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Increases and decreases in marine disease reports in an era of global change

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Outbreaks of marine infectious diseases have caused widespread mass mortalities, but the lack of baseline data has precluded evaluating whether disease is increasing or decreasing in the ocean. We use an established literature proxy method from Ward and Lafferty (Ward and Lafferty 2004 *PLoS Biology* **2**, e120 (doi:10.1371/journal.pbio.0020120)) to analyse a 44-year global record of normalized disease reports from 1970 to 2013. Major marine hosts are combined into nine taxonomic groups, from seagrasses to marine mammals, to assess disease swings, defined as positive or negative multi-decadal shifts in disease reports across related hosts. Normalized disease reports increased significantly between 1970 and 2013 in corals and urchins, indicating positive disease swings in these environmentally sensitive ectotherms. Coral disease reports in the Caribbean correlated with increasing temperature anomalies, supporting the hypothesis that warming oceans drive infectious coral diseases. Meanwhile, disease risk may also decrease in a changing ocean. Disease reports decreased significantly in fishes and elasmobranchs, which have experienced steep human-induced population declines and diminishing population density that, while concerning, may reduce disease. The increases and decreases in disease reports across the 44-year record transcend short-term fluctuations and regional variation. Our results show that long-term changes in disease reports coincide with recent decades of widespread environmental change in the ocean.

1. Introduction

Infectious diseases have severely impacted marine organisms across a wide variety of taxa, from wasting diseases in sea stars to fibropapilloma tumours in turtles and phocine distemper in seals [1–3]. Mass mortalities and morbidity from these recent disease outbreaks raise the question of whether the risk of infectious disease is different in contemporary oceans relative to the past, and if so, what the causes might be. Both increases and decreases of infectious disease may be important signals of anthropogenic effects on ocean biota. However, in the absence of long-term records on marine disease and a true baseline, it has been difficult to quantify whether marine disease has increased, decreased or remained stable in recent decades [4,5], and the degree to which anthropogenic change drives disease risk across multiple decades. This challenge exists for marine and terrestrial systems alike, and stems from the absence of baseline data on disease levels prior to the present era of rapid global change.

Infectious diseases occur in all ecosystems, both healthy and unhealthy, and play an important role in structuring biological communities [6]. Abiotic and biotic components of environmental change can disrupt host–parasite interactions and lead to an increase or decrease in disease risk. In recent decades, ocean conditions have shifted dramatically owing to anthropogenic stressors, including climate change, invasive species, pollution and overfishing. Climate

change drivers known to influence marine disease include warming temperatures, increasing acidification, changes in precipitation and storm damage [7]. For example, bleaching events on coral reefs, which occur during prolonged periods of elevated temperature, have been closely linked to subsequent outbreaks of disease in many coral species [8,9]. However, climate change may not always lead to increases in disease, as when increasing temperatures exceed a parasite's thermal optimum or lead to parasite range contractions [10,11]. Likewise, invasive species can increase disease by introducing new parasites or acting as reservoir hosts for existing diseases, but they can also decrease the parasite load in native species if they are unfit hosts for specialist native parasites [12]. Despite its many ecological costs, overfishing is generally expected to decrease disease risk because it can directly kill parasites, increase host immunity by preventing overcrowding, and limit disease transmission by isolating host metapopulations and decreasing host density [13–16]. Whether the outcome is an increase or decrease in disease risk, anthropogenic stressors that modify host–parasite interactions can lead to drastic ecological consequences.

Amidst a shifting ocean environment, several ecological foundation and keystone species have been driven to endangerment by disease, including two species of Caribbean coral [17], three species of California abalone [18], and at least one species of west coast sea star [19]. The recent disease-driven decline of the sea star *Pycnopodia*, a pivotal predator, has caused a trophic cascade through exploding urchin abundance and consequent declines in kelp [20–22]. Disease outbreaks can thus imperil both host and non-host populations. Indeed, the first recorded historical extinction of a marine invertebrate, the extirpation of the limpet *Lottia alveus* across its entire western Atlantic range, was caused by the eelgrass wasting epidemic in the 1930s [23]. Meanwhile, decreases in disease can disrupt an ecosystem if parasites regulate host populations and their absence allows the host species to increase unfettered [24]. Increases in the host population may restructure the ecological community through trophic cascades and other food web interactions, potentially altering energy flow and species richness. The decline in one parasite may also exacerbate the spread of a second parasite [6]. The ecological consequences of increases or decreases in disease are especially disruptive during a disease swing, which we define as a long-term (multi-decadal) shift in disease impact across an entire group of related hosts. However, there have been few opportunities to test whether disease swings are occurring because of the requirement for long-term data on multiple species.

In their 2004 paper, Ward and Lafferty (hereafter WL04) tackled the question of whether marine disease has been increasing or decreasing. They conducted a Web of Science literature search for each year from 1970 to 2001 and normalized the number of wild disease reports by research effort through dividing by the number of papers published on that taxonomic group in that year. The analyses showed that disease reports increased in mammals, urchins, turtles and molluscs [5]. Although disease reports decreased in fishes, Wood *et al.* [13] revisited the analysis and found it was a non-significant decreasing trend [13]. Still, WL04 identified non-significant decreases in disease in fishes, decapods and sharks, all of which had been subject to high fishing pressure [5,13].

The period 2001–2013 was one of intense anthropogenically driven change. At the time, these were 13 of the 14 warmest years on record [8] that precipitated ecological disasters such as mass coral bleaching in 2002 (Great Barrier Reef, [25]), 2005 (Caribbean, [26]) and 2010 (global event, [27]). Meanwhile, there has been a continued onslaught of invasive species under increased globalization [28], and overfishing pressure has expanded to new species and geographical regions [29]. These anthropogenic stressors are expected to disrupt stable, long-term patterns of disease, and can result in increase or decrease in different taxonomic groups. We tested the hypothesis that disease reports have shifted across nine marine taxa from 1970 to 2013, and also during the most recent 13-year period (2001–2013). We included the 2001–2013 period to assess the potential for changes on the scale of a single decade, based on the precipitous environmental changes since 2001 and the mechanistic links between anthropogenic drivers and disease outbreaks. We assessed long-term change in the health of marine biota by replicating the method developed in WL04: we conducted a Web of Science literature search for disease reports in each year and calculated trends in normalized disease reports using taxon results. The consistent method allowed us to combine the dataset from WL04 (1970–2001) with our updated records (2001–2013). Thus, our analyses test for positive and negative disease swings over a 44-year period using the longest and most up-to-date record of marine disease reports across multiple taxa.

The literature proxy method used in this paper and in WL04 is a useful approach, but has limitations. It may reflect trends that are artefacts of the research process (e.g. in the identification and subsequent reporting of a new finding) or miss true trends in nature (e.g. cryptic increases in disease that are masked by other disturbances). The method may suffer from biases, which we investigate and address where possible. However, the literature proxy provides information on decades-long trends in marine disease that is not available from any other source. For all the analyses, we (i) used only records from natural populations to provide the best estimate of disease, (ii) normalized disease reports, and (iii) incorporated a 3-year time lag. These approaches should minimize the effects of funding cycles, research effort and time delays on patterns in disease records [30,31]. Grouping hosts into high-level taxa also reduces the influence of fluctuations in publication rates that may occur in a single host–parasite system or for a specific disease type [30,32]. Still, bias probably remains in some capacity from factors that are difficult to account for, such as the emphasis on novel results and taxon-specific biases (e.g. the influence of stranding on data for marine mammals, [30]).

Whereas WL04 compared literature results to the generally increasing trend in rabies reported in Rupprecht & Smith [33], we include a comparison of actual rabies records and the literature proxy results to ground truth the method. Owing to the strong observational and mechanistic connections between temperature and coral disease, we also expanded beyond describing temporal trends in disease reports to test the hypothesis that reports of disease in corals track warming oceans in the Caribbean, a hotspot for coral disease [34]. This enhanced analysis considers warm temperature anomalies on reefs in the Caribbean from 1970 to 2013, capitalizing on high-resolution environmental data for a sessile host species. By leveraging the longest existing record of marine disease reports across multiple taxa, we advance the understanding of disease swings across a period of intense globalization and anthropogenic change.

2. Methods

(a) Web of science searches

We searched all databases on Web of Science (Institute for Scientific Information, v. 5.27) for papers published from 2001 to 2015 with titles containing specific host taxonomic strings, alone and in combination with the disease string (hereafter 'taxon' and 'disease' searches, respectively). Modifications to the WL04 search strings were made to further exclude irrelevant articles ([5], electronic supplementary material, table S1).

In both taxon and disease searches, we read 50 records or 20% of the total number of results (to a maximum of 200) per year, whichever was greater, replicating the approach in WL04. In selecting 20% of titles, we first sorted the results pages by 'Times Cited' and randomly selected a subset of pages to avoid clustering by author. Page selections with the same author or journal listed more than twice were not chosen and a different page was randomly generated. We believe this random sorting method achieves the same goal as the random sorting by titles in WL04, which is no longer available as a feature in Web of Science [5]. The total number of relevant reports was calculated as the proportion of relevant articles in the subsample multiplied by the total number of titles returned.

We read titles to determine relevance for the taxon searches, but read both titles and abstracts for the disease searches to ensure accuracy. This improvement does not alter the overall results relative to reading titles alone, the method in WL04 (electronic supplementary material table S6 and figure S1). As in WL04, we only included reports of disease in nature and thus excluded literature about disease in aquaculture, farms, zoos, aquariums, transplants, and hatcheries, or about experimental or laboratory infections. Additionally, we did not include articles from post-harvest studies (i.e. frozen foods, fish markets, etc.), freshwater systems, reviews, or studies that reported on the microbiome without signs of disease. Likewise, reports of antibodies to a pathogen (in marine mammals) had to be associated with a current disease state to avoid counting immunity as current infection. Articles pertaining to stranding (in marine mammals) were also not included unless the study provided evidence of disease. Reports of coral bleaching were not included in the analysis unless bleaching was linked to the presence of a parasite, as coral bleaching is typically not an infectious disease.

We used the dataset from WL04 for 1970–2001 for all taxa except for fishes, for which we used the revised dataset in Wood *et al*. [5,13].

(b) Data analyses

The number of disease reports per year was normalized for research effort in each taxon by dividing by the number of taxon results for that year. We calculated the time lag between the year in which a disease report occurred and the year of publication and determined that three years was an accurate approximation (average 3.1, s.d. = 0.51, electronic supplementary material, table S5). This also matched the 3-year time lag WL04 used in their calculations [5]. Given this 3-year lag, our dataset runs through to 2015 and translates to analysis of reports through to 2013. A 3-year running mean was used to account for variability in time lags between the initial observation and the publication. For the 3-year averages that included 2001, the point at which our data overlap with that of WL04, we used an average of the values from the two studies (spanning 2000–2002). Finally, we analysed change over time by testing the correlation between time in years and normalized disease reports using Spearman's ρ ($\alpha = 0.05$). We used Holm's sequential Bonferroni adjustments to account for multiple testing using p.adjust in the R package 'stats' with $n = 10$ for the 2001–2013 dataset and $n = 9$ for 1970 to 2013. WL04 also used Spearman's ρ and Holm's Bonferroni. We added echinoderms for 2001–2013 given recent outbreaks in sea stars. In addition to testing change over time, we used Spearman's ρ to test the correlation between the temperature anomalies and normalized disease reports in corals in the Caribbean from 1970 to 2013.

To determine which diseases could be driving temporal patterns, we identified years of high disease 'peaks' as those with normalized diseased reports greater than one standard deviation from the 1970 to 2013 mean for each taxon. For 2001–2013, we separately tallied instances of hosts and parasites/diseases in the titles and abstracts of each peak's articles. We report the number of instances as distinct from the number of records, as multiple hosts or parasites could be described in a single article and specific host–pathogen systems could not always be ascertained.

We calculated confidence intervals for each year of data for each taxon by using the ratio of successes to total experiments using 'confint' with 'method = exact' in the R package binom [35].

(c) Addressing potential biases

Firstly, as suggested by WL04, the literature proxy calculations could be skewed by prolific authors. This is particularly the case if papers by prolific authors are numerous relative to the total number of reports and clustered in time. WL04 found that removing the most common author did not affect the trends in any of the taxa from 1970 to 2001 [5]. To assess the potential bias for 2001–2013, we repeated analyses for corals and molluscs with the most common author removed, and for elasmobranchs with the two authors tied for the most common removed.

Secondly, results were checked for duplicate titles within and between all years for each taxon search. Though relatively uncommon, any duplicates were omitted to avoid skewing results. Disease results were also checked for duplicate titles. WL04 evaluated the influence of multiple reports of the same disease event from 1970 to 2001 and found no effect in turtles, corals, urchins, mammals, elasmobranchs and seagrass, justifying the use of normalized disease reports for all taxa. We conducted a similar analysis from 2001 to 2013 in corals by checking all disease results for reports of the same disease in the same species at a single time and place. Many records were about multiple diseases in multiple species, in which case the record was only retained if it contained a unique species-disease combination. If uncertainties arose owing to a lack of information, we excluded the record to err on the side of caution.

(d) Rabies analyses

We evaluated the accuracy of our method by comparing the results of the literature proxy method with actual reports of disease for rabies in the United States (US), one of the only long-term datasets on wildlife disease. We acquired data on the number of rabies cases from the Centers for Disease Control and Prevention (CDC) from 1970 to 2013 [36]. WL04 illustrated the accuracy of their dataset by showing that their data recapitulated the general increase in rabies from 1970 to 2001 [5]; we extended upon this approach by comparing literature proxy results for 1970 to 2013 to trends from actual records. Only US cases were included in order to match the scope of the CDC case data.

(e) Temperature anomalies

We assessed the correspondence between anomalous temperature conditions and disease reports for Caribbean corals using heat stress derived from sea surface temperature (SST) and the literature proxy method. Given the widespread availability of abstracts and the frequency of location information in titles, we were able to determine which disease reports were from the Caribbean for the entire time period (1970–2013). For the period 1985–2013, we used the CoralTemp SST data product (0.05° daily resolution; [37]) from the 623 satellite pixels identified as containing coral reefs by the National Oceanic and Atmospheric Administration

(NOAA) Coral Reef Watch (described in [27,38]). Heat stress events in which the degree heating weeks (DHW) metric reached or exceeded 4°C weeks have shown statistically significant correlation with bleaching events [26,38,39]. For the pre-satellite era, (1970–1984), SST data from the HadISST dataset (1° monthly resolution; [40]) were used to calculate degree heating months (DHM) for each of the 134 reef-containing pixels, following Donner *et al.* [41]. The DHM threshold associated with bleaching is 1°C month [41]. For each dataset and year, the proportion of reef pixels for which the annual maximum heat stress exceeded the threshold was determined for comparison with the record of disease.

3. Results

(a) Changes in disease reports over time

The patterns in normalized disease reports over time varied among taxa and temporal intervals. From 1970 to 2013, normalized disease reports significantly increased in both corals ($\rho = 0.55$, $p = 6.4 \times 10^{-4}$) and urchins ($\rho = 0.83$, $p = 3.9 \times 10^{-11}$; figure 1*a*). Normalized disease reports significantly decreased in elasmobranchs ($\rho = -0.59$, $p = 2.5 \times 10^{-4}$) and fishes ($\rho = -0.66$, $p = 1.6 \times 10^{-5}$), and decreased non-significantly (p-value between 0.05 and 0.10) in decapods ($\rho = -0.36$, $p = 0.089$; figure 1*b*). Other taxa did not significantly change over that period (figure 1*c*). Statistically significant changes in the period 2001–2013 were apparent only for marine mammals (increase, $\rho = 0.84$, $p = 5.9 \times 10^{-3}$; figure 1*a*) and fishes (decrease, $\rho = -0.85$, $p = 3.5 \times 10^{-3}$; figure 1*b*), with a non-significant decrease in molluscs ($\rho = -0.69$, $p = 0.089$).

While we focused on directional trends, there were clearly defined peaks in disease in the 1970–2001 period for corals (1982–1983), mammals (1987–1992), decapods (1970–1975, 1992, 1994), elasmobranchs (1975–1977, 1998–2001), fishes (1973–1978), turtles (1992–1999), molluscs (1997–2000) and seagrasses (1982–1984, 1989–1991). In the recent period (2001–2013), peaks occurred only for corals (2007–2010), urchins (2007–2013), mammals (2009–2011), and molluscs (2003–2005).

We investigated the results of disease searches for the recent peaks (2001–2013) to determine which infectious agents were driving observed trends. No single disease represented the majority of instances for any of the recent peaks. The peak of coral disease (2007–2010) encompassed 27 diseases (82 instances), spread across the Caribbean Sea and Pacific Ocean. Black band disease accounted for most of the instances ($n = 13$, 16%), followed by white syndrome ($n = 11$, 13%) and white plague ($n = 7$, 8.5%). Urchin disease peaked from 2006 to 2013 with 15 total instances, primarily owing to mass mortalities (eight instances, 53%). Mass mortalities included five amoebic infections (33%), all of which were infections of *Strongylocentrotus droebachiensis* in Nova Scotia. There were four instances of parasitic eulimid snails. Mammals experienced a peak from 2009 to 2011. Of 97 instances, 59 were of metazoan parasites (61%), many of which were detected in regional descriptive assessments of parasites found in stranded animals (e.g. [42]). The bottlenose dolphin, *Tursiops truncata*, was the most commonly reported mammal host but without a predominant disease. The next most common host, the striped dolphin, *Stenella coerulealba*, was predominately reported with morbillivirus infections. The mollusc peak (2003–2005) was predominated by 11 instances (48% of 23) of protozoan infections of bivalves, including four of *Perkinsus* spp. and one or two of each of *Bonamia ostreae*, *Mikrocystos mackini*, *Haplosporidium nelsoni* and quahog parasite unknown.

For all taxa, the confidence intervals for the literature proxy method improved in later years, as evidenced by decreasing error bounds (electronic supplementary material, figure S2). Fishes have the highest total number of normalized disease reports across the entire time period, while urchins and seagrass have the lowest (electronic supplementary material, figure S3).

(b) Potential biases

We considered whether a prolific research group could bias the results towards detecting more disease outbreaks than actually occurred. Removing the most common author did not change the overall result (the trend in normalized disease reports over time) for corals and elasmobranchs, but the result for molluscs changed from a non-significant ($p = 0.089$) to a significant decrease ($p = 1.9 \times 10^{-4}$, electronic supplementary material, table S2). Given that the most common author in molluscs was not much more common than in corals and less common than the most common author for elasmobranchs (electronic supplementary material, table S3), it is more likely that the result changed because of removing reports from a dataset that counteracted the negative trend, rather than owing to author commonness alone.

Multiple reports of the same disease event also did not skew the results. As in WL04, the results of this analysis did not differ from the original analysis; i.e. with repeat reports of the same event included (electronic supplementary material, table S4).

(c) Rabies analysis

We evaluated how well the literature proxy tracks disease outbreaks by comparing the results for raccoon rabies literature to actual data reports from the CDC. We found that the literature proxy reliably captured the direction and significance of the trend from 1970 to 2013, an overall increase. Moreover, the literature proxy correlated significantly with the CDC rabies records ($\rho = 0.58$, $p = 3.8 \times 10^{-5}$). Both the literature proxy and the CDC records showed a decrease from 2001 to 2013. Though this recent decrease in the literature proxy was not significant (figure 2, $p = 0.16$), it was significantly correlated with the CDC rabies records ($\rho = 0.57$, $p = 0.042$). This suggests that the trends were reliably captured, and that non-significant trends in the proxy method might merit additional attention. Thus, we also report results that had p-values between 0.05 and 0.1. Most importantly, there was no bias towards false positives (i.e. detecting significant trends that are not present), which supports the validity of increases detected in this study.

(d) Temperature anomalies

Heat stress extent from coral reef-containing SST pixels was correlated with normalized coral disease reports for the Caribbean for the time period 1970–2013 ($\rho = 0.55$, $p = 1.0 \times 10^{-4}$; figure 3). A peak in disease reports followed the 1998 bleaching event, and disease reports increased in general in a period of increased heat stress that included the 1998, 2005 and 2010 bleaching events in the Caribbean [26,43].

4. Discussion

Quantifying anthropogenic drivers of infectious marine disease is challenging owing to the lack of long-term records

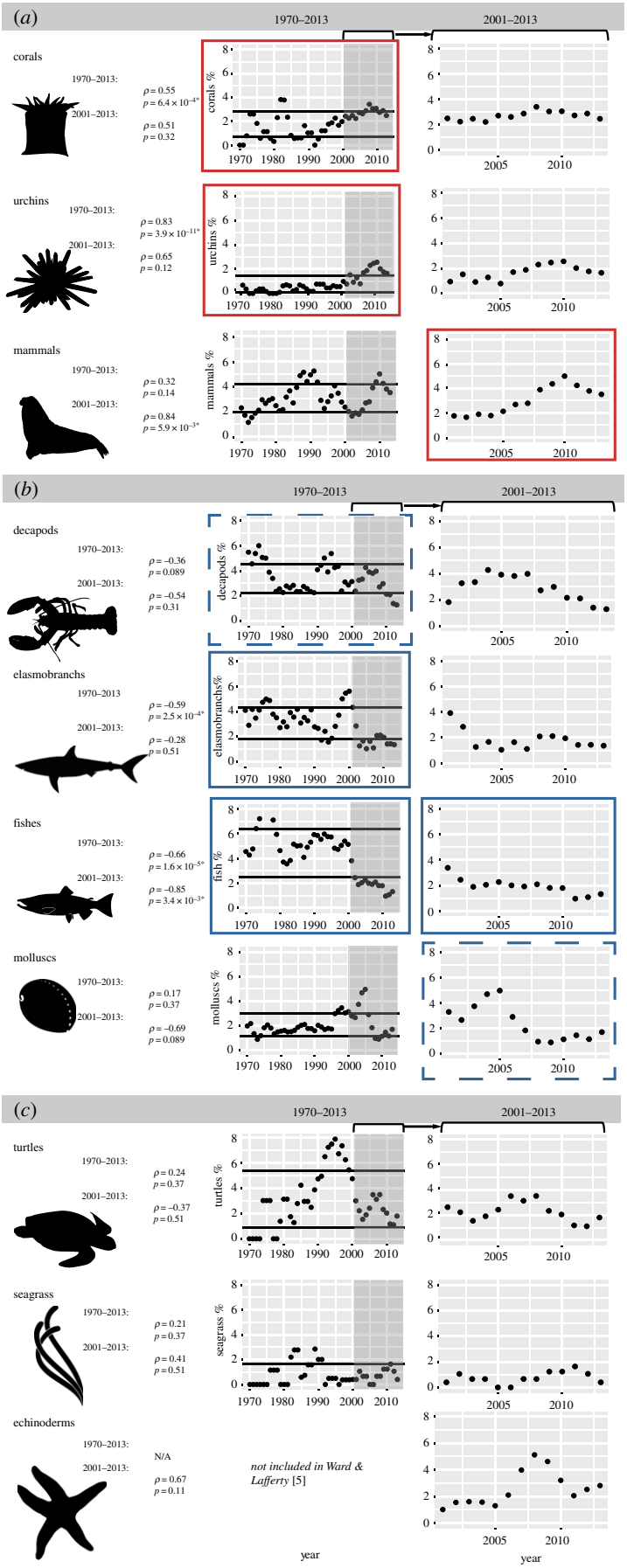

Figure 1. Comparison of trends over time for reports of disease in focal marine taxa from 1970 to 2013, with the right panel zoomed to the more recent 2001–2013 time period and divided into groups with (*a*) increases, (*b*) decreases and (*c*) neither increases nor decreases. Correlations with time were evaluated using Spearman's ρ, with horizontal lines indicating ±1 standard deviation about the mean of the dataset (1970–2013). The percentage on the vertical axes refers to the per cent of total reports per taxon per year that reported disease. Red (blue) outlines denote significant positive (negative) correlations (ρ) with time (significance level 0.05); dashed lines indicated non-significant correlations (significance level between 0.05 and 0.1). Significance (p) values were adjusted for multiple testing using Holm's Bonferroni method ($n = 10$ for 2001–2013, $n = 9$ for 1970–2013). (Online version in colour.)

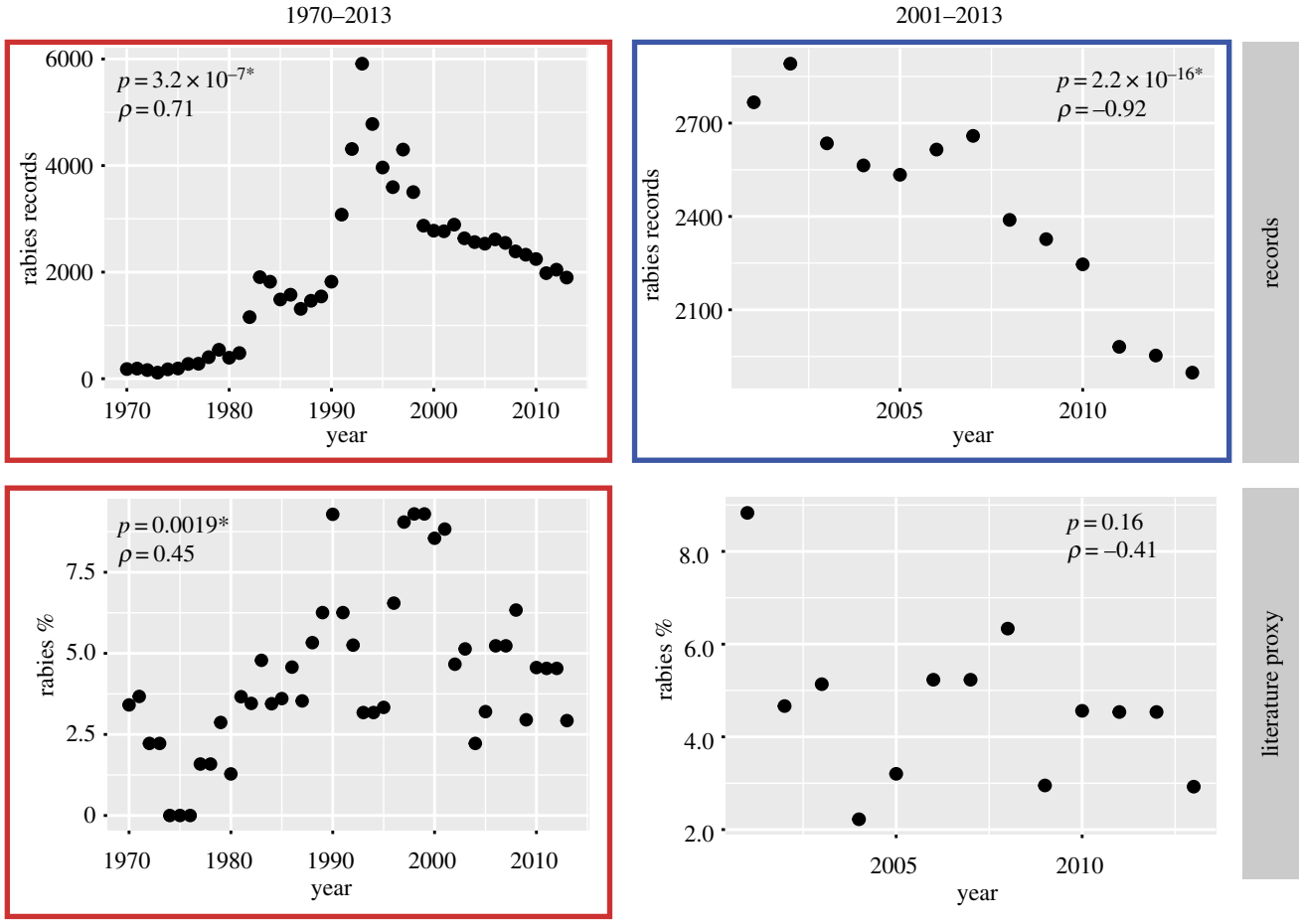

Figure 2. Comparisons between rabies records and the rabies literature proxy for 1970–2013 and 2001–2013 based on correlations using Spearman's ρ. Rabies records is the number of reports per year, while rabies % is the per cent of total reports per taxon per year that report an incident of disease. Red outlines denote positive correlations with time and blue outlines denote negative correlations with time. (Online version in colour.)

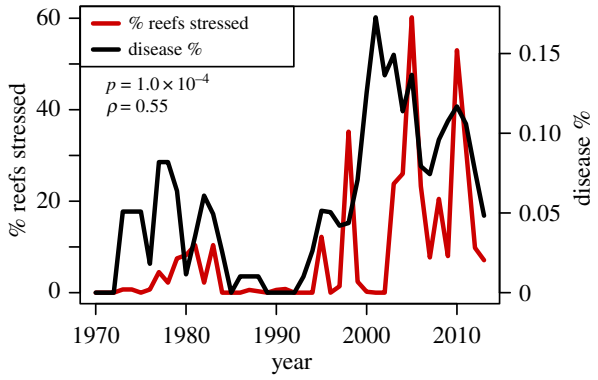

Figure 3. The proportion of Caribbean reef pixels exposed to bleaching-level heat stress (DHM ≥ 1°C-months, 1970–84; DHW ≥ 4°C-weeks, 1985–2013) and normalized disease reports for corals in the Caribbean both increase over time. (Online version in colour.)

on these globally distributed, often inaccessible underwater ecosystems. However, understanding these trends is critical for evaluating threats to ocean ecosystems. Using a proxy method based on literature reports of disease, we detected several taxa with decades-long shifts in disease reports. The changes in disease reports from 1970 to 2013 provide a novel perspective on how anthropogenic pressures may disrupt species interactions on a global scale.

The increases in disease reports over 44 years in corals and urchins may result from increasing temperatures, as these ectothermic marine invertebrates are particularly sensitive to temperature. Urchin disease outbreaks have been previously linked to elevated temperatures [44]. This is also the case for corals, for which elevated temperatures have repeatedly triggered outbreaks across host species and geography [8]. Underlying mechanisms for temperature-driven disease outbreaks in corals include shifts in pathogen virulence, microbial communities and host defences [45–47]. Our annotation of the peak in coral disease reports encompasses over 30 disease syndromes in a variety of host species, indicating that disease reports are not limited to one host, or even one parasite. We tested the hypothesis of a link between heat stress and coral disease using temperature data, and identified a significant positive correlation between the frequency of Caribbean heat stress and reports of Caribbean coral disease from 1970 to 2013. This supports the hypothesis that increases in coral disease are linked to warming oceans. Although WL04 did not find an increase in coral disease from 1970 to 2001, Lafferty *et al.* [48] show that two peaks in global coral disease reports before 2001 occurred soon after two large El Niño events and the accompanying climatic shifts [48]. While the role of temperature in coral disease is well established for shorter time scales, our analysis suggests that temperature is also an important, long-term driver of increasing disease across multiple coral species. The finding is noteworthy because the correspondence between warming and coral infectious disease is more difficult to detect than the connection between warming and coral bleaching, owing in part to the longer time lag. Yet mass mortality and loss of coral cover from infectious disease may equal or exceed the consequences

of bleaching-related mortalities on a reef, an important consideration for reef management [49,50].

We detected long-term decreases in disease reports in fishes and elasmobranchs, which align with the non-significant decreases in disease detected in WL04 [5,13]. The presence of both increases and decreases in disease reports provides an opportunity to investigate how anthropogenic pressures may disrupt host-parasite interactions on a global scale. In either case, these swings in multiple taxonomic groups of ecologically important hosts are likely to impact ecosystems. In detecting disease swings, our results offer a rare glimpse into long-term trends and indicate that changing environmental conditions often lead to directional change in disease risk.

Though the mechanisms behind long-term decreases in disease reports are less clear, one hypothesis is that overfishing accounts for the reductions in disease reports in fishes and elasmobranchs from 1970 to 2013. Overfishing could reduce disease risk by decreasing host population size and/or density and supporting less disease transmission, though this effect is only expected for parasites with certain life histories and transmission modes. Decreased density from overfishing could also improve nutrition of remaining fishes and thus bolster immune defences against infections, indirectly reducing disease. In regard to fishing practices, disease risk can decrease if fishing preferentially removes diseased individuals [13]. The significant decrease in normalized disease reports for fishes and elasmobranchs from 1970 to 2013 could result from any of these mechanisms. In fishes, the decline was also significant more recently from 2001 to 2013, confirming our hypothesis that changes in disease levels can be strong enough to be detected within a single decade. Decapod disease reports exhibited a non-significant decrease over the entire 44-year period, providing additional evidence for declining disease reports in fished species that could result from reduced population density owing to overfishing [13]. Negative trends in disease reports were also observed for fished taxa (i.e. fishes, elasmobranchs, and decapods) from 1970 to 2001 in WL04, though they were non-significant [5,13]. Although the decreases in disease in fish and elasmobranchs may initially seem beneficial, the loss of parasites could alter food webs and ecosystem function [6].

While the 44-year disease swings in four of the nine taxonomic groups highlight long-term changes in disease reports, other taxa have also experienced important disease impacts between 1970 and 2013. All of the taxa experienced disease impacts, as indicated by the total normalized disease reports (electronic supplementary material, figure S3). The higher total reports in fishes and the lower reports in urchins and seagrass may be owing to true differences in disease burden, or to overall greater investment in and knowledge of disease in certain species. Outbreaks in an individual species may be swamped out by prevailing trends, and localized or acute disease outbreaks are probably not reflected at the scale of our analyses owing to the grouping across geography and host species. For example, turtle fibropapilloma tumours occur in all turtle species and across all major ocean basins, but outbreaks were highly localized and asynchronous [3]. This may account for the lack of a significant trend in turtles from 1970 to 2013. There were also no detectable, significant trends in marine mammal diseases from 1970 to 2013. Simeone *et al.* [30] previously determined that a literature-based metric did not capture the impacts of disease on marine mammals using a comparison of literature and hospital records for neoplasia [30]. They suggest that the discrepancy between the two sources results from the emphasis on novel findings, the impact of new diagnostic methods, and other biases in the literature [30]. However, the Simeone *et al.* [30] study did not account for research effort or time delays in publication [30], and the focus on neoplasia in one host species probably leads to greater variability than is present using broader host groupings with more data. By grouping hosts more broadly and normalizing disease reports, we detected a significant increase in disease reports in marine mammals from 2001 to 2013 that may reflect increased impacts in the last decade. However, given that strandings provide much of the data on marine mammal disease, this finding could also result from increases in strandings and number of stranding facilities [30].

Warming temperatures are expected to continue influencing marine diseases. Just as extending the WL04 analysis to 2013 revealed new insights on temporal disease patterns, disease events since 2013 also have the potential to influence our results. For example, echinoderms on the Pacific coast of the US experienced a devastating, multi-species outbreak of sea star wasting disease from 2013 to 2015 [2,19], which continues for some species [22]. While our analyses include some of the earliest papers on the outbreak and thus contribute to the non-significant increasing trend in echinoderm disease from 2001 to 2013 (figure 1), the sea star outbreak is not fully captured by our time period, accounting for the 3-year time lag. The 2016 and 2017 mass bleaching events on the Great Barrier Reef caused broad-scale mortality of corals [8,51], and there are preliminary reports of an increase in disease associated with heat stress [50]. It will be particularly relevant to further extend the literature proxy analysis given that the years since 2013 have each been warmer than all years from 1880 to 2013, with 2018 currently ranked fourth warmest behind 2016, 2015 and 2017 [8].

The literature proxy is a valuable method that provides insight into patterns not available from any other source. We confirm the validity of the literature proxy established by WL04 using the concordance between rabies reports in the literature and actual records of raccoon rabies in the US. Targeted analyses of prolific authors and repeat reports indicate that these potential limitations of the dataset are unlikely to affect the results. However, the literature proxy method has natural limitations. It cannot distinguish between an instance of disease that was not recorded and the absence of disease. In previous work, Fernández Robledo *et al.* [32] report that publications on a given host-parasite system follow a pattern of an initial lag after the discovery of the disease aetiology, followed by increased publication rates if and when the pathogen is available in culture [32]. As the literature proxy method herein generalizes across all the hosts in a taxonomic group (including their many parasites) and does not include laboratory studies, we do not expect this phenomenon to influence our results. It is also possible that increased research effort on disease, in general, could drive increases in the literature proxy. Koprivnikar & Johnson [31] conducted a literature analysis from 1970 to 2014 and determined that the per cent of publications on disease has increased in recent years, as has funding for disease research [31]. However, their search includes all publications about disease, not just the reports of disease in wild marine populations, as in the present study. Moreover, there are other hot topics

and increasingly popular study organisms that could simultaneously inflate the denominator in our calculations of normalized disease reports. The demonstrated capacity to detect negative disease swings in the midst of these biases confirms the minor role of these phenomena in our data. Ultimately, the 44-year dataset is the longest available record of disease reports for multiple marine taxa and constitutes a valuable resource given the impossibility of retrospectively obtaining true baseline measurements.

Our study uncovers both positive and negative long-term trends in marine infectious diseases. Decades-long trends in corals, urchins, fishes and elasmobranchs eclipse spatial variation and impacts restricted to individual host species, and coincide with a period of marked anthropogenic change that can act both directly and indirectly to alter disease in wild hosts. We link the increase in disease reports in corals to warming oceans with analyses of SST data in the Caribbean. This long-term link between warming and coral disease suggests new management priorities for coral reef resilience that focus not only on bleaching, but also on infectious disease. Our findings indicate that rapidly changing oceans have diverse but potent effects on disease. Unfortunately, these effects will probably intensify in the coming years. While the direct and indirect impacts of anthropogenic change on organisms are threatening many species, disease swings affecting multiple related hosts have the capacity to fundamentally alter marine ecosystems.

Data accessibility. All data and R code for analyses are available through the Dryad Digital Repository: https://doi.org/10.5061/dryad.nb372tc [52]. This excludes CDC rabies data that are not public and were obtained with permission.

Authors' contributions. C.D.H., A.M.T., R.M.Y. and M.L.P. designed the study. M.L.P. and A.M.T. conducted the Web of Science searches, A.M.T. conducted R analyses, R.M.Y. collated rabies reports and annotated disease peaks, and S.F.H. conducted analyses on temperature anomalies. A.M.T. wrote the first draft of the manuscript and all authors contributed substantially to revisions.

Competing interests. We declare we have no competing interests.

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