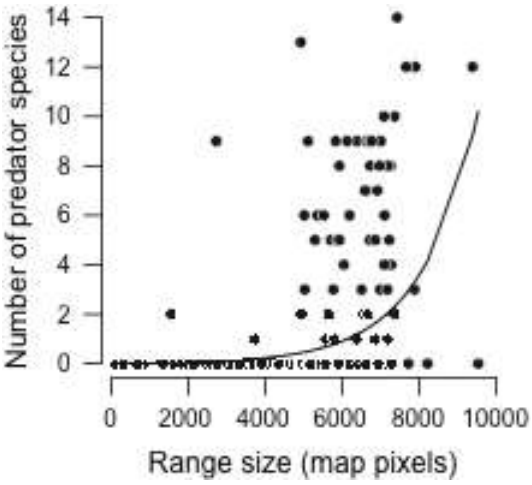

Figure 5. The triangular relationship between geographic range size of coral species and number of predator species. A generalised linear model (with a poisson error distribution) illustrates the underlying trend.

Supplementary Material

Disease data Summary table

Summary of information captured in the Great Barrier Reef coral disease literature, illustrating the high level of data heterogeneity and our decision to use a binomial response variable (disease potential). Only species-level observations could be used in analyses (last table column).

Reference	Presence-only data	Proportion of colonies infected	All diseases reported	1 disease reported	1- 5 host species	All host species	Host identified to species
Ainsworth et al. 2007	x			x	x		x
Antonius and Lipscomb 2001	x			x		x	x
Bourne 2005	x				x		
Boyett et al. 2007	x			x	x		x
Bruno et al. 2007				x			
Cole et al. 2009							x
Dalton and Smith 2005		x	x			x	x
Dinsdale 2000		x		x		x	x
Jones et al. 2004	x			x	x		x
Page and Willis 2006		x		x		x	x
Page and Willis 2008		x		x		x	x
Palmer et al. 2008	x			x	x		x
Patten et al. 2008	x			x	x		x
Roff et al.	x		x		x		x

2008a							
Roff et al. 2008b	x		x		x		x
Sato et al. 2009	x			x	x		x
Selig et al. 2006		x		x		x	
Sutherland et al. 2004	x		x				
Ulstrup et al. 2007							x
Willis et al. 2004	x		x			x	x

Reference list (Summary table)

Ainsworth TD, Kvennefors EC, Blackall LL, Fine M, Hoegh-Guldberg O (2007) Disease and cell death in white syndrome of Acroporid corals on the Great Barrier Reef. Mar Biol 151: 19-29

Antonius A, Lipscomb D (2001) First protozoan coral-killer identified in the Indo-Pacific. Atoll Res Bull 481: 1-21

Bourne DG (2005) Microbiological assessment of a disease outbreak on corals from Magnetic Island (Great Barrier Reef, Australia). Coral Reefs 24: 304-312

Boyett HV, Bourne DG, Willis B (2007) Elevated temperature and light enhance progression and spread of black band disease on staghorn corals of the Great Barrier Reef. Mar Biol 151: 1771-1720

Bruno J, Selig E, Casey K, Page C, Willis B, Harvell D, Sweatman H, Mekendy AM (2007) Thermal stress and coral cover as drivers of coral disease outbreaks. PLoS Biology 5: e124, 1220-1227

Cole AJ, Chong Seng KM, Pratchett MS, Jones GP (2009) Coral-feeding fishes slow progression of black band disease. Coral reefs 28: 965

Dalton SJ, Smith SD (2006) Coral disease dynamics at a subtropical location, Solitary Islands Marine Park, eastern Australia. Coral Reefs 25: 37-45

Dinsdale EA (2000) Abundance of black-band disease on corals from one location on the Great Barrier Reef: a comparison with abundance in the Caribbean region. Proc 9th Int Coral Reef Symp 2:1239–1243

Jones R, Bowyer J, Hoegh-Guldberg O, Blackall L (2004) Dynamics of a temperature-related coral disease outbreak. Mar Ecol Prog Ser 281: 63-77

Page C, Willis B (2006) Distribution, host range and large-scale spatial variability in black band disease prevalence on the Great Barrier Reef, Australia. Dis Aquat Org 69: 41-51

Page C, Willis B (2008) Epidemiology of skeletal eroding band on the Great Barrier Reef and the role of injury in the initiation if this widespread coral disease. Coral Reefs 27: 257-272

Palmer CV, Mydlarz LD, Willis B (2008) Evidence of an inflammatory-like response in non-normally pigmented tissues of two scleractinian corals. Proc R Soc Lond, Ser B 275: 2687-2693

Patten NL, Harrison PL, Mitchell JG (2008) Prevalence of virus-like particles within a staghorn scleractinian coral (*Acropora muricata*) from the Great Barrier Reef. Coral Reefs 27:569-580

Roff G, Kvennefors ECE, Ulstrup KE, Fine M, Hoegh-Guldberg O (2008a) Coral disease physiology: the impact of Acroporid white syndrome on *Symbiodinium*. Coral Reefs 27: 373-377

Roff G, Ulstrup KE, Fine M, Ralph PJ, Hoegh-Guldberg O (2008b) Spatial heterogeneity of photosynthetic activity within diseased corals from the Great Barrier Reef. Journal of Phycology 44: 526-538

Sato Y, Bourne D, Willis B (2009) Dynamics of seasonal outbreaks of black band disease in an assemblage of *Montipora* species at Pelorus Island (Great Barrier Reef, Australia). Proc R Soc Lond, Ser B 276: 2795-2803

Selig E, Harvell D, Bruno J, Willis B, Page C, Casey K, Sweatman H (2006) Analyzing the relationships between ocean temperature anomalies and coral disease outbreaks at broad spatial scales. In: Phinney J, Hoegh-Guldberg O, Kleypas J, Skirving W, Strong A (eds) Coral reefs and climate change: science and management. American Geophysical Union, Washington, DC. pp 111-128

Sutherland K, Porter J, Torres C (2004) Disease and immunity in Caribbean and Indo-Pacific zooxanthellate corals. Mar Ecol Prog Ser 266: 273-302

Ulstrup K, Kühl M, Bourne D (2007) Zooxanthellae harvested by cilliates associated with brown band syndrome of corals remain photosynthetically competent. Appl Environ Microbiol 73: 1968-1975

Willis B, Page C, Dinsdale E (2004) Coral disease on the Great Barrier Reef. In: Coral health and disease (ed. Rosenberg E, Loya Y). Springer-Verlag. Berlin, pp. 69-104