Response to the Reviewers.

Major outstanding points on 2nd round review.

Following the 2nd round of reviews at *Nature*, all reviewers accepted the fundamental scientific conclusions of the paper, although reviewer 1 has many requests for clarification of specific points in order to make these conclusions more transparent. The most important of reviewer 1's concerns was that we elaborate further on temperature data, and how it relates to standard measures of coral thermal stress. Reviewer 3 felt that all scientific points were well addressed, but recommended that further attention to the proposed role of algal DOC in coral mortality should be given in the manuscript, and that the results should be more fully related to major qualitative and quantitative models of coral microbiology in general. We appreciate these suggestions, and have addressed each of them.

Our revised manuscript addresses reviewers' remaining points by:

1) Clarifying the relationship between temperature, and measures of coral thermal stress. We added an extended data figure summarizing raw temperature values and standard derived measures of coral thermal stress. This new figure (Supplemental Figure 10) shows the absolute temperature track over the experiment, the maximum of monthly means value (MMM), a climatological baseline used in calculation of stressful temperatures for corals, and NOAA's degree-heating-weeks (DHW) value, which measures cumulative thermal stress. The raw data has also added to a supplemental data table for use by interested readers, and we have added text in the methods and results providing additional details for the calculation of coral thermal stress.

2) Adding substantially to our coverage of research on the coral microbiome in the introduction. The revised manuscript includes a succinct description of the algal dissolved organic carbon hypothesis as requested by reviewer 3 (DDAM; dissolved organic carbon, disease, algae, and microbes). We also now highlight some additional results of ours that were pertinent to this hypothesis, such as increases in the abundance of bacterial sugar metabolism pathways with algal competition in the main text. We have extensively revised our discussion to place our findings in the larger context of this field.

3) Clarifying other minor points raised by Reviewer 1 in the text where appropriate (see below), and making many small changes in order to reformat the paper to *Nature Communications* standards, including addition of new separate introduction and discussion sections, and removing references from the abstract.

Referees' comments:

Referee #1 (Remarks to the Author):

Although the authors have clarified how they define thermal stress, the issue remains a weakness. The authors calculate the average maximum summer temperature, and assert that anything warmer than the mean constitutes "stress". They cite an obscure paper (line 615, reference 24) on microbiology to support this.

By definition, the mean lies near the middle of a range - so temperatures slightly above the mean are not exactly unusual or unexpected.

We thank the reviewer for the careful reading, and have revised our presentation to further clarify this issue.

As background, the reviewer had previously asked for us to quantify thermal stress, and we did so using the only accepted definition for thermal stress to corals in the literature. This definition, used by NOAA and CoralReefWatch, relies on (i) a climatological baseline temperature for the warmest month, which is the maximum of monthly means (MMM) in climatological data, and (ii) accumulation of cumulative thermal stress based on the amount of time during which temperatures exceed this value by at least one degree (i.e. the MMM + 1 °C) during a study period, expressed as degree-heating-weeks (DHWs). The maximum of monthly means (MMM) value that we use as the climatological baseline underlying calculations of thermal stress is not our own invention, but is the basis for how degree-heating-weeks (DHWs), which the reviewer accepts, are calculated. We brought on an expert from this field (Jeff Maynard) as a co-author specifically to address this point.

In our calculation of Degree Heating Weeks, we have recalculated the MMM to exclude the study years (i.e. we use climatological data from 1982-2009), avoiding any concerns about circularity of the type raised by the reviewer. This altered the MMM, MMM + 1, and DHW values slightly, but didn't otherwise affect the conclusions. We have also made it more clear that while studies of coral bleaching do often discuss the MMM as a threshold for thermal stress, the calculation of thermal stress in Degree Heating Weeks is usually performed on the MMM + 1 °C. In practice, all of our thermal stress values use the MMM + 1 °C, so it is not the case that half of measured temperatures will accumulate thermal stress. For reference, we have contrasted the standard DHW (MMM + 1 °C) values we used in our calculations with what one would get using just the MMM (that is DHW(MMM)) in Extended Data Fig. 10a (this may be of interest to some readers since this alternative formulation is sometimes used in the literature).

The major source of confusion on this point seems to be that the only definition for coral thermal stress used widely in the field is designed to study coral bleaching. Separately, meta-analysis of microbiological studies of bacterial opportunism (see Maynard et al *Nature Climate Change*) has led to predictions of bacterial opportunism on corals in terms of one the same climatological baseline temperatures used in the calculation of coral thermal stress (the MMM). The reviewer is correct that, if current temperatures reflect historical climatology, about half the weeks in the warmest month will exceed the MMM in any given year. We agree that this previously published prediction would imply that, in general, corals may be more vulnerable to bacterial pathogens in the warmest couple of weeks of the year, but that is not a conclusion that is formulated here. In this study, we simply compare microbiological data to this proposed temperature threshold (the MMM), as well as to the MMM +1 °C and standard DHW values based on the MMM +1 °C used in studies of coral bleaching.

More broadly, we appreciate the reviewer's concern expressed here and in previous reviews that observed temperatures and coral thermal stress be reported in language that is as transparent and neutral as possible. We have substituted the term 'above-average summertime temperatures' or 'above-average temperatures' in the main manuscript to refer to temperatures above the MMM (by definition temperatures greater than the

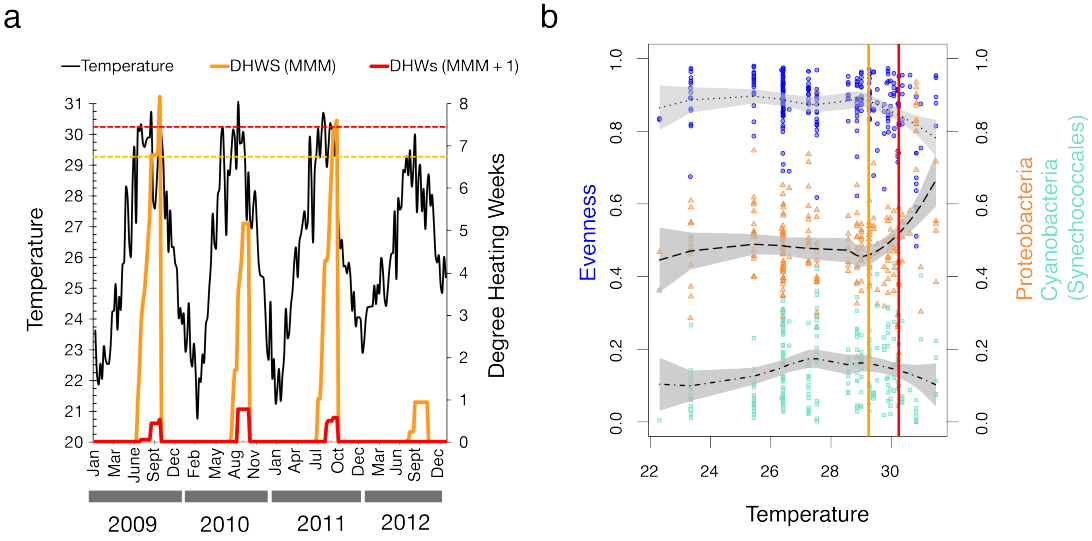
MMM are above long-term averages for the warmest month at a particular site). This hopefully addresses some of the questions raised by reviewer, as well as concerns in previous rounds of revision, about other possible ways of describing these temperatures such as ‘elevated temperatures’ or ‘warm temperatures’. We carefully distinguish this from ‘coral thermal stress’, which is used in the standard sense of DHW values > 0. At some points in the text, we make this more specific, referring to ‘sub-bleaching thermal stress’, meaning DHW values >0 and < 4 (a standard threshold for the onset of coral bleaching).

To further clarify this issue, we have included additional text explaining how DHWs are calculated in the main text, and added an additional two-part figure to show these values for our dataset (see Supplemental Fig 10). Part b of this figure shows how the MMM and MMM +1 temperatures relate to the microbiology. We make the raw data and intermediate steps in the thermal stress calculation available as Supplementary Table S6a-e, each with accompanying notes.

We hope that together these changes fully satisfy the reviewer’s concern that these data are now presented in a transparent and complete way.

We're not told when, how often or for how long were temperatures above the mean observed.

We added a new figure to clarify this point (Supplemental Fig. 10a). This figure shows temperature values for our site, the MMM and MMM + 1 climatological baselines, and the accumulation of thermal stress in units of Degree Heating Weeks based on temperatures above the MMM + 1. For the sake of completeness, we also show DHWs based on the MMM (sometimes used in the literature) in Extended Data figure 10a, but all calculations in the rest of the paper use the more standard DHW (MMM + 1) value (the red line in the figure). These values are reported in raw form in Supplementary Table 6.



Extended Data Figure 10. Temperature, thermal stress, and effects on the coral microbiome. a, Temperature time-series based on the Pathfinder V5.2 dataset (Methods). The left vertical axis and thin solid black lines shows temperatures in °C. The horizontal orange dotted line indicate the maximum monthly mean (MMM) temperature of 29.26 °C. This is calculated as the average temperature of the warmest month in available climatological data (1982-2008; i.e. excluding the study period). The red horizontal dotted line indicates the MMM +1 °C (30.26 °C), which is often used as a temperature threshold at which coral thermal stress begins to accumulate in predictions of coral bleaching. For purposes of predicting coral bleaching, coral thermal stress is typically measured in units of Degree Heating Weeks (DHWs, °C-weeks). DHWs are usually calculated relative to the MMM +1 °C, and are the accumulation of temperatures above this threshold. As a hypothetical example, if temperatures exceeded the MMM + 1 by 0.2 C for 3 weeks, 0.6 DHWs would accumulate. The accumulation of degree heating weeks due to temperatures above the MMM + 1 °C is shown by the solid red line and the secondary vertical axis on the right hand side of the plot (DHWs (MMM +1)). Because vulnerability to pathogenic bacteria is thought to occur at lower levels of thermal stress than coral bleaching, we also plotted DHWs calculated relative to the MMM. This is shown by the orange line (DHWs (MMM)). We emphasize that all DHW values presented in the main text are calculated based on the MMM + 1 °C (red lines). **b,** Coral microbiomes vs. temperature. Microbial community evenness (left axis, blue circles) and the relative abundance of *Proteobacteria* and *Synechococcus* cyanobacteria (right axis, orange triangles and cyan squares, respectively). The x-axis shows sea-surface temperatures. Regression lines show the loess regression (span=0.25) for each data series (evenness, dotted lines; *Proteobacteria*, dashed line; *Synechococcus* dot-dashed line). Gray shading around each line indicates twice the standard error of the regression. Vertical lines indicate metrics from the thermal stress calculation. The orange vertical line is the MMM, the red one is the MMM + 1 °C. Notably, the abundance of *Proteobacteria* increase, and overall community evenness decrease around the MMM of 29.26 °C.

We have also updated and expanded the overall summary of the methods used to quantify thermal stress (Methods, lines 768-792):

> **Quantification of sub-bleaching thermal stress**
> We calculated cumulative thermal stress using the Degree Heating Week (DHW) metric of NOAA's Coral Reef Watch[52]. These calculations rely on a climatological baseline for the mean temperature of the warmest month, known as the maximum monthly mean (MMM). We calculated the maximum monthly mean (MMM) for our site using the NOAA Pathfinder v5.2 dataset, the U.S. official climate data record for sea surface temperature[50], and found that the MMM for our site was 29.26 ºC (Database S6). Data used spanned 1982-2008, and excluded study dates. Temperatures that exceed the maximum monthly mean (MMM) by +1 ºC are generally regarded as constituting thermal stress[52]. We calculated an MMM + 1 ºC threshold of 30.26 ºC for our site. Temperatures above the 30.26 ºC threshold for accumulation of DHWs only occurred at our site during the warm seasons (3 months centered on the warmest month, August).
>
> Degree Heating Weeks (DHWs) represent the extent to which temperatures exceeded the MMM +1 ºC in a given season. Temperatures were above 30.26 ºC for 7 weeks (total) during all sampling years, resulting in the accumulation

> of <1 DHW during each of the study years (Extended Data Figure 10a). In predictions of coral bleaching, accumulation of 4 DHWs is often associated with minor to moderate bleaching[21]. We saw little bleaching within experimental plots, consistent with sub-bleaching levels of thermal stress. Sub-bleaching thermal stress may nonetheless negatively affect reef health if it increases the abundance and/or virulence of bacterial pathogens. For example, one recent model[22] proposes that bacterial pathogens may become problematic for corals at temperatures exceeding the MMM (a lower threshold than the MMM + 1 ºC threshold used in coral bleaching studies). We therefore related our microbiological data to both the MMM + 1 ºC for coral thermal stress during coral bleaching, and the MMM threshold proposed for bacterial pathogens (Extended Data Figure 10b).

Additionally, all metadata used in the manuscript is reported across all microbial samples in the revised supplemental data table S2c. This includes a complete record of raw temperature data used in comparisons with the microbiome.

The appendix (lines 618-624) also refers to degree heading days (DHW), but I can't find any analyses based on this approach.

This analysis is now more completely described in the introduction, results and the supplementary results. We also include some additional microbiological and coral mortality results using DHW values directly.

In the previous manuscript we used Degree Heating Week values only to make a simple point: that thermal stress, under standard definitions, accumulated at our site, but this thermal stress did not rise to levels that would cause mass coral bleaching. Therefore, both the microbial shifts we document happen, and the large bias in coral mortality towards the warm season, occur well before corals bleach. This is consistent with the predictions of recent climate models (Maynard *et al*., Nature Climate Change, 2015) that inferred thresholds for pathogen blooms based on laboratory studies. However, the combined effects of seasonal temperature variation and local overfishing and nutrient pollution on coral microbiomes have not previously been measured empirically in field experiments.

We had previously used the climatological baselines for coral thermal stress (the MMM + 1 C) or bacterial opportunism (the MMM) from the literature as reference points on our temperature scale (i.e. for reader's information). However, the suggestion of more formally relating our findings on coral microbiological and coral health to the accumulation of coral degree heating weeks (rather than temperature at the time of sampling) is well taken. We think it is likely that both affect the microbiome to some degree. In the revised manuscript we conduct this analysis, and show that there are some effects of cumulative thermal stress (DHWs) on coral microbiomes even after accounting for temperature on the day of sampling. We also tested whether there was any correlation between the sub-bleaching levels of coral thermal stress observed during each year of the experiment and coral mortality due to local stressors in that year. These new analyses are reported in the main-text (Results, lines 268-324):

> Surprisingly, coral mortality caused by simulated overfishing and nutrient pollution was strongly seasonal. Although we observed minimal coral bleaching, ~80% of coral mortality was concentrated in summer and fall (Fig.

3d), which include the warmest months of the year. Yet this seasonal mortality occurred only when herbivores were removed or nutrients increased, suggesting that local stressors and temperature interact to kill corals. If seasonal temperature variation and local stressors do interact to drive coral mortality, we might expect years in which corals suffer greater sub-bleaching thermal stress to correspond to greater coral mortality.

To put treatment outcomes in the context of seasonal temperature variation and coral thermal stress, we calculated raw sea-surface temperatures for our study site using both the Pathfinder v5.2[50] and HYCOM Gulf of Mexico resources[51] (Methods; Extended Data Figure 10). From these we calculated several standard derived measures of coral thermal stress[52]. The maximum monthly mean (MMM) is the average temperature of the warmest month in long-term climate data for our site, excluding the study period (i.e. 1982-2009). In predictions of coral bleaching, temperatures that exceed the MMM by ≥1 °C (hereafter MMM + 1 °C) are considered 'thermal stress'[52]. The level of thermal stress is then measured in degree-heating-weeks (DHWs), which accumulate weekly temperatures above the MMM + 1 within a 3-month timeframe (see Methods). Because the threshold for increased pathogenesis may be lower than that for coral bleaching[17], we also calculated DHWs based on the MMM (DHW-MMM) for reference, although the more standard DHW-MMM + 1 °C was used in all calculations.

Although summertime temperatures during our experiment exceeded the MMM + 1 °C, and positive DHW values accumulated – indicating some thermal stress – in no cases did these values rise to levels that would predict increased bleaching risk (Dataset S6). However we did observe correlations between sub-bleaching thermal stress and warm-season coral mortality during 2009, 2010, 2011 and 2012, despite limited replication of seasons across years (i.e. n=4). Across all treatments, the extent of mortality in summer and fall was positively correlated with the extent of sub-bleaching thermal stress in Degree Heating Weeks ($r = 0.960$, $p = 0.03$), an effect strengthened when control corals were excluded ($r = 0.993$, $p = 0.007$).

Differences in microbiome stability between healthy and unhealthy corals were exaggerated at temperatures above 30 °C (Fig. 3e), as was the displacement of likely beneficial *Cyanobacteria* by opportunistic *Proteobacteria* (Fig. 3f). Regressions using local temperature data (Extended Data Figure 10b) indicated that both increasing abundance of *Proteobacteria* relative to *Cyanobacteria* and reduced microbial community evenness became noticeable around the MMM (29.26 °C) before becoming pronounced around the MMM +1 °C (30.26 °C). This finding suggests that the local MMM may be a critical temperature threshold for the onset of bacterial opportunism.

Temperature variation explained differences in microbial community structure over time better than other measured seasonal parameters (Supplementary Text). The accumulation of thermal stress due to periods of above-average temperature also appeared to influence microbial communities. Samples in which sub-bleaching thermal stress had accumulated (i.e. DHWs > 0) showed increased β-diversity (permutational t-test on Weighted UniFrac distance matrices; p =

0.001) relative to samples from periods without thermal stress (DHWs = 0). Cumulative thermal stress also had small but significant effects on microbial communities after accounting for daily temperature variation. As the difference in thermal stress between microbiome samples increased, the Weighted UniFrac distances between microbial communities also increased (Mantel test p=0.01, r=0.176; see Methods), even after accounting for the temperature on the day the sample was taken (partial Mantel test, p=0.01, r=0.17). These microbiological changes were probably not due to confounding effects of coral bleaching, as cumulative thermal stress stayed well below the 4 Degree Heating Week threshold for mass coral bleaching[5] (Extended Data Fig 10a; Supplementary Text), and little visible bleaching was observed.

There does not appear to have been any coral bleaching during the experiment, suggesting that temperatures exhibited their normal seasonal cycle.

We've tried to emphasize throughout the revised manuscript that we are looking at the interaction of local stressors with seasonal temperature variation. This may, in typical years, include some thermal stress to corals, even if those corals don't visibly bleach. Coral thermal stress is typically measured in units of degree heating weeks. Substantial, sustained thermal stress is required before corals bleach. Thus thermal stress can occur in the absence of the signs of bleaching. Typical thresholds for moderate coral bleaching are observed for many reefs at 4 DHWs, while 8 DHWs is often used in climate modeling papers as the threshold at which nearly all corals bleach. Thus, the absence of bleaching does not preclude the accumulation of thermal stress, and an absence of coral bleaching is consistent with the DHW values that we report (always < 1, but >0 in 2009, 2010 and 2011).

Previous climate models have proposed, based on extrapolations from smaller laboratory studies, that even seasonal peak temperatures too mild to trigger coral bleaching could nevertheless increase the abundance and/or virulence of coral opportunistic pathogens (Maynard *et al*., Nature Climate Change, 2015). We were actually surprised to see, during revision of the manuscript, how well the empirical threshold at which we started to see major shifts towards pathogenesis in our microbiomes corresponded to *a priori* predictions for bacterial pathogenesis (the MMM).

The 30C estimate of the summer maximum (where stress kicks in) is based on 30 years of satellite data.

Yes, the Degree Heating Week metric uses historical data to set its climatological baseline.

It's not clear if the authors measured temperature in situ during their experiment, or if it too is derived from coarse-scale Pathfinder sources.

For the main analysis we used Pathfinder and HYCOM datasets, since these will be most comparable with what climate models use. Showing an on-the-ground relationship between satellite-measured sea-surface temperatures and microbial ecology/ coral health seems most useful. Note that the possible negative effect of using a course-scale sea-surface temperature would be to generate a false-negative relationship with temperature. We see strong trends with temperature, so we can rule out the possibility that satellite data obscured an existing relationship.

As discussed above, we have now expanded the introductory and methods text regarding temperature to clarify this point, and added a figure showing the raw temperature data.

I can't find any information on how temperature (e.g. in Figure 3F) was measured, or how often during 2009-2012. That figure has only one data point above the "threshold" of 30C.

As discussed above, we have added additional text to the methods to clarify the details of our temperature measurements. Temperature data used here are from the hybrid coordinates ocean model (HYCOM) data for our grid square which includes direct measurements from a local buoy, interpolated by the model with other local buoys. These data also agreed well with Pathfinder satellite data. All temperature values and other metadata used in microbial analysis are presented for each DNA sample in Supplemental Table 2c.

The reason the figure has only one point for a temperature of 30 C is that the x-axis is temperature aggregated in units of degrees. The error bars indicate that the presented data is a mean value for multiple data points. We have clarified these points in the legend for Fig. 3f (lines 564-570):

> Temperature effects on coral microbial variability, evenness, and relative abundance of *Proteobacteria* or *Cyanobacteria*. Evenness and β-diversity data are means ± SEM. Microbial and coral health data are averaged within each 1 degree Celsius interval on the x-axis. The vertical red line at 30 ºC indicates the point nearest to the MMM +1 ºC value for our site (30.26 ºC); temperatures beyond this result in accumulation of degree heating weeks of coral thermal stress (see Methods).

The authors are trying to make a climate change story, referring to fishing and nutrients as local stressors (line 55) whereas temperatures are rising globally. There is a sizeable coral reef literature on how local and global stressors interact to affect resilience, which is not cited.

We describe how local stressors reduce the ability of reefs to recover from large disturbances (i.e. resilience) in the opening lines of the paper (lines 56-64):

Tropical reefs continue to lose coral cover worldwide due to a variety of factors, including warming ocean temperatures, nutrient pollution, sedimentation, and overfishing[1-3]. Thermal stress from sustained periods of high temperature can kill corals by driving widespread coral bleaching events and coral disease outbreaks[2,4,5]. Localized stressors can also greatly impact corals. For example, elevated nutrients and overfishing of herbivorous fishes increase algal abundance on reefs[6-8]. Competition with these algae can compromise coral recruitment, growth, and survivorship[9-11]. The intersecting impacts of these local stressors may ultimately compromise the resilience of reefs to disturbances such as hurricanes and thermal anomalies[1,1213].

A couple of the relevant references we previously cited on this point are:

Jackson, J., Donovan, M. K., Cramer, K. L. & Lam, V. V. Status and Trends of Caribbean Coral Reefs: 1970-2012., (Global Coral Reef Monitoring Network, IUCN, Gland, Switzerland, 2014).

Adam, T. C., Burkepile, D. E., Ruttenberg, B. I. & Paddack, M. J. Herbivory and the resilience of Caribbean coral reefs: knowledge gaps and implications for management. *Marine Ecology Progress Series* **520**, 1-20 (2015).

Hughes, T. P. *et al.* Phase shifts, herbivory, and the resilience of coral reefs to climate change. *Current Biology* **17**, 360-365, doi:10.1016/j.cub.2006.12.049 (2007).

Birrell, C. L., Mccook, L. J., Willis, B. L. & Diaz-Pulido, G. A. Effects of benthic algae on the replenishment of corals and the implications for the resilience of coral reefs. *Oceanography and Marine Biology: An Annual Review* **46**, 25-63 (2008).

Graham, N. A., Jennings, S., MacNeil, M. A., Mouillot, D. & Wilson, S. K. Predicting climate-driven regime shifts versus rebound potential in coral reefs. *Nature* **518**, 94-97, doi:10.1038/nature14140 (2015).

However, the reviewer's broader point is well taken – this is a large literature and we are happy to credit it appropriately to the greatest extent possible given citation limits. We have added additional references from the reef resilience literature in the introductions and expanded discussion section:

Edwards, H. J. *et al.* How much time can herbivore protection buy for coral reefs under realistic regimes of hurricanes and coral bleaching? *Global Change Biology* **17**, 2033-2048 (2011).

Anthony, K. *et al.* Ocean acidification and warming will lower coral reef resilience. *Global Change Biology* **17**, 1798-1808 (2011)

Hoegh-Guldberg, O. *et al.* Coral reefs under rapid climate change and ocean acidification. *Science* **318**, 1737-1742 (2007)

My interpretation of the author's results is that there is a seasonal signal in the responses of microbes to added nutrients and simulated overfishing. This is potentially publishable in a top journal, if it was presented much more transparently (see below). The seasonal shifts in assemblages is shown most clearly in Extended Data Figure 3; microbe assemblages are stable from 24-30C, and they change by a modest amount at 22 and 31C at the height of winter and summer.

We broadly agree. We have attempted to present the data more transparently now. In regards to why we tend to talk about temperature rather than seasonal shifts more broadly, we conducted 4 separate analyses to address the question of whether a seasonal factor other than temperature (e.g. algal community changes, water chemistry) might account for changes in microbial community structure, and in each case the available evidence seemed to point primarily towards temperature rather than other seasonal factors that we tested. That said, we did uncover some other seasonal factors that may play secondary roles. We report these results, and the necessary caveats regarding interpretation, in the Supplementary Text (lines 160-219):

Comparison of temperature vs. seasonal effects on microbial beta diversity

Temperature was an important correlate of many aspects of microbial community structure. Because temperature was not experimentally manipulated, and many other oceanographic parameters fluctuate seasonally, we sought to test whether apparent changes in temperature might be due to unrelated seasonal changes.

We first tested whether temperature increased microbial β-diversity within seasons as well as between seasons. We reasoned that if overall seasonal changes drove microbial β-diversity, and correlations with temperature were an incidental byproduct, then within-season effects of temperature on microbial β-diversity should be weak or non-existent. Instead, we found that even within summer and fall samples (considered separately), high temperatures (>30°C) resulted in greater β-diversity than non-stressful temperatures (24-29°C), indicating that thermal stress influences microbial communities within as well as between seasons (Summer: $p = 0.001$, permutational t-test; Fall $p = 0.001$, permutational t-test). Consistent with either warm or cold temperatures disrupting coral microbiomes, low (< 24° C) temperatures in winter were associated with significantly greater β-diversity than 24-29° C winter samples ($p=0.003$, permutational t-test).

To address the possibility that some other seasonal environmental factor besides short-term temperature changes might be the main driver of microbial β-diversity, we examined 40 environmental parameters measured seasonally by the SERC Water Quality Monitoring Network in South Florida, including dissolved inorganic nitrogen, total organic carbon, chlorophyll a, turbidity, SiO_2, etc (Supplemental Table S3k). To test whether differences in these parameters might explain microbial β-diversity, we constructed Euclidean distance matrices for each parameter across samples, and used Mantel tests, a permutational procedure for comparing two distance matrices, to test whether any of these environmental parameters significantly correlated with microbial β-diversity. No correction for multiple comparisons was performed in this instance, since we did not wish to miss a relevant parameter that might falsify our interpretation of the role of temperature.

Among the measured parameters and temperature, only three correlated significantly with microbial β-diversity. Daily temperature had the greatest influence ($r=0.127$, $p = 0.01$), with surface measurements of total organic carbon (TOC; $r =0.045$, $p = 0.02$), and turbidity ($r=0.09$, $p=0.04$) playing secondary roles. Notably, although daily temperature measurements (HCOM_temp_0m_degrees) were well correlated with microbial community structure, seasonal temperature measurements (collected by SERC quarterly at a single time point per season) were not (SERC_TEMP_B). This may indicate that short-term changes in temperature are important, above and beyond typical seasonal temperature trends.

A final possibility that we considered was that the apparent effects of temperature on β-diversity might be explained by the relatively modest seasonal variation in upright algal cover (which includes both tall turf algae and macroalgae). To disentangle the effects

of temperature from upright algal cover on microbial β-diversity, we conducted a partial Mantel test examining the relationship between temperature and microbial β-diversity while normalizing for the effect of upright algal cover. We found that temperature was still significantly correlated with β-diversity (Partial Mantel test ; r=0.12 p= 0.01) after accounting for the effects of upright algal cover (Supplemental Table S3k).

We do not interpret these results to mean that temperature is the only influence on microbial communities. For example, oceanographic parameters not significant on a quarterly basis may have important short-term or spatially localized effects that could be uncovered with high-resolution sampling. However, these results taken together argue against the possibility that the observed correlations between temperature and microbial community structure are artifacts of seasonal fluctuations in water chemistry or algal cover. Instead, short-term temperature variation appears to be an important factor influencing coral microbiome stability. This analysis also identified seasonal variation in dissolved organic carbon and turbidity as additional influences on coral microbial community structure, consistent with the effects of these parameters on corals and coral microbiomes in laboratory experiments[26,27].

Specific comments:

Line 37-38. The latter half of the sentence should have references.

The *Nature Communications* abstract is unreferenced, so this is no longer an issue. However, we've added some extra citations from the reef resilience literature to the discussion section.

Reference #3 is inappropriate here and on line 65- it does not provide any data on losses of corals or diversity.

We have replaced this reference.

Line 55. The authors' use of the term "stress" is inconsistent. On line 74, we're told that "microbiomes are sensitive to multiple stressors, including algal completion, elevated temperatures, and disease. Arguably, algal blooms and disease are ecosystem responses, arising from stresses such as pollution and overfishing.

The sentence in question from the previous revision (line 55) was: "Thermal stress exacerbated the impact of local stressors, further disrupting microbiomes of unhealthy corals and concentrating 80% of coral mortality in the warmest seasons."

We agree that the repetition of local stressors with thermal stress was a bit awkward, and have replaced 'local stressors' with 'local overfishing or nutrient pollution' in the corresponding line of the revised manuscript:

"Above-average temperatures exacerbated the impact of local overfishing and nutrient pollution, further disrupting microbiomes of unhealthy corals and concentrating 80% of coral mortality in the warmest seasons."

More broadly, the literature accommodates notions of stress at both organismal and ecosystem scales. In both cases, we think the meaning is clear. Overfishing and pollution (as the reviewer notes) are often discussed as 'stressors' to a coral reef ecosystem. High temperatures or competition from algae can certainly generate physiological stress in corals (this seems to be the usage that bothers the reviewer?). The notion of thermal stress has specific technical meaning that can't easily be replaced. Many factors, including disease, temperature, sedimentation, etc, are thought to 'stress' microbial ecosystems, in that they shift them from normal, healthy compositions to less desirable configurations for coral hosts.

The author's data also show that microbes respond to low temperatures.

We agree, and think it likely that both upper and lower physiological temperature limits for corals, if exceeded, reduce host control of the microbiome. In the Florida Keys, cold snaps like that of 2010 can be associated with significant coral mortality. We don't emphasize our findings regarding low temperatures strongly in the manuscript, however, because low-temperature effects (i) often did not (quite) rise to the level of significance, (ii) tended to be of smaller magnitude than microbial changes with high temperature, (iii) summarize fewer data points (there were not as many very low-temperature days in this dataset), (iv) were not associated with disproportionate coral mortality in the same way that high temperature periods were (only ~20% of coral mortality in our plots occurred in the cooler winter/spring seasons), (v) no formal definition for the lower physiological limit (e.g. cold thermal stress) is available that we could quantify in the same way as we have quantified degree heating weeks for high temperatures.

Exploring quantitative measures of the lower temperature limit for corals could be of interest in some ecosystems, especially at the outer edges of coral's ranges where cold temperatures are likely an important boundary. Our data suggest that in such systems it would be worth checking for disruption of the microbiome by cold temperatures. One could imaging calculating the minimum monthly mean and degree-cooling-weeks by analogy with existing maximum monthly mean and degree-heating-weeks measures, and comparing with microbiome data, but such a measure would need to be carefully validated, and we therefore think this interesting research direction is best left to other work.

Line 94. The authors state that they measured benthic cover by eye.

Benthic cover was assessed with standard quadrat surveys (Methods, Lines 651-671):

> **"Quantification of benthic cover**
> At least once every season (e.g. Spring, Summer, Fall, Winter at 12-14 week intervals), we visually quantified benthic cover within four, 50 cm X 50 cm quadrats in each of the 1 m^2 treatment areas. These quadrats were divided into 49 points, and benthic organisms under each point were identified to species or genus. Algae that are challenging to identify taxonomically under field conditions (e.g. crustose coralline algae, filamentous algae) were classified into algal functional groups e.g. [18]. Filamentous algae were classified into short algal turf (< 0.5 cm in

height) or algal turf (> 0.5 cm in height) e.g. [11] given that taller, thicker algal turf can often be deleterious to coral health and growth[19].”

I've never seen estimate so far in excess of 100%.

Algal cover can exceed 100% due to the overlapping, 3D nature of the algal canopy- if multiple algae are over a particular grid point in the quadrat survey, each is counted, which can result in >100% cover.

We note this in the figure legend (lines 511-512):
“Total cover often exceeds 100% due to the 3-D algal canopy.”

One reason that cover may be higher here than in some other surveys is that the treatments are extended far longer than past interventions. In Fig 1a., average cover does not exceed 100%, even in the caged and nutrient enriched plots, until more than one year into the study. Thus, if we had ended our intervention after less than 1 year, as previous studies have done, we would not have seen the full trajectory of algal succession that we document here.

How and why was the data "normalized" to 100%? Wouldn't that affect the structure of the data?

The statement in question was:

“Herbivore exclusion rapidly increased algal cover and species richness (Fig. 1a,b), and altered algal community composition (Extended Data Fig. 2, Supplementary Table S1a-c).”

As the reviewer notes above, data in Fig 1ab was not normalized to 100%. We do present an area-plot representation of the data showing the relative abundance of algal groups across treatments that is normalized to 100% for those who are interested (Extended Data Fig 2). So we feel that we have fairly covered both the overall increase in algal cover and diversity, and also the relative shifts in proportional representation of different algal taxa that accompany this increase. We considered a plot showing the absolute abundance of all algal taxa over time, but found that line plots with that many series rapidly become unreadable, and do not show anything that can’t be seen more clearly in existing figures. Nonetheless, we also present the raw data values for all algal taxa in the supplemental data without normalization (Supplemental Table S1a-c) for interested readers.

Line 103. It's not clear what "harm" means - elevated mortality, physiological responses, recruitment failure, etc.
The statement in question was:

“Overall, removing herbivorous fishes, and to a lesser extent nutrient pollution, facilitated growth of algae that harm corals via shading, abrasion, and allelopathy[7,9].”

We have changed ‘harm’ to: “known to increase coral tissue loss or mortality”

Line 113. Here and elsewhere (e.g. line 143, 145) it would be clearer to say "algal abundance" rather than competition.

We substituted ‘algal competition’ with ‘algal abundance’ when referring to quadrat survey data. We also changed ‘algal competition’ to ‘algal contact’ for photo-series data indicating that a particular coral is in frequent direct contact with macroalgae. We retain ‘algal competition’ at several places in the introduction and discussion to refer to the broader process of competition inferred from our data (including coral mortality data) and the broader literature.

ED Figure 3 shows only a small change occurred, in the top decile.

We agree that the upper end of the range of algal cover has the most dramatic changes. However, we tested for linear correlation between microbial abundance across all deciles of algal cover, and found highly significant linear correlation values for 11 bacterial orders while using FDR control for multiple comparisons (Figure 2a). This rules out the idea that the only significant effect is change at a single decile of algal cover.

The supplemental table, which summarizes the fold-change for orders significantly enriched by herbivore exclosure or nutrient pollution also shows that treatment effects produced both highly significant and sometimes quite large shifts in the relative abundance of particular microbial lineages. For example, Chlamydiales increased by 149% in the exclosure treatment relative to controls.

Extended Data Figure 3 is summarized at a phylum level, so it would be surprising if enormous shifts occurred. The original version of the manuscript presented order level results, which show more dramatic percent increases at the highest temperatures and/or levels of algal cover in key groups, but we removed these at reviewer request during previous rounds of revision. If there is any doubt that substantial microbial changes occurred in particular lineages we can certainly add them back in to Extended Data Figure 3.

Line 151. It's unconventional to equate same-to-sample variation with beta-diversity, which is normally spatial.

We acknowledge that the notion of beta-diversity is used in slightly different ways in ecology and microbial ecology. The original definition of beta-diversity, was sample-to-sample turnover in species composition, along a gradient (Whittaker 1972). Typically, these gradients were spatial. In microbial ecology, this definition has been generalized slightly, to include turnover in species between host samples separated by any gradient or discrete category of interest. It is very typical in that literature to use measures of beta-diversity to examine microbial community change over time (e.g. in studies of how the human gut microbiome matures in diverse human populations) or disease states (e.g. within samples from the lung microbiomes of smokers vs. non-smokers).

To clarify this somewhat different usage between fields for readers who come from a reef ecology background, we have adding the following sentences to the main text (lines 204-217):

> “Stressful conditions may shift coral microbiomes from one stable state to another[27]. We had originally hypothesized that algal competition would produce such a shift in the structure of coral microbiomes.

However, we did not find any evidence for such treatment-induced shifts between alternative stable states (Extended Fig. 6a). Rather, we found that algal contact and increased ambient temperatures reduced the overall stability of the microbiome as a whole. This type of microbiome destabilization was manifest as increased sample-to-sample variability and quantified using measures of microbiome β-diversity (e.g. [47]). While β-diversity was originally used to describe species turnover among habitats[48], in microbial ecology host organisms are often treated as the relevant habitat. This allows β-diversity measures to be used to compare inter-individual variation in the microbiome. This notion has also been generalized to measure species turnover within individuals over time (intra-individual variation). However, few studies (notably [47]) have emphasized the relationship between the health of animal hosts and the variability of their microbiomes."

Repeated samples are temporally auto-correlated.

We agree that repeated samples are partially auto-correlated within individuals. Both inter-individual and temporal shifts in the microbiome contribute to increased microbial variability in stressful conditions. As discussed above, we measured that increased variability using a beta-diversity distance metric (Weighted UniFrac) often used in this way in microbial ecology studies. We therefore interpret increased microbiome variability with e.g. algal contact to indicate some combination of increased inter-colony microbiome variation, or increased within-colony temporal variation

We should note that we report reanalysis of older work that placed algae in contact with corals, and recovered the same trend (increased microbiome variability with algal contact) in a setting where algal contact was experimentally manipulated over a fixed time period (Extended Data Fig. 7). This suggests that there isn't anything special about our particular experimental setup that might have caused an increase in microbiome variability artefactually.

As an informal point of information, we also have seen this increase in a follow-up experiment at the same site that also manipulated algal contact experimentally for a fixed period. A significant increase in beta-diversity with algal contact vs. controls/plastic mocks was also recovered there, consistent with the previous study analyzed in Extended Data Fig. 7 (a manuscript describing that experiment is *in prep*).

Therefore we are confident that this effect is real, and agree with reviewer that it should be interpreted as an increase in temporal variability and/or inter-colony variability. We agree that exploring these subtleties is an important future direction, and papers approaching these questions using stochastic modeling and meta-analysis of microbiome time-series data from several host-microbial systems are in prep.

The box plots in ED Figure 7 indicate that the differences among treatments are quite small (i.e. strongly overlapping upper and lower quartiles).

We report strongly significant differences between corals exposed or not exposed to algal competition in two experiments. We agree that in Extended Data Figure 7a, algal contact overall only modestly increases beta-diversity. In Extended Data Figure 7b, we break this down by the type of algae contacted, and show more pronounced effects

wherein the mean value for corals exposed to multiple algae or to *Dictyota* macroalgae exceeds the 3rd quartile value for control corals. Finally, in Extended Data Figure 7c, we re-analyze a previous algal competition experiment in which algal contact was manipulated directly. In that experiment, in which algal contact was manipulated specifically, the effect is even clearer, and there is no overlap between the upper and lower quartiles of control *Porites* corals and *Porites* corals exposed to macroalgae.

It is not yet known what levels of microbiome instability are biologically relevant. Few published studies document this type of dispersion effect that we can use to establish a baseline for what a biologically meaningful difference in beta-diversity looks like. A brief examination of disturbed vs. normal beta-diversity in the work of Mueller et al 2013 in SIV^+ chimpanzees suggests that the absolute magnitude of the changes in corals are large relative to at least one other condition that we know influences host immunity, vulnerability to opportunistic pathogenesis and fitness. There, the difference between control vs. disease conditions was approx. +0.05 in units of Bray-Curtis dissimilarity and +0.04 in units of Unweighted UniFrac. Our Weighted UniFrac values here were approximately +0.15 for all corals contacting *Dictyota* algae vs. those not contacting any algae and +0.10 for corals contacting any algae vs. those that did not. Weighted UniFrac accounts for phylogeny whereas Bray-Curtis dissimilarity does not, but both measure microbial beta-diversity on the same scale otherwise. As a back of the envelope calculation, this suggests that microbiome disruption in corals due to algal competition is, if anything, larger in magnitude than those in systems where we understand the mechanism by which hosts lose control of their microbiome in more detail.

We think a more systematic comparison of beta-diversity across many disturbed host-associated microbiomes would be needed to examine this point further, but feel that this would be outside the scope of the present manuscript. Indeed, we are planning to examine this in future work to allow for development of the relevant theory and background, and to do more extensive comparisons (e.g. by getting the raw data and calculating a suite of consistent diversity metrics across many studies).

Line 224. Reference #35 is less relevant than several empirical coral reef experiments.

We thank the reviewer for this suggestion, and have replaced the citation

Figure 1 shows that algal cover was 35-40% before the experiment began. So, is this reef already polluted?

These values and associated interpretation are reported in detail in the Supplemental Results (lines 28-47):

> "The reefs of the Florida Keys have robust herbivorous fish populations1 and are relatively oligotrophic2. Coral cover on most reefs in the Florida Keys, including our site, is 5-10%, while macroalgal cover averages ~15%, but ranges from 0-70% depending on location and season[3-5]. Parrotfishes (Scaridae) and surgeonfishes (Acanthuridae) are the dominant herbivores on these reefs as fishing for them was banned in 1981. The other important herbivore on Caribbean reefs, the urchin Diadema antillarum, remains at low densities across the Florida Keys following the mass mortality event in 1982-36."

The values observed in this site are somewhat above average for the Keys, but well within the ranges of 0-70% that are typically observed. It is worth noting that the first pretreatment timepoint came at a seasonal peak of algal abundance (see control time-series in Fig 1a) that recurred each year. At other times, the algal cover values in control plots were substantially lower (Fig 1a).

Figure 2 is hard to read - symbols are too small and the colors are too similar.

We tried many possible versions of this figure, and feel that this presentation best captures the main trend in the data. The main intent of the figure is to show how dominance by mostly *Synechococcus* (left side of figure) transitions to dominance by a wide array of other microbes (many colors, right side of figure), and this trend is the main one that separates microbial diversity across all of our coral microbiome samples. Other presentations that we tried (e.g. just highlighting *Synechococcus* dominance vs. dominance by any Proteobacterium using 2 colors, or coloring just the 4 most common orders) did not capture the variability in dominant taxon on the right end of PC1.

Given that this variability is a major part of our story (since it connects to our findings regarding stochastic blooms of different opportunists), we propose keeping the figure, but have added a line to the legend making it clear to readers that (unless they're specifically interested in one microbe), there's no need to squint at all the colors in order to get the key point that we're trying to make:

"The main pattern we seek to show here is a shift from *Synechococcus* dominated communities (cyan) to dominance by a wide variety of other orders as one moves from left to right along PC1"

What does "most dominant" mean?
This is stated in the figure legend, immediately after the quoted phrase:
"Points are colored to reflect the most dominant (numerically abundant) microbial order in each sample." As an example, if we have three orders of microbes (say A,B, and C) in a sample and A is 10% of bacterial sequences detected, B is 20% and C is 1%, we would call B dominant in that sample.

In 2c, are these mean temperatures?
Yes, these are the mean temperatures at which each bacterial order dominated the coral microbiome, where dominance is defined (as in ecology) as greatest numerical abundance. The idea is to show differences in conditions that favor different bacterial types.

Figure 3c - what are algal types or taxa?

Figure 3c shows number of algal contacts overall (the types used are those in Extended Data Fig. 2 and throughout the analysis). Simulated overfishing and nutrient pollution induced growth of many different combinations of algae. In Figure 3c we show that, on average, touching two or three species of macroalgae induces more tissue loss and greater microbiome disruption than touching a single species. We describe average coral mortality in categories based on contact with the most common algal types in Extended Data Fig. 8, and the effects of these algal types on the microbiome in Extended Data Fig. 7.

Figure 3d shouldn't stack multiple treatments. See earlier comment on 3f.

The Chi-squared test in 3d is by season overall. Colors are shown for information on the contribution of each treatment to mortality in each season. Overall mortality numbers are too low to run separate Chi-squared tests across each treatment.

Referee #2 (Remarks to the Author):

After re-reviewing this manuscript I am satisfied that the authors have made sufficient efforts to consider my comments and modify the MS accordingly. I believe that the manuscript delivers some interesting new insights into coral reef health that will be of interest to the readership of Nature.

We thank the reviewer for their constructive suggestions and these kind words.

Referee #3 (Remarks to the Author):

The authors have done a long-term experiment that represents a lot of work. They have done a good job of analyzing the data and addressed many of the technical concerns raised by the reviewers.

Many thanks for these comments, and for the suggestion of using machine-learning analysis in order to put a capstone on the novelty of the results. We are glad to see that broad agreement has been reached on the substance of the key conclusions put forward in the manuscript.

The problem that still remains is whether this represents a significant step forward in our understanding of coral reefs and their threats.

We have expanded the discussion to highlight the significance of this work, and state more clearly how it relates to the field as a whole, given the more generous word limit at *Nature Communications.*

Even without any novel findings this would represent by far the most comprehensive field test of the long-term interactive effects of nutrient pollution and herbivory in the coral literature. However the combination of this with an extensive time-series of microbial community changes in three genera of corals allowed us to uncover several novel features of how reefs respond to overfishing and nutrient pollution:

a. The 3-year duration of this study allowed us to capture novel temporal dynamics invisible in shorter-term studies. By extending this manipulation of herbivore pressure and nutrient pollution to twice the length of the longest previous study (and >2 years longer than the majority of field studies that address these questions), we were able to track the effects of experimental manipulations on the benthos, coral health, and coral microbiology over multiple thermal stress events that typically occurred only every summer and early fall. This allowed us to detect and quantify a novel and surprising linkage between local stressors and coral mortality during sub-bleaching thermal stress. Manipulating herbivore access and nutrient abundance on the reef made corals more vulnerable to blooms of harmful bacteria, and caused even sub-bleaching thermal stress to be lethal for many corals. However, sub-bleaching thermal stress was lethal only

when combined with nutrient pollution and/or overfishing - thermal stress during summer months did not increase coral mortality under control conditions. The idea that protecting herbivorous fishes would protect corals from temperature-induced mortality is not new, but we are the first to support it with experimental data. More importantly, we are the first to show that one potentially important mechanism whereby herbivores protect corals is that they prevent destabilization of coral microbiomes by algal competition.

b. We provide direct experimental evidence that overfishing and nutrient pollution destabilize coral microbiomes by driving high macroalgal and turf algal cover. We also show that these microbiological changes are tightly linked to coral tissue loss, disease, and mortality. These linkages have been suggested by previous large-scale correlative, but confounded, work across gradients of anthropogenic impact and small-volume, bench-top lab experiments, which, although carefully controlled, have been criticized as lacking ecological realism. Thus, it has never been clear if this mechanism is really important for impacting corals on ecologically relevant temporal and spatial scales. Our work not only shows that this mechanism is an important driver of coral mortality in an ecologically realistic setting but also shows that there is a strong interaction with thermal stress in driving coral mortality. Thus, these results connect key ideas about coral microbiology proposed based on laboratory studies to broader ecological processes. We agree with Reviewer 3 that the impacts of herbivores and nutrients on algae, and even corals, have been well tested over shorter time-scales. However, this is the first time that these experiments have been married to microbial analysis and extended long enough to allow for examination of the interactive effects of nutrients, herbivory, and temperature.

c. In addition to the novelty of pairing microbiology to this type of study, the outcome of the microbial analysis was itself quite surprising. While models of the coral microbiome have predicted that stress would shift coral microbiomes towards pathogen dominance, we show that the specific pathogens that come to dominate under algal competition or thermal stress were very unpredictable. Instead of shifting communities to a new stable state, as current theory would predict, algal competition and thermal stress destabilized the microbiome (increasing community dissimilarity) by driving communities away from a relatively stable configuration dominated by *Synechococcchus* towards unstable community structures dominated by stochastic blooms of multiple opportunistic microbial assemblages. This novel finding, confirmed by reanalysis of previous studies, contrasts with prevailing theories and mathematical models of the coral microbiome, which predict shifts to particular new stable states. Instead, our data mirror recent important findings in disturbed human and primate microbiomes, suggesting an underexplored paradigm that may be common to many disturbed host-microbe systems.

d. Lastly, our long-term field experiments uncovered an entirely unexpected effect of nutrient enrichment that fundamentally altering the outcome of normal trophic interactions on reefs. Under ambient nutrient conditions parrotfish predation was not harmful to corals. However, nutrient enrichment turned parrotfish predation deadly for corals by shifting coral microbiomes towards potential pathogens after predation. An interaction between parrotfish predation and nutrient loading on coral health has never previously been reported or

hypothesized. This unexpected observation is especially worrisome as it shows that nutrient pollution turns parrotfishes, which normally promote coral health by consuming macroalgae, into agents of coral mortality. This is especially important given that nutrient pollution is a problem on many reefs and parrotfish restoration is an important goal as part of coral conservation and management. Our data suggest that restoring parrotfish without controlling nutrient pollution could be ineffective, or even a recipe for enhanced coral death.

1) The major hypothesis about how bacteria cause coral mortality is based on dissolved organic carbon, which was not measured (Google Scholar 84 references, including 2 by the senior author). Direct contact mediated coral mortality also includes several references to the microbiology (e.g., Morrow et al., hydrophilic versus hydrophobic compounds); none of these compounds were measured in the current study. The authors are treating the expected effects of overfishing and nutrient enrichment as if we are extremely naive instead of putting their results into the current context of field.

We are familiar with the hypothesis raised by Reviewer 3, as Dr. Rohwer was Dr. Vega Thurber's mentor and she was an author on two of the papers that tested it. We agree that given the expanded word count available at *Nature Communications*, an expanded introduction to current work in the coral microbiome, including the DDAM hypothesis, benefits the manuscript. We have greatly expanded our treatment of existing theories of coral microbiology in the introduction, emphasizing literature on the effects of algae on the microbiome (both the DDAM hypothesis and some others), as well as some major ideas about how temperature affects the microbiome. We thank the reviewer for this suggestion.

We have also greatly expanded our discussion section in order to be able to highlight some of our findings that speak to these ideas. However, while debates over whether specific algal species alter bacterial growth on corals mostly through dissolved organic carbon, or mostly through other proposed mechanisms (e.g. hydrophobic allelochemicals, direct transfer of microbes, abrasion, or shading) are worth acknowledging, in our view they are not essential to the novelty of our results. The goal of this experiment was not to test all mechanisms of coral-algal interaction individually: many of these appear to be at least partially species specific within coral-algal pairs. Instead, we sought to definitively test the idea that removing herbivorous fishes and increasing nutrient pollution could cause coral microbiome disruption, and ultimately coral death over ecologically realistic temporal and spatial scales, and if so, to trace the benthic and microbiological steps by which these negative effects occurred.

2) There are over 25 studies that use nutrient enrichment and/or caging on coral reefs. They all find about the same thing as reported in this manuscript; remove of grazers is the most important controller of most algae and there is an additive effect of nutrient additions.

We agree that many studies have assessed the role of nutrient pollution or herbivory on algal growth. Fewer studies have extended manipulations across multiple seasons to determine interactions with seasonal temperature variation (most have covered weeks or months), and none have combined such a multi-year field manipulation with microbial time-series data.

This integrative approach allowed us to observe that even in the absence of coral bleaching, overfishing and pollution sensitize corals to warm temperatures, which combines with local stressors to increase coral microbiome disruption and coral mortality. To date there have been no field experiments other than ours that empirically test this idea (nor are any cited by the reviewer). We agree with Reviewer 3 that the literature contains much discussion of the idea that protecting herbivorous fishes improves reef health in general, and that this might help ameliorate coral decline from global warming. However, much of this work is from modeling exercises or mere speculation. In this study, we not only show that protecting herbivorous fishes prevents coral mortality under stressful temperatures, but also demonstrate that a likely mechanism of this protection is prevention of algal-induced disruption of the coral microbiome. This empirical demonstration that overfishing, through its intermediate effects on benthic communities, makes corals vulnerable to pathogens during periods of above-average temperature is fundamentally new to the field.

That said, we appreciated the reviewer's point regarding the additive effects of overfishing and nutrient pollution. One finding we had previously not emphasized was that, while the effects of nutrient pollution and overfishing on benthic communities may be roughly additive, their effects on coral mortality were decidedly not additive. We have added substantial discussion of how the data violated our expectation that local stressors would have additive effects on coral mortality, and now connect this back to some of the new mechanisms of coral death that we uncovered (e.g. the interaction between corallivory, nutrients, and microbial opportunism).

The only thing novel is that parrotfish seem to be targeting nutrient-enriched coral, but this has been shown for the algae.

Our field experiment uncovered an extremely strong and unexpected interaction between nutrient pollution and parrotfish predation that has never previously been even hypothesized, but is likely to have important consequences for reef conservation. Corals bitten by parrotfish in ambient conditions experienced 0% mortality, while those bitten in nutrient enriched conditions suffered 60% mortality (Fig 4). Thus the novelty of this aspect of the story is not so much about alteration of parrotfish behavior (which has indeed been discussed elsewhere), but on what happens to corals after they are bitten by parrotfish. We documented significant shifts in the coral microbiota towards opportunists prior to coral death only in corals exposed to bites + nutrient pollution. The microbiological data suggest that increased nutrient-fueled susceptibility to infection is a likely mechanism underlying this surprising and unexpected finding. This outcome matters because parrotfish restoration is championed as a key step to reef restoration. These findings suggest that this may be either ineffective or a recipe for coral death in polluted areas unless nutrient levels are also lowered.

We have expanded our discussion of these results on parrotfish predation in the discussion to better connect these findings to the broader literature.

The most unique aspect of this work is the long term study with some microbiology.

We thank the reviewer for these kind words, and agree that the combination of long-term experimental intervention with microbial time series are a key part of the novelty of this study.

The authors need to rewrite the manuscript reflecting our current understanding and mechanistic models of how coral reefs are influenced by overfishing and eutrophication, with particular attention to the microbiology (that is, the major novelty of their study).

We have taken reviewers suggestions in this regard very seriously, and have undertaken a substantial revision of our presentation to better reflect these considerations.

Therefore in the new introduction and discussion we have greatly expanded our treatment of the proposed role of algal DOC in coral-algal competition (and related, non-mutually exclusive hypotheses of how specific algae may affect coral microbiomes). We have also expanded our coverage of current research and quantitative models on bacterial opportunism during periods of warm seawater temperature. In the results section, we have elaborated on some of our predictive functional profiling results (from PICRUSt) regarding bacterial sugar vs. lipid and amino acid metabolism that bear on the DDAM hypothesis (corals competing with algae have microbiomes with more predicted sugar metabolism genes, consistent with enrichment of specific lineages by algal neutral sugars). Finally, we have also greatly expanded our discussion section in order to better describe how we see these results in relationship to the broader field.

We thank the reviewer for their suggestions, which have improved the manuscript.