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Effects of a changing climate on the dynamics of coral infectious disease: a review of the evidence

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ABSTRACT: A close examination of the coral disease literature reveals many hypothesized mechanisms for how coral disease may be linked to climate change. However, evidence has been largely circumstantial, and much uncertainty remains. Here, I review the latest information on both the predicted effects of climate change in coastal marine ecosystems and current research on coral-pathogen dynamics in relation to climate variables. The published evidence supports the hypothesis that coral infectious diseases are emerging and demonstrates that coral disease research has been exponentially expanding over the last few decades. Current research suggests that environmental factors, such as ocean warming, altered rainfall, increased storm frequency, sea level rise, altered circulation, and ocean acidification may play a role in coral disease. These climate variables likely alter coral epidemiology through effects on pathogen growth rates, transmission, virulence, and susceptibility. Despite recent advances, discovering the causes of coral disease emergence at large spatial and temporal scales has been hindered by several factors including (1) the inability to rely on Koch's postulates for diseases with multifactorial etiologies, (2) the paucity of long-term, coordinated, coral disease data, and (3) the difficulty in detecting correlations in inherently non-linear, dynamic disease systems. In a rapidly changing global environment, the consequences of increasing coral disease may be severe, leading to elevated extinction risk and loss of critical reef habitat. Current evidence is still preliminary but is increasingly suggestive that mitigating the effects of climate change may help reduce the emergence of disease and improve the health of coral reef ecosystems.

KEY WORDS: Coral diseases · Emerging infectious diseases · Environmental stress · Epidemiology · Temperature · Marine · Global warming · Climate change · Causation

INTRODUCTION

There is no doubt that we live in a rapidly changing world. The average global temperature increased 0.6 ± 0.2°C during the last century (Fig. 1) and is predicted to further increase another 1.5 to 4.5°C during the next, while at the same time, atmospheric CO_2 concentrations have increased 100 ppm compared with pre-industrial levels (Bindoff et al. 2007, Rogner et al. 2007, Rosenberg et al. 2007). The global mean sea level has been rising over the last century, with the rate of rise hastening significantly during recent decades (Bindoff et al. 2007). Ocean salinity has significantly changed over the last half century, with freshening near the poles and salinification at shallower tropical latitudes, and global oceanic pH has dropped approximately 0.1 pH units since 1750 (Bindoff et al. 2007). There is wide acceptance that human activities are currently adding greenhouse gases to the atmosphere, and that these gases contribute to both warming (Crowley 2000) and chemical alteration of the oceans (Kleypas et al. 1999, Caldeira & Wickett 2003). There is also compelling evidence that coral reefs are in precipitous decline worldwide (Hughes 1994, Hughes & Tanner 2000, Gardner et al. 2003, Pandolfi et al. 2003). By one recent estimate, nearly one-third of reef-building corals face elevated extinction risk from climate change and other impacts (Carpenter et al. 2008). However, like many emerging fields of research, both global climate change research and coral reef science are fraught with uncer-

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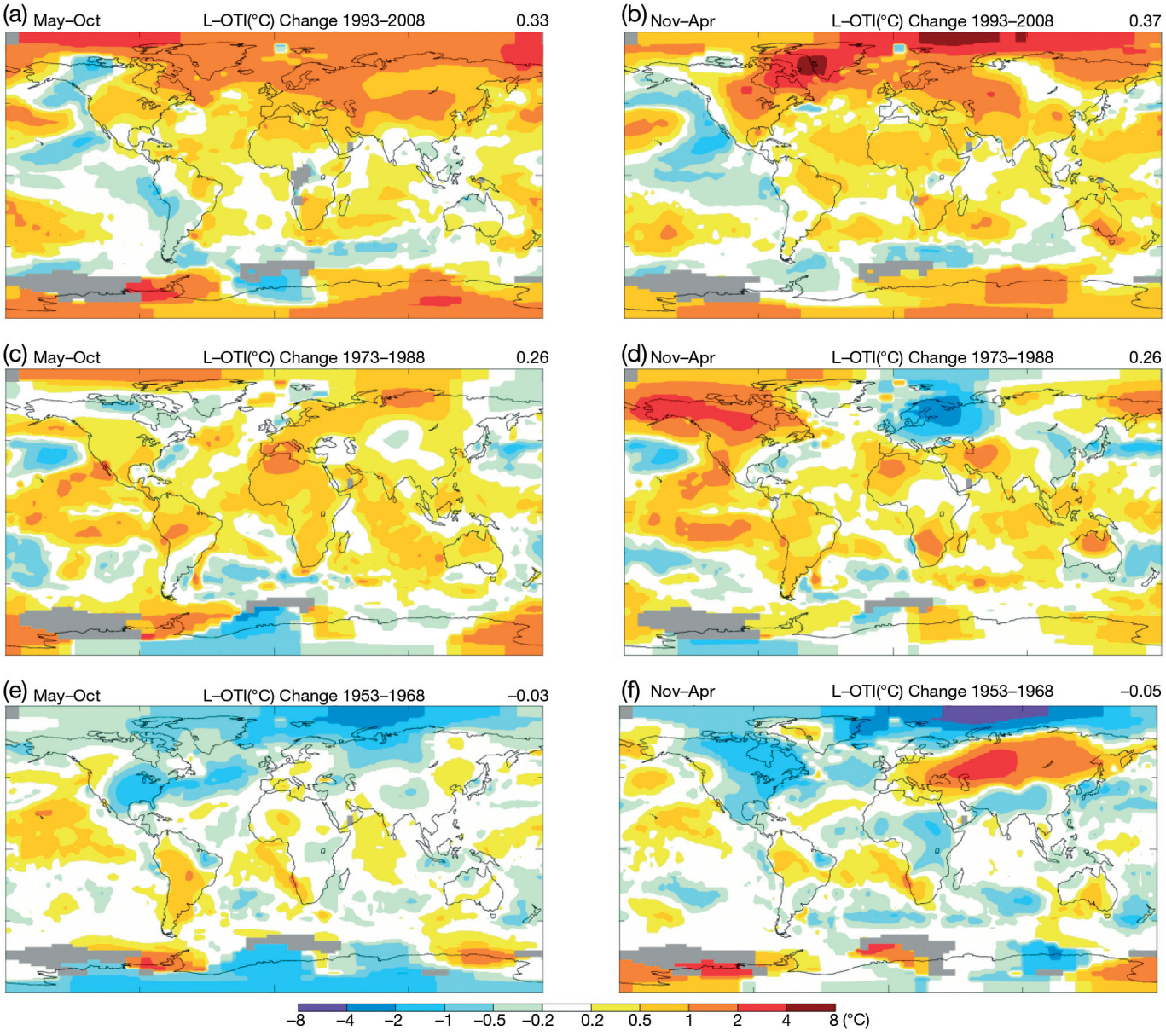


Fig. 1. Global temperature trends for the Northern Hemisphere warm (May to October averages; left side) and cold season (November to April averages; right side) for different 15 yr intervals from (a,b) 1993–2008, (c,d) 1973–1988, and (e,f) 1953–1968. Colors represent average changes over the time interval mapped. L-OTI = land-ocean temperature index. Note the accelerated warming over successive periods, with substantial global variation, and the greatest increases seen at high latitudes. Maps obtained from the NASA Goddard Institute for Space Studies (http://data.giss.nasa.gov/gistemp/maps/, accessed December 2008)

tainty, and thus, the net effects of the predicted environmental changes on the survival, health, and resilience of coral reefs are not yet fully understood (Ward et al. 2007, Maynard et al. 2008).

The most commonly cited environmental factors implicated as causes of declining coral health include: increasing ocean temperature (Harvell et al. 2002), nutrient pollution (Bruno et al. 2003), and over-fishing (Jackson et al. 2001, Hughes et al. 2003). Evidence for these factors as causative agents has been largely circumstantial (Rosenberg et al. 2007), in part because of the difficulty in trying to demonstrate causation in large and complex ecological systems (Plowright et al. 2008).

An examination of the coral disease literature reveals many hypothesized mechanisms for why increasing rates of coral disease have been reported over the last few decades, and there is much controversy. It is difficult to measure and predict the cumulative effects of global environmental change on coral health. Here, I review the hypotheses and the evidence regarding coral infectious disease emergence and potential climatic drivers. I begin by examining the evidence for the recent emergence of many coral infectious diseases. I

then review some of the climate variables likely to change over the coming decades and how these variables may affect disease-related processes, including disease transmission, host dynamics, pathogen growth, and pathogen virulence. Finally, I discuss some current and future challenges to investigating the role of climatic factors in coral disease and decline.

HOW COMMON ARE INFECTIOUS DISEASES OF CORAL TODAY, AND ARE THEY 'EMERGING?'

'Emerging' diseases are those that have recently expanded their incidence, host range, or spatial extent (Daszak et al. 2000). A lack of underwater baseline data has hindered progress in assessing whether coral diseases are emerging. Some paleontological data suggest that the rapid loss of *Acropora* spp. coral in the Caribbean, beginning in the late 1970s and due in part to white band disease outbreaks, was unprecedented in the Holocene (Aronson & Precht 2001). Data for other diseases and regions have been scarce. Ward & Lafferty (2004) cleverly calculated a proxy measure for disease emergence in the absence of baseline data. They adjusted the number of publications related to disease within a group/taxon by the total number of reports (related to disease or not) regarding that group/taxon. Admittedly, this proxy has drawbacks because it does not explicitly control for biases resulting from increased interest or funding within subdisciplines. However, in the absence of baseline data, this proxy represents some of the only available evidence for the emergence of multiple diseases across large spatial and temporal scales. By this proxy, Ward & Lafferty (2004) concluded that disease is significantly increasing for marine mammals, turtles, urchins, and mollusks, and significantly decreasing for fish. For coral, they concluded that the proxy measure could not detect a significant increase in diseases other than coral bleaching.

Several studies have demonstrated increasing coral diseases within sites that have been quantitatively monitored for multiple years (Porter et al. 2001, Lafferty et al. 2004, Borger & Steiner 2005, Francini-Filho et al. 2008) and long time-series compiled for multiple diseases in the ocean collectively suggest an increasing trend (Harvell et al. 1999). However, it has been unclear whether coral diseases, other than bleaching, are increasing beyond expectations based on increasing investigative effort.

Ward & Lafferty's (2004) assessment reviewed articles published up until 2001. A substantial amount of additional coral disease research has amassed since their analysis. Revisiting their techniques, I compiled a list of all articles about coral disease, published up to December 2008, since the first coral disease report in 1965 (excluding those pertaining only to stress-induced bleaching). This was compared to all reports from the same time period retrieved on the ISI Web of Science (Science Citation Index Expanded v. 4.4, accessed December 2008) using the search word 'coral' (limited to subject areas that relate to ecological, oceanographic, or biological sciences, excluding human biomedical research). Both disease and non-disease reports exponentially increased over the observation period. Data were log-transformed and the slopes compared using analysis of covariance (ANCOVA), with time as a covariate. Indeed, when only records up until 2001 were included, there was no detectable increase in infectious disease reports beyond that expected by the rate of increase in all coral reports ($p = 0.69$); however, including reports from the most recent 7 yr revealed a rate of increase that was significantly greater than the background rate of increase for all coral reports (Fig. 2, $p = 0.01$). Thus, the most current available evidence suggests that coral diseases (not just bleaching) are emerging, and coral disease research is rapidly expanding.

What remains a mystery is whether these emerging diseases are predominantly infectious, whether they are manifestations of physical or physiological stress, or whether both infection and stress are involved. An infectious disease is defined as an illness caused by a transmissible biological agent, rather than by genetic, physical, or chemical agents. Transmissible agents can include viruses, bacteria, parasites, fungi, algae, and prions. Most coral diseases, other than some forms of bleaching and certain growth anomalies, are presumed to be caused by infectious pathogens, although this remains to be conclusively demonstrated for most described diseases (Richardson 1998, Lesser et al.

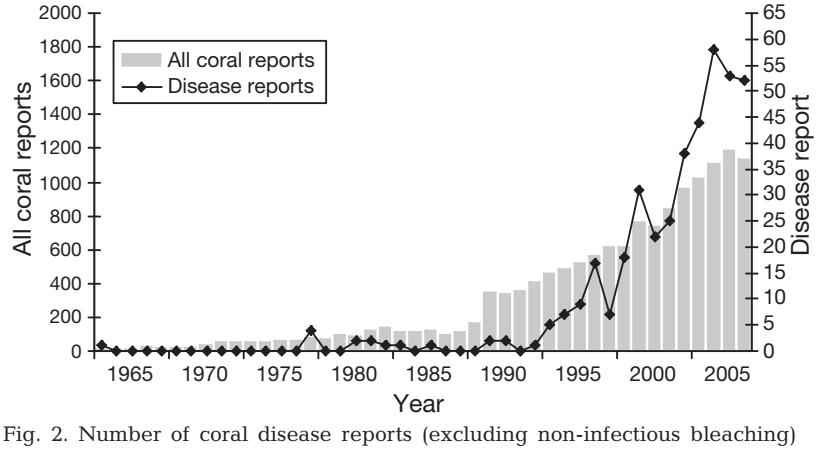


Fig. 2. Number of coral disease reports (excluding non-infectious bleaching) compared with all coral reports over time

2007, Rosenberg et al. 2007). In a few cases, the pathogen or group of pathogens identified as causal during initial studies are no longer capable of producing disease in laboratory challenges (Rosenberg et al. 2007). Also, many coral diseases display similar signs, leading to confusion. For example, several 'white plague-like' diseases of coral have been reported in disparate regions (Croquer et al. 2003, Miller et al. 2003, Pantos et al. 2003, Dalton & Smith 2006). Despite these obstacles, our understanding of microbial causes of coral disease is improving.

At least 6 and up to a dozen pathogens have been implicated as causes of disease in coral. This estimate varies based on different interpretations of what constitutes causation, such as different interpretations about the types of pathogens considered (e.g. whether opportunistic pathogens are included) and what types of evidence (e.g. controlled laboratory studies, field data, Koch's postulates) are sufficient for confirmation (Weil et al. 2006, Lesser et al. 2007, Rosenberg et al. 2007). The implicated pathogens, thus far, form a taxonomically diverse group including marine bacteria (Ritchie & Smith 1995, Kushmaro et al. 1996, 1997, Ben-Haim & Rosenberg 2002, Ben-Haim et al. 2003, Denner et al. 2003, Cervino et al. 2004, Thompson et al. 2006, Bally & Garrabou 2007, Sussman et al. 2008), cyanobacteria and associated members of microbial consortia (Myers et al. 2007, Voss et al. 2007), bacteria from presumed terrestrial sources (Patterson et al. 2002), a ubiquitous fungus (Smith et al. 1996, Geiser et al. 1998), a protozoan (Antonius & Lipscomb 2000), and an alga (Goldberg et al. 1984). The role of viruses as agents of coral disease remains virtually unexplored (Davy et al. 2006, Patten et al. 2008). It is not parsimonious to suppose that this diverse group of coral pathogens emerged coincidentally, although this hypothesis has not been completely ruled out. Thus, what remains in question is, why have all of these diseases emerged simultaneously?

It may be tempting to point towards anthropogenic causes of disease emergence, since coral diseases appear to have emerged concurrently over time with anthropogenic alteration of the global environment. However, a thorough review of the evidence and more basic information about disease pathogenesis will be necessary before causation can be established.

CLIMATE CHANGE IN COASTAL MARINE ECOSYSTEMS

There are numerous simultaneous manifestations of climate change that may theoretically affect coastal marine ecosystems and disease parameters, including changes in seasonality, atmospheric CO_2 concentrations, sea surface temperature, sea level, rainfall, storm intensity, storm frequency, wave climate, and run-off (Table 1). These changes have been summarized recently by the Intergovernmental Panel on Climate Change (Bindoff et al. 2007, Nicholls et al. 2007, Trenberth et al. 2007). Precisely how these climatological changes may influence the rise and fall of coral disease outbreaks remains uncertain. Summarized in the next 4 subsections are some recently recognized climate changes and their theoretical effects on coral infectious disease (Table 1, Fig. 3), and subsequently the section 'Coral disease dynamics and climate' discusses the current evidence for environmental influences on epidemiological parameters including transmission, host population dynamics, pathogen population dynamics, and disease virulence.

Seasonality and increasing sea surface temperature

Ocean temperatures vary over space and time on seasonal, inter-annual, and decadal (or longer) time scales (Fig. 1). Many coral diseases display seasonal variation in prevalence, with higher prevalence in warm summer months (see Rosenberg et al. 2007 for examples). Strong seasonality can be taken as evidence that pathogens and/or hosts are strongly influenced by changes in seasonally fluctuating environmental variables such as temperature, but other variables such as rainfall, light levels, water clarity, run-off, ocean circulation, and nutrients can also fluctuate seasonally, along with temperature (Delcroix & Henin 1991, Lima et al. 1996, Poulos et al. 1997). Showing that prevalence fluctuates seasonally does not conclusively demonstrate a link between temperature and coral disease.

Besides apparent seasonality, empirical evidence linking increasing ocean temperature to coral infectious disease is compelling, but not absolute. Several studies have shown temperature-regulated growth and virulence of pathogens in the laboratory (Banin et al. 2000, Alker et al. 2001, Ben-Haim et al. 2003, Remily & Richardson 2006, Ward et al. 2007). There is some evidence that temperature-regulated bleaching events can be followed by outbreaks of infectious diseases (Guzman & Guevara 1998, Muller et al. 2008), and some mass mortalities in coral caused by disease were coincident with periods of anomalously high water temperatures (Cerrano et al. 2000, Riegl 2002, Bruno et al. 2007). Caribbean yellow band disease prevalence around Puerto Rico, for example, was shown to significantly correlate with temperature over the past decade (Harvell et al. 2009). For some other diseases, average inter-annual changes in prevalence were not directly correlated with ocean temperature; in fact, for aspergillosis in the Florida Keys, USA, the

Table 1. Main climate drivers for coastal marine systems, their trends due to climate change, and their possible effects on the dynamics of coral infectious diseases. ↑ = increase; ↓ = decrease; ? = uncertain; R = regional variability. Adapted from the Intergovernmental Panel on Climate Change 2007 reports: Table 6.2 in Nicholls et al. (2007) and the main text in Bindoff et al. (2007) and Trenberth et al. (2007)

Climate change	Effects on marine and coastal systems	Effects on coral infectious disease dynamics
CO_2 concentration (↑)	Increased CO_2 fertilization; decreased seawater pH (or 'ocean acidification')	Slower accretion/regrowth/recovery of coral hosts May increase coral stress and disease susceptibility Pathogens will respond to pH change based on optimal growth curves
Sea surface temperature (↑, R)	Increased stratification/changed circulation; poleward species migration; increased algal blooms	Increased host susceptibility due to temperature stress Pathogens will respond to temperature based on optimal growth curves
Sea level (↑, R)	Increased erosion; wetland loss (and change)	Possible increased movement of particulates, may increase exposure to land-based pathogens and pollutants 'Drowning' of hosts if accretion rates cannot keep up
Storm intensity (↑, R)	Increased extreme water levels and wave heights; increased episodic erosion, storm damage	Increased intensity of physical damage to coral hosts Possible increased disease inoculation through physical lesions Decrease in coral abundance/density due to increased storm-related mortality, especially near reef crests
Storm frequency (?, R) Storm track (?, R)	Altered surges and storm waves and hence risk of storm damage	Increased frequency of physical damage to coral by storms Possible increased frequency of disease inoculation through physical lesions Disruption of recovery and recolonization after disease events
Wave climate (?, R)	Altered wave conditions, including swell; altered patterns of erosion and accretion	Possible shifts in movement of particulates, may increase exposure to pollutants or pathogens Possible increased sediment stress
Run-off (R)	Altered water quality/salinity; altered fluvial sediment supply; altered circulation and nutrient supply	Increased disease susceptibility from sediment stress Possible increased exposure to land-based pathogens or pollutants Possible increased pathogen growth rates and disease severity due to eutrophication
Seasonality (R)	Lessening of cool season temperature extremes; increase in warm season extremes	Variable effects on host susceptibility and pathogen growth based on optimal temperature response curves Possible increase in chronic, rather than epizootic or seasonal, disease
Overland rainfall (R)	Rainfall (↑) >30° latitude and (↓) in tropics; general increase in droughts and 'heavy' rain events	Possible increased exposure to land-based pathogens or pollutants Possible increased micronutrient supply from atmospheric dust leading to faster rates of pathogen growth Changes in surface salinity with probable, but unknown, effects on disease
Ocean and air circulation (?, R)	Changes in El Nino Southern Oscillation (↑), North Atlantic Oscillation (?), and Meridional Overturning Circulation (?)	Unknown effects on coral infectious disease Possible, but unclear, changes in ocean productivity, reservoirs of infection, or movements of pathogens

opposite relationship was seen, where disease levels steadily decreased after the outbreak peaked, even as ocean temperatures increased (Kim & Harvell 2004). This seems confusing, since laboratory evidence shows positive associations between pathogen growth and temperature (Alker et al. 2001). The phenomenon may be explained by epidemiological dynamics such as the removal of susceptible individuals over time through death or induced disease resistance, leading to a waning of the outbreak, despite increasing temperatures.

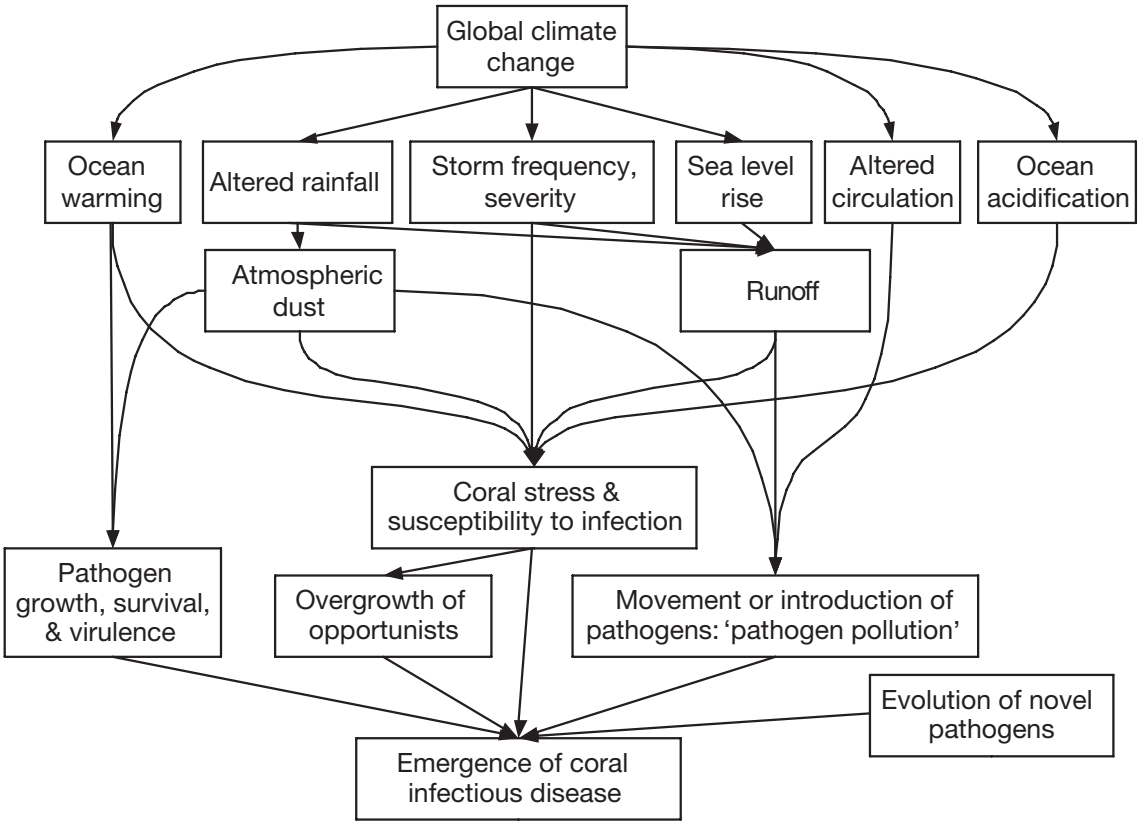


Fig. 3. Causal diagram outlining the hypothesis space for climate change as a cause of coral infectious disease emergence (not considering stress-related bleaching and other non-infectious diseases): a compilation of proposed causal pathways from the coral disease literature

For coral, there may be complex interactions between extrinsic forcing (due to temperature and environmental effects) and intrinsic dynamics (due to the interplay of epidemiological variables such as susceptibility and transmission). For aspergillosis, one laboratory study found that from 27 to 30°C, pathogen growth increased and anti-fungal activity decreased, while at 31°C, pathogen growth decreased and anti-fungal activity sharply increased, demonstrating the complex, non-linear relationship between temperature and epidemiological processes (Ward et al. 2007). These complications demonstrate why direct correlations between disease and temperature are not always obvious in real ecosystems, and how elucidating epidemiological dynamics, along with environmental variables, may be essential in predicting long-term disease trends.

Metapopulation models of disease have demonstrated that seasonal forcing for disease transmission (i.e. wider high and low seasonal extremes in transmission) means a higher probability of disease fade-out (or regional disease disappearance) than is predicted without seasonality (Grenfell et al. 1995). Conversely, if seasonal fluctuations in disease levels decrease as a result of less severe winters and more over-winter pathogen survival — the lessening of cold extremes has been a documented global trend accompanying global warming (Trenberth et al. 2007) — regional coral disease fade-out may become less common, and more endemic disease may occur.

If average temperatures steadily increase in the next few decades, following the recently observed trend — with 1998 to 2005 containing the top 5 warmest years on record since 1850 (Trenberth et al. 2007) — thresholds for pathogen growth and coral susceptibility may become chronically exceeded, leading to more frequent, longer outbreaks (Hughes et al. 2003). There is a limit to this effect: as temperature increases beyond the upper tolerance limits for pathogen survival, then pathogens may begin to die out during periods of maximal temperature (Lafferty et al. 2004) and outbreaks could once again become more scarce. These predictions are complicated by the likelihood that both coral hosts and pathogens will adapt over time, and current observed temperature relationships may represent

snapshots within a shifting baseline (Done 1999, Hughes et al. 2003, Edmunds & Gates 2008).

Increased CO_2 concentrations and ocean acidification

Dissolved CO_2 forms a weak acid so that the uptake of anthropogenic CO_2 leads to chemical alteration of the ocean, and global mean ocean pH has decreased by about 0.1 pH units since 1750 (Bindoff et al. 2007). The uptake of CO_2 also shifts the balance of carbon species (CO_2–carbonate–bicarbonate) and leads to decreased ability of coral to accrete $CaCO_3$ into their skeletons (Kleypas et al. 1999). The mean pH of ocean surface waters is 7.9 to 8.3 (Bindoff et al. 2007), compared to 5.8 for a typical coral surface mucous layer (Remily & Richardson 2006). Coral pathogens have different growth rates in different pH conditions, and temperature can change the pH tolerance of pathogens (see 'Pathogen dynamics — pathogen reservoirs in the environment'). Experimental evidence and modeling predictions preceded observational evidence for changes in ocean chemistry, but acidification is now unequivocally observed in the global records (Bindoff et al. 2007). Acidification may act synergistically with other environmental stressors to increase the probability of coral species extinctions (Jackson 2008). Other than direct effects on pathogen growth and coral accretion rates, the effects of ocean acidification on coral infectious disease are relatively unknown.

Storm frequency and intensity

Globally, estimates of the frequency and potential destructiveness of hurricanes, cyclones, and typhoons show a significant upward trend: a large increase in numbers and proportion of hurricanes reaching Categories 4 and 5 has been observed since 1970, with trends strongly correlated with changes in sea surface temperature (Trenberth et al. 2007). The effects of increasing storm frequency and intensity on coral infectious disease dynamics are not completely clear. One recent study documented 'disease-like' mortality on remnant populations of *Acropora palmata*, a coral species federally listed as threatened, after a severe hurricane season in the Florida Keys, USA (Williams et al. 2008). Diseases that kill large branching coral, such as white band on *Acropora* spp., leave skeletons weakened, which can increase rates of bioerosion during storms (Riegl 2002). Increased fragmentation of weakened, diseased coral could potentially increase pathogen transmission, and stress from hurricane damage and physical lesions acquired during storms could increase coral susceptibility to infectious disease, but empirical evidence is scarce.

Sea-level rise, rainfall, and increased run-off

Sea level rose at an average rate of 1.7 ± 0.5 mm yr^{-1} during the 20th century, and during the period between 1993 and 2003, the rate of rise was nearly double that average (Bindoff et al. 2007). Overland rainfall has been decreasing in the tropics and increasing in temperate zones, and the frequency of drought and extreme rain events has been increasing in all areas (Trenberth et al. 2007). A combination of sea-level rise and changes in rainfall could synergistically alter run-off and salinity in coastal ecosystems.

There are multiple examples that demonstrate that increased sedimentation, nutrients, and run-off affect coral health. Experimental nutrient enrichment increases the spreading rate of several coral diseases (Bruno et al. 2003, Voss & Richardson 2006), and nutrient limitation has been hypothesized to control microbe and algal growth in oligotrophic reef ecosystems (Szmant 2002). Lesions and tissue necrosis on *Porites* spp. coral were shown to positively correlate with increased wave exposure and sedimentation, although specific diseases were not distinguished (Wesseling et al. 2001). Increased sedimentation has been implicated as a driver of coral mortality through smothering and facilitating algal competition (Nugues & Roberts 2003), and spatial analyses reveal that coral reefs are currently much less likely to exist near river run-off (McLaughlin et al. 2003). Sediment damage to coral can be decreased in an experimental setting by treatment with antibiotics (Hodgson 1990), indicating a link with bacterial overgrowth. Sea level rise can result in 'drowning' of coral due to light limitations with increasing depth (Knowlton 2001), and increased run-off may elevate exposure to land-based pollutants and pathogens (Patterson et al. 2002, Lafferty et al. 2004).

CORAL DISEASE DYNAMICS AND CLIMATE

Transmission rates

Infectious coral pathogens presumably invade susceptible colonies (which become infected) and then spread to other susceptible colonies either directly through the water column or through a vector or an intermediate host. Mounting evidence has demonstrated that many, but not all, coral diseases appear to fit a contagion model of transmission (i.e. can be transferred locally from host to host), including white plague type II (Richardson et al. 1998), some growth

anomalies (Kaczmarsky & Richardson 2007), bacterial bleaching (Ben-Haim & Rosenberg 2002), and black band disease (Kuta & Richardson 1996, Bruckner et al. 1997, Page & Willis 2006). Spatial data for other diseases, such as sea fan aspergillosis and Caribbean yellow band disease, are not consistent with contagion as the sole mode of transmission (Jolles et al. 2002, Foley et al. 2005). Evidence exists for vectored transmission of some coral diseases (Sussman et al. 2003, Nugues et al. 2004, Grober-Dunsmore et al. 2006, Miller & Williams 2007), although vectored transmission is generally considered rare in marine compared with terrestrial systems (McCallum et al. 2004). There is no evidence of vertical transmission (spread from parent to offspring through gametes) for any described coral disease, but this may be due to a detection bias, because planulae and recruits are rarely directly studied and are not typically examined for pathogens.

Transmission rates could theoretically be affected by all the climate drivers discussed above, although empirical data are scarce, in part because transmission is difficult to measure. It was recently shown that attachment of *Vibrio shiloi* to coral hosts was inhibited when the bacteria were grown at low temperatures and fostered when bacteria were grown at high temperatures, indicating that transmission may be facilitated by temperature (Banin et al. 2000). Remily & Richardson (2006) showed that increasing water temperature changed the pH tolerance of *Aurantimonas coralicida*, allowing growth to occur at acidic conditions comparable to coral mucus, suggesting that temperature may regulate pH tolerance and infectivity of this pathogen. Transmission is likely to be increased when hosts are stressed (Lafferty & Holt 2003), and extreme temperature, sediment, and UV exposure can stress coral and damage zooxanthellae (Anderson et al. 2001, Sutherland et al. 2004).

Host dynamics — coral recruitment and mortality rates

Coral establishment and survival are highly dependent on available space and appropriate environmental conditions (Harrison & Wallace 1990). Recruitment, survival, connectivity, and density of susceptible hosts are important determinants of infectious disease risk (Kermack & McKendrick 1927, Bartlett 1957, Hess 1996, Bruno et al. 2007). Thus, stressors that affect host population dynamics may also affect infectious disease dynamics. Yet, there is very little known about the recruitment and dispersal of coral propagules, and even less is known about the effect of climate change on these processes, although coral are known to have a narrow temperature tolerance (Barber et al. 2001). Some data support a trend of decreasing recruitment over time, coincident with collapsing coral populations, increasing temperatures, and increasing disease burdens (Connell et al. 1997). A recent study demonstrated that temperature affects larval survival in a similar manner for a wide range of marine taxa with planktonic larvae, demonstrating lower larval dispersal potential at higher temperatures, although this analysis of 72 different species of marine animals did not include any coral (O'Connor et al. 2007).

There is some evidence that coral fecundity is affected by bleaching and disease (Petes et al. 2003). For example, mean polyp fecundity has been shown to decrease with bleaching, with severely affected polyps producing as few as zero gametes for the reproductive season directly after a bleaching event (Michalek-Wagner & Willis 2001). Larger colonies within a species are typically more fecund, and so partial mortality or changes in population size-class distributions caused by disease may decrease coral recruitment (Richardson & Voss 2005). Conversely, dead coral skeletons exposed by infection are rarely observed hosting new coral recruits; rather, coral substrate liberated by disease mortality is usually rapidly colonized by fleshy algae (Miller et al. 2003, Borger & Steiner 2005), although one study demonstrated no difference in recruitment to areas killed by black band disease compared with areas killed by hurricane damage (Edmunds 2000).

There is an important circular feedback predicted for coral populations affected by disease and climate change: temperature influences both host dynamics and disease, while in turn, disease influences host recruitment and mortality rates, which influences disease risk. Thus, the cumulative effects of climate change and disease on coral population dynamics over time are neither trivial to predict nor additive (Lafferty & Holt 2003).

Pathogen dynamics — pathogen reservoirs in the environment

The relationship between environmental variables and the growth and survival of several coral pathogens have been described, including the agent of white plague type II (Remily & Richardson 2006), the agent of sea fan aspergillosis (Alker et al. 2001, Ward et al. 2007), and several agents of bacterial bleaching (Banin et al. 2000, Ben-Haim et al. 2003). These studies report, in common, that higher temperatures lead to higher pathogen growth rates, within certain optimal growth bounds. That is, within the bounds at which most of these pathogens grow optimally, between typical tropical ocean winter (15 to 25°C) and summer temperatures (27 to 35°C), pathogen growth rates are positively

correlated with temperature. However, for several of these pathogens, it was also demonstrated that the relationship between growth and temperature was reversed when temperatures exceeded a maximum threshold of approximately 31 to 35°C (Remily & Richardson 2006, Ward et al. 2007).

Disease virulence

There is evidence that the temperature regulation of coral infectious disease can occur through temperature-sensitive induction of virulence factors, resulting in higher disease severity, transmission, or rates of disease progression. For bacterial bleaching of *Oculina patagonica* corals, the growth rate of the implicated pathogen *Vibrio shiloi* (called *Vibrio* strain AK1 in earlier studies) was not substantially hindered at low temperatures (16°C), but virulence factors expressed by the pathogen were down-regulated (Kushmaro et al. 1998). Adhesion of the *V. shiloi* bacteria to *O. patagonica* hosts did not occur at either 16 or 25°C when the bacteria were grown at 16°C, but did occur at both temperatures when bacteria were grown at 25°C, suggesting that factors up-regulated in the pathogen grown at higher temperatures affected infectivity or virulence (Banin et al. 2000). Similarly, bacterial bleaching of *Pocillopora damicornis* corals by *V. coralyticus* resulted in coral tissue lysis at 27°C, but not at lower temperatures; the pathogen was shown to express a proteinase (a virulence factor) at much higher levels when grown at 27°C than at lower temperatures (Rosenberg & Ben-Haim 2002).

CURRENT AND FUTURE CHALLENGES TO INVESTIGATION OF CLIMATE DRIVERS OF CORAL DISEASE

Rise and fall of coral disease outbreaks

By nature, population-level fluctuations of infectious disease are highly dynamic: disease incidence rises as pathogens spread among susceptible hosts and then falls as infected hosts are removed by death or recovery. These systems are inherently non-linear and they pose challenges for traditional (often static) statistical analyses. More appropriate dynamic epidemiological models may assist investigation of the effects of environmental variables on disease emergence. Many insights about the dynamics of infectious disease have resulted from careful application of epidemiological models, beginning with seminal work on human diseases and terrestrial host-parasite systems nearly a century ago (e.g. Kermack & McKendrick 1927, Bailey 1957, Bartlett 1957, Anderson & May 1979). However, there is a clear paucity of studies applying epidemiological models to coral infectious disease, and this represents a promising research frontier both for understanding coral disease dynamics and for elucidating the connections between disease outbreaks and environmental drivers (Harvell et al. 2004). Because of the prolonged nature of coral disease outbreaks, with each outbreak lasting up to a decade (Kim & Harvell 2004, Sokolow et al. 2009), longer-term studies are essential.

Issues of scale — local management vs. global climate change

The dynamics of coral infection (or pathogen colonization of coral) can be studied at various spatial scales, from infection of an individual polyp, to a colony, the holobiont, a local population, regional communities, and finally, the entire globe. Several coral diseases have been shown to spread widely across vast areas of reef (a few km to hundreds of km) in relatively short periods of time (weeks to months) (Richardson et al. 1998, Kim & Harvell 2004). Because of the potential for widespread movement of disease agents, coral infection dynamics focusing at the level of individual polyps or colonies must be complemented by studies at larger spatial scales. Scales relevant to management of coral populations range from local to regional politically or geographically defined areas. Changes in climate, on the other hand, are global, although there is wide variability among regions (Fig. 1), and discovering how these global processes affect local or regional coral health is not trivial. In order to detect climate effects on coral disease, data must be collected at much larger spatial scales than currently available. This will require cooperation among investigators in disparate regions and across political boundaries.

Is Koch 'dead'?

In 1969, at the annual conference of the Wildlife Disease Association, Dr. Robert Hanson, a distinguished virologist and then the director of the International Center for the Control of Animal Disease at the University of Wisconsin, gave a speech entitled 'Koch is dead' (reprinted in Hanson 1988) wherein he outlined the reasons why 'the ritual which has evolved [from Koch's original sound postulates] into present day experimental studies [of disease causation] has obscured almost completely what occurs in natural processes outside the laboratory' (p. 193). Hanson's speech nearly 40 yr ago has relevance for the modern problem of assessing the influence of climate change on the emergence of coral disease.

Many diseases recognized by modern medicine have multifactorial etiologies, meaning that multiple factors are required to produce disease signs. Coral research is likewise beginning to reveal that most coral diseases probably have multifactorial etiologies. For example, Bruno et al. (2007) demonstrated that the coral disease 'white syndrome' occurred most commonly where both temperatures and coral cover were high, while Sussman et al. (2008) reproduced this disease in laboratory aquaria by introducing any 1 of 6 genetically distinct bacterial isolates. Kushmaro et al. (1997) showed that bacterial-mediated bleaching of *Oculina patagonica* was enhanced in warmer water and prevented when temperatures were decreased to 16°C or when antibiotics were applied, meaning that both appropriate temperature and bacteria were required for disease to occur.

In assessing complex diseases, it is useful to distinguish whether the many factors involved in disease causation are 'necessary', or 'sufficient', or both (Box 1). Known as the 'disease triad', specific host–pathogen–environment combinations may influence disease. Demonstrating that certain pathogens must be present does not preclude the requirement that specific environmental, physical, or host-immunological-status factors must also be met for disease signs, nor vice versa. Rather, the perspective of multifactorial etiologies requires synthesis of the many drivers of coral disease emergence into a unified framework, and calls for the measurement of participation of each factor in natural ecosystems, before reaching conclusions about causation.

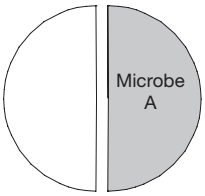
Koch's postulates entail 3 main criteria for establishing causality: (1) a suspected pathogen must be present in every case of the disease, (2) it should occur in no other disease as a non-pathogenic parasite, and (3) it must be isolated in pure culture, repeatedly passed, and induce the disease anew (Koch 1942). Multifactorial etiologies render Koch's postulates impossible to apply, since the postulates do not consider non-infectious factors and rely on the simplifying assumption of one-pathogen-one-disease (Wobeser 1994). Likewise, Koch's postulates are impossible to fulfill when disease is caused by non-culturable pathogens (Evans 1978); this includes many viruses and marine bacteria. For example, Israely et al. (2001) showed that 99 to 99.9% of *Vibrio shiloi* bacterial pathogens were in a viable-but-not-culturable state. These challenges have been recognized by many coral disease research groups, and some have offered robust biomedical research tools to overcome them (Richardson et al. 2001, Ritchie et al. 2001, Downs et al. 2005, Plowright et al. 2008, Work et al. 2008).

Although Koch's postulates can be useful, as Hansen warned in 1969, failure to fulfill the postulates does not *disprove* causation, nor does the reproduction of disease signs in a controlled laboratory setting demonstrate all factors capable of causing a disease. Caution

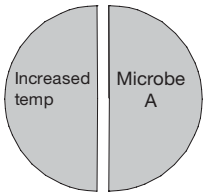
Box 1. Epidemiology of multiple causes: the concepts of necessary and sufficient. Disease signs occur only when the circles are completely shaded

In epidemiological terms, 'necessary causes' are factors that must be present for disease to occur but which alone may or may not be capable of causing disease. 'Sufficient causes' contain a complete set of factors capable of producing a disease in question. For example, in terms of coral disease, the presence of a particular microbe may be necessary but not sufficient for disease to occur (A), whereas the presence of that microbe along with an increase in water temperature may together comprise a sufficient cause of disease (B). However, if other stressors besides the increase in water temperature could produce a similar effect, then alternative stressors, such as increased sediment or ultraviolet radiation or nutrient stress, together with the presence of the microbe, may comprise another sufficient cause of the same disease (C). Similarly, if there are multiple strains or species of microbe that can fill functional groups within a pathogenic consortium, the presence of each single microbial species may not be strictly necessary to produce disease signs (D and E). The existence of these complexities highlights the importance of identifying all alternative individual factors involved in each coral disease, such as microbial pathogens or temperature, and determining causal roles as 'necessary,' 'sufficient,' or both. Experiments and field observations to assess alternative causes can clarify the significance of a single causal factor within the wider context of regional or global disease emergence.

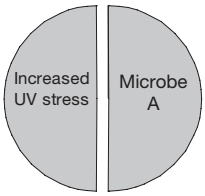
(A) necessary but insufficient cause of disease X



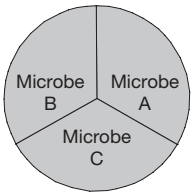
(B) sufficient cause of disease X



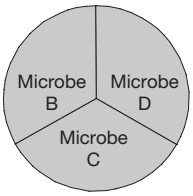
(C) alternative sufficient cause of disease X



(D) sufficient cause of disease X



(E) alternative sufficient cause of disease Y



in interpreting initial challenge studies is recommended because alternative 'sufficient causes' may exist (Box 1), and factors not identified by laboratory challenge studies may nevertheless be relevant in real ecosystems.

Ultimately, a clearer understanding of all 'sufficient causes' of a disease, including all combinations of pathogens and non-infectious factors, will facilitate investigations of climatic drivers of disease emergence at the population, ecosystem, and global scales, and will guide management options. It is becoming increasingly clear that multifactorial etiologies are common among coral diseases, and that active cooperation among coral scientists and managers will be invaluable for unraveling the causal web.

CONCLUSIONS

Coral disease research has increased exponentially in the last several decades. Evidence is mounting that many coral diseases are caused by microbial pathogens and outbreaks of infectious disease are increasingly common. Researchers have recognized some of the complexities in coral disease dynamics and have expanded beyond the simple prediction that climate warming equals increased disease, but a lot of unknowns remain. Despite great strides over the last decade, much uncertainty still exists regarding the mechanisms of coral disease emergence, connections with climate variables, and the best management options. Coral reefs will inevitably continue to experience disease outbreaks, and consequently, it is feared that the synergistic effects of many compounding stressors may push coral reefs to a tipping point, propelling some species towards extinction and leading to loss of critical reef habitat (Jackson 2008). Current evidence is still preliminary but is increasingly suggestive that mitigating the effects of climate change may help reduce the emergence of disease and improve the health of coral reef ecosystems. In this rapidly changing world, discovering the environmental drivers of coral disease emergence at large spatial and temporal scales remains a challenge and a priority.

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