Projections of climate conditions that increase coral disease susceptibility and pathogen abundance and virulence

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Running title: Climate projections of coral disease conditions

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Rising sea temperatures are likely to increase the frequency of disease outbreaks affecting reef-building corals through impacts on coral hosts and pathogens. We present and compare climate model projections of temperature conditions that will increase 1) coral susceptibility to disease, 2) pathogen abundance, and 3) pathogen virulence. A moderate (RCP 4.5) and fossil fuel aggressive (RCP 8.5) emissions scenario are examined. We also compare projections for the onset of disease-conducive conditions and severe annual coral bleaching, and produce a disease risk summary that combines climate and anthropogenic stress. There is great spatial variation in the projections both among and within the major ocean basins in conditions favouring disease development. Our results indicate disease is as likely to cause coral mortality as bleaching in the coming decades. These projections identify priority locations to reduce anthropogenic stress and test management interventions to reduce disease impacts.

The 2014 boreal summer was the warmest on record [1], breaking air temperature records in hundreds of cities and causing unprecedented highs in sea surface temperatures in the North Pacific [2]. Concurrently, a catastrophic outbreak of starfish wasting disease decimated American west coast populations of ~20 starfish species [3] and outbreaks of eelgrass wasting disease resulted in declines in habitat area as high as 90% in parts of California and Washington (Wyllie-Echeverria pers obs). Pathogens causing these wasting disease outbreaks have been in the environment for at least decades [4], although the causative virus for seastar wasting is newly described [3]. These recent examples serve as reminders that disease outbreaks can rapidly and extensively devastate populations of keystone species and key habitat builders. Both events also caught the scientific and management communities by surprise, underscoring the importance of developing forecasts and long-term projections of conditions that increase outbreak likelihood.

Forecasts of conditions conducive to disease onset have been most extensively developed for the agricultural crop sector [5,6] because of the economic value of optimising the timing of pesticide application. Studies presenting longer-term, climate-model-based projections of conditions that promote disease onset for other plants and animals are far more rare. To date, climate models driven by Intergovernmental Panel on Climate Change (IPCC) emissions scenarios have only been used to develop projections of conditions related to the causative agents and vectors of human diseases [7], such as malaria [8-10] and Chikungunya [11]. Overall, the science of developing forecasts and projections for wildlife diseases is in its infancy and warrants much greater research focus [7], especially in the marine environment where disease outbreaks have been increasing in frequency and severity over recent decades [12].

Climate-related diseases have already severely impacted the primary framework builders of coral reef habitats [12-15]. Of the range of bacterial, fungal, and protozoan diseases known to affect stony corals [16], many have explicit links to temperature, including black band disease [17], yellow band disease [18,19], and white syndromes [13,20,21]. Here, we apply the climate models used in the IPCC 5th Assessment Report (see Table S1 for list) to project three temperature conditions that increase the susceptibility of coral hosts to disease or increase pathogen abundance or virulence.

We posit that temperature conditions that increase host susceptibility, pathogen abundance and pathogen virulence will substantially increase the likelihood of disease outbreaks once the set threshold frequencies and stress levels are surpassed. The output from the climate model ensemble for each of these three conditions is a projected year by which the target frequency or stress level is reached. All projections are presented for RCP8.5, the emissions scenario that best characterises current conditions and emission trends, and for RCP4.5, which represents a pathway to stabilisation at 4.5 W/m^2 (~650 ppm CO_2 equivalent) after 2100 [22]. Along with the individual projections, we present maps of the earliest and latest projected year one of these three conditions favourable to disease development is projected to occur. We also present: a) comparisons between the projected timing of these conditions and annual severe coral bleaching, b) a map of a composite metric of anthropogenic stressors that can also increase host susceptibility to disease, and c) a map of disease risk under RCP8.5 that combines climate and anthropogenic stress.

Projections of disease conditions

The year in which host susceptibility is projected to exceed the set threshold (i.e., sub-lethal bleaching stress 3 times per decade) varied spatially throughout all reef regions, but with a clear latitudinal trend. Reef locations in the tropics (<23° latitude) suffered thermal stress conducive to disease before sub-tropical reefs (23-32.5° latitude), a pattern that was similar under both RCPs (Fig. 1a and Fig. 2a). There was little variation (<5 years) in the projected timing of this condition among locations in the tropics (Fig. 1a). In contrast, some northern hemisphere sub-tropical reefs, such as in the Red Sea and Persian Gulf, were projected to experience these conditions ~20 years later than sub-tropical reefs in the south of Australia and Madagascar. Overall, under both RCP8.5 and RCP4.5, the median year this threshold will be surpassed was 2011; most (~76% as of 2014) of the world's reefs are already experiencing thermal stress potentially conducive to disease outbreaks. Under both RCP8.5 and 4.5, the metric for increased host-susceptibility will be reached at >90% of reef locations by 2020 (Fig. 2a).

In contrast to patterns for the host susceptibility metric, there was no clear latitudinal gradient in the projections for increased pathogen abundance (i.e., when cool season temperatures have warmed by ≥0.5°C) (Fig. 1b). Additionally, greater variation in the projected timing of this condition among reefs within both the tropics and sub-tropics was observed, as well as between the RCPs, than was seen for the host susceptibility metric. Under both RCPs, the threshold set for increased cool season temperatures will be reached by 2014 in the southern Red Sea, southern India, the province of Papua in Indonesia, and in the Bahamas (Fig. 1b). In contrast, under RCP8.5, increased cool season temperatures were not projected to occur until the 2030s and 2040s for much of the Coral Triangle, Madagascar and Hawaii, and not until the 2050s and 2060s for locations throughout the far south Pacific, such as French Polynesia (Fig. 1b). The projected years for these locations were all roughly a decade later under RCP4.5 (Fig. 1b). The median years for the projections were 2036 (RCP8.5) and 2043 (RCP4.5). Under RCP8.5, the threshold set for increased cool season temperatures is reached at 20% of reef locations by 2020 and for 17% after 2050 (remaining 63% fall between 2020-2050) (Fig. 2e).

Spatial patterns for projections of the pathogen virulence metric (i.e., for *Vibrio corallilyticus*, when the number of months that temperatures are ≥ the MMM is double that observed on average from 2006-2011) were similar to those found for the host susceptibility metric. Reefs in the tropics will experience this condition earlier than sub-tropical reefs (Fig. 1c), with little variation between the two RCPs. The Caribbean was an exception to this latitudinal pattern; the years that sub-tropical reefs in the Caribbean were projected to experience a doubling of months at or above MMM (i.e., 2020) were among the earliest projected under both RCPs. For sub-tropical reefs in the south Pacific and Red Sea, the target stress level will be reached 20 or more years later, in the mid 2040s. The median years for this projection were 2031 (RCP8.5) and 2030 (RCP4.5), ~20 years later than the median for the host susceptibility metric.

For most reef locations (~80% for both RCPs), the models projected timing of increased host-susceptibility to occur earliest (Fig. 1a) and for increased pathogen virulence (for *Vibrio corallilyticus)* to occur latest (Fig. 1c). Under RCP8.5 at least one of the three types of temperature conditions favouring disease development were projected to be surpassed at all reef locations by 2031, and 80% of reefs will have experienced one of the conditions by 2020 (Fig. 3a,b; S1a,b).

There was limited variation between the two RCPs in the projected year that the three conditions favouring disease development would be reached (Fig. 3b and Fig. S1b). Across all reef locations, the average difference in projections between RCP8.5 and RCP4.5 was less than 1 year for the host susceptibility and pathogen virulence thresholds. For the pathogen abundance metric, the average difference between the two RCPs was ~6 years. This difference is likely inconsequential given the standard deviation of model outputs is ~6 years for both scenarios (Fig. 2e,h). The minor nature of differences in the projection outputs for the two RCPs reflects the slow divergence of RCP4.5 from RCP8.5 over the coming two decades [22]. Even drastic cuts to emissions outputs and emissions growth required to achieve the CO_2 concentrations characteristic of RCP4.5 do not prevent all of the disease conditions set here from being surpassed at >75% of reef locations by 2090 (Fig. 2 and Fig. S1b).

Comparing coral disease and bleaching

The same model ensemble for RCP8.5 was used to project the onset of annual severe bleaching conditions, defined as the year in which 8 DHWs is exceeded annually during the warm season [23]. Currently, most corals will bleach once 8 DHWs is reached (Fig. 3), and coral diversity and cover are likely to decline dramatically when temperature stress of this severity begins to recur with insufficient time for recovery [23]. We sought to determine whether temperature conditions that favour disease development are projected to occur earlier or later than annual severe coral bleaching. To make this comparison, we calculated the difference in the number of years between the projected timing of any two of the three temperature conditions set here for coral disease and the onset of annual severe bleaching conditions (Fig. 3d). Under RCP8.5, at least two of the three disease-favouring temperature conditions occurred at 96% of reef locations (Fig. 3d) before the onset of annual severe bleaching (98% under RCP4.5, Fig. S1d). All three conditions occur before the onset of annual severe bleaching at 40% of locations. The comparisons of projected timing of disease versus bleaching conditions offered here suggest disease outbreaks will be at least as great a driver of future coral reef condition and community composition as bleaching.

Anthropogenic stress patterns and disease risk

Anthropogenic stress is likely to be as important a driver of coral disease dynamics over the coming decades as the temperature conditions presented here [24-27]. The Integrated Local Threat (ILT) Index [28] combines four threats that increase disease susceptibility: increased sedimentation and nutrients associated with coastal development [27,29,30], watershed-based pollution [26,29-32], marine-based pollution and damage [25,33,34], and injuries associated with fishing activities, particularly destructive fishing [12]. The ILT index (500-m resolution) results are resampled here to match the climate model grid used for the temperature projections and the highest threat level within each model pixel is displayed (Fig. 4a). This ensures the global patterns can be seen at the resolution the figure is printed within the article.

Anthropogenic stress and climate stress are combined here in a disease risk summary, as both are likely to drive future patterns in disease outbreak likelihood. Ecosystem impacts from coral disease have the potential to be equal to or exceed those of severe bleaching stress when two (or all three) of the disease-favouring conditions occur before the onset of annual severe bleaching. Outbreak likelihood is also higher when anthropogenic stress is either high or very high. This logic was applied to produce 5 criteria for relative outbreak likelihood over the coming 20-30 years, which we describe as 'disease risk' (Fig. 4b). Locations with greater relative risk (#'s 2-5 in Fig. 4b; 22% of locations) were southern Florida, the southern and eastern Caribbean, Brazil, the province of Papua in Indonesia, Philippines, Japan, India, northern Maldives, the Persian Gulf and the Red Sea (Fig. 4b). For the combined disease risk metric, relative risk was considered lower for locations where anthropogenic stress was low or medium, a condition found for 78% (see

Fig. 4 caption) of reef locations. Some of these locations included Hawaii, the central and south Pacific, Australia, Thailand and Madagascar (Fig. 4b). The disease risk summary can be seen at the resolution of the anthropogenic stress data (500 m) in a high-resolution image presented in the electronic supplementary material (see Fig. S2). The high-resolution image complements Fig. 4 enabling viewers to zoom into reef locations to interpret disease risk in relation to the actual rather than resampled anthropogenic stress data. The disease risk summary reflects that anthropogenic stress is only high or very high at 22% of locations. However, at almost all reef locations (>95%), 2 of the 3 temperature conditions conducive to disease development occurred before the onset of annual severe bleaching. The risk of coral diseases due to climate change (ignoring anthropogenic stress) is high at nearly all reef locations.

Future applications and conclusions

These are the first climate-model-based projections of conditions that influence the likelihood of marine disease outbreaks. Some important complexities are necessarily excluded here so that global-scale conservative projections could be produced. The main examples are: 1) variation among and within coral communities and species in host susceptibility due to variation in genetics related to immunity, the expression of immunity genes, and exposure to environmental disturbances and anthropogenic stress, 2) the potential for coral evolution of resistance, which will be highly variable among and even potentially variable within species, 3) the relationships between temperature conditions and the virulence of other pathogens that cause diseases in stony corals, which are not as well known or understood as *Vibrio coralliilyticus* and white syndromes, and

4) extreme stochastic events such as extreme climatic events or the evolution of new 'super' pathogens, which could invalidate some of the presented conclusions. Other possible conditions that can increase disease susceptibility and pathogen abundance and virulence that are not included here are: sediment runoff and lowered salinity following monsoonal rain events, and coral injuries from cyclones [35,36] or predation by coral-feeding gastropods [37], crown-of-thorns starfish [38], and reef fish [39,40]. Future scenarios that include ocean acidification projections would also be valuable for understanding conditions that increase coral disease susceptibility and pathogen virulence. Members of the research community can use the data presented here to refine or produce higher-resolution projections for areas for which spatially explicit data on some or all of the information described above becomes available.

The standard caveats and assumptions related to the use of climate models also apply [41,42], and two are especially pertinent. Firstly, model resolution is coarse and a 1x1° cell can contain many individual coral reefs, a fact related to the computational-intensiveness of climate modeling and to modeling uncertainties (see below). While spatial variation within single model cells is not resolved here, there is considerable variation within reef regions in the projected timing of all three temperature conditions for disease and in anthropogenic stress. Therefore, even at this resolution, the results can be used to target applied research and management actions. Secondly, all climate models have uncertainties and vary greatly in their capacity to project trends in key drivers of climate in the tropics, such as the El Niño Southern Oscillation and its global teleconnections. We include the standard deviation around the ensemble average (the 'model spread') for each temperature condition (Fig. 2d-i). The spread in the model results is small (standard deviation of 2-6.5 years), which increases confidence in the major conclusions presented based on the ensemble results and supports use of the ensemble rather than one or more of the individual models. A review of the robustness and uncertainties in the new CMIP5 climate model projections (used here) suggests that climate models are improving, representing more climate processes in greater detail, and that the *"uncertainties should not stop decisions being made"* [41]. For this study, the relevant decisions involve the targeting of actions to reduce anthropogenic stress and trials of the efficacy of interventions that reduce disease impacts and support recovery.

Currently, the role of disease as a significant driver of future reef community composition is under-appreciated, especially in the Indo-Pacific, and needs to be given greater consideration for at least two reasons. Disease has a tendency to result in greater coral mortality than bleaching [14,43,44]. Secondly, given the strong links between anthropogenic stress and disease susceptibility [24,26,29,30], management actions that reduce anthropogenic stress are probably more likely to reduce the prevalence and severity of coral diseases than reduce the impacts of thermal bleaching. Immediate actions to reduce anthropogenic stress are needed at locations with high or very high anthropogenic stress (Fig. 4a), and are especially urgent at locations also predicted to experience all three temperature conditions set here in the coming two decades (Fig. 4b). These sets of conditions apply to ~20% of the reef locations (Fig. 4b, categories 4 and 5). These locations are priority targets for proactive conservation efforts to reduce anthropogenic stress, such as managing watersheds and coastal development, reducing destructive fishing, and addressing other extractive practices. Furthermore, there is a need for collaborative efforts between researchers and managers to both better understand disease outbreaks and test reactive management interventions that reduce disease transmission rates. Examples include quarantining or culling infected corals, which could be followed by actions that mitigate impacts and support recovery such as managing human activities through temporary closures or other use restrictions. Many of these actions (reviewed in [45,46]) are currently experimental and only feasible at small local scales. Trials of the efficacy of these actions can lead to broader implementation in the coming decades.

There is also a need for researchers and managers to expand upon the currently very limited suite of tools that forecast conditions conducive to coral disease outbreaks [20,47]. New early warning systems will need to be built into coral disease response plans. Such plans can help managers consider and justify various decisions and investments in both targeted monitoring and trials/implementation of actions to reduce disease impacts and support recovery. A coral disease response plan framework has been developed for the Great Barrier Reef in Australia [39] and for Hawaii but coral disease response plans have not been as widely adopted as coral bleaching response plans [46].

Perhaps more than any findings to date, the results presented herein indicate that increases in the prevalence and severity of coral diseases will be a major future driver of decline and changes in coral reef community composition, and at least as great a driver as coral bleaching. Elevated temperatures that increase host susceptibility, pathogen abundance or virulence are either already occurring or are projected to occur in the coming decades at almost all reef locations. This is true irrespective of whether nations are able to sufficiently cut emissions such that RCP4.5 better characterizes our emissions trajectory than RCP8.5. There is great spatial variation in the projected timing of the disease-favouring conditions, which is in keeping with much new research highlighting that the impacts of climate change will not be spatially uniform. The spatial variation in the projections we present also emphasises the value for decision-making of developing near real-time early warning systems and seasonal outlooks for marine diseases.

Methods

Climate model data: Monthly sea surface temperature (SST) data were retrieved for each available GCM from the World Climate Research Programme's CMIP5 data set (from http://www.esg.llnl.gov) for RCP8.5 (n=33) and RCP4.5 (n=35, see Table S1 for list of models). Methods for matching the start of each model with the observed climatology used (1982-2005), correcting model means, replacing annual cycles, and interpolation routines are all as per [48]. Projections produced are based on model runs that are then averaged, rather than on ensemble means [23,48], ensuring variance among models is examined and presented for each projection output (Fig. 2d-i).

Temperature conditions that increase disease susceptibility and/or pathogen virulence: Three temperature conditions are examined that increase the susceptibility of coral hosts to disease or increase pathogen abundance or virulence.

For all of the projected conditions, results are shown for reef locations only (also as per [48]) rasterised to match the climate model grid (n=1748 pixels or 'reef locations').

Histograms and plots of the spread in results (average ±1 stdev) from the climate models are presented for each temperature condition with percentages based on the total number of reef locations.

(1) **Host susceptibility:** Field studies from all reef regions have shown that coral diseases often follow sub-lethal bleaching, presumably when energy and resources required for the maintenance of disease resistance are reduced [14,16,49-51]. For sensitive species globally, thermal stress represented by 4 Degree Heating Weeks (DHWs) is a conservative threshold for predicting the presence of sub-lethal bleaching, since the global optimum predictor of bleaching is slightly higher at ~6 DHWs [52]. Here, our 'host susceptibility' metric identifies when a decade starts in which thermal stress is projected to exceed 4 DHWs at least 3 times. This frequency was selected because the return period is so short (~3 years) that corals will likely struggle to recover between bleaching events thus remaining in a weakened and therefore susceptible state.

(2) **Pathogen abundance:** Research on diseases affecting agricultural crops indicates that survival rates of both causative agents and disease vectors increase during anomalously warm winters (called 'overwintering' [6]). While many long-term studies of coral diseases detect higher levels of disease prevalence when temperatures peak [12], a common group of coral diseases, white plague and white syndromes, have been found in higher abundances during warm summers that follow mild winters (neither excessively cool or warm [20]). This is likely due to a combination of overwintering and increased host susceptibility because warmer winters provide less of a reprieve for corals between warm seasons. Here, the

'pathogen abundance' metric indicates the first year in which the means of the three months centered on the coolest month are $\geq$0.5°C above the minimum monthly mean (coolest month) calculated from a 1982-2008 climatology. This roughly equates to the thermal stress associated with mild winters in [20] of 2-6.5 °C-weeks, which are calculated from a higher baseline than is used here and which resulted in an increased abundance of white syndromes in Australia during the following summers.

(3) Pathogen virulence: The model coral pathogen used here, *Vibrio coralliilyticus*, is the causative agent of a number of virulent white syndromes on Indo-Pacific corals, causing progressive tissue loss and ultimately, whole colony mortality. We reviewed experimental studies and related the temperatures at which the virulence and host-seeking motility behaviors (i.e., chemotaxis and chemokinesis) of this pathogen are augmented to the maximum monthly mean (MMM, warmest month) at each sampling location (see Table S2). For each of three strains of *V. coralliilyticus*, the pathogen becomes virulent within 2.5 °C of the MMM calculated for the period 1982-2008 at the respective sampling location, so we conservatively set the threshold as MMM (1982-2008). Here, the metric 'pathogen virulence' identifies when the number of months in which temperatures exceed the MMM becomes twice that observed, on average, during 2006-2011. This represents the timing of anticipated increases in virulence and in the projected number of months corals are exposed to the virulent pathogen.

Maps and histograms (standardised to the total number of reef locations) are presented for: a) the earliest year by which at least one disease condition will be met, b) the year by which all three disease conditions will be met, (c) the year from which annual severe bleaching stress is projected, and (d) the difference between the year by which at least two of the three disease conditions are met and the onset of annual severe bleaching (8 DHWs).

A map is also presented of anthropogenic stress using the Integrated Local Threat (ILT) index developed for Reefs at Risk Revisited [28], as is described in the paper. We resample these data to our climate model grid by taking the highest level of stress within each model pixel to produce a visual summary interpretable at article-resolution. The disease risk summary presented for RCP8.5 grades risk based on 5 criteria: 1) none of the following criteria apply; 2) two of three climate stressors occur before the onset of annual severe bleaching and anthropogenic stress is high; 3) as for criterion 2 but anthropogenic stress is very high; 4) all three climate stressors occur before the onset of annual severe bleaching and anthropogenic stress is high; and 5) as for criterion 4 but anthropogenic stress is very high. A 500-m resolution image of the disease risk summary is provided as electronic supplementary material enabling readers to zoom into reefs of interest to see which reefs meet the criteria set. The percentage values cited in the paper for reef pixels that meet each of the five criteria are derived at 500-m resolution rather than from the resampled data.

References

1. National Climatic Data Center. Global Analysis - August 2014 | State of the Climate. *ncdc.noaa.gov* at <http://www.ncdc.noaa.gov/sotc/global/2014/8>
2. Slezak, M. Oceans get into hot water. *New Scientist* **224,** 8–9 (2014).
3. Hewson, I. *et al.* Densovirus associated with sea-star wasting disease and mass mortality. *Proc Natl Acad Sci* **111,** 17278–17283 (2014).
4. Groner, M. L. *et al.* Host demography influences the prevalence and severity of eelgrass wasting disease. *Dis. Aquat. Org.* **108,** 165–175 (2014).
5. Savary, S., Nelson, A., Sparks, A. H. & Willocquet, L. International agricultural research tackling the effects of global and climate changes on plant diseases in the developing world. *Plant Disease* **95,** 1204–1216 (2011).
6. Garrett, K. A., Dendy, S. P. & Frank, E. E. Climate change effects on plant disease: genomes to ecosystems. *Annu Rev Phytopathol* **44,** 489–509 (2006).
7. Altizer, S., Ostfeld, R. S., Johnson, P. T. J., Kutz, S. & Harvell, C. D. Climate change and infectious diseases: from evidence to a predictive framework. *Science* **341,** 514–519 (2013).
8. Rogers, D. J. & Randolph, S. E. The Global Spread of Malaria in a Future, Warmer World. *Science* **289,** 1763–1766 (2000).
9. van Lieshout, M., Kovats, R. S., Livermore, M. T. J. & Martens, P. Climate change and malaria: analysis of the SRES climate and socio-economic scenarios. *Global Environ Change* **14,** 87–99 (2004).
10. Béguin, A. *et al.* The opposing effects of climate change and socio-economic development on the global distribution of malaria. *Global Environ Change* **21,** 1209–1214 (2011).
11. Ruiz-Moreno, D., Vargas, I. S., Olson, K. E. & Harrington, L. C. Modeling Dynamic Introduction of Chikungunya Virus in the United States. *PLoS Negl Trop Dis* **6,** e1918 (2012).
12. Burge, C. A. *et al.* Climate change influences on marine infectious diseases: implications for management and society. *Annu Rev Marine Sci* **6,** 249–277 (2014).
13. Bruno, J. F., Selig, E. R., Casey, K. S., Page, C. A. & Willis, B. L. Thermal stress and coral cover as drivers of coral disease outbreaks. *PLoS Biol* **5,** e124 (2007).
14. Miller, J. *et al.* Coral disease following massive bleaching in 2005 causes 60% decline in coral cover on reefs in the US Virgin Islands. *Coral Reefs* **28,** 925–937 (2009).
15. Harvell, D., Altizer, S., Cattadori, I. M., Harrington, L. & Weil, E. Climate change and wildlife diseases: when does the host matter the most? *Ecology* (2009). doi:10.1890/08-0616.1
16. Harvell, D., Jordán-Dahlgren, E. & Merkel, S. Coral disease, environmental drivers, and the balance between coral and microbial associates. *Oceanography* **20,** 172–195 (2007).
17. Sato, Y., Bourne, D. G. & Willis, B. L. Dynamics of seasonal outbreaks of black band disease in an assemblage of Montipora species at Pelorus Island (Great Barrier Reef, Australia). *Proc Biol Sci* **276,** 2795–2803 (2009).
18. Riegl, B. Effects of the 1996 and 1998 positive sea-surface temperature anomalies

on corals, coral diseases and fish in the Arabian Gulf (Dubai, UAE). *Mar Biol* **140,** 29–40 (2002).

19. Cervino, J. M. *et al.* Relationship of Vibrio species infection and elevated temperatures to yellow blotch/band disease in Caribbean corals. *App Environ Microbiol* **70,** 6855–6864 (2004).
20. Heron, S. F. *et al.* Summer Hot Snaps and Winter Conditions: Modelling White Syndrome Outbreaks on Great Barrier Reef Corals. *PLoS ONE* **5,** e12210 (2010).
21. Maynard, J. A. *et al.* Predicting outbreaks of a climate-driven coral disease in the Great Barrier Reef. *Coral Reefs* 1–11 (2010). doi:10.1007/s00338-010-0708-0
22. van Vuuren, D. P. *et al.* The representative concentration pathways: an overview. *Climatic Change* **109,** 5–31 (2011).
23. van Hooidonk, R. J., Maynard, J. A., Manzello, D. & Planes, S. Opposite latitudinal gradients in projected ocean acidification and bleaching impacts on coral reefs. *Glob Change Biol* **20,** 103–112 (2014).
24. Aeby, G. S. *et al.* Patterns of Coral Disease across the Hawaiian Archipelago: Relating Disease to Environment. *PLoS ONE* **6,** e20370 (2011).
25. Lamb, J. B. & Willis, B. L. Using Coral Disease Prevalence to Assess the Effects of Concentrating Tourism Activities on Offshore Reefs in a Tropical Marine Park. *Conservation Biology* **25,** 1044–1052 (2011).
26. Vega Thurber, R. L. *et al.* Chronic nutrient enrichment increases prevalence and severity of coral disease and bleaching. *Glob Change Biol* **20,** 544–554 (2013).
27. Pollock, F. J. *et al.* Sediment and Turbidity Associated with Offshore Dredging Increase Coral Disease Prevalence on Nearby Reefs. *PLoS ONE* **9,** e102498 (2014).
28. Burke, L. M., Reytar, K., Spalding, M. & Perry, A. *Reefs at risk revisited*. (World Resources Institute, 2011).
29. Kline, D. I., Kuntz, N. M. & Breitbart, M. Role of elevated organic carbon levels and microbial activity in coral mortality. *Marine Ecology* (2006).
30. Bruno, J. F., Petes, L. E., Drew Harvell, C. & Hettinger, A. Nutrient enrichment can increase the severity of coral diseases. *Ecol Letters* **6,** 1056–1061 (2003).
31. Haapkylä, J. *et al.* Seasonal Rainfall and Runoff Promote Coral Disease on an Inshore Reef. *PLoS ONE* **6,** e16893 (2011).
32. Sutherland, K. P., Shaban, S., Joyner, J. L., Porter, J.W., & Lipp, E.K. Human Pathogen Shown to Cause Disease in the Threatened Eklhorn Coral Acropora palmata. PLoS ONE 6(8): e23468. doi:10.1371/journal.pone.0023468 (2011).
33. Lamb, J. B., True, J. D., Piromvaragorn, S. & Willis, B. L. Scuba diving damage and intensity of tourist activities increases coral disease prevalence. *Biological Conservation* (2014). doi:10.1016/j.biocon.2014.06.027
34. Hawkins, J. P. *et al.* Effects of recreational scuba diving on Caribbean coral and fish communities. *Conservation Biology* **13**, 888-897 (1999).
35. Miller, M. W., & Williams, D. E. Coral disease outbreak at Navassa, a remote Caribbean island. *Coral Reefs*, **26**, 97-101 (2007).
36. Bruckner, A. W., & Bruckner, R. J. Outbreak of coral disease in Puerto Rico. *Coral Reefs*, **16**, 260-260 (1997).

37. Nicolet, K., M. Hoogenboom, N. Gardiner, M. Pratchett, and B. Willis. The corallivorous invertebrate Drupella aids in transmission of brown band disease on the Great Barrier Reef. *Coral Reefs* **32**:585-595 (2013).
38. Katz, S. M., Pollock, F. J., Bourne, D. G., & Willis, B. L. (2014). Crown-of-thorns starfish predation and physical injuries promote brown band disease on corals. *Coral Reefs*, **33**, 705-716 (2014).
39. Aeby, G. S., & Santavy, D. L. (2006). Factors affecting susceptibility of the coral Montastraea faveolata to black-band disease. *Marine Ecology Progress Series*,*318*, 103-110.
40. Casey, J. M., T. D. Ainsworth, J. H. Choat, and S. R. Connolly. Farming behaviour of reef fishes increases the prevalence of coral disease associated microbes and black band disease. *Proceedings of the Royal Society B: Biological Sciences* **281**: 20141032 (2014).
41. Knutti, R. & Sedláček, J. Robustness and uncertainties in the new CMIP5 climate model projections. *Nature Clim Change* – (2012). doi:10.1038/nclimate1716
42. van Hooidonk, R. & Huber, M. Effects of modeled tropical sea surface temperature variability on coral reef bleaching predictions. *Coral Reefs* **31,** 121–131 (2012).
43. Cróquer, A. & Weil, E. Changes in Caribbean coral disease prevalence after the 2005 bleaching event. *Dis Aquat Org* **87,** 33–43 (2009).
44. Aeby, G. S., Ross, M., Williams, G. J., Lewis, T. D. & Work, T. M. Disease dynamics of Montipora white syndrome within Kaneohe Bay, Oahu, Hawaii: distribution, seasonality, virulence, and transmissibility. *Dis Aquat Org* **91,** 1–8 (2010).
45. Beeden, R., Maynard, J. A., Marshall, P. A., Heron, S. F. & Willis, B. L. A Framework for Responding to Coral Disease Outbreaks that Facilitates Adaptive Management. *Environmental Management* **49,** 1–13 (2012).
46. Maynard, J. A. *et al.* A Strategic Framework for Responding to Coral Bleaching Events in a Changing Climate. *Environmental Management* **44,** 1–11 (2009).
47. Maynard, J. A., Anthony, K., Harvell, C. D. & Burgman, M. A. Predicting outbreaks of a climate-driven coral disease in the Great Barrier Reef. *Coral Reefs* (2011). doi:10.1007/s00338-010-0708-0
48. van Hooidonk, R., Maynard, J. A. & Planes, S. Temporary refugia for coral reefs in a warming world. *Nature Clim Change* **3,** 1–4 (2013).
49. Miller, J., Waara, R., Muller, E. & Rogers, C. Coral bleaching and disease combine to cause extensive mortality on reefs in US Virgin Islands. *Coral Reefs* **25,** 418 (2006).
50. Brandt, M. & McManus, J. Disease incidence is related to bleaching extent in reef-building corals. *Ecology* **90,** 2859–2867 (2009).
51. Muller, E. M., Rogers, C. S., Spitzack, A. S. & Van Woesik, R. Bleaching increases likelihood of disease on Acropora palmata (Lamarck) in Hawksnest Bay, St John, US Virgin Islands. *Coral Reefs* **27,** 191–195 (2008).
52. van Hooidonk, R. & Huber, M. Quantifying the quality of coral bleaching predictions. *Coral Reefs* **28,** 579–587 (2009).

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Author contributions

JM, CDH, CME, SFH, RvH, BW, MG, JL and GW designed the study. RvH compiled and analysed the climate model data in collaboration with JM. MP conducted the spatial analysis required to build the maps upon which Figs. 3 and 4 and Figs. S1 and S2 are based in collaboration with JM. JM, CDH, CME and BW wrote the manuscript with assistance from all other authors.

Competing financial interests statement

The authors declare no competing financial interests.

Supplementary Information

Supplementary Information is linked to the online version of the paper at www.nature.com/nature and includes a table listing the climate models used, a review of experimental studies that examined the effects of temperature on *Vibrio coralliilyticus,* a panel figure for RCP4.5 that matches Fig. 3 here, and a 500-m resolution disease risk summary figure that complements Fig. 4 here.

Fig. Legends

Figure 1. Projections of temperature conditions that increase host susceptibility (a), pathogen abundance (b), and pathogen virulence (c) under RCPs 8.5 and 4.5. The conditions and condition thresholds are: (a) *Host susceptibility* – year in which thermal stress first exceeds 4 DHWs 3x per decade; (b) *Pathogen abundance* – first year in which the 3 cool season months exceed 0.5 °C above the minimum monthly mean (1982-2008); (c) *Pathogen virulence* – year in which the number of months of temperatures $\geq$max monthly mean (1982-2008) is twice that observed on average from 2006-2011. See Table S1 for a list of climate models.

Figure 2. Histograms and model means and spreads for the projections of temperature conditions under RCPs 8.5 and 4.5. For the histograms (top row), bins are 5-year intervals and n=1748 reef locations. For model means and spreads (2 bottom rows), means are shown as the bold line and spreads are the mean $\pm$1 stdev (grey shade). These data correspond to the model projections shown as maps in Fig. 1. See Table S1 for a list of climate models.

Figure 3. Summaries of projections for disease and bleaching conditions under RCP8.5. The earliest year (a) is the first year in which at least one of the three temperature conditions for disease shown in Fig. 1 will be reached. The year in which all three temperature conditions will be reached is shown in (b). The onset of annual severe bleaching is shown in (c), defined as temperature stress annually exceeding 8 DHWs (from [23]). The difference in timing between when at least 2 of the 3 temperature conditions for disease shown in Fig. 1 will be reached and (c) is shown in (d). Negative values in (d) mean at least 2 of the 3 temperature conditions for disease are projected to occur before annual severe bleaching conditions (96% of reef locations).

Figure 4. Anthropogenic stress patterns and disease risk based on exposure to anthropogenic and climate stress. Anthropogenic stress (a) is a resampling of the Reefs at Risk Revisited [28] Integrated Local Threat index to the climate model grid used in Figs. 1 and 3; the highest value for stress within each model pixel is retained so that approximate global patterns can be interpreted at this resolution. Disease risk (b), in relative terms, relates to whether: 2 or 3 of the temperature conditions (from Fig. 1) occur before annual severe bleaching (ASB) (see Fig. 3c), and anthropogenic stress is high or very high. Reef location (model cell) counts and percentages are as follows and are from the 500-m resolution data, which are presented within Fig. S2: 1 (353485, 78%), 2 (35975, 8%), 3 (23378, 5%), 4 (25184, 6%), 5 (13375, 3%).