Bacterial Communities Associated with *Porites* White Patch Syndrome (PWPS) on Three Western Indian Ocean (WIO) Coral Reefs

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Abstract

[illegible] *Porites lutea*, [illegible] (WIO), [illegible] (WS) [illegible] *Porites* [illegible] (PWPS). [illegible] 16S RNA [illegible] PWPS-[illegible] *P. lutea*, [illegible] (BBD), [illegible] (WBD) [illegible] (WPD). [illegible] *Porites lutea* [illegible] WIO [illegible] *Vibrionaceae* [illegible] *Rhodobacteraceae* [illegible] PWPS-[illegible] *Shimia marina* (NR043300.1) [illegible] *Vibrio hepatarius* (NR025575.1) [illegible] PWPS [illegible]

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Introduction

The scleractinian *Porites lutea*, commonly found on back reefs, lagoon and fringing reefs [1], is an important reef-building coral in the Western Indian Ocean (WIO) reefs. Despite its widespread distribution, this hermatypic coral has shown a particular susceptibility to natural pressures such as predation [2,3] and infestation by parasites [4,5]. Moreover, it seems to be more vulnerable to infectious disease than many other coral species [6].

Of the 30 coral diseases described to date [7,8], eight are known to affect *P. lutea* world wide. On Indo-Pacific reefs, colonies of *P. lutea* have been recorded with signs of black band disease (BBD), white plaque syndrome (WPL), growth anomalies (GA), yellow band disease (YBD) and pink line syndrome (PLS) [8]. Surveys conducted in the Gulf of Kutch [9], Papua New Guinea [10] and Philippines [11] have recorded BBD outbreaks in this scleractinian coral. In addition, a study performed on coral health and diseases in the northern Egyptian Red Sea has revealed two other syndromes: *Porites* ulcerative white spot (PUWS) and a white syndrome (WS) so far unreported on *P. lutea* [12]. More recently, a white syndrome (WS) named *Porites* white patch syndrome (PWPS) was described on massive colonies of *P. lutea* on Western Indian Ocean (WIO) reefs [13]. This syndrome was characterised by diffuse, medium to large (50–300 mm diameter), circular to oblong tissue loss, surrounded by swollen white tissue. The dead skeleton was progressively colonised by opportunistic algae and *Cyanobacteria* [13].

To date, nothing is known about the aetiology of PWPS. Previous studies on other white syndromes (white band disease (WBD), white plague disease (WP), progressive white syndromes (PWS), Australian subtropical white syndrome (ASWS), *Acropora* white syndrome in American Samoa (AWS), and *Porites* bleaching with tissue loss (PBTL)) have characterized organisms (bacteria, ciliates, helminths, fungi, algae) associated with both healthy and diseased coral colonies. These investigations have allowed identification of a number of putative pathogens [14–22]. Evidence of the involvement of bacteria as causative agents has been suggested in some studies on several of the WS observed on scleractinian corals [23–26]. For example *Serratia marcescens* has been reported to be linked with white pox disease (WPD) in the Elkhorn coral *Acropora palmata* [25] and *Vibrio owensii* to be the aetiological agent of *Montipora* white syndrome (MWS) in the Hawaiian coral *Montipora capilata* [27]. However, some of these

potential causative agents have i) not been fully characterised in terms of fulfilling all Koch's postulates [28] or ii) have been biased by the execution of infection trials with a specific pathogen rather

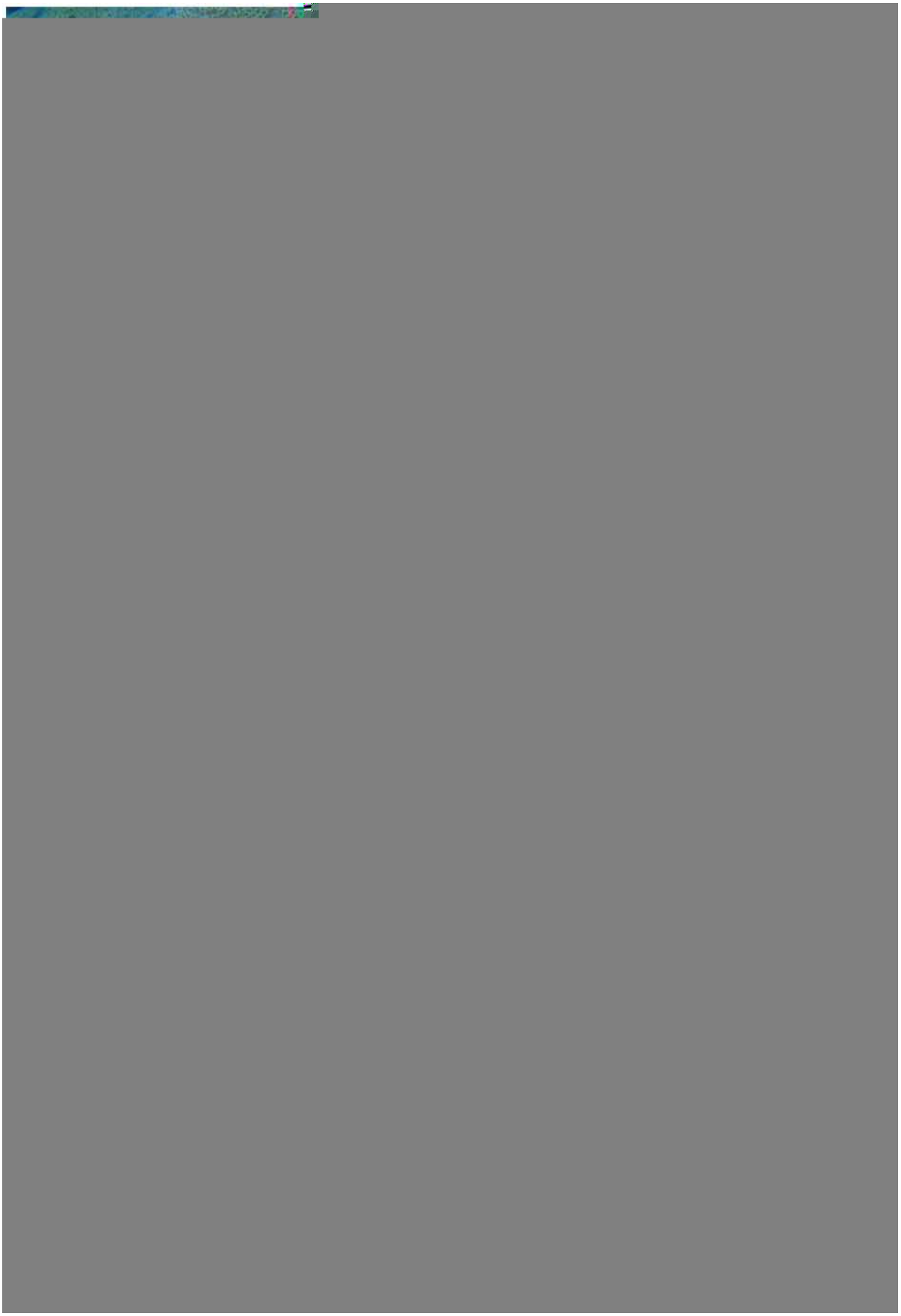

Figure 1. PWPS on *Porites lutea* (A) and map of the Western Indian Ocean showing the sampling locations (B).
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alignment and rearranged manually. A phylogenetic tree was built using the Neighbor-Joining method of GENEIOUS^{TM}Pro (V.5.6.3). All 16r RNA gene sequences are accessible through the NCBI GeneBank database under accession numbers KF179641-KF180135.

Sta

Multidimentional Scale (MDS) analysis of bacterial communities associated with healthy and PWPS-affected tissues of Porites lutea collected at Reunion (R), South Africa (RSA) and Mayotte (M) was performed using PRIMER (V.6.1.14). Data were square root-transformed and MDS analysis was carried out using the Bray–Curtis similarity coefficient. Finally, the Shannon-Weaver index (H) was calculated for each tissue category to characterise pooled bacterial diversities in healthy and diseased coral samples

Results

Mi

Histological cross-sections of PWPS revealed extensive tissue breakdown and necrosis within the lesion area between the exposed skeleton and living tissue (Fig. 2A). Ovoid basophilic bodies resembling bacterial aggregates were visible within the mesogloea of the body wall, mainly in DT (Table 1), especially in the area of tissue fragmentation (Fig. 2B, D). These aggregates were seen in nine of the 15 samples collected from corals showing signs of PWPS at all three sampling locations. Among the 15 samples of HT, only one was found with such aggregates (Table 1). Other organisms, including *Cyanobacteria* (Fig. 2C, F), Nematoda (Fig. 2E), Ciliata (Fig. 2A) and algae (Fig. 2F) were also observed but only within dead tissue.

Ba

A total of 91, 74 and 100 16S rRNA sequences (818–1627 bp), subdivided into seven, six and four classes (Table 2 and Fig. 3) were obtained from healthy tissues collected in Mayotte (HT-M), South Africa (HT-RSA) and Reunion (HT-R) respectively (Table 2). Sequences retrieved from HT-M (Table 2 and Fig. 3) were mainly identified as members of the *γ-proteobacteria* (42.0%), *α-proteobacteria* (19.0%), *Cyanobacteria* (11.0%), *Firmicutes* (6.0%), *Cytophagia* (6.0%) and *?-proteobacteria* (3.0%). Of all sequences, 14.0% had no close relatives in the NCBI database and could only be classified as unknown bacterial clones. The bacterial diversity associated with HT-RSA (Table S1) was also dominated by sequences closely related to *γ-proteobacteria* (88.0%) followed by *Cyanobacteria* (5.0%), *α-proteobacteria* (2.0%), *Spirochaetes* (1.0%) and *Actinobacteria* (1.0%). HT-R (Table S1) seemed to contain less group diversity, mainly dominated by *γ-proteobacteria* (67.0%), *Cyanobacteria* (11.0%) and *Firmicutes* (6.0%). Unknown bacterial clones constituted 17.0% of all analysed sequences. The *γ-proteobacteria* retrieved from HT-M, HT-RSA and HT-R were dominated by bacterial species closely related to *Endozoicomonas elysicola* (accession no. NR041264), comprising 53.1%, 67.8% and 81.8% of the total *γ-proteobacteria* respectively. Sequences closely-related to *Vibrio fortis* (accession no. NR025575) were also common to bacterial communities associated with HT from all three sampling locations.

Table 1. Nb

	Ri		SbAfi		My	
Tissue categories	**HT**	**DT**	**HT**	**DT**	**HT**	**DT**
Samples	5	5	5	5	5	5
Cross-sections/sample	10	10	10	10	10	10
Samples with Ciliata	0	1	0	0	1	0
Samples with endophytic algae	0	5	0	5	0	5
Samples with *Cyanobacteria*	0	3	0	0	0	2
Samples with Nematoda	0	5	0	4	0	0
Samples with bacterial aggregates	0	4	0	2	1	3

HT = ; DT =
doi:10.1371/journal.pone.0083746.t001

Bb

A total of 91, 145 and 60 16S rRNA gene clones (527–1602 bp), subdivided into 10, 12 and 5 classes (Table 2 and Fig. 3), was obtained from diseased tissues collected in Mayotte (PWPS-M), South Africa (PWPS-RSA) and Reunion (PWPS-R) respectively (Table S1). PWPS-M samples exhibited high diversity (Fig. 3), dominated by members of the *γ-proteobacteria* (38.0%) and *α-proteobacteria* (23.0%), followed by *Cyanobacteria* (6.0%), *Cytophagia* (6.0%), *Firmicutes* (3.0%), *Bacteroidetes* (3.0%), *β-proteobacteria* (3.0%), *Chloroplasts* (3.0%), *Planctomycetes* (3.0%) and *Flavobacteriia* (3.0%). Of the analysed sequences, 17.0% had no close relatives in the NCBI database and could only be classified as unknown bacterial clones. Similar trends emerged for PWPS-RSA (Fig. 3), the bacterial classes being dominated by *γ-proteobacteria* (56.9%) and *α-proteobacteria* (14.6%), followed by *Cyanobacteria* (3.5%), *Firmicutes* (3.5%), *Planctomycetes* (2.1%), *Bacteroidetes* (0.7%), *Cytophagia* (0.7%), *Delta-proteobacteria* (0.7%), *Flavobacteriia* (3.0%) and *Sphaerobacteridae* (3.0%). Again, some sequences (16.0%) had no close relatives in the NCBI database and could only be classified as unknown bacteria. PWPS-R (Table S1) samples were similarly dominated by members of the *γ-proteobacteria* (38.0%) and *α-proteobacteria* (23.0%), but contained only three other classes belonging to genera of *Bacteroidetes* (15.0%), *Cyanobacteria* (8.0%) and *Firmicutes* (4.0%).

Among the several bacterial classes found in this study, the *γ-proteobacteria Vibrio parahaemolyticus* (accession no. NR041838.1; n = 15), *V. fortis* (accession no. NR025575.1; n = 5) and *V. rotiferianus* (accession no. NR042081.1; n = 4), as well as the *α-proteobacteria Paracoccus yeei* (accession no. NR029038.1; n = 4), *Pseudoruegeria aquimaris* (accession no. NR043932; n = 2) and *Shimia marina* (accession no. NR043300.1; n = 3) were the best represented ribotypes in PWPS-M. The predominant bacterial ribotypes in PWPS-R were the *γ-proteobacteria E. elysicola* (accession no. NR041264; n = 12), *Photobacterium damselae* (accession no. NR042975.1; n = 4) and *Photobacterium* sp. (accession no. HQ697926; n = 3). The next most abundant sequences were closely related to the *α-proteobacteria P. yeei* (accession no. NR029038.1; n = 5), *Ruegeria pomeroyi* (accession no. NR028727; n = 3), *S. marina* (accession no. NR043300.1; n = 3) and *Silicibacter lacuscaerulensis* (accession no. NR029197; n = 3). Among the bacterial strains retrieved from PWPS-RSA, the *γ-proteobacteria*, *E. elysicola* (accession no. NR041264; n = 41) and *Oceanospirullum beijerinckii* (accession no. NR040784; n = 5), the *α-proteobacterium S. marina* (accession no. NR043300.1; n = 3) and the *Cyanobacterium*

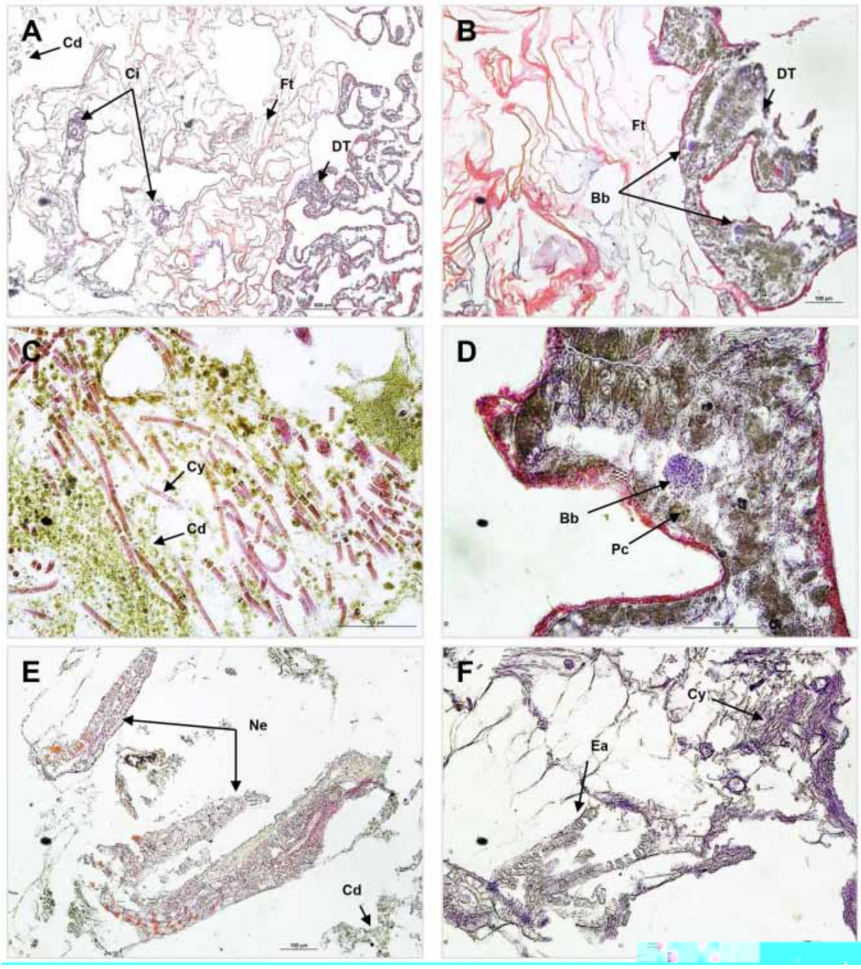

Figure 2. Photomicrographs of diseased *Porites lutea* coral tissues: *Porites* white patch syndrome (PWPS). A) C[illegible] (FT) [illegible] (DT); Cd = [illegible] Ci = C[illegible] **B**) *P. lutea* [illegible] PWPS. N[illegible] Ft = [illegible] **C**) C[illegible] *P.lutea* [illegible] PWPS [illegible] (Cd) [illegible] *Cyanobacteria* (Cy). **D**) C[illegible] (Bb) [illegible] **E**) N[illegible] (Ne) [illegible] **F**) E[illegible]
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Table 2. N[illegible] (SW) [illegible]

	Reunion		South Africa		Mayotte	
	HT	**DT**	**HT**	**DT**	**HT**	**DT**
Samples collected	3	3	3	3	3	3
Pooled samples	1	1	1	1	1	1
Random clones from pooled samples	92	100	101	150	94	100
Consensus sequences	74	60	100	145	91	91
Sequence lengths (bp)	905±498	904±490	842±50	850±498	1240±483	855±477
Sequence quality (%)	77.8±00	72.2±00	74.2±00	66.2±00	78.7±00	67.1±00
Class/subdivision	4	5	6	11	8	9
Species	12	26	21	53	36	39
Diversity (Shannon-Weaver index)	1.20	**2.83**	1.70	**3.37**	2.86	**3.29**

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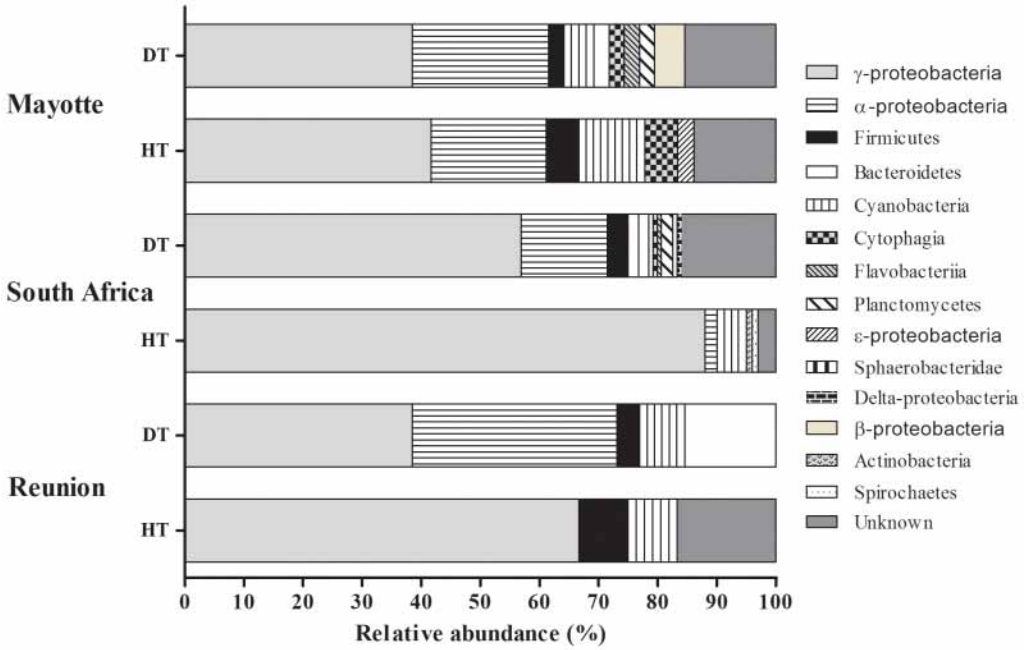

Figure 3. Relative abundance (%) of bacterial phyla retrieved from three diseased (DT) and three healthy (HT) samples of *Porites lutea* collected in Mayotte (MAY), South Africa (RSA) and Reunion (REU).
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Prochlorococcus marinus (accession no. NR028762; n = 5) were the most representative species.

Comparison of bacterial communities

Distinctly partitioned ribotypes were detected among diseased and healthy tissues samples. In total, 31 (77.8%), 54 (90.0%) and 24 (92.3%) bacterial ribotypes were exclusively associated with PWPS-M, PWPS-RSA and PWPS-R respectively, while 28 (77.8%), 17 (73.9%) and 10 (83.3%) were found only in HT-M, HT-RSA and HT-R respectively. Multidimentional scaling (MDS, Fig. 4B) analysis performed on the composition of bacterial 16S rRNA gene of each tissue categories revealed four distinct clusters representing four distinct bacterial communities. Similarities in bacterial composition in RDT and MDT were detected but SAHT, RHT, SADT and MHT samples exhibited more variability (Fig. 4). In addition, the bacterial diversity identified in PWPS tissues collected on the three WIO coral reefs was higher than in HT (Table 2). For instance, 39, 60 and 26 16S rRNA gene sequences affiliated to bacterial genera/species were identified in PWPS-M, PWPS-RSA and PWPS-R respectively, whereas only 36, 23 and 12 were obtained in HT-M, HT-RSA and HT-R respectively (Table 2). Among these, only six ribotypes were commonly detected in PWPS samples from the three sampling localities and were closely related to *P. yeei* (accession no. NR025491), *P. aquimaris* (accession no. NR029038.1), *S. marina* (accession no. NR043300.1), *V. fortis* (accession no. NR025575.1), *V. hepatarius* (accession no. NR025575.1) and *V. parahaemolyticus* (accession no. NR041838.1). In HT, only 16S rRNA gene sequences affiliated to bacterial species *E. elysicola* (accession no. NR041264) and *V. fortis* (accession no. NR025575.1) were common in samples collected at the three sampling localities.

Discussion

Histopathology

Corals exhibiting signs of PWPS revealed extensive tissue fragmentation, generally associated with ovoid basophilic bodies resembling bacterial aggregates within the mesoglea of the body wall. These aggregates were seen in 60% of all samples collected from corals with signs of PWPS. However, these aggregates could not be directly linked with the pathology as there was no clear evidence of inflammatory response or tissue lysis associated with these ovoid bodies. In addition, some clusters of basophilic bodies were also observed in sections of one healthy sample of *P. lutea*, preventing definitive conclusion that they constituted a bacterial infectious agent in the PWPS lesions. Similar observations on bacterial aggregates have been previously reported in several histopathological studies, in both healthy and WS-infected colonies of *Acropora* spp., *Pocillopora meandrina* and *Porites compressa* [22,30]. Direct identification from formalin-fixed, paraffin-embedded coral tissue combined with descriptions of cellular changes over time may be a viable option to identify the role of these aggregates in PWPS. Other organisms, including *Cyanobacteria*, Nematoda, Ciliata and endophytic algae, were also observed on diseased tissues and were generally associated with the dead epidermis and cell debris. No obvious evidence of the direct physical ingress of these organisms into the tissue in cross-sections was observed suggesting that these were potentially opportunistic invaders.

Variability in PWPS and HT

The bacterial communities in both healthy and PWPS-infected tissues of *P. lutea* were dominated at all three sampling locations by members of the *α-proteobacteria*, *γ-proteobacteria* and *Cyanobacteria* (Fig. 3). However, diseased corals exhibited higher bacterial diversity compared to healthy ones (Table 2). These results are consistent with recent studies which have reported lower bacterial abundance and diversity in healthy corals than those displaying signs of WPD

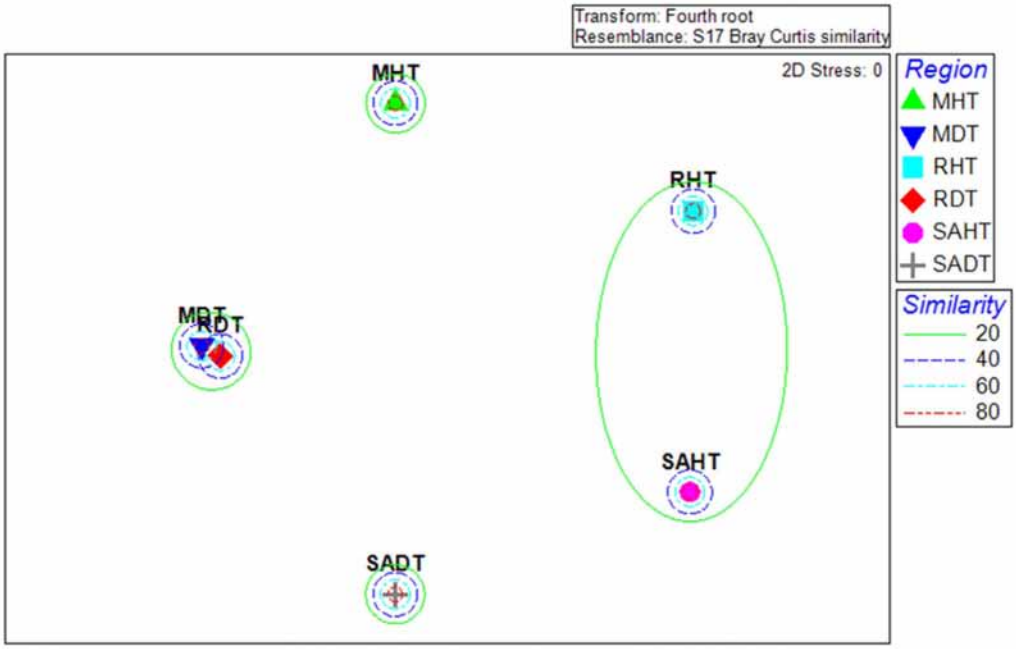

Figure 4. Multidimensional scaling (MDS) ordination of bacterial communities associated with healthy (HT) and PWPS-affected tissues (DT) of *Porites lutea* collected at Reunion (R), South Africa (RSA) and Mayotte (M).
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[18,19,31] and WBD [14]. Examination of 16S rRNA gene sequences using cloning as a culture-independent molecular technique, revealed high variability between bacteria associated with PWPS-infected and healthy tissues of *Porites lutea*, with only a few ribotypes commonly found in both diseased and healthy tissues (Table S1). Among them, ribotypes similar to *Pseudoalteromonas* spp., *Paracoccus yeei* and *Amphritea balenae* have been previously identified in sea water [32], soil [33] and sediment [34] respectively, suggesting that these bacteria were present in the environment and opportunistically became resident in the coral mucus or associated with the healthy coral microbiota. Similar variations in the bacterial communities have been reported in several coral species affected by BBD [35], WPD [17], WBD in the Caribbean [14] and other WS in Australia and American Samoa [15,20,36]. The bacterial diversity found in PWPS was higher than in HT at all three localities. Our results were similar to those reported for white syndromes including *Acropora* white syndrome (AWS) on American Samoan reefs [20] and white plague disease in the Caribbean coral *Montastrea annularis* [17]. This difference in the composition of bacterial communities may suggest that disease agents impair the structure of natural bacterial communities. It is likely that compromised or dead tissues represent a "micro-niche" that can be colonised by more competitive and opportunistic bacteria in the surrounding water and sediments or transmitted by other marine organisms [14,15,17,18,37].

Interestingly, comparisons of bacterial communities associated with both PWPS-infected and healthy tissues also revealed distinct populations at the three sampling locations (Table S1, Fig. 4). This may suggest that no specific bacterial communities are associated with *P. lutea* on the WIO reefs. However some exceptions were recorded. For example, *E. elysicola* (accession no. NR041264) and *V. fortis* (accession no. NR025575.1) were found in both PWPS and HT collected at all the localities and seemed to be coral-specific. Another species, *V. rumoiensis* (accession no. NR024680), seemed to develop the same specific bacterial-coral association but was only found in HT sampled on South Africa and Mayotte reefs and not those at Reunion. These bacterial strains, apparently ubiquitous in HT, may play an important role in coral health and growth [14]. For instance the genus *Endozoicomonas*, found in several marine organisms [38–41], seems to play an important role in corals, notably in the biogeochemical cycling of sulphur [42]. *V. fortis* was initially described as a probiotic that out-competes pathogen strains [43,44] or is involved in the recycling of dimethylsulfoniopropionate (DMSP), which may be detrimental to coral health [42]. However, further studies are needed to elucidate the ecological function of these genera in corals.

Pathogens in PWPS

In our study, several 16S rRNA gene sequences were closely related (97–100% similarity) to bacteria associated with coral diseases or known pathogens. Interestingly, one sequence was detected in PWPS from all three sampling localities but absent in healthy corals. Blast identification associated with phylogenetic analysis (Fig. 5) showed it to be closely related to the *γ-proteobacteria V. hepatarius* (accession no. NR025575.1), isolated for the first time from the healthy wild white shrimp *Litopenaeus vannamei* in Ecuador [43]. Other 16S rRNA gene sequences affiliated to the family *Vibrionaceae* were associated with PWPS-infected tissues (Fig. 5). For instance, *V. fortis* was detected at all three sampling locations. This bacterium was first isolated from various marine organisms and has been reported to be pathogenic in corals [45], fish and crustacea [46], and is associated with several coral diseases including yellow band disease (YBD) in *Montastrea faveolata* [47] and BBD in the Red sea [48]. However, sequences affiliated with this species were also found in HT from all three localities, making this a less likely candidate for PWPS pathogenesis. In addition, ribotypes similar to *V. parahaemolyticus*, known to induce disease in humans [49] and many aquatic organisms [50], were identified as well as *V rotiferanus* associated with YBD in several Caribbean and Indo-Pacific scleractinian species [51]. However, similar sequences were found in healthy coral tissues or were not represented in

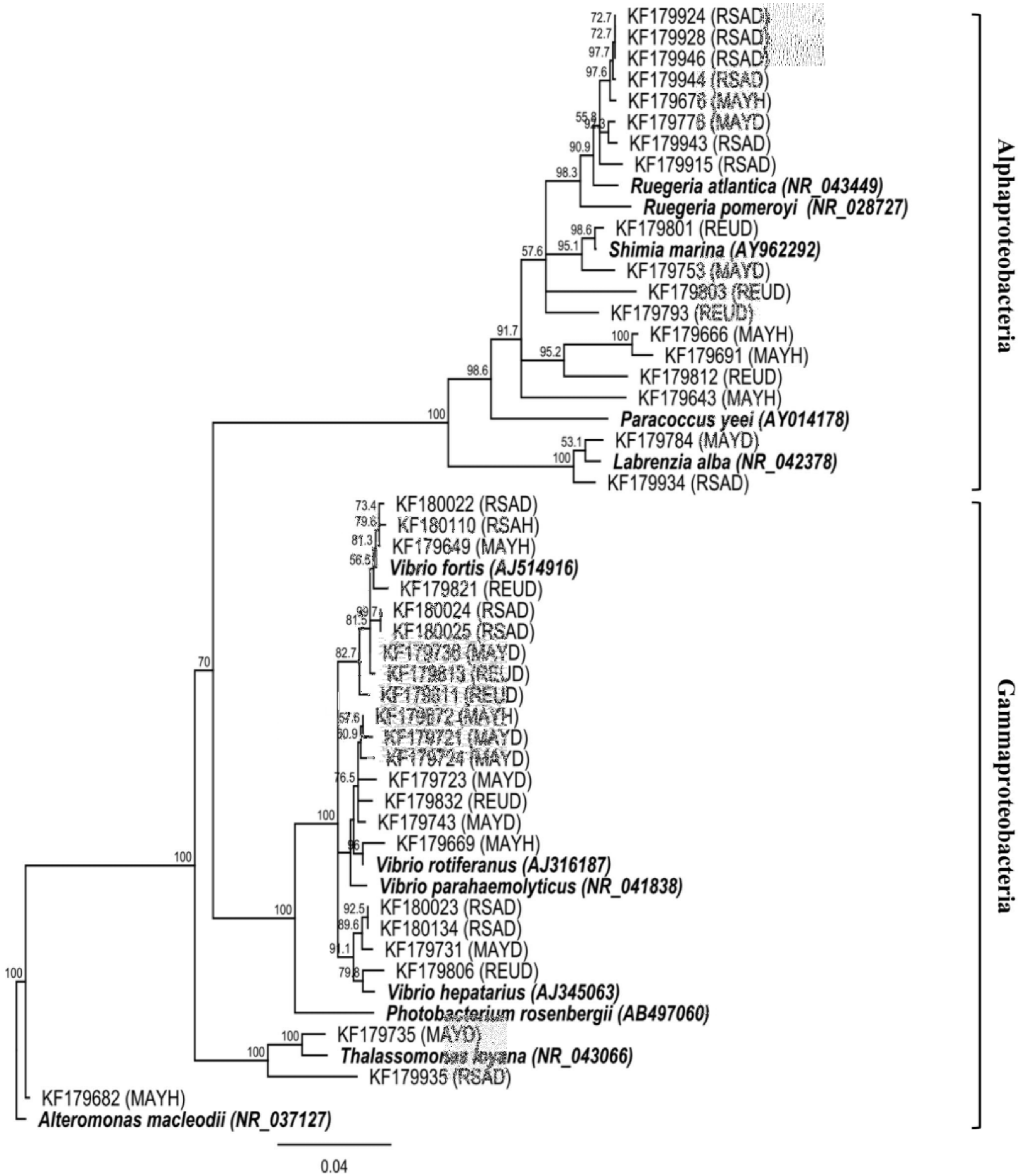

Figure 5. Neighbour-joining phylogenetic tree for the 16SrRNA gene sequences that were closely related to known and putative pathogens found in both healthy (HT) and *Porites* white patch syndrome (PWPS)-infected tissues (DT) of *Porites lutea* from Mayotte (MAY), South African (RSA) and Reunion (REU) corals. Nbeeba(%) tf1000 b

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diseased tissues at all three localities. Finally, sequences affiliated to *Shimia marina* (accession no. NR043300.1) were recorded only in PWPS-infected corals at all three sampling localities. This *roseobacter* was previously reported in the coral *Turbinaria mesenterina* infected by ASWS [15] but no evidence of its pathogenicity has been established in previous studies. The potential pathogens related to the sequences obtained in this study thus need to be

isolated, cultured and inoculated in laboratory corals to ascertain their ability to induce disease in corals.

Conclusion

This is the first study characterising bacterial communities associated with healthy and PWPS-infected *Porites lutea* coral colonies on WIO coral reefs. Microscopy revealed the inclusion of basophilic bodies like bacterial aggregates in the coral epidermis within the lesion area. We established that the structure of the microbial communities is different between diseased and healthy coral tissues, and between localities, by cloning the 16S rRNA gene as a culture-independent molecular method. Furthermore, higher bacterial diversity was observed in PWPS-infected tissues. This shift may be explained by a perturbation of the natural bacterial communities associated with coral holobionts which are progressively replaced by a succession of opportunistic bacteria including potential pathogens. Since the bacterial diversity at each of the three sites was assessed by analysing pooled samples, additional replicates including seasonal monitoring is needed to confirm the heterogeneity of bacterial species associated with PWPS in the areas studied. Several bacterial ribotypes affiliated to potential putative pathogens were consistently found among the 16S rRNA sequences derived from the PWPS lesions, and absent and/or poorly represented in HT. Isolation, culture and subsequent infection trials to satisfy Henle-Koch's postulates would be needed to prove their pathogenicity.

Supporting Information

Table S1 Bacterial 16S rRNA gene sequences from samples of apparently healthy (HT) and PWPS-diseased (DT) *Porites lutea* tissues collected at Mayotte (M), Reunion (R) and South Africa (SA).
(DOCX)

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Author Contributions

Conceived and designed the experiments: MGS PT JPQ PC MHS JT. Performed the experiments: MGS. Analyzed the data: MGS PT MHS. Contributed reagents/materials/analysis tools: MGH PT JT JPQ PC MHS. Wrote the paper: MGS PT MHS.

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