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# Identification and prevalence of coral diseases on three Western Indian Ocean coral reefs

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ABSTRACT: Coral diseases have caused a substantial decline in the biodiversity and abundance of reef-building corals. To date, more than 30 distinct diseases of scleractinian corals have been reported, which cause progressive tissue loss and/or affect coral growth, reproductive capacity, recruitment, species diversity and the abundance of reef-associated organisms. While coral disease research has increased over the last 4 decades, very little is known about coral diseases in the Western Indian Ocean. Surveys conducted at multiple sites in Reunion, South Africa and Mayotte between August 2010 and June 2012 revealed the presence of 6 main coral diseases: black band disease (BBD), white syndrome (WS), pink line syndrome (PLS), growth anomalies (GA), skeleton eroding band (SEB) and *Porites* white patch syndrome (PWPS). Overall, disease prevalence was higher in Reunion (7.5 ± 2.2%; mean ± SE) compared to South Africa (3.9 ± 0.8%) and Mayotte (2.7 ± 0.3%). Across locations, *Acropora* and *Porites* were the genera most susceptible to disease. Spatial variability was detected in both Reunion and South Africa, with BBD and WS more prevalent on shallow than deep reefs. There was also evidence of seasonality in 2 diseases: the prevalence of BBD and WS was higher in summer than winter. This was the first study to investigate the ecology of coral diseases, providing both qualitative and quantitative data, on Western Indian Ocean reefs, and surveys should be expanded to confirm these patterns.

KEY WORDS: Coral diseases · Western Indian Ocean · Scleractinian corals · Seasonality · Spatial variability



## INTRODUCTION

The emergence and spread of infectious diseases has caused substantial declines in the biodiversity and abundance of reef-building corals during the last 4 decades (Garzón-Ferreira et al. 2001, Weil et al. 2006). To date, more than 30 distinct diseases, affecting at least 150 scleractinian corals, have been reported worldwide (Sutherland et al. 2004, Weil et al. 2006). Several of them are known to generate tissue loss and subsequently affect growth rate, reproductive capacity and the competitive ability of corals. For instance, several studies have shown that black band disease (BBD) can generate tissue loss of up to 2 cm $d^{-1}$ by producing high concentrations of sulphide that kill the coral tissue (Boyett et al. 2007, Haapkylä et al. 2009, Sato et al. 2009). Petes et al. (2003) showed that the sea fan *Gorgonia ventalina*, when infected by the fungal pathogen *Aspergillus sydowii*, is reproductively compromised. Similarly, yellow band disease (YBD) compromises the reproductive output of the Caribbean reef-building coral *Montastraea faveolata* (Weil et al. 2009).

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While coral bleaching has received intense attention since the 1998 El Niño Southern Oscillation (ENSO) event (Bigot & Quod 2000, Cole et al. 2000, Goreau et al. 2000, McClanahan 2000, Spencer et al. 2000, Celliers & Schleyer 2002, Chabanet 2002, McClanahan et al. 2004a, 2007, Obura 2005), no in-depth studies quantifying the current status of coral diseases have been performed on Western Indian Ocean reefs (McClanahan et al. 2004a). Bacteria-induced bleaching (Ben-Haim & Rosenberg 2002), BBD, white band disease (WBD) and YBD have been observed in isolated outbreaks in Zanzibar (McClanahan et al. 2004b). In Kenya and Tanzania, a white syndrome associated with infection by fungal hyphae has been reported on *Montipora* and *Astreopora* (McClanahan et al. 2004b). In South Africa, BBD and a yellowing disease were noted during a coral reef monitoring programme (Jordan & Samways 2001), and an increased incidence of BBD and cyanobacterial films associated with coral bleaching was recorded by Celliers & Schleyer (2002). Thus, the goals of our study were to (1) identify the main coral diseases by systematically describing gross lesions in scleractinian corals and (2) investigate their prevalence and variability at temporal and spatial scales, focusing on 3 target coral reefs in Reunion, South Africa and Mayotte (see Fig. 1 in Séré et al. 2013).

## MATERIALS AND METHODS

### Study areas

Surveys were conducted in 3 areas, viz. Reunion, Sodwana Bay in South Africa and Mayotte. At Reunion, corals form fringing reefs at Reunion that are 12 km$^2$ in area along 25 km of the coastline, mainly on the dry west coast. Three geomorphological zones are evident (Montaggioni & Faure 1980): (1) an outer reef slope (5–30 m) exposed to high turbulence and characterised by a basaltic substratum in alternating spurs and grooves, mostly covered by massive and encrusting corals, (2) a reef flat (0.5–2 m), generally composed of branching corals, and (3) an inner back-reef covered with sand and rubble (0.5–1 m). Reefs at Sodwana Bay (1.9 km$^2$) are not typically accretive (Schleyer 2000); the corals grow on late-Pleistocene beach rock, originating from submerged, fossilised coastal sand dunes (Ramsay 1996). In topography, the reefs consist of shallow pinnacles (8–10 m), extensive deep subtidal reef flats (14–18 m) and a sloping fore-reef edge (24–27 m; Celliers & Schleyer 2008). Mayotte reefs are characterised by a large (15 km wide), deep (30–35 m) lagoon surrounded by a long barrier reef (150 km), which is 1.5 km wide in some areas and interrupted by 12 deep channels. Fringing reefs are also present along 210 km of the coastline of the island. A discontinuous, inner secondary barrier reef system (12 km long) is located on the south-west coast.

### Disease surveys

In total, we conducted 76 coral disease surveys at 22 sites within the 3 locations between September 2010 and March 2012, covering an area of 7920 m$^2$ of reef. Surveys in South Africa were conducted on Two-mile Reef (TMR) in the central Maputaland reef complex at Sodwana Bay in northern KwaZulu-Natal (27.31° S, 32.41° E; see Table S1 in the Supplement, available at www.int-res.com/articles/suppl/d114p249_supp.pdf, and Fig. 1 in Séré et al. 2013). Surveys were conducted at 7 sites along a north–south gradient on TMR at 2 depth intervals: 8–10 m (shallow inshore region) and 12–16 m (deeper offshore region). On Mayotte, surveys were conducted at 8 latitudinal sites on the barrier and fringing reef (12.82° S, 45.17° E; see Table S1, and Fig. 1 in Séré et al. 2013). In Reunion, surveys were undertaken at 4 latitudinal sites (21.12° S, 55.25° E; see Table S1, and Fig. 1 in Séré et al. 2013) on the outer reef slope and reef flat. Protocols were adapted to the different geomorphological zones. Five 10 × 2 m (1 m on each side of the transect line) transects were laid parallel to depth contours at each site at Sodwana Bay and Mayotte. A gap of 20 m was left between transects to ensure independence in the data for statistical analysis. At Reunion, the outer reef slope is characterised by a succession of spurs and grooves that represent different habitats. Spurs are covered mainly by hard corals, whereas grooves are often filled with sand and coral rubble. In order to stay within the coral community, 5 belt transects (10 m × 2 m) were laid along different spurs at the same depth. Surveys on the inner reef flat were conducted along 3 belt transects (20 m × 2 m) positioned parallel to the coastline, again to avoid crossing different coral communities. At each location, transects were haphazardly laid, and starting GPS coordinates were recorded to locate the survey sites.

Scleractinian corals displaying evidence of disease were identified to the genus level and counted within each transect. Additionally, all coral genera exhibiting comparable gross lesions were considered to have the same disease (e.g. BBD, white syndrome [WS], etc.). Bleaching and compromised tissue (CT)

such as tissue discolouration and unusual tissue loss were considered an impairment of normal function and were also recorded. The prevalence of diseases was estimated as (number of diseased colonies)/(total number of coral colonies >2 cm) × 100. Coral colonies were identified to genus level and counted within each transect in 1 × 1 m photoquadrats which covered the transect area (20 images transect$^{-1}$). Each image (taken at a fixed height) was analysed to determine the number of coral colonies in the respective taxa. Surveys in both Reunion and South Africa were conducted over 2 consecutive summers (December–February) and winters (June–October) to gain a measure of seasonality in the prevalence of the diseases (Table 1). Average sea surface temperatures (SSTs) ranged from 23.5°C in winter to 31.6°C in summer in Reunion and from 22°C in winter to 27°C in summer at the locations in South Africa (see Fig. 1 in Séré et al. 2013). In Mayotte, coral diseases could only be monitored during the summer (March) and winter (August) of 2012. SSTs during cooler months ranged from 23 to 24°C, and during the rainy summer reached 27 to 30°C.

Table 1. Mean coral disease prevalence (±SD) in 3 Western Indian Ocean regions during successive winters and summers in 2010 to 2012

| Location | Date | Season | Prevalence (%) |
|---|---|---|---|
| Reunion | September 2010 | Winter 1 | 6.8 (6.7) |
| | December 2010 | Summer 1 | 7.2 (6.4) |
| | October 2011 | Winter 2 | 8.3 (7.1) |
| | January 2012 | Summer 2 | 7.8 (6.2) |
| | **Total** | | **7.5 (0.6)** |
| South Africa | February 2011 | Summer 1 | 3.9 (3.6) |
| | July 2011 | Winter 1 | 1.9 (1.2) |
| | February 2012 | Summer 2 | 4.1 (2.0) |
| | June 2012 | Winter 2 | 5.7 (3.2) |
| | **Total** | | **3.9 (1.1)** |
| Mayotte | August 2011 | Winter | 2.3 (3.4) |
| | March 2012 | Summer | 3.1 (2.3) |
| | **Total** | | **2.7 (0.6)** |

### Disease identification

Gross lesions observed during the surveys were photographed and identified using the Underwater Cards for Assessing Coral Health on Caribbean and Indo-Pacific Reefs (Beeden et al. 2008) and illustrations/descriptions available in the literature. Similar gross lesions can be manifested by multiple microscopic pathologies and/or different causal agents (Work & Rameyer 2005). Therefore, to avoid subjective interpretations and to verify field observations, each coral disease was described according to the systematic nomenclature developed by Work & Aeby (2006).

### Statistical analysis

Disease prevalence, calculated per transect, was averaged for each site at each location. Overall prevalence was expressed as a proportion of the total infected coral colonies surveyed, all surveys being combined at each location. Coral disease prevalence was calculated according to disease, e.g. prevalence of *Porites* white patch syndrome (PWPS) = (number of coral colonies with PWPS)/(total number of coral colonies surveyed) × 100 and coral genus, e.g. prevalence of *Porites* with BBD = (number of *Porites* with BBD)/(total number of *Porites* surveyed) × 100. Coral genus susceptibility was assessed using a chi-squared test to compare observed with expected disease prevalence in each genus according to its abundance in the field (Aeby et al. 2010, 2011b). Variations in the prevalence of coral disease over the 2 survey years were tested in the consecutive summers and winters and across reef zones (shallow vs. deep). Due to variation in the survey protocols, dates and reef morphotypes, statistical analyses were undertaken within locations. Data were tested prior to analysis for homoscedasticity (Levene's test) and normality of variance (Kolmogorov-Smirnov and Lilliefors tests) and were then log-transformed [$\log_{10}(x)$] for analysis of variance (ANOVA). Analyses of seasonality and spatial variations were performed for the most prevalent diseases and most susceptible coral genera using 2-way factorial ANOVA (STATISTICA 8). Finally, post hoc Fisher LSD tests were performed for multiple group comparisons.

## RESULTS

### Description of disease gross lesions *in situ*

The photographs and samples taken from the reefs revealed the presence of 6 main coral diseases manifesting discoloration, tissue loss and growth anomalies (Fig. 1). They included WS, BBD, pink line syndrome (PLS), skeletal eroding band (SEB), growth anomaly (GA), PWPS (Séré et al. 2012, 2013) and CT. These are characterised in Table S2 in the Supplement.

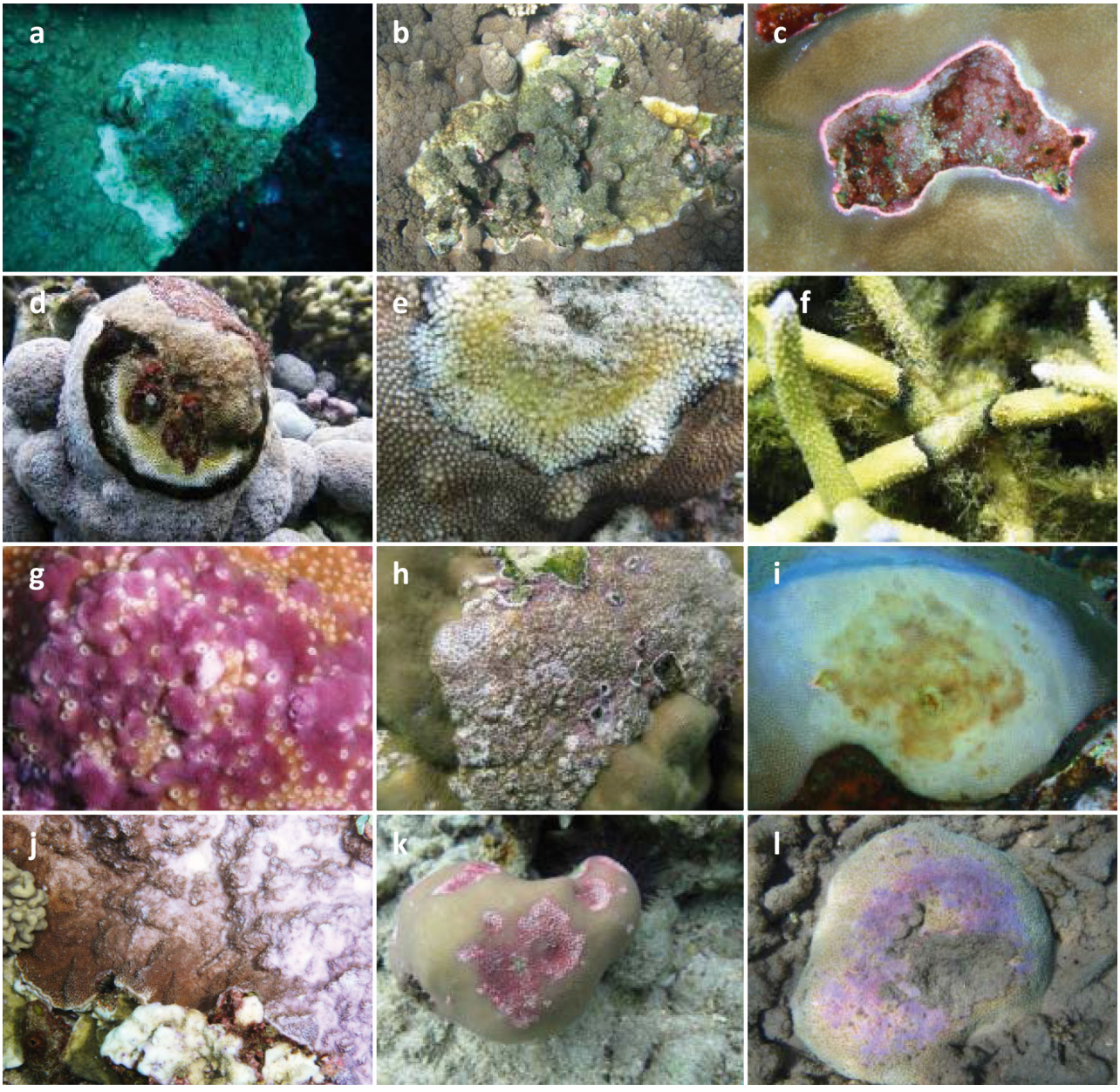


Fig. 1. Principal coral diseases observed on 3 Western Indian Ocean reefs. White syndrome (WS) on (a) *Montipora* sp. and (b) *Acropora* sp. Pink line syndrome (PLS) on (c) *Porites lobata*. Active black band disease (BBD) on (d) *Goniopora djiboutinensis* and (e) *Hydnophora* sp. Skeleton eroding band (SEB) on (f) *A. muricata*. Growth anomalies (GA) on (g) *Astreopora* sp. and (h) *P. lobata*. *Porites* white patch syndrome (PWPS) on (i) *P. lutea*. Bleaching on (j) *Montipora* sp. Compromised tissue (CT) on (k) *P. lobata* and (l) *Astreopora* sp.

### Coral disease prevalence and susceptibility

The prevalence of all the coral diseases encountered varied among the 3 locations; the overall prevalence recorded in Mayotte was 2.7 ± 0.6% (mean ± SD) in 2011 and 2012, while in Reunion 7.5 ± 0.6% of all coral colonies were affected by diseases between 2010 and 2012 (Table 1). In South Africa, the proportion of infected coral colonies was low but quite variable between the 2 survey periods, with the average disease prevalence higher in 2011–2012 (4.9 ± 1.3%) than in 2010–2011 (2.9 ± 1.4%). The most prevalent coral disease recorded in Reunion was PWPS (2.3 ± 2.0%). This was closely followed by PLS (2.0 ± 3.9%), WS (1.5 ± 3.9%) and BBD (1.3 ± 1.8%), but the most common diseases on both South African and Mayotte

reefs were accompanied by a much lower prevalence of any other diseases (Table 2).

Disease susceptibility varied between coral genera, but no clear relationship was found between disease prevalence and the abundance of affected coral genera at each location (Reunion: $\chi^2 = 146.01$, df = 5, $p < 0.001$; South Africa: $\chi^2 = 248.60$, df = 6, $p < 0.001$; Mayotte: $\chi^2 = 115.68$, df = 6, $p < 0.001$). *Acropora*, *Goniopora*, *Hydnophora* and *Porites* were the most susceptible coral genera to disease on both the reef slope and reef flat in Reunion (Fig. 2, Table 3). WS was the most common disease affecting branching colonies of *Acropora*, but only in the shallowest zone of the reef. Colonies of *Goniopora*, *Hydnophora* and *Porites* were most susceptible to BBD, especially those on the reef flat. Massive colonies of *Porites* appeared to be the most vulnerable to disease, exhibiting multiple infections, including PLS, PWPS, BBD, WS, GA and CT. In South Africa, 11 coral genera were observed with signs of disease (Fig. 3, Table 3). The most susceptible coral genera were *Astreopora*, *Hydnophora*, *Pocillopora* and *Porites*. GA, WS and CT were more prevalent on encrusting and massive *Astreopora* spp. in summer and winter during both survey years. Both BBD and WS were the most prevalent diseases on *Hydnophora* on shallow reefs. Colonies of *Pocillopora* exhibited high susceptibility to WS, whereas the massive corals *Porites lutea* and *P. lobata* were more vulnerable to PWPS and PLS. Of the 8 genera observed with diseases on both the barrier and fringing reefs of Mayotte (Fig. 4, Table 3), *Acropora*, *Astreopora* and *Porites* seemed to be the most susceptible to disease. Colonies of *Astreopora* were highly susceptible to GA and WS. *Acropora* appeared to be particularly vulnerable to WS, whereas *Porites* showed a particular susceptibility to GA, BBD, PLS and CT.

Table 2. Overall prevalence (= number of diseased coral colonies divided by the total number of colonies identified to the genus level within each transect; ± SD) of the main coral diseases in coral genera on reefs in Reunion (n = 23 562 coral colonies), South Africa (n = 17 140 coral colonies) and Mayotte (n = 19 426 coral colonies) between 2010 and 2012

| Disease | Reunion | South Africa | Mayotte |
|---|---|---|---|
| Bleaching (Ble) | – | 0.4 (1.3) | – |
| White syndrome (WS) | 1.5 (3.9) | 2.1 (2.5) | 1.0 (1.4) |
| Pink line syndrome (PLS) | 2.0 (3.9) | 0.5 (0.9) | 0.1 (0.4) |
| *Porites* white patch syndrome (PWPS) | 2.3 (2.0) | 0.2 (0.5) | 1.0 (0.4) |
| Black band disease (BBD) | 1.3 (1.8) | 0.4 (0.6) | 0.1 (0.9) |
| Compromised tissue (CT) | 0.1 (0.4) | 0.8 (0.3) | 0.5 (1.3) |
| Growth anomaly (GA) | 0.1 (0.7) | 0.1 (0.5) | 0.01 (0.15) |
| Skeletal eroding band (SEB) | 0.2 (0.7) | – | – |
| ∑ disease | **7.5 (1.2)** | **3.9 (0.8)** | **2.7 (0.4)** |

### Seasonal and spatial variations in coral disease prevalence

Among coral disease states recorded in Reunion, the prevalence of only BBD varied between the reef zones and seasons (Table 4). Its prevalence was significantly higher on the reef flat, with the percentage of infected colonies being significantly higher in summer than winter (Fisher LSD, $p < 0.001$; Table 4). This spatial and seasonal pattern was observed particularly on *Porites* colonies, which exhibited a higher mean BBD prevalence in summer (Fisher LSD, $p < 0.001$), and at the shallowest sites (Fisher LSD, $p < 0.05$; Table 4, Fig. 2). PWPS and WS varied significantly between reef zones (Table 4), but no significant difference was found between seasons (Table 4). Among diseases recorded on South African reefs, WS was seasonal on *Acropora* and *Pocillopora*, with a higher percentage of infected colonies in summer than winter (Table 4, Fig. 3). A similar, seasonal trend was evident for PWPS but the difference between summer and winter was not significant (Table 4). A significantly higher prevalence of WS was recorded on *Pocillopora* (Table 4) in the shallow South African reef zones (Fisher LSD, $p < 0.001$). At Mayotte, WS varied significantly between seasons, especially on *Acropora* (Table 4), with a higher prevalence in winter than in summer (Fisher LSD, $p < 0.05$; Table 4, Fig. 4). However, no significant difference was recorded between the fringing and barrier reef (Table 4).

## DISCUSSION

This study provides baseline information on the incidence of coral disease with qualitative and quantitative data for 3 Western Indian Ocean localities, viz. Reunion, South Africa and Mayotte. Surveys revealed the presence of 6 main coral diseases: WS, BBD, SEB, PLS, GA and PWPS. Except for PWPS, all diseases recorded during this study have been previously reported within other regions across the Indian Ocean, including the Chagos Archipelago (Sheppard et al. 2012), Republic of Maldives (Onton et al. 2011), Southern India (Thinesh et al. 2009, 2011) and the Indo-Pacific region (Willis et al. 2004, Raymundo et al. 2005, Aeby et al. 2006). PWPS has to date been reported on only one species of massive

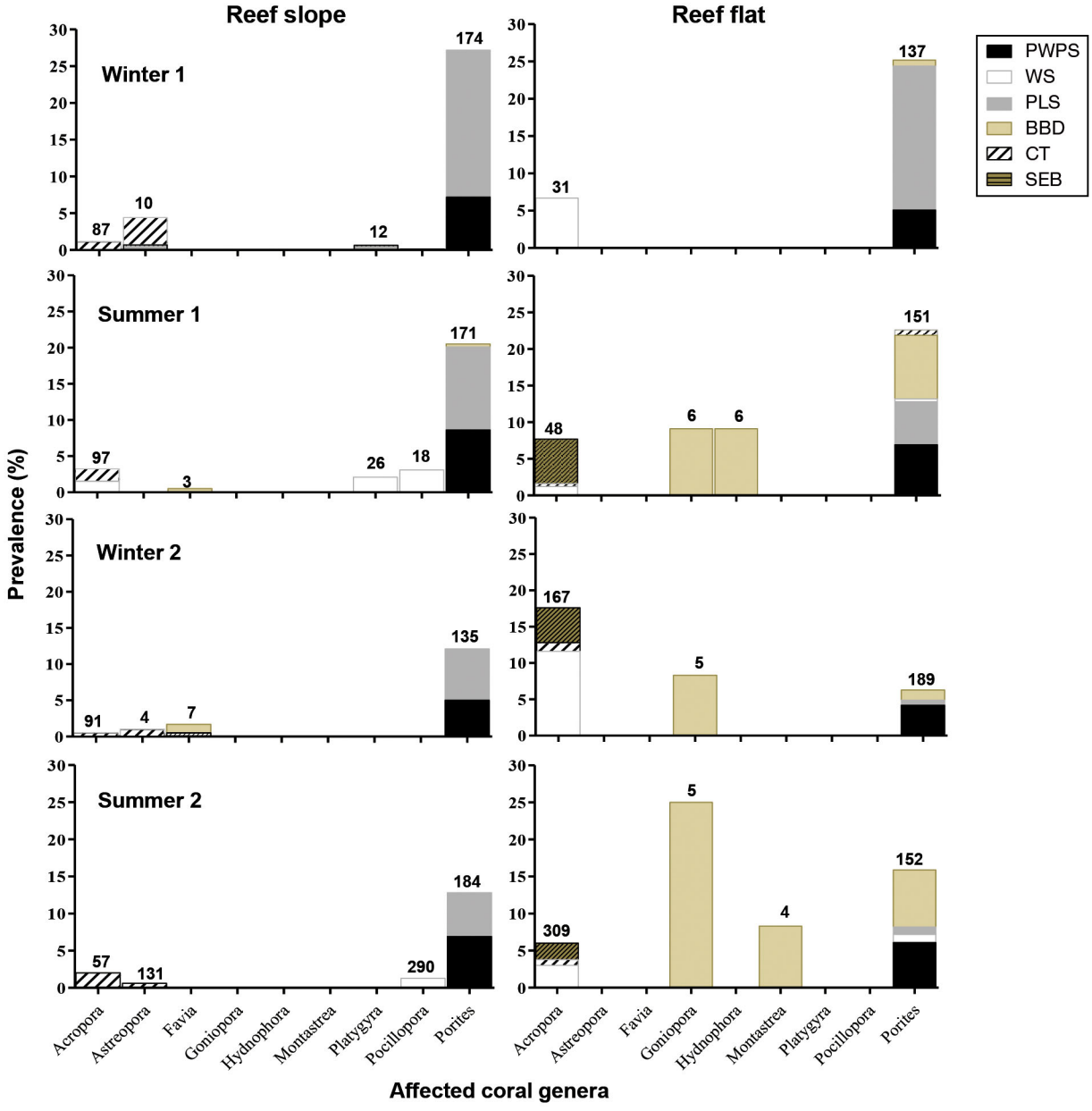


Fig. 2. Prevalence (%) of the main coral diseases in 9 scleractinian genera in Reunion: *Porites* white patch syndrome (PWPS), white syndrome (WS), pink line syndrome (PLS), black band disease (BBD) and skeleton eroding band (SEB). Prevalence is calculated relative to the total number of colonies in the respective taxa, per coral genus and per reef zone (reef flat and reef slope) for 2 consecutive summers and winters. Note that compromised tissue (CT) is included in the analysis (growth anomalies were not found). Numbers above bars represent the N of each affected coral genus

coral, *Porites lutea*, which in itself is among the most important reef-building corals throughout the Western Indian Ocean (Séré et al. 2012). While WS is generally characterised by acute to sub-acute, diffuse, irregular to distinct tissue loss exposing denuded skeleton (e.g. Aeby et al. 2010, Work & Aeby 2011, Work et al. 2012), PWPS is manifested by patches of circular to oblong tissue loss (5–30 cm diameter), the lesions being surrounded by a front of swollen and bleached tissue (1–20 cm width). Additionally, PWPS-infected colonies exhibit a clearly visible scar in the middle of the lesion, probably resulting from feeding by corallivorous organisms.

The overall disease prevalence in Reunion (7.5%) was higher than that found in either South Africa (3.9%) or Mayotte (2.7%). Disease levels on Reunion

Table 3. Relative abundance (% ± SD) of the main coral genera displaying disease signs and mean prevalence (% ± SD) of all diseases calculated relative to the total number of colonies in the respective genera at Reunion, South Africa and Mayotte

| Genus | Reunion | | | | South Africa | | | | Mayotte | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Reef slope | | Reef flat | | Shallow inshore region | | Deeper offshore region | | Fringing reef | | Barrier reef | |
| | Abundance | Prevalence | Abundance | Prevalence | Abundance | Prevalence | Abundance | Prevalence | Abundance | Prevalence | Abundance | Prevalence |
| *Acropora* | 12.0 (22.7) | 1.7 (1.2) | 20.4 (30.3) | 9.6 (5.4) | 23.8 (21.4) | 4.1 (4.2) | 23.0 (30.5) | 2.7 (2.9) | 41.4 (24.2) | 1.6 (1.3) | 56.3 (38.3) | 1.9 (1.8) |
| *Astreopora* | 10.0 (12.2) | 1.5 (2.0) | 0.9 (0.1) | – | 2.3 (4.5) | 5.8 (5.5) | 3.0 (4.1) | 10.9 (7.1) | 0.4 (0.5) | 9.0 (12.7) | 0.5 (0.7) | 10.8 (15.3) |
| *Favia* | 6.9 (8.0) | 0.5 (0.8) | 1.1 (0.8) | – | 4.4 (4.4) | – | 4.2 (5.3) | – | 5.5 (6.6) | 1.3 (1.8) | 3.4 (3.2) | |
| *Fungia* | 0.4 (1.0) | – | – | – | – | – | 2.7 (2.2) | – | 6.2 (10.7) | 0.9 (1.2) | 6.0 (8.3) | 1.5 (2.1) |
| *Galaxea* | 2.3 (2.9) | – | – | – | 12.7 (12.4) | – | 11.8 (9.1) | – | 6.2 (8.1) | 0.1 (0.1) | 4.6 (6.4) | 2.0 (1.8) |
| *Goniopora* | 1.1 (0.5) | – | 1.8 (2.6) | 10.6 (10.4) | 0.1 (0.6) | – | 1.6 (0.1) | – | 0.1 (0.3) | – | 0.2 (0.5) | |
| *Hydnophora* | 0.8 (0.6) | – | 1.2 (0.9) | 2.3 (4.6) | 1.8 (3.4) | 11.9 (10.8) | 2.0 (2.5) | 13.4 (15.4) | 0.7 (0.9) | – | 0.2 (0.5) | |
| *Montastrea* | – | – | 0.9 (0.1) | 2.1 (4.2) | – | – | – | – | 0.1 (0.2) | – | 0.2 (0.3) | |
| *Montipora* | 4.1 (4.9) | – | 1.3 (0.9) | – | 11.2 (7.9) | 0.5 (0.9) | 14.6 (13.9) | 2.6 (2.7) | 1.2 (2.3) | 0.8 (0.1) | 0.7 (1.0) | 1.0 (1.3) |
| *Pavona* | 1.0 (1.2) | – | 4.9 (5.5) | – | 0.1 (0.6) | – | – | – | 0.8 (1.8) | – | 0.9 (1.7) | |
| *Platygyra* | 2.5 (2.1) | 0.7 (1.0) | 6.7 (8.6) | – | 5.7 (6.6) | 2.0 (2.3) | 4.1 (4.5) | 0.2 (0.3) | 1.7 (2.0) | – | 0.3 (0.7) | |
| *Pocillopora* | 29.8 (22.2) | 1.1 (1.4) | 8.1 (14.6) | – | 18.0 (15.9) | 16.4 (14.0) | 8.9 (9.1) | 8.2 (6.1) | 0.2 (0.7) | – | 0.8 (1.5) | |
| *Porites* | 16.0 (9.3) | 18.1 (7.1) | 35.6 (21.5) | 17.5 (8.4) | 4.4 (4.5) | 22.4 (18.8) | 4.0 (3.0) | 12.0 (11.6) | 11.1 (7.7) | 8.4 (0.4) | 4.9 (4.3) | 9.5 (2.6) |

reefs were also higher than those reported on other Indian Ocean reefs such as Ningaloo Reef in Australia (2.3%; Onton et al. 2011), the Chagos Archipelago (5.2%; Sheppard et al. 2012) or the Maldives (<2%; Montano et al. 2012). However, values obtained for Reunion were similar to those on Mandapam reefs in Southeastern India (8.9%), but were substantially lower than those recorded at Palk Bay (21.0%), also in Southeastern India (Thinesh et al. 2009, 2011; Table 5). This relatively high disease level may be attributable to the fact that fringing reefs in Reunion are young and adjacent to areas of high coastal development, and are thus subjected to stressors such as poor water quality caused by anthropogenic activities (urbanisation and agricul-

Table 4. Summary of factorial ANOVA testing of seasonal and spatial variations in coral diseases on reefs in Reunion, South Africa and Mayotte across reef zones over 2 consecutive summers and winters. Analyses were performed on the mean prevalence of coral disease within each location, the most prevalent diseases (black band disease, BBD; *Porites* white patch syndrome, PWPS; and white syndrome, WS) and the most susceptible coral genera including *Acropora* (*Acr*), *Pocillopora* (*Poc*) and *Porites* (*Por*). $^{*}p < 0.05$; $^{**}p < 0.01$

| Location | Disease | Factor tested | df | *F* | p |
|---|---|---|---|---|---|
| Reunion | WS | Season | 3 | 0.01 | 0.97 |
| | | Zone | 1 | 80.14 | ** |
| | | Season × Zone | 3 | 64.05 | ** |
| | BBD | Season | 3 | 5.34 | * |
| | | Zone | 1 | 6.75 | * |
| | | Season × Zone | 3 | 1.44 | 0.24 |
| | PWPS | Season | 3 | 0.43 | 0.76 |
| | | Zone | 1 | 5.56 | * |
| | | Season × Zone | 3 | 1.47 | 0.23 |
| | *Por*-BBD | Season | 3 | 5.07 | * |
| | | Zone | 1 | 38.67 | ** |
| | | Season × Zone | 3 | 2.73 | * |
| South Africa | BBD | Season | 3 | 0.05 | 0.83 |
| | | Zone | 1 | 4.93 | * |
| | | Season × Zone | 3 | 1.82 | 1.19 |
| | *Acr*-WS | Season | 3 | 4.42 | * |
| | | Zone | 1 | 1.12 | 0.30 |
| | | Season × Zone | 3 | 0.71 | 0.55 |
| | *Poc*-WS | Season | 3 | 14.15 | ** |
| | | Zone | 1 | 17.87 | ** |
| | | Season × Zone | 3 | 4.26 | * |
| Mayotte | WS | Season | 1 | 5.27 | * |
| | | Zone | 1 | 0.34 | 0.55 |
| | | Season × Zone | 1 | 2.52 | 0.12 |
| | *Acr*-WS | Season | 1 | 4.85 | * |
| | | Zone | 1 | 0.49 | 0.48 |
| | | Season × Zone | 1 | 0.10 | 0.74 |

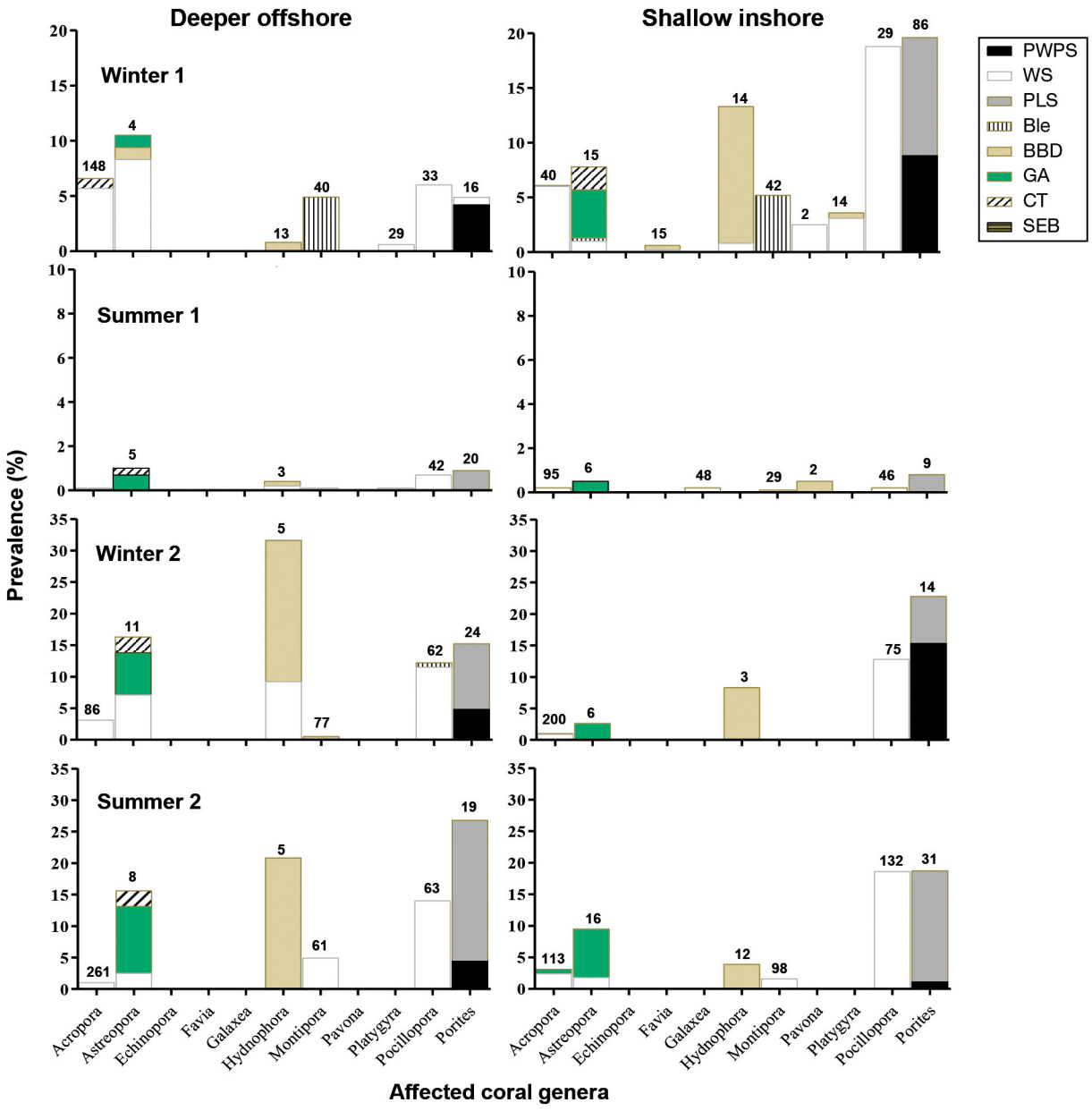


Fig. 3. Prevalence (%) of the main coral diseases in 11 scleractinian genera at Sodwana Bay, South Africa: *Porites* white patch syndrome (PWPS), white syndrome (WS), pink line syndrome (PLS), black band disease (BBD) and growth anomalies (GA). Prevalence is calculated relative to the total number of colonies in the respective taxa, per reef zone for 2 consecutive summers and winters. Note that bleaching (Ble) and compromised tissue (CT) are included in the analysis. Numbers above bars represent the N of each affected coral genus

ture in watersheds, wastewater discharge, sedimentation, over-exploitation and over-frequentation of reefs). This assumption was also proposed by Thinesh et al. (2011) to explain diseases patterns in Palk Bay and on Mandapam reefs. Nevertheless, more investigations are needed to identify factors that may facilitate disease outbreaks or exacerbate their effects on coral reefs.

Among the coral communities at the 3 localities, the genera most vulnerable to disease were generally *Acropora* and *Porites*. These genera, commonly found on back, lagoon and fringing reefs, are important reef-building corals in Reunion, South Africa and Mayotte (Turner & Klaus 2005). *Acropora* was mainly represented by the species *A. muricata* in Reunion and Mayotte and exhibited signs of WS,

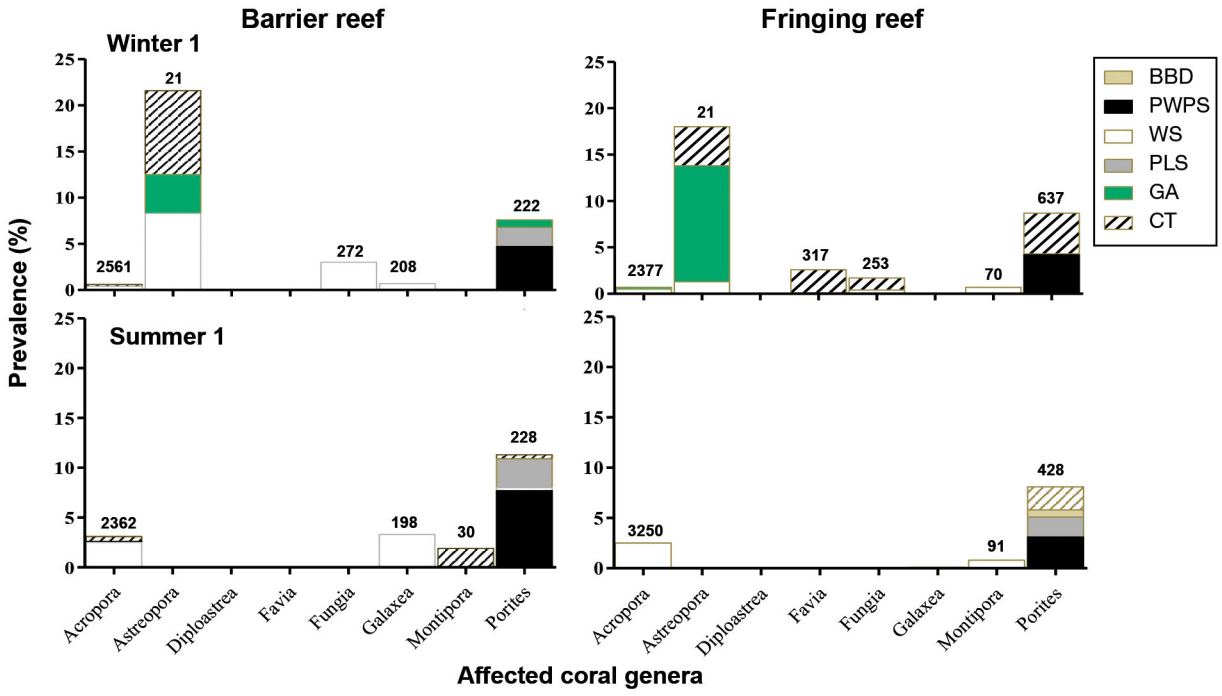


Fig. 4. Prevalence of the main coral diseases in 8 scleractinian genera in Mayotte: *Porites* white patch syndrome (PWPS), white syndrome (WS), pink line syndrome (PLS), black band disease (BBD) and growth anomalies (GA). Prevalence is calculated relative to the total number of colonies in the respective taxa per coral genus for 1 summer (March 2012) and 1 winter (August 2011). Note that compromised tissue (CT) is included in the analysis. Numbers above bars represent the N of each affected coral genus

Table 5. Overall coral disease prevalence (± SE) and diseases recorded on Indian Ocean coral reefs. WS: white syndrome; BBD: black band disease; PWPS: *Porites* white patch syndrome; PLS: pink line syndrome; SEB: skeleton eroding band; CT: compromised tissue; GA: growth anomaly; BrB: brown band disease, WBD: white band disease; WP: white plague disease; YBD: yellow band disease; PS: pink spot; UWS: ulcerative white spot; n-BCI: non-black cyanobacterial infections; AN: atramentous necrosis; PDDr: *Porites* dark discolouration response. ND: no data

| Geographic distribution | Prevalence % | Coral diseases observed | Source |
|---|---|---|---|
| **Western Indian Ocean** | | | |
| Reunion (2010–2012) | 7.5 (2.2) | WS, BBD, PWPS, PLS, SEB, CT, GA | Present study |
| South Africa (2010–2012) | 3.9 (0.8) | WS, BBD, PWPS, PLS, CT, GA | Present study |
| South Africa (1998) | 0.4 ± ND | BBD, YBD | Jordan & Samways (2001) |
| Mayotte (2011–2012) | 2.7 (0.3) | WS, BBD, PWPS, PLS, CT, GA | Present study |
| Tanzania to Kenya | ND | WS | McClanahan et al. (2004b) |
| **Eastern Indian Ocean** | | | |
| Ningaloo Reef | 2.3 (0.39) | WS, BBD, SEB, BrB | Onton et al. (2011) |
| Christmas and Coco Islands | 1–13 | WS | Hobbs & Frisch (2010) |
| Pilbara, Western Australia | | BBD, n-BCI, WS, GA, AN | Page & Stoddart (2010) |
| Montebello and Barrow Islands, Western Australia | 7.26 (1.56)–3.1 (0.6) | GA, BrB, SEB, BBD, WS | Pollock et al. (2014) |
| **Central Indian Ocean** | | | |
| Chagos Archipelago | 5.2 (0.2) | WS, GA | Sheppard et al. (2012) |
| Republic of Maldives | < 2 | WS, BBD, SEB, PDDr, BrB, UWS | Montano et al. (2012) |
| **Northern Indian Ocean** | | | |
| Mandapam (South India) | 8.9 ± ND | WS, BBD, WBD, WP, YBD, GA, PS | Thinesh et al. (2009) |
| Palk Bay (South India) | 21.0 ± ND | WS, BBD, WBD, PS, WP, YBD | Thinesh et al. (2011) |

SEB, GA and CT. This high level of susceptibility is consistent with other studies which revealed that *Acropora* species are particularly vulnerable to disease on Indian Ocean (McClanahan et al. 2004b, Thinesh et al. 2009, 2011) and Indo-Pacific reefs (Willis et al. 2004, Aeby et al. 2006, 2011a, Haapkylä et al. 2010). It has been suggested that corals which allocate more energy to growth and reproduction (e.g. Acroporidae and Pocilloporidae) are more susceptible to disease than massive corals; the latter seem to have greater resistance as they allocate more energy to colony maintenance (Haapkylä et al. 2010, Diaz & Madin 2011). However, our results have shown that massive colonies of *P. lutea* and *P. lobata*, generally considered robust and slow-growing (Raymundo et al. 2005), were prone to multiple infections and exhibited signs of BBD, PLS and PWPS. The susceptibility of this genus to disease has also been reported worldwide (Table S3 in the Supplement), notably on southeastern Indian (Thinesh et al. 2009, Onton et al. 2011, Montano et al. 2012), Philippine (Santavy et al. 2001, Raymundo et al. 2005) and other Indo-Pacific reefs (Sutherland et al. 2004, Haapkylä et al. 2009). Therefore, these results seem to contradict the assumptions of Haapkylä et al. (2009) and Palmer et al. (2008) that disease vulnerability is related to life history traits, with the investment of energy into growth by fast-growing species being to the detriment of their pathogen resistance. Alternatively, the high susceptibility of massive *Porites* colonies to disease may be attributed to predation that compromises their health (Diaz & Madin 2011). During this study, fish bites were observed on almost every colony of *P. lutea* and *P. lobata*. Corallivorous fishes are considered potential vectors of coral disease (Aeby & Santavy 2006, Raymundo et al. 2009, Chong-Seng et al. 2011), and Chong-Seng et al. (2011) found that fishes belonging to the families Blennidae, Chaetodontidae and Pomacentridae feed preferentially on infected coral colonies and may spread coral diseases. Finally, *Hydnophora* spp. and *Goniopora* spp., representing a minor component in both Reunion and South African reef communities (Table 3), exhibited particularly high susceptibility/sensitivity to BBD. For instance, all infected colonies of *Hydnophora* sp. recorded on the reef flat in Reunion died and, in subsequent surveys, were recorded as being colonised by opportunistic algae; no further *Hydnophora* colonies were encountered. This may suggest that BBD can restructure reefs at the local scale, highlighting the importance of frequent monitoring to assess disease-related shifts in coral community structure. The threat posed by BBD was also reported in the Caribbean, contributing to long-term mortality of susceptible coral species. For instance, an important BBD outbreak in the Florida Keys (USA) in 1993 has been identified as a major contributor to the decline in *Montastrea annularis* populations (Green & Bruckner 2000).

Spatial variability was detected in BBD and WS on both Reunion and South African reefs. For instance, the incidence of BBD on *Porites* spp. in Reunion seemed to be depth-related, with more diseased cases observed in shallow than deep habitats. Similar trends were found in South Africa in colonies of *Pocillopora* sp. infected by WS. These results are consistent with patterns found in the Caribbean (Weil & Cróquer 2009), Republic of Maldives (Montano et al. 2012) and Southern India (Thinesh et al. 2009, 2011), where both WS and BBD are more abundant at shallow than deep sites. However, in contrast, no spatial variation was observed in Mayotte, despite fringing reefs being exposed to increasing and greater anthropogenic pressures than the barrier reef. This may be attributable to an earlier suggestion that corals may develop resistance to the same stressors when frequently and continuously exposed to them (Weil et al. 2000).

Of the diseases found on shallow reefs in Reunion, BBD in particular seemed to manifest seasonality. For instance, when recorded on massive *Porites* spp., it was first observed during the summer of 2010. It decreased significantly during the winter of 2011 and then reappeared the next summer (Fig. 2). In South Africa, a higher prevalence of disease seemed to be linked to warmer water temperatures in the first year during summer. This was the case for WS-infected *Acropora* spp. and *Pocillopora* spp. However, no variations were observed for the same syndromes between the summer and winter of 2012. PWPS was also more prevalent in summer on both the reef slope and reef flat but the results were not statistically significant. On Mayotte reefs, WS syndromes on *Acropora* spp. varied between seasons, with their prevalence being higher during the warmer months. Similar seasonal patterns have been reported for both BBD and WS in Australia (Willis et al. 2004, Page & Willis 2006, Boyett et al. 2007, Bruno & Selig 2007, Haapkylä et al. 2010, Onton et al. 2011) and the Caribbean (Bruckner et al. 1997). These patterns may be due to impairment of the host's disease resistance under summer conditions, generating a shift in the natural bacterial communities in the coral holobionts towards opportunistic pathogens. Previous work has shown that elevated seawater temperatures increase disease progression (Willis et al. 2004, Bruno & Selig

2007) and tissue mortality (Boyett et al. 2007, Haapkylä et al. 2010) by stimulating the growth of putative pathogens (Patterson et al. 2002, Ben-Haim et al. 2003, Cervino et al. 2004, Rosenberg & Falkovitz 2004, Boyett et al. 2007). This alters the structure of the coral-associated bacterial population (Reshef et al. 2006, Mouchka et al. 2010) which may have an important role in disease-resistance (Ritchie 2006).

Although this study fills a gap in the knowledge on coral disease prevalence in the Western Indian Ocean, it constitutes preliminary work based on surveys conducted only at 3 geographically distant localities and during 2 consecutive years. More investigations are needed, notably on the drivers (factors) and vectors (e.g. corallivorous organisms) of the diseases to improve our understanding of coral diseases and our ability to mitigate their impacts at local and regional scales. Therefore, long-term disease surveys should be incorporated and standardised within existing monitoring programmes regularly conducted by both scientists and volunteers on the Western Indian Ocean reefs such as the Global Coral Reef Monitoring Network (GCRMN).

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