

Assessing threats from coral and crustose coralline algae disease on the reefs of New Caledonia

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Abstract. The present study reports the results of the first quantitative survey of lesions on coral and crustose coralline algae (CCA) on reefs in the lagoon of New Caledonia. Surveys on inshore and offshore reefs were conducted at 13 sites in 2010, with 12 sites resurveyed in 2013. Thirty coral diseases affecting 15 coral genera were found, with low overall disease prevalence (<1%). This study extends the known distribution of growth anomalies to the coral genera *Platygyra* and *Hydnophora*, endolithic hypermycosis to *Platygyra*, *Leptoria* and *Goniastrea* and extends the geographic range of three CCA diseases. We found the first trematode infection in *Porites* outside of Hawaii. Disease prevalence differed among coral genera, with *Porites* having more lesions, and *Acropora* and *Montipora* fewer lesions, than expected on the basis of field abundance. Inshore reefs had a lower coral-colony density, species diversity and reduced CCA cover than did the offshore reefs. Disease prevalence was significantly higher on inshore reefs in 2013 than in 2010, but did not change on offshore reefs. The potential ecological impact of individual coral diseases was assessed using an integrative-scoring and relative-ranking scheme based on average frequency of occurrence, prevalence and estimated degree of virulence. The top-five ranked diseases were all tissue-loss diseases.

Additional keywords: CCA disease, coral disease, ecological impact of coral disease, endolithic hypermycosis, growth anomalies, trematode infection, white syndrome.

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Introduction

Recently, coral disease has emerged as a serious threat to coral reefs worldwide and the numbers of diseases, species affected and the distributions of diseases have all increased within the past decade (Green and Bruckner 2000; Sutherland *et al.* 2004). Diseases have already dramatically affected the reefs of the Caribbean (Gardner *et al.* 2003; Sutherland *et al.* 2004; Schutte *et al.* 2010) and are now being reported with increasing frequency from across the Indo-Pacific (Willis *et al.* 2004; Vargas-Angel and Wheeler 2009; Aeby *et al.* 2011a). Increased anthropogenic stress on near-shore environments, overfishing and environmental conditions associated with global climate change have all been implicated as factors contributing to increases in observed disease levels (Colwell 2004; Harvell *et al.* 2007).

The Indo-Pacific region poses a serious challenge for the management of emerging coral diseases because it is much larger than the Caribbean and supports entire communities that depend on reefs for their livelihood (Hughes *et al.* 2003). Baseline disease surveys provide a basis to assess status and

trends of coral-reef health over time, yet numerous regions throughout the Indo-Pacific have not been surveyed. New Caledonia forms a large group of islands in the South-west Pacific that has one of the longest continuous barrier reefs and the largest lagoon in the world, including fringing reefs, patch reefs and barrier reefs (Laboute and Richer de Forges 2004; Andréfouët *et al.* 2006). The reefs of New Caledonia have a fairly high diversity of corals (Payri and Richer de Forges 2006; Fenner and Muir 2009; Fenner 2011), and this unique resource has received international recognition and has been designated a UNESCO World Heritage site.

However, reefs of New Caledonia are prone to both natural and man-made threats. Cyclones have been known to severely reduce coral cover (Wilkinson 2008), and New Caledonia has active open-surface nickel mining (1/4 of the global nickel resources are located in New Caledonia) and mining pits are not re-vegetated. They also have agricultural industries that result in extensive runoff, laden with pollutants that are particularly severe during the wet season (Fernandez *et al.* 2006; Bonvallot *et al.* 2012). In addition, some reefs could be subject



Fig. 1. Sites surveyed for coral and crustose coralline algae (CCA) disease in 2010 and 2013 around the largest island of New Caledonia, Grande Terre. Six inshore–offshore pairs of reefs were surveyed ($n = 12$ sites) and one lagoonal reef. 1, Ilôt Casy (inshore reef); 2, Gué reef (offshore barrier reef); 3, Baie des Citrons (inshore reef); 4, Séche Croissant reef (lagoonal patch reef); 5, Mbere reef (offshore barrier reef); 6, Chenal de Teremba (inshore reef); 7, Passe de Ouarai (offshore barrier reef); 8, Kreliat reef (inshore reef); 9, Passe de Koné (offshore barrier reef); 10, Bouerabate reef (inshore reef); 11, Passe de la Gazelle (offshore barrier reef); 12, Neongaon reef* (inshore reef); 13, Balade reef (offshore reef in front of the Col d'Amos). Sites with asterisks (*) were surveyed in 2010 only.

to episodic industrial chemical spills and sewage because of insufficient treatment infrastructures. A few coral diseases have been reported from the reefs of New Caledonia (McKenna 2009, 2011), yet no quantitative baseline disease data exist for these reefs. The objectives of the present study were to determine the health status of corals and crustose coralline algae in the lagoon of New Caledonia, in nearshore reefs subject to terrigenous inputs and anthropogenic activities, compared with more remote offshore barrier reefs in a 2-year time period.

Materials and methods

Study site

New Caledonia is a special collectivity (semi-independent status) of France located in the Melanesian region of the south-western Pacific Ocean, 1210 km east of Australia (Fig. 1). The archipelago includes the main island of Grande Terre, the Loyalty Islands, the Chesterfield Islands, the Belep archipelago, the Isle of Pines and other assorted islets (Lasne 2007).

Disease surveys

Thirteen reefs were surveyed in the lagoon surrounding the main island of Grande Terre, including one mid-lagoonal reef, six inshore and six offshore reefs (Fig. 1). Owing to logistical constraints and difficulties in accessing eastern Grande Terre, most surveys were conducted on the western coast of Grand Terre, with only two sites on the north-eastern coast. Inshore–offshore comparisons were made to examine potential anthropogenic effects on coral reef health. The locations of inshore reef

sites ranged between 1- and 8-m depth and were situated 1–3 km from the shore. Outer barrier reefs were located ~10–16 km offshore on the outer reef slope at depths between 6 and 19 m and the lagoonal reef was located at 1–2-m depth, a few kilometers north of the city of Nouméa (south lagoon). The baseline levels of coral and crustose coralline disease at each of the selected sites were documented using two 25-m belt transects laid end-to-end along depth contours, separated by ~5 m. All coral colonies within each 1-m-wide belt (25 × 1 m) were identified to genus and enumerated. Substrate was quantified by point-intercept method, whereby the substratum underlying the tape measure was recorded at 50-cm intervals. The average percentage coral cover and colony density per square metre were determined from the diver surveys. Lesions in corals and CCA were quantified along wider 25 × 6 m transects (total of 300-m² area of reef). All lesions encountered during the surveys were photographed and representative samples collected for histopathology and surveys of microboring organisms, which can be involved in coral diseases (e.g. Work *et al.* 2008a; see also Tribollet 2008). Surveys were conducted in the summer season, January 2010 ($n = 13$ sites) and February 2013 ($n = 12$ sites). The GPS coordinates were recorded for each site in 2010 and were used to relocate sites in 2013.

Gross lesions were classified into three broad categories including tissue loss, discoloration and growth anomaly (Work and Aeby 2006). Nomenclature for lesions was based on the host affected and lesion type (e.g. *Porites* growth anomaly; Work and Aeby 2006). Tissue-loss lesions can be caused by biological factors, such as predation or sedimentation, as well as infectious

disease, and it can be challenging to discriminate between these processes in the field. However, valuable clues can be gained from looking at the lesion size, shape, presence of predators, knowledge of what common predation marks look like and evidence of lesion progression based on degree of algal colonisation onto the bare coral skeleton. We conducted our disease surveys using the aforementioned criteria but also collected samples for follow-up histological analyses. Histology allows you to further differentiate between potentially infectious versus non-infection processes (Work *et al.* 2012). After histological examination of lesions, tissue-loss lesions not found associated with an obvious pathogenic organism, usually ciliates or cyanobacteria, were termed 'white syndrome'. If ciliates or cyanobacteria were found microscopically invading coral tissue in the lesion, then they were termed ciliate or cyanobacterial infections. Results of histological analyses for cnidaria are reported in Work *et al.* (2014). Most disease surveys have not included histological findings in the interpretation of their survey results.

Data analysis

For corals, prevalence of lesions was calculated by extrapolating colony counts within the 25×1 m transect to the wider 25×6 m disease survey area and by using this as the denominator of prevalence calculations (e.g. (number of colonies with lesions \div total number of estimated colonies) \times 100). CCA substrate cover was estimated within each survey area by determining the percentage cover by point-intercept and extrapolating that out to the spatial area of the belt transects. Abundance of CCA disease was then determined by calculating the number of CCA lesions per estimated square metre of CCA surveyed. The frequency of disease occurrence (FOC) was defined as the number of sites having corals or CCA with lesions divided by total number of sites surveyed. FOC reflects the spatial distribution of diseases on reefs.

Data were not normally distributed, even with transformation, so non-parametric analyses were used. A matched-pairs signed-ranks test was used to examine differences in coral disease prevalence and CCA disease abundance within sites between years. A Wilcoxon two-group test was used to test for differences between regions (offshore vs inshore) in coral cover, CCA cover, coral disease prevalence, number of coral genera and colony density (number of coral colonies per square metre). For each survey period (2010 and 2013), a chi-square goodness-of-fit test examined differences in the distribution of the number of diseased versus healthy colonies among the scleractinian genera affected by disease. Non-parametric statistics were performed using JMP statistical software (v. 10.0.2, SAS Institute Inc., Buckinghamshire, UK).

To examine differences in coral community structure between regions (offshore vs inshore), the data were square-root transformed and an analysis of similarity (ANOSIM), a non-parametric permutation procedure, was performed. A non-metric multi-dimensional scaling (nMDS) plot based on Bray–Curtis similarity was used to illustrate the differences in coral communities among sites and a SIMPER analysis examined the percentage contribution of each genus towards the groupings (PRIMER v. 6.1.12, Ivybridge, UK).

The potential for damage to coral reefs from diseases would depend on the spatial distribution (frequency of occurrence), prevalence (proportion of colonies surveyed that were affected) and degree of virulence (harm to host) of the disease. These three metrics were used to develop an integrative-scoring and relative-ranking scheme to compare the potential ecological impact of each disease. Ranking scales have been used successfully in other studies to examine coral-ecosystem health (Jokiel and Rodgers 2007). For each disease, the average prevalence and frequency of occurrence was calculated across both years. On the basis of what is known about the ecology of each disease, potential virulence was categorised as low (reduced growth or reproduction), medium (chronic tissue loss) or high (acute or subacute tissue loss). The scores were scaled from 0 (not harmful) to 10 (very harmful). To get each metric under the same 0–10 scale, the average frequency of occurrence data were divided by 10, prevalence data were multiplied by 10 and the prevalence categories were assigned scores of 1 for low virulence, 5 for medium virulence and 10 for high virulence. A mean score for each disease was calculated as the average of the three metrics.

Results

Coral-reef characteristics

We identified 44 coral genera, within transects, and four of those coral genera were numerically dominant. These were *Acropora* (2010: ~43% of the coral community; 2013: ~31.9%), *Porites* (2010: ~11.7%; 2013: ~16.1%) *Montipora* (2010: ~11.0%; 2013: ~16.0%) and *Pocillopora* (2010: ~6.8%; 2013: ~4.5%). Coral cover ranged from 7.8–83.3% (~35.3%) in 2010 and 13.9–63.4% (~44.6%) in 2013. CCA cover ranged from 0% to 38.2% (~9.9%) in 2010 and from 0% to 24.7% (~8.6%) in 2013.

Spatial and temporal occurrence of disease

Coral diseases

We examined a total of 47 166 scleractinian corals from 3780-m² reef area in 2010 and 38 251 corals from 3600-m² reef area in 2013. Thirty different coral diseases were found, affecting 15 coral genera on the reefs of the New Caledonian lagoon. Ten diseases were observed in both years surveyed, 11 and 9 diseases exclusively in 2010 and 2013 respectively (Table 1). Average prevalence of disease differed among disease types, but was overall low (<1%) in both years surveyed (Table 2). Average disease prevalence was 0.165% (s.e. $\pm 0.05\%$) in 2010 v. 0.287% (s.e. $\pm 0.1\%$) in 2013 (matched-pairs sign test, $t = 2.1$, $n = 12$, $P = 0.06$). Frequency of occurrence (FOC) of the different diseases varied between years (Table 1) but the most common diseases (>15% of sites in both years) were *Acropora* white syndrome, *Acropora* growth anomalies, *Porites* multi-focal pink spot, *Porites* growth anomalies, *Montipora* growth anomalies and *Pavona* dark spot.

CCA disease

The following three CCA diseases were documented: CCA white syndrome, CCA concentric white discoloration (target phenomenon) and coralline lethal orange disease (CLOD). CCA

Table 1. Frequency of occurrence (FOC, %) of coral diseases surveyed in New Caledonia in 2010 and 2013

Thirteen sites were surveyed in 2010 and 12 sites in 2013. White syndrome indicates a tissue-loss disease not found associated with an obvious pathogen on histological analysis

Disease	FOC (%)	
	2010	2013
<i>Acropora</i> growth anomalies	23.1	50
<i>Acropora</i> white syndrome	46.2	25
<i>Acropora</i> ciliate infection	7.7	25
<i>Astreopora</i> white syndrome	7.7	0
<i>Astreopora</i> cyanobacterial infection	0	8.3
Faviid dark spot	23.1	0
Faviid growth anomalies	7.7	8.3
<i>Galaxea</i> white syndrome	7.7	8.3
<i>Goniastrea</i> dark spot	7.7	0
<i>Goniastrea</i> growth anomalies	7.7	0
<i>Hydnophora</i> growth anomalies	0	8.3
<i>Hydnophora</i> white syndrome	0	8.3
<i>Leptoria</i> dark spot	7.7	0
<i>Montipora</i> growth anomalies	23.1	25
<i>Montipora</i> cyanobacterial infection	7.7	8.3
<i>Pachyseris</i> bleached patch	0	8.3
<i>Pachyseris</i> white syndrome	7.7	0
<i>Pavona</i> dark spot	15.4	16.7
<i>Pavona</i> white syndrome	7.7	0
<i>Pectinia</i> white syndrome	7.7	0
<i>Platygyra</i> growth anomalies	0	16.7
<i>Platygyra</i> dark spot	7.7	0
<i>Porites</i> bleached patch	0	16.7
<i>Porites</i> growth anomalies	38.5	25
<i>Porites</i> multi-focal pink spot	53.8	41.7
<i>Porites</i> white syndrome	0	25
<i>Porites</i> cyanobacterial infection	0	8.3
<i>Symphyllia</i> dark spot	7.7	0
<i>Turbinaria</i> cyanobacterial infection	7.7	0
<i>Turbinaria</i> white syndrome	15.4	0

diseases were found predominantly on offshore reefs, with frequency of occurrence of CCA white syndrome 46.2% in 2010 and 33.3% in 2013; CCA concentric white discoloration was 0% in 2010 and 8.3% in 2013 and CLOD was 30.8% in 2010 and 16.7% in 2013. The number of CCA lesions ranged from zero to 13 per site. At sites where CCA disease occurred, the average number of lesions was 0.11 m^{-2} CCA (s.e. ± 0.05) in 2010 and 0.09 m^{-2} CCA (s.e. ± 0.03) in 2013.

Differences in disease prevalence among coral genera

Out of 44 genera surveyed, only 15 showed signs of coral disease. Prevalence of disease differed among the coral genera within each year surveyed (2010: $\chi^2 = 411.9$ d.f. = 9, $P < 0.001$; 2013: $\chi^2 = 174.5$, d.f. = 12, $P < 0.001$). Of the four most abundant scleractinian coral genera found within transects, *Porites* had the highest overall prevalence (2010: 0.73%; 2013: 1.5%), followed by *Montipora* (2010: 0.07%; 2013: 0.1%), *Acropora* (2010: 0.06%; 2013: 0.1%) and *Pocillopora* (0% both years). Of the main coral genera affected by disease, *Acropora* and *Montipora* had fewer diseased colonies than was expected

Table 2. Mean (\pm s.e.) disease prevalence of coral diseases surveyed in the Lagoon of New Caledonia in 2010 and 2013

Thirteen sites were surveyed in 2010 and 12 sites in 2013. Data include diseased colonies only within transects and so will differ from frequency of disease-occurrence data. White syndrome indicates a tissue-loss disease not found associated with an obvious pathogen on histological analysis

Disease	Average prevalence (%)	
	2010	2013
<i>Acropora</i> growth anomalies	0.02 (0.01)	0.06 (0.03)
<i>Acropora</i> white syndrome	0.04 (0.02)	0.03 (0.02)
<i>Acropora</i> ciliate infection	0.004 (0.004)	0.04 (0.03)
<i>Astreopora</i> white syndrome	0.25 (0.25)	0
<i>Astreopora</i> cyanobacterial infection	0	1.39 (1.39)
Faviid dark spot	0.21 (0.14)	0
Faviid growth anomalies	0.03 (0.03)	0.02 (0.02)
<i>Galaxea</i> white syndrome	0.04 (0.04)	0.02 (0.02)
<i>Goniastrea</i> dark spot	7.69 (7.69)	0
<i>Goniastrea</i> growth anomalies	0.25 (0.25)	0
<i>Hydnophora</i> growth anomalies	0	0.46 (0.46)
<i>Hydnophora</i> white syndrome	0	0.05 (0.05)
<i>Leptoria</i> dark spot	0.32 (0.32)	0
<i>Montipora</i> growth anomalies	0.04 (0.03)	0.06 (0.04)
<i>Pachyseris</i> bleached patch	0	0.35 (0.35)
<i>Pavona</i> dark spot	0.12 (0.09)	0.06 (0.04)
<i>Pavona</i> white syndrome	0.04 (0.04)	0
<i>Platygyra</i> dark spot	0.32 (0.32)	0
<i>Porites</i> bleached patch	0	0.65 (0.64)
<i>Porites</i> growth anomalies	0.27 (0.10)	0.23 (0.14)
<i>Porites</i> multi-focal pink spot	0.3 (0.13)	0.4 (0.19)
<i>Porites</i> white syndrome	0	0.29 (0.20)
<i>Porites</i> cyanobacterial infection	0	0.01 (0.01)
<i>Turbinaria</i> cyanobacterial infection	0.43 (0.43)	0
<i>Turbinaria</i> white syndrome	0.19 (0.19)	0

on the basis of their abundance in surveys, and *Porites* had more diseased colonies than expected (Fig. 2).

Ecological rank of potential damage from coral diseases

On the basis of the average score from the three metrics (average FOC, average prevalence and virulence category) the top-five ranked diseases were all tissue-loss diseases and included white syndrome in *Acropora*, *Porites*, *Astreopora* and *Turbinaria*, and cyanobacterial infection in *Turbinaria* (Fig. 3). FOC and prevalence of diseases were low in surveys and none of the average scores was above 5 in this 0–10 ranking scale.

Coral-reef health state of offshore versus inshore reefs

In 2010, we surveyed six inshore–offshore profiles and found that average coral cover did not differ significantly between offshore and inshore reefs (Wilcoxon two-group test, $Z = 0.40$, $n = 12$, $P = 0.7$; Table 3). However, offshore reefs had a significantly higher average colony density (Wilcoxon two-group test, $Z = 2.8$, $n = 12$, $P = 0.005$), number of coral genera (Wilcoxon two-group test, $Z = 2.7$, $n = 12$, $P = 0.008$) and crustose coralline algal cover (Wilcoxon two-group test, $Z = 2.1$, $n = 12$, $P = 0.04$; Table 3). There was a shift in the abundance and composition of coral genera found in the

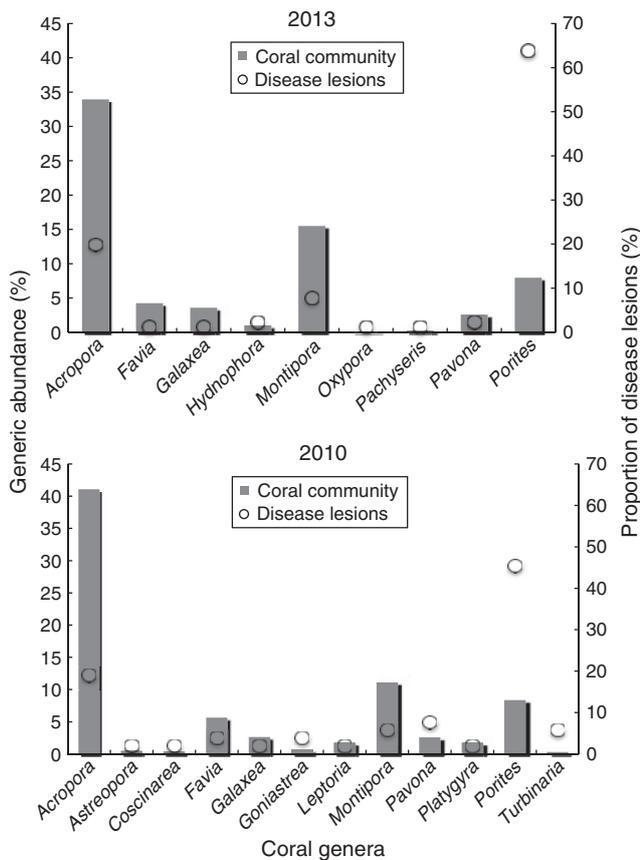


Fig. 2. Relationship between overall abundance of coral genera within transects and proportion of corals with signs of disease (lesions). Top graph shows data from surveys conducted in 2013 and bottom graph from surveys in 2010.

different habitats (ANOSIM, $r = 0.33$, $P < 0.02$; Table 4). SIMPER analysis showed that the coral genus, *Porites*, explained the highest percentage (9.9%) of the differences between inshore and offshore reefs. *Porites* comprised an average of 21.4% of the coral community on inshore reefs, as compared with 5.1% on offshore reefs. An nMDS plot (Fig. 4) showed two groupings (inshore vs offshore) and also revealed several inshore survey sites that were distinct. For example, the inshore reef, Chenal de Teremba, had eight scleractinian genera found within transects, compared with the other five inshore sites with coral diversity ranging from 12 to 24 genera. The two inshore sites, Bouerabate and Casy, had a low abundance of *Porites* (3.1 and 3.2% respectively) compared with the other four sites, with *Porites* abundance ranging from 15.6% to 61.3% of the coral community.

Average disease prevalence did not differ between inshore (mean $0.26\% \pm 0.09$ s.e.) and offshore reefs (mean $0.10\% \pm 0.02$ s.e.) (Wilcoxon two-group test, $Z = 0$, $n = 12$, $P = 1.0$). However, inshore reefs had a significant increase in disease prevalence from 0.17% (s.e. $\pm 0.06\%$) in 2010 to 0.57% (s.e. $\pm 0.18\%$) in 2013 (matched-pairs sign test, $t = 3.13$, $n = 5$, $P = 0.03$), whereas offshore reefs had no significant change, with disease prevalence being 0.10% (s.e. $\pm 0.02\%$) in 2010

and 0.09% (s.e. $\pm 0.02\%$) in 2013 (matched-pairs sign test, $t = -0.59$, $n = 6$, $P = 0.58$; Fig. 5).

Discussion

Here, we report on the first quantitative coral- and CCA-disease study to be conducted on the reefs of the largest lagoon in the world, i.e. the lagoon of New Caledonia. During disease surveys in 2010 and 2013, we found a total of 30 different coral diseases affecting 15 coral genera and three CCA diseases. The number of diseases we found is higher than reported from other regions of the Indo-Pacific. Vargas-Angel and Wheeler (2009) reported 10 diseases from across the USA Pacific territories and affiliated states (Hawaii, American Samoa, Pacific Remote Island Areas, Guam, northern Mariana Islands) and Muller *et al.* (2012) reported five diseases from Sulawesi, Indonesia. Differences in numbers of reported diseases are, in part, a result of differences in nomenclature used among researchers, making regional comparisons difficult, and this has been pointed out elsewhere (Work *et al.* 2008a; Aeby *et al.* 2011b). Assigning a specific name to a disease lesion *in situ* is difficult because there is limited information on coral-disease etiologies, ecologies and pathogen specificities. The 'white syndromes' are the classic example of a lesion type that is now known to have different underlying etiologies, ecologies and differences in host specificity, even though the gross lesions (i.e. lesion determined underwater) may look similar. For example, tissue-loss diseases (white syndromes) in corals are associated with bacterial pathogens (Sussman *et al.* 2008; Luna *et al.* 2010; Ushijima *et al.* 2012, 2014), chimera parasites (Work *et al.* 2011), programmed cell death perhaps in response to pathogens (Ainsworth *et al.* 2006), as well as a variety of other organisms such as algae, sponges, endolithic fungi and helminths (Work and Aeby 2011; Work *et al.* 2012). In some regions, white syndrome can be seasonal (Willis *et al.* 2004; Dalton and Smith 2006; Bruno *et al.* 2007), whereas this is not the case in other regions (Aeby *et al.* 2009, 2010). From field patterns of disease occurrence and evidence of direct transmission in the field, it is suggested that some white syndromes are host generalists affecting numerous coral genera (Willis *et al.* 2004; Dalton and Smith 2006), whereas others affect a single coral genus (Aeby *et al.* 2010, 2011a). Thus, white syndrome is a non-specific lesion with multiple potential causes and host responses (Work *et al.* 2012). Other diseases, such as black-band disease (BBD), have a more consistent microscopic presentation, with tissue damage being associated with a microbial consortium visually dominated by filamentous cyanobacteria, which is consistent across numerous regions in both the Indo-Pacific (Frias-Lopez *et al.* 2003; Barneah *et al.* 2007; Sato *et al.* 2009) and the Caribbean (Rützler and Santavy 1983; Richardson 2004). BBD consistently affects a wide range of coral genera (Sutherland *et al.* 2004) and becomes more prevalent during warm-water months (Voss and Richardson 2006; Sato *et al.* 2010). Until more is known about the aetiology and ecology of other coral diseases, researchers are advised to report diseases by host genera affected and lesion type (Work and Aeby 2006).

Some diseases in New Caledonia, such as *Porites* and *Acropora* growth anomalies, are widespread across the Indo-Pacific (Aeby *et al.* 2011c); however, our findings represent a

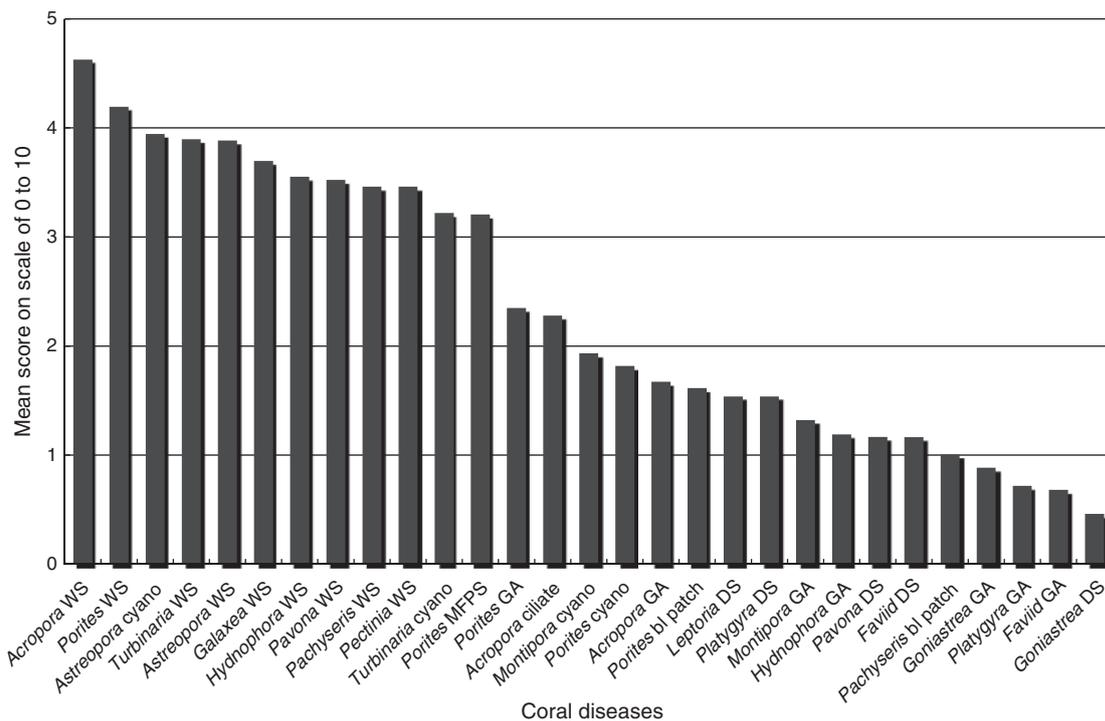


Fig. 3. Mean score on a scale of 0–10 of the potential ecological impact for each coral disease found on the reefs of New Caledonia. Scores were the average of three metrics (frequency of occurrence, prevalence, virulence) relevant in determining the harm each disease could cause on coral reefs. (WS, white syndrome; cyano, cyanobacterial infection; MFPS, multi-focal pink spot; GA, growth anomaly; ciliate, ciliate infection; bl patch, bleached patch; DS, dark spot.)

Table 3. Comparisons of average benthic cover of inshore versus offshore sites surveyed in 2010 in New Caledonia

Two 25-m belt transects were surveyed at each site; CCA, crustose coralline algae

Site	Percentage coral cover	Colonies per square metre	Percentage CCA cover	Number of coral genera
Offshore				
Ouarai	46.1	12.5	19.6	24
Koné	46.1	13.1	31.4	28
Gazelle	46.1	21.0	38.2	21
Balade	27.5	13.9	13.7	27
Gué	48.0	13.6	1.0	29
Mbere	8.8	14.3	2.9	27
Average	37.1	14.7	17.8	26
Inshore				
Chenal de Teremba	17.6	2.1	8.8	8
Kreliat	11.8	5.1	2.9	14
Bouerabate	7.8	5.2	0.0	14
Neongaon	47.1	7.5	2.0	17
Ilôt Casy	83.3	10.0	0.0	19
Baie des Citrons	33.3	10.3	0.0	23
Average	33.5	6.7	2.3	15.8

range extension for other diseases. For example, this is the first report of growth anomalies on *Platygyra* and *Hydnophora* (host-range extension). We also discovered the first cases of trematode infection in *Porites* outside of Hawaii (biogeographic range

extension). In Hawaii, *Porites* trematodiasis is a disease caused by a parasitic digenetic trematode that presents as swollen, pink spots on poritid corals (Aeby 1998, 2003). Not all cases of multi-focal pink spots on *Porites* are due to trematode infections (Benzoni et al. 2010), and so, we initially described lesions as *Porites* multi-focal pink spot. Subsequently, using histology, Work et al. (2014) found that some, but not all, cases of *Porites* multi-focal pink spot from our surveys contained trematodes. Interestingly, compared with *Porites* trematodiasis in Hawaii, lesions on *Porites* in New Caledonia had reduced tissue swelling and frequently had a small bit of sediment adhering to the centre of the pink spot, which seldom occurs in Hawaii (Fig. S1, available as Supplementary Material for this paper). Work et al. (2014) also found that the trematodes in *Porites* in New Caledonia were not encysted, as were those found in Hawaiian corals (Cheng and Wong 1974; Aeby 1998); therefore, it is unknown whether the species of trematodes or disease ecology are similar among regions.

Another disease that has been found in few other places is dark spot, which we found in six different coral genera. In other regions, this lesion is associated with overgrowth of coral tissue by euendolithic fungi and was termed endolithic hypermycosis (Work et al. 2008b). Work et al. (2014) found that the dark-spot samples collected in New Caledonia also had a consistent diagnosis of euendolithic fungal invasion across the coral genera we sampled (endolithic hypermycosis). Our report has increased both the number of regions where this disease been found to occur as well as the number of genera affected. Prior studies

Table 4. Overall coral community composition of six inshore and six offshore sites surveyed in New Caledonia in 2010

Data reflect the proportion (%) of the coral community represented by each coral genus

Coral genus	Inshore						Offshore					
	Chenal de Teremba	Kreliat	Bouerabate	Neongaon	Îlot Casy	Baie des Citrons	Ouarai	Koné	Gazelle	Balade	Gué	Mbere
<i>Acanthastrea</i>	0	0.78	0	0	0.20	0	0	0.61	0.10	0.43	0.59	0.98
<i>Acropora</i>	23.58	37.35	59.77	32.36	57.60	32.30	41.79	31.35	57.63	48.13	15.04	25.00
<i>Anacropora</i>	0	0	0	0	4.00	0	0	0	0	0	0	0
<i>Archrelia</i>	0	0	0	0	0.20	0	0	0	0	0	0	0
<i>Astreopora</i>	0	0.39	0	0.27	3.60	0.19	0	0.61	0	0.29	0.29	0.70
<i>Barabattoia</i>	0	0.39	0.38	0.27	0	0	0	0.61	0	0.29	0	0
<i>Coscinaraea</i>	0	1.17	0	0.53	0	0.19	0.32	0.30	0.57	0.14	1.18	0.28
<i>Cyphastrea</i>	0.94	1.17	0	0	3.40	0.58	0.64	0.46	0.57	0.43	7.37	0.98
<i>Diploastrea</i>	0	0	0	0	0	0	0	0	0	0.58	0	0
<i>Echinophyllia</i>	0	0	0	0	1.80	0.78	0	0.30	0.38	1.44	0.59	0.70
<i>Echinopora</i>	0	0	0.38	0.27	0.40	3.11	0.80	1.67	1.05	0.72	0	0.14
<i>Favia–Favites</i>	2.83	1.95	3.83	6.10	1.60	5.45	5.58	8.52	4.29	6.20	10.47	7.68
Fungiidae	0	0	1.92	0.80	4.20	0.58	0	2.44	0.10	2.02	5.01	0.70
<i>Galaxea</i>	1.89	0.39	2.30	3.18	3.00	0.97	5.26	3.35	1.43	1.59	2.21	5.87
<i>Gardinoseris</i>	0	0	0	0	0	0	0.16	0.30	0.10	0	0	0
<i>Goniastrea</i>	0	0	7.28	0	0	0.58	0.16	0.46	0.67	0.86	0.74	0.14
<i>Goniopora</i>	0	0	0	0	0.20	0	0.64	0	0	0	0.59	0.56
<i>Hydnophora</i>	0	0	0.77	0	0	0.19	2.07	3.81	0.48	1.15	0	2.37
<i>Leptastrea</i>	0	0.78	0	0	0	0.19	0	0.30	0	0.29	1.92	0.84
<i>Leptoria</i>	0.94	0.39	2.30	1.06	0	0.39	3.51	4.26	0.57	2.59	2.80	2.37
<i>Lobophyllia–Symphyllia</i>	0	0	0.77	0	0.80	0.97	2.39	1.67	0	1.44	3.24	0.56
<i>Merulina</i>	0	0	0	0	0	0.39	0	3.50	0.38	0.43	1.03	0.28
<i>Millepora</i>	0	0	0	0.27	0	0.97	1.91	1.37	0.67	0.86	0.15	2.65
<i>Montastrea</i>	0	0	0.77	0.80	0	0.78	1.44	1.67	0.67	0.29	2.51	5.31
<i>Montipora</i>	2.83	12.84	13.03	24.14	8.00	14.98	15.63	8.83	5.34	7.20	14.31	11.59
<i>Mycidium</i>	0	0	0	0	0	0	0	0	0	0	0.29	0
<i>Oxypora</i>	0	0	0	0	0	0	0.32	0	0	0.14	1.18	0
<i>Pachyseris</i>	0	0	0	0	2.40	0	0	0	0	0.14	1.18	0
<i>Pavona</i>	0	0	0	0.53	3.80	2.92	1.59	5.63	3.05	1.59	5.01	2.09
<i>Pectinia</i>	0	0	0	0.27	0	0.19	0	0	0	0	0	0
<i>Pocillopora</i>	5.66	20.62	2.68	3.18	0.20	6.23	11.00	7.76	14.12	10.37	5.60	6.28
<i>Porites</i>	61.32	21.01	3.07	15.65	3.20	24.12	0.96	1.07	2.86	4.32	4.72	16.90
<i>Psammocora</i>	0	0.78	0.38	2.65	0.80	1.95	0.48	0.30	0.67	0.58	1.47	0.56
<i>Seriatopora</i>	0	0	0	0	0.20	0	0.16	0.46	0	0	2.21	0
<i>Stylocoeniella</i>	0	0	0	0	0	0	0	0	0	0	0.59	0
<i>Stylophora</i>	0	0	0	6.90	0	0.97	2.87	7.76	4.29	4.47	5.31	3.35
<i>Scolymia</i>	0	0	0	0	0.40	0	0.16	0	0	0	2.21	0.28
<i>Scapophyllia</i>	0	0	0	0	0	0	0	0	0	0	0	0.28
<i>Turbinaria</i>	0	0	0.38	0.80	0	0	0.16	0.61	0	1.01	0.15	0.56

have found this disease affecting only *Pavona*, *Psammocora* and *Montipora* in American Samoa and *Pavona* in Hawaii (Work *et al.* 2008b). The present report has extended affected coral genera to *Platygyra*, *Leptoria* and *Goniastrea*.

The present study has also extended the biogeographic range of the following three diseases of crustose coralline algae: coralline lethal orange disease (CLOD), CCA white syndrome and CCA concentric white discoloration (target phenomenon). Little is known about CCA white syndrome or concentric discoloration, but CLOD is well studied and is widespread across the Indo-Pacific, from the coral triangle (Littler and Littler 1995; Vargas-Ángel 2010) out to the remote north-western Hawaiian Islands (Aeby 2007; Vargas-Ángel 2010). CLOD was not common on the reefs of New Caledonia, with

4 of 13 sites in 2010 and 2 of 12 sites in 2013 having infections at low abundances (0.01–0.33 lesions m⁻² CCA). Low CLOD levels have also been reported from other regions, such as the north-western Hawaiian Islands where only a few cases of CLOD have been reported despite extensive surveys (Aeby 2007; Vargas-Ángel 2010). In contrast, Vargas-Ángel (2010) found CLOD to be abundant in American Samoa (0.5–3.2 lesions m⁻² CCA), the Pacific Remote Island Areas (0.1–1.4 lesions m⁻² CCA) and the southern Mariana Islands (7.1–19.0 lesions m⁻² CCA). CCA are the second major reef framebuilders, contributing to reef accretion, cementation, sedimentation, primary production and coral settlement (Aeby 1998; Harrington *et al.* 2004). Our documentation of CCA diseases on the reefs of New Caledonia highlight the global

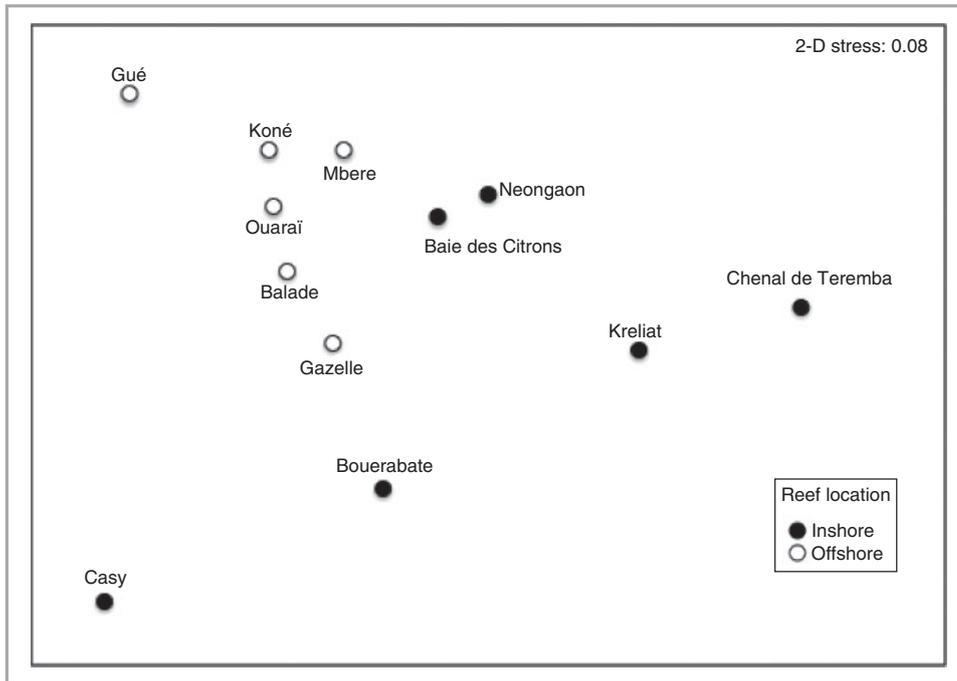


Fig. 4. A non-metric multi-dimensional scaling (nMDS) plot illustrating the differences in coral communities between inshore and offshore sites surveyed for coral disease.

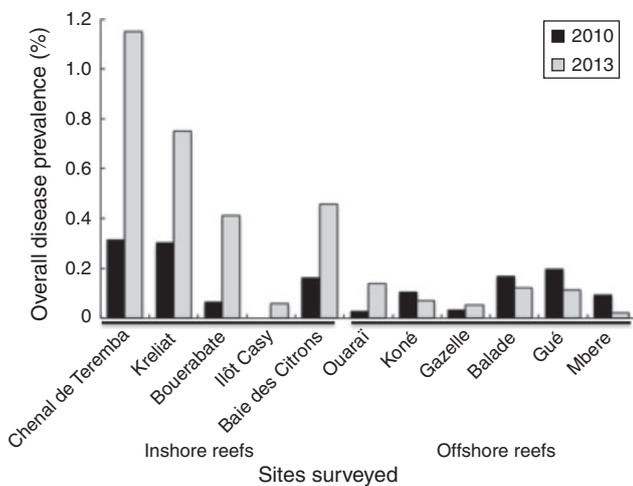


Fig. 5. Differences in coral-disease prevalence from surveys in 2010 versus 2013. Six offshore and five inshore sites were surveyed in both years. Arrow indicates the split between inshore and offshore reefs.

distribution of CCA diseases and the need for a better understanding of disease dynamics, pathogen identification and implications in maintenance of coral reefs in the context of climate change.

Of 44 coral genera surveyed, we found signs of disease only in 15 genera. Numerous other studies have reported disease prevalence in specific coral genera as different from what was expected on the basis of coral abundance in the field (Willis *et al.* 2004; Aeby *et al.* 2009; Myers and Raymundo

2009). Acroporids have been reported as especially vulnerable to disease in the Indo-Pacific (Willis *et al.* 2004; Aeby *et al.* 2009; Myers and Raymundo 2009) as well as in the Caribbean (Sutherland *et al.* 2004). In contrast, in New Caledonia, we found lower disease levels in *Acropora* than what was expected on the basis of their abundance. Although we found some isolated cases of tissue-loss diseases in *Acropora* (*Acropora* white syndrome), we found no localised outbreaks, as has frequently been reported elsewhere (Willis *et al.* 2004; Heron *et al.* 2010; Aeby *et al.* 2011a). It has been suggested that coral reefs in species-rich regions should be more resistant or resilient to coral-disease outbreaks than are reefs in species-poor regions, because low host richness can, through relaxed interspecific competition, increase relative abundances of one or more susceptible hosts (Keesing *et al.* 2010). Aeby *et al.* (2011a) found that an outbreak of *Acropora* white syndrome in the species-poor region of the north-western Hawaiian Islands was more severe and prevalent than was an outbreak in the more species-rich region of American Samoa. New Caledonia is known to have ~320 species of hard corals, including 21 species of *Acropora* (Laboute and Richer de Forges 2004), and so could be more resistant to developing disease outbreaks. Alternatively, because disease outbreaks are usually localised and ephemeral, our 2-year study at a limited number of sites may not have been adequate to locate them.

Overall, coral-disease prevalence was low, making New Caledonia well placed to proactively protect their reefs from local stressors (such as e.g. overfishing, shoreline development, land-based sources of pollution) as well as from problems anticipated from global climate change, both of which can lead to increased disease levels. There was a significant increase in

disease on inshore reefs between surveys in 2010 and 2013; however, it is unclear whether this increase could be ascribed to background temporal or spatial variation, because surveys were not on permanently marked transects. The consistent increase in disease between 2010 and 2013 across numerous sites was noticeable, and it would be useful if disease surveys were incorporated as part of current or future monitoring efforts to get a more concrete assessment of longer-term trends. This also points out the critical need to document baseline disease levels on reefs before changes occur from chronic anthropogenic stress and climate change, with the latter imposing its own set of impacts on coral reefs (Hoegh-Guldberg *et al.* 2007; Pandolfi *et al.* 2011). For example, Willis *et al.* (2004) found that the number of cases of tissue-loss diseases (white syndrome) at monitoring sites on the Great Barrier Reef increased 20-fold between 1998 and 2002–2003. Hence, the definition of ‘normal’ disease levels on those reefs would be very different had they conducted their very first disease survey in 2002, and this highlights the problem of ‘shifting baselines’. Numerous regions throughout the Indo-Pacific still lack baseline disease studies and this is problematic.

The potential ecological impact of disease on coral reefs would depend on the spatial distribution, prevalence and the degree of virulence of specific diseases. Chronic diseases, such as growth anomalies or *Porites* trematodiasis, can be widespread and prevalent, but result only in reduced growth or reproduction (Aeby 1993; Stimson 2011), whereas other diseases, such as black-band disease, result in slow but recurrent tissue loss (Kuta and Richardson 1996; Sato *et al.* 2009). Acute and subacute tissue-loss diseases are the most virulent, resulting in significant colony mortality, reported as white syndromes across the Indo-Pacific (Aeby 2005; Dalton and Smith 2006; Sussman *et al.* 2008) and white plague or white band from the Caribbean (Richardson 1998; Aronson and Precht 2001). The reefs of the Caribbean have already lost significant coral cover (Gardner *et al.* 2003) in large part from coral disease (Sutherland *et al.* 2004), and a similar pattern of coral loss is arising in the Indo-Pacific (Bruno and Selig 2007; De’ath *et al.* 2012). Knowing which coral diseases may be most damaging to reefs would be an important first step in focussing limited resources and directing future studies. We used integrative scoring of three disease metrics (frequency of occurrence, prevalence, virulence) and a relative ranking scheme to compare the potential ecological impact of the diseases encountered in New Caledonia. On the basis of this metric, the top five diseases of concern were white syndrome in *Acropora*, *Porites*, *Astreopora* and *Turbinaria* and cyanobacterial infection in *Turbinaria*. Not surprising, all five are tissue-loss diseases, resulting in partial to total colony mortality. Use of a ranking scheme to communicate relative threat from different diseases facilitates reporting threats to resource managers and allows cross-regional comparisons.

We found that inshore reefs had lower densities of coral colonies, lower species diversity and a reduced CCA cover. Fenner (2011) also found lower coral-species richness on inshore reefs than on outer barrier reefs in New Caledonia. Inshore reefs also showed a shift in the structure of coral communities, with sediment-tolerant corals such as *Porites* found to be more abundant. Evidence of sediment stress was common in inshore reefs, along with reduced water quality

(poor visibility and layers of sediments accumulated on the top of coral colonies), but was never observed on the offshore reefs. The serious threat that sedimentation poses to New Caledonia’s inshore reefs has been mentioned elsewhere (Lasne 2007; Fenner and Muir 2009; McKenna 2011), with open-surface nickel mining being identified as one of the major sources of sediment (Fenner and Muir 2009). Many studies have shown that land-based pollution, such as sedimentation, nutrient enrichment and turbidity, can severely degrade coral reefs at the local scale (reviewed by Fabricius 2005). Improvement in water quality may be an effective way to improve overall coral-reef integrity and should be considered a priority for reef management.

The degree of environmental stress to inshore reefs would vary depending on, for example, their proximity to river mouths, adjacent shoreline development, density of human populations in adjacent watersheds. Accordingly, we found that not all inshore reefs appeared to be affected by terrestrial runoff and this was reflected in the higher variability in the structure of coral communities found among the inshore reefs than among the offshore reefs. However, inshore reefs, by spatial proximity, would also be more prone to other types of anthropogenic damage. For example, one of our survey sites, Casy, showed few signs of sediment stress and was dominated by branching acroporids. Disease levels were also low; however, in 2013, we found a large area of the reef reduced to a pile of rubble, from what appeared to be mechanical breakage (probably anchor damage). Coral cover declined at Casy from 83.3% in 2010 to 63.4% in 2013.

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Supplementary material

Assessing threats from coral and crustose coralline algae disease on the reefs of New Caledonia

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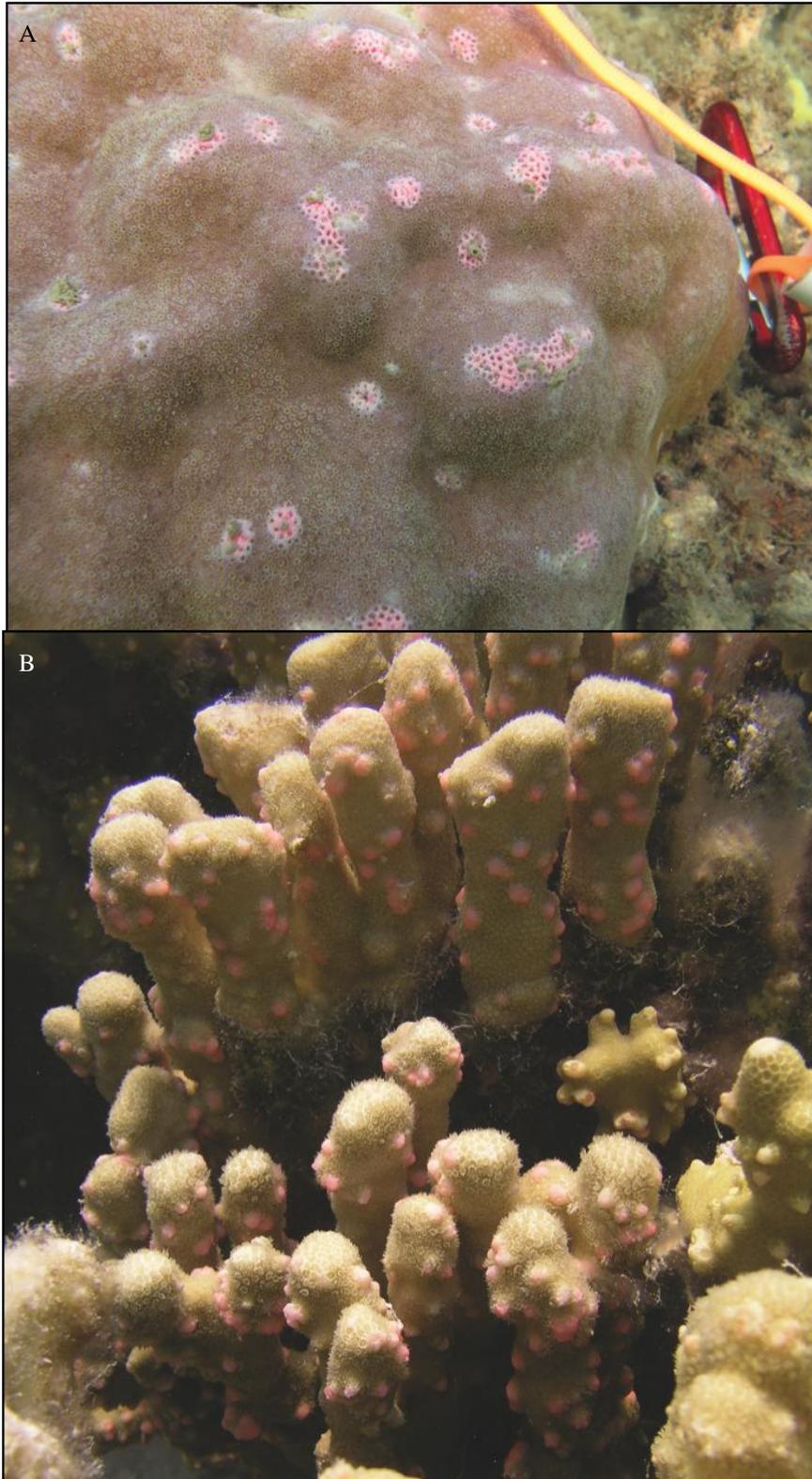


Fig. S1. (a) Photograph of multi-focal pink spot on *Porites* in New Caledonia. Note the reduced swelling and sediment adhering to the pink spots. (b) Photograph of *Porites* trematodiasis in Hawaii. Note the protruberant swelling of the pink spots.