Tissue mortality by Caribbean ciliate infection and white band disease in three reef-building coral species (#8615)

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Alejandra Verde, Carolina Bastidas, Aldo Croquer

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Tissue mortality by Caribbean Ciliate Infection and White Band Disease in three reef-building coral species

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ABSTRACT

Caribbean ciliate infection (CCI) and white band disease (WBD) are diseases that affect a multitude of coral hosts and are associated with rapid rates of tissue losses; thus, contribute with declining coral cover in Caribbean reefs. In this study we compared tissue mortality rates associated to CCI in three species of corals with different growth forms: *Orbicella faveolata* (massive-boulder), *O. annularis* (massive-columnar) and *Acropora cervicornis* (branching). We also compared mortality rates in colonies of *A. cervicornis* bearing WBD and CCI. The study was conducted at two locations in Los Roques Archipelago National Park between April 2012 and March 2013. In *A. cervicornis*, the rate of tissue loss was similar between WBD (0.8±0.2 mm/day, mean SD) and CCI (0.7±0.2 mm/day). However, mortality rate by CCI in *A. cervicornis* was faster than in the massive species *O. faveolata* (0.4±0.1 mm/day) and *O. annularis* (0.3±0.05 mm/day). Tissue regeneration was at least fifteen times slower than the mortality rates for both diseases regardless of coral species. This is the first study providing coral tissue mortality and regeneration rates associated to CCI in colonies with massive morphologies, and it highlights the risks of further cover losses of the three most important reef-building species in the Caribbean.

Keywords: coral diseases, Caribbean ciliate infection, white band disease, ciliates, corals

INTRODUCTION

During the past few decades Caribbean coral reefs have declined partly due to the increasing prevalence of emergent and highly virulent coral diseases (Goreau et al. 1998; Harvell et al. 1999; Richardson & Aronson, 2000). Coral diseases, defined as a transitory or permanent alteration of the host physiology (Sutherland, Porter & Torres 2004), have been often associated to bacteria (Garrett & Ducklow 1975; Ritchie & Smith 1995; Richardson 1998), fungi (Le Champion-Alsumard, Golubic & Priess 1995; Morrison-Gardiner 2001; Ravindran, Raghukumar & Raghukumar 2001) or consortia of different microorganisms (Ducklow & Mitchell 1979; Richardson 1996). However, fewer diseases have been associated to protozoan infections (Antonius & Liscomb 2000; Cróquer et al. 2006).

Among protozoan infections, brown band (BB), skeletal eroding band (SEB) and Caribbean ciliate infections (CCI) are the ones with wider geographical distribution; the first two affecting a myriad of Indo-Pacific coral hosts (Page & Willis 2008) and the latter more than 25 out of the approximately 60 scleractinian species in the Caribbean (Cróquer et al. 2006). Based on microscopic examination, Rodriguez et al. (2009) suggested the name Caribbean ciliate infections (CCI) for describing *Halofolliculina* on Caribbean corals (Weil & Hooten 2008; Rodríguez et al. 2009). Here, ciliate infections by *Halofolliculina* were first reported in 10 coral species from Venezuela (Cróquer, Bastidas & Lipscomb2006) but soon after that it was observed throughout the wider Caribbean (Cróquer et al. 2006). Among affected corals, *Acropora palmata*, *A. cervicornis*, *Diploria labyrinthiformis*, *D. strigosa*, *Colpophyllia natans*, *Montastraea faveolata*, *M. annularis*, *M. franksi*, *Agaricia tenuifolia* and *Porites porites*, appeared particularly vulnerable to *Halofolliculina* infections (Cróquer, Bastidas & Lipscomb 2006; Page et al. 2015). In Venezuela, CCI mostly affects species of *Acropora* and *Orbicella*, reaching a prevalence of up

to 85% of colonies of *A. cervicornis* in Los Roques (Cróquer, Bastidas & Lipscomb 2006; Rodríguez et al. 2009). The relatively recent discovery of CCI in the Caribbean, despite disease surveys dating back to the 1970s, suggested that either the disease has recently emerged or it has been overlooked or confounded with Black Band Disease (Cróquer, Bastidas & Lipscomb 2006; Page et al. 2015).

Experimental studies in the Caribbean demonstrated that *Halofolliculina spp.* transmits directly and horizontally from infected to susceptible host (Rodríguez et al. 2009). Also, the presence of lesions in corals facilitates the colonization by folliculinid ciliates (Rodríguez et al. 2009). Thus, it has been suggested that *Halofolliculina* infections in the Caribbean and in the Indo-Pacific (CCI and SEB, respectively) are opportunistic since they are more likely to invade damaged tissues. Aggregations of folliculinid ciliates forming scattered or dense clusters are often found in corals affected by WPD and WBD. However, factors involved in the formation of pathogenic aggregations of *Halofolliculina* species in CCI remain poorly understood. Seasonal environmental changes seem to affect the rate of tissue mortality of infected hosts. For instance, Rodríguez (2008) found differences in the rate of tissue mortality of CCI in *Acropora palmata* and *A. cervicornis*, being significantly higher between July and December when temperature and wind speed are higher. Moreover, Hernández (2009) found a positive and significant correlation between the rate of tissue mortality of CCI and the concentration of suspended solids in the coral *A. cervicornis*.

Recent studies also show that ciliates are common organisms thriving in lesions produced by other coral diseases including WBD, and whether they are scavengers or pathogens in corals with white syndromes remain controversial (Sweet & Bythell 2012; Randall, Jordán-Garza & van Woesik 2015; Sweet & Séré 2015). White band disease was first noticed in the early 1970s (Gladfelter 1982) and was the first coral disease to cause widespread mass mortality (Gladfelter 1982; Green & Bruckner 2000). Multiple bacteria have been associated as the primary cause of WBD infections: a) Ritchie & Smith (1998) and Gil-Agudelo, Smith & Weil (2006) identified *Vibrio harveyi* as the putative pathogen of WBD; b) Sweet, Cróquer & Bythell (2014) identified three bacteria *V. harveyi*, *Lactobacillus suebicus* and *Bacillus sp.* as possible putative pathogens; and c) Gignoux-Wolfsohn & Vollmer (2015) proposed various strains of Flavobacteriales as a new causative pathogen of WBD, although it is unknown if WBD is caused by a single or a consortium of bacteria. WBD has only been found to affect acroporid corals in the Caribbean, and two types of WBD have been described based on short-term observations of specific features of lesions (Ritchie & Smith 1998; Bythell, Pantos & Richardson 2004). Likely, WBD is the most detrimental disease on Caribbean coral reef ecosystems as it has decimated populations of the reef building corals *Acropora palmata* and *A. cervicornis* to critical levels (Goreau et al. 1998; Richardson 1998; Richardson & Aronson 2000) and the presence and rapid spreading of CCI could be aggravating this plight by producing further tissue loss and hampering recovery of populations.

For CCI, no studies have compared the rate of tissue mortality among coral host with different morphologies, growth forms and life strategies under natural conditions. The ability to recover and/or to heal CCI injuries is also poorly understood. In this study we estimated the rate of tissue mortality by CCI in two massive and one branching Caribbean coral species in the field (i.e., *Orbicella faveolata*, *O. annularis* and *Acropora cervicornis*) and their rate of tissue regeneration. We also compared the rates of tissue mortality associated to CCI and WBD in *A. cervicornis*.

MATERIALS AND METHODS

Study Site

Los Roques National Park (LRNP) is an oceanic archipelago located 160 km north of the Venezuelan coast (N 11° 44′ 26”-11° 58′ 36”, W 66° 32′ 42”-66° 57′ 26”; Figure 1). The reef system encompasses more than 50 coralline cays with fringing reefs, patch reefs, over 200 sand banks, and extensive mangrove forests and seagrass beds (Weil, 2003). The study was conducted in two sites: Dos Mosquises Sur and Cayo de Agua (Figure 1).

Estimation of mortality rates of WBD and CCI

A total of 106 coral colonies of *Acropora cervicornis*, *Orbicella faveolata* and *O. annularis* were tagged and observed during four field trips: April, May and November 2012 and March 2013. Estimations of mortality and regeneration were obtained from two sets of independent observations (April-May 2012 and November 2012-March 2013). The first set of colonies showing the classic signs of WBD (only *A. cervicornis*) and CCI (all three species) were tagged in April 2012 and measured in May 2012. The

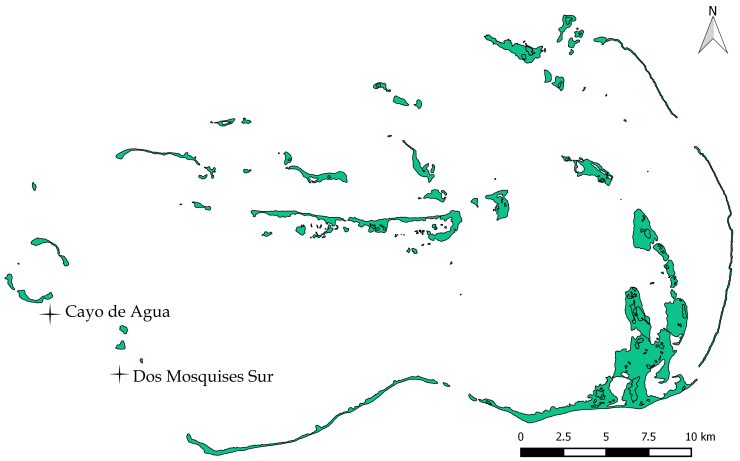

Figure 1. Study sites Dos Mosquises Sur and Cayo de Agua. Map provided by Francoise Cavada and Laboratorio de Sensores Remotos.

second group of colonies were tagged in November 2012 and measured on March 2013. All permits necessary to conduct this work were processed and accepted by the Governmental Venezuelan authorities (i.e., Ministerio del Poder Popular para el Ambiente-Oficina de Diversidad Biológica) and the Instituto Nacional de Parques Nacionales. PAA-123-2012.

Each coral colony was identified using aluminum tags with three stamped digits hammered with nails into dead areas in the case of massive corals and with t-raps for branching *Acropora*. Each colony was photographed at the start and at the end of an observation period (April-May 2012 or November 2012-March 2013). Linear rates of tissue mortality were calculated from these pictures, and for each picture a metric scale was used to convert pixels to mm.

Pictures were analyzed using the software GIMP 2.8. For this, we calculated the distance between living tissue and a reference point at each sampling time. When the difference between distances in a time period (April vs May 2012 or November 2012 vs March 2013) was positive, the disease had caused mortality (Figure 2 and 3). When this difference was negative, the disease had arrested and the coral had recovered tissue from the infection (Figure 4). Because lesions may progress in different directions, particularly in corals with massive morphologies, three measures were taken for each colony: (1) the distance from the reference point to the location of living tissues at a perpendicular angle, (2) 2.5 cm to the right and (3) 2.5 cm to the left. CCI progression occurred regardless of the position where measurements were taken (Factor "Position", Table 1 ; therefore, we won't refer to this factor hereafter.

Statistical analyses

The null hypothesis of no difference in the rate of tissue mortality produced by WBD and CCI among coral species was tested using a permutation-based analysis of variance based on Euclidean distances (PERMANOVA, Anderson 2001). For the data analysis we used a two factor design for *Acropora cervicornis*: (1) Location (random) with two levels (Cayo de Agua and Dos Mosquises Sur), (2) Disease (fixed and orthogonal to Location) with two levels (WBD and CCI). For the *Orbicella* species analysis we used a three factor design: (1) Location (random) with two levels (Cayo de Agua and Dos Mosquises Sur), (2) Species (fixed and orthogonal to Location) with two levels (*O. annularis* and *O. faveolata*) and (3) Position (random, nested within Species) with three levels (1, 2 and 3 concerning the three measurements made on each lesion). The analyses were performed with PRIMER + PERMANOVA V. 6.1.

RESULTS

Comparison of CCI and WBD in *Acropora cervicornis*

Tissue mortality of Acropora cervicornis differed significantly between diseases from April to May 2012 but showing opposite trends between sites (Figure 5, Table 1). There colonies with WBD in Cayo de Agua lost their tissues three-fold faster than colonies with CCI (1.5±0.5 mm/day, n=9 versus 0.5±0.1

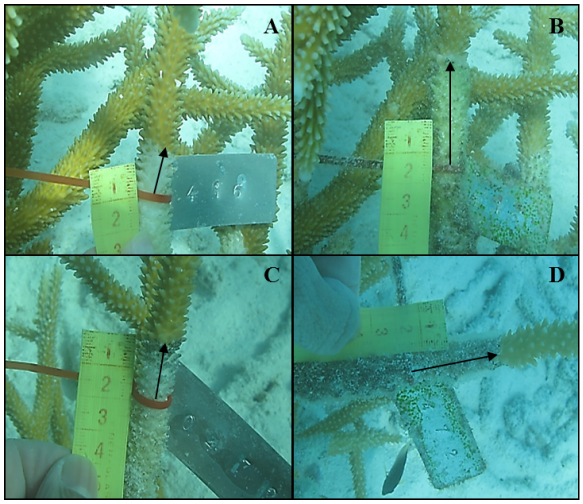

Figure 2. Tissue mortality of *Acropora cervicornis* with CCI on April 2012 (A) and May 2012 (B) and with WBD (C and D, respectively).

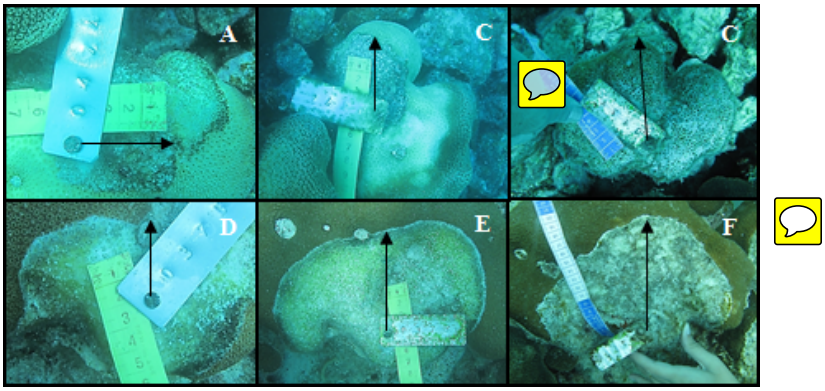

Figure 3. Tissue mortality of *Orbicella annularis* with CCI on April 2012 (A), May 2012 (B) and November 2012 (C) and of *Orbicella faveolata* (D, E and F, respectively).

mm/day, n=8). The opposite occurred in Dos Mosquises Sur, where mortality in corals with CCI was seven-fold faster compared to colonies with WBD (Figure 5, Table 1). Between November 2012 and March 2013 the rate of tissue mortality in colonies with WBD was slightly higher than in colonies with CCI at both sites (Figure 5). Also, lesions of corals located in Cayo de Agua seemed to move faster regardless the pathology (Figure 5). For this period (Nov 2012-March 2013), however, there were no significant differences in mortality rates between diseases or between sites for a given disease (Table 1).

Comparison of CCI mortality between *Orbicella faveolata* and *O. annularis*

Orbicella faveolata was more vulnerable to the presence of CCI as the rate of tissue loss was 0.8 to 3-fold faster than in *O. annularis*. This result was consistent at both reef sites and during the two sampling periods (Figure 6).

Rates of recovery from CCI and WBD lesions

The tissue regeneration rate of CCI lesions was significantly different between species (Figure 7, Table 1). *Acropora cervicornis* regeneration and mortality rate were higher compared to *Orbicella annularis*; while *Orbicella faveolata* regeneration and mortality rates were intermediate among species (Figure 7). For both massive species, the rate of tissue mortality was 15-40 faster than the rate of lesion regeneration, whereas for *A. cervicornis* mortality occurred 15 times faster than regeneration (Figure 7). Finally, the rate of tissue regeneration and mortality in WBD lesions was similar than in CCI lesions in *A. cervicornis* (Figure 7, Table 1).

Table 1. Univariate PERMANOVA based on Euclidean distance for the rate of tissue mortality on *Acropora cervicornis*, *Orbicella faveolata* and *Orbicella annularis* during April-May 2012 and November 2012-March 2013. Bold indicates significant source of variation.

Source of variation	df	MS	F	p-value	Coefficient of variation (%)
Rate of tissue mortality of CCI and WBD in *Acropora cervicornis* at Cayo de Agua and Dos Mosquises Sur					
April-May 2012					
Location	1	0.0016	0.1452	0.692	0.000
Disease	1	0.0005	0.005	1	0.000
Location* Disease	1	0.1085	9.597	**0.009**	51.000
Residual	28	0.0113			49.000
Total	31				
November 2012 - March 2013					
Location	1	1.383	82.323	0.317	3.956
Disease	1	97.725	58.172	0.177	3.127
Location* Disease	1	1.680	664.930	0.782	0.000
Residual	20	25.265			92.916
Total	23				
Rate of tissue mortality of CCI in *O.annularis* and *O. faveolata* at Cayo de Agua and Dos Mosquises Sur					
April-May 2012					
Location	1	21.041	25.387	0.323	1.169
Species	1	215.730	51.397	**0.001**	12.310
Position (Species)	4	3.409	23.614	0.214	0.340
Location*Species	1	0.829	0.530	0.514	0.000
Location*Position (Species)	4	0.144	461.290	0.999	0.000
Residual	84	31.304			86.182
Total	95				
November 2012 - March 2013					
Location	1	1.126	34.718	0.505	3.109
Species	1	18.823	24.332	**0.003**	71.203
Position (Species)	4	0.046	14.618	0.369	1.700
Location*Species	1	0.324	0.999	0.365	0.000
Location*Position (Species)	4	0.318	0.157	0.958	0.000
Residual	51	0.202			23.988
Total	62				
Rates of tissue mortality and regeneration of CCI and WBD in *Acropora cervicornis*, *Orbicella annularis* and *O. faveolata*					
Tissue Mortality					
A. cervicornis, *O. annularis*, *O. faveolata* with CCI					
Species	2	0.012	3.077	**0.046**	7.4
Residual	75	0.004			92.6
Total	77				
A. cervicornis with CCI, WBD					
Disease	1	0.003	0.365	0.545	0.0
Residual	54	0.009			100.0
Total	55				
Tissue Regeneration					
A. cervicornis, *O. annularis*, *O. faveolata* with CCI					
Species	2	0.00003	4.252	**0.027**	29.29
Residual	24	0.00001			70.71
Total	26				
A. cervicornis with CCI, WBD					
Disease	1	0.00001	0.682	0.411	0.0
Residual	9	0.00002			100.0
Total	10				

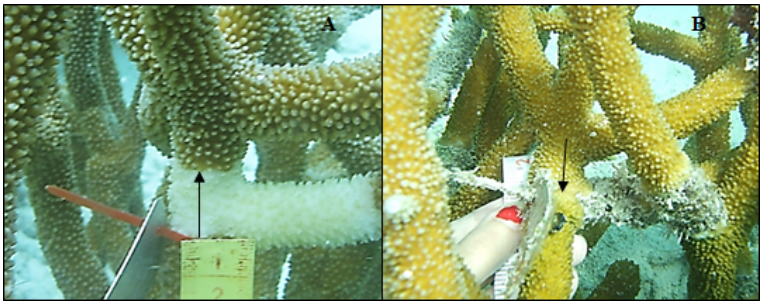

Figure 4. Tissue regeneration of *Acropora cervicornis* with WBD on April 2012 (A) and November 2012 (B).

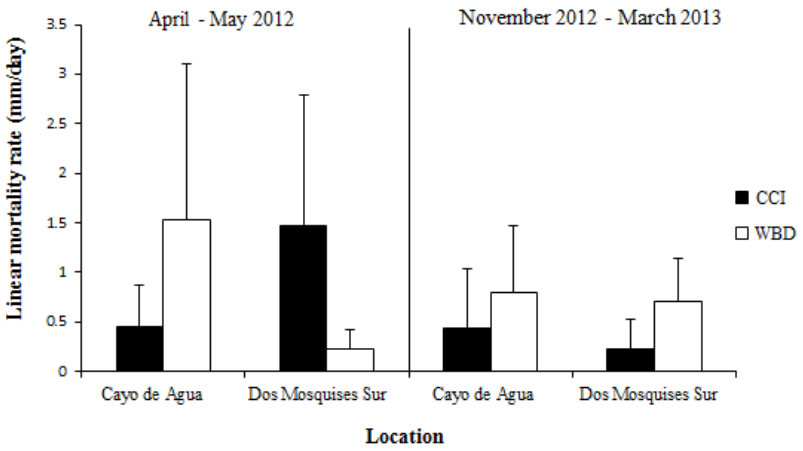

Figure 5. Rate of tissue mortality (mm/day±SD) of CCI and WBD in Acropora cervicornis at Cayo de Agua and Dos Mosquises Sur during April-May 2012 and November 2012 –March 2013.

DISCUSSION

This study provides the first estimation of mortality rates in massive Caribbean coral species with *Halofolliculina* infection (CCI). The results showed that CCI is more virulent in *Orbicella faveolata* than in *O. annularis*; this pattern being consistent between sites and periods of observation. Our results also indicated that CCI and White Band Disease may cause similar rates of tissue mortality in *Acropora cervicornis*, which is of concern as WBD is considered highly virulent.

Among species, tissue mortality in the presence of CCI was at least 2.5-fold faster in branching *Acropora cervicornis* than in the two massive coral species *Orbicella faveolata* and *O. annularis*. *Halofolliculina ciliate* infections show variations in rates of tissue loss, particularly among Caribbean coral species, supporting that taxa vary in their susceptibility as suggested by Page et al. (2015). This idea is supported by observations showing that CCI is more prevalent in species of certain genera (e.g. *Diploria* and *Orbicella*) than others (Cróquer & Weil 2009). This is further supported with results from this study, where tissue mortality by CCI was strikingly different in two species of the genus *Orbicella*: tissue mortality in *Orbicella faveolata* was at least two-fold faster than in *O. annularis*. This could be due to the level of integration of the colony as the boulder type of *O. faveolata* seems more integrated than the columnar type of growth of *O. annularis*. In addition to coral morphology, pathogen virulence and host resistance to diseases also depend on intrinsic mechanisms of defense and a suite of immune responses which may be more or less efficient among coral species (Sutherland, Porter & Torres 2004; Cróquer & Weil 2009).

Caribbean acroporids are highly susceptible to disease epizootics, particularly to WBD which reduced their population number to critical levels on a regional scale (Goreau et al. 1998; Richardson 1998; Richardson & Aronson 2000). Our study supports a high susceptibility of *Acropora cervicornis* to WBD,

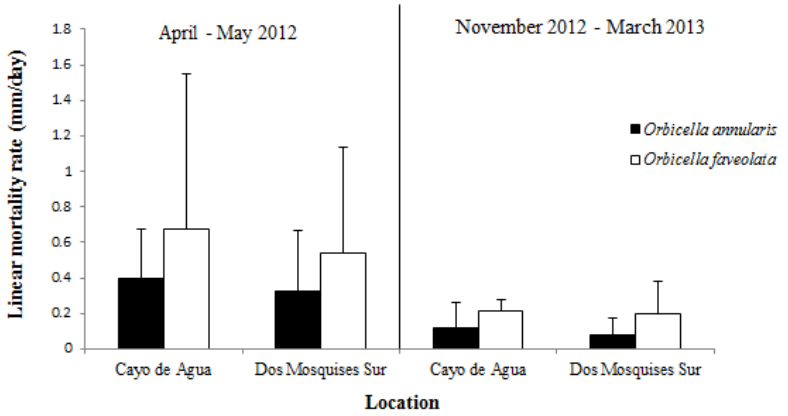

Figure 6. Rate of tissue mortality (mm/day±SD) of CCI in *O.annularis* and *O. faveolata* at Cayo de Agua and Dos Mosquises Sur during April-May 2012 and November 2012-March 2013.

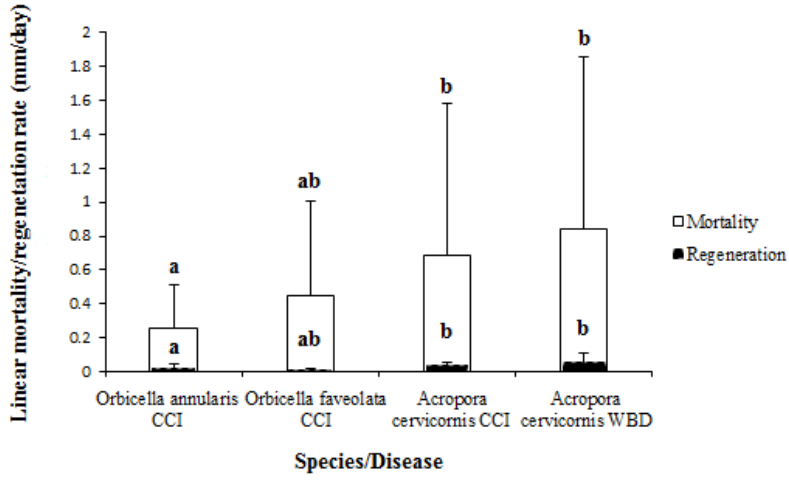

Figure 7. Rates of tissue mortality and regeneration (mm/day±SD) of CCI and WBD in *Acropora cervicornis*, *Orbicella annularis* and *O. faveolata*. The different letters indicates significant differences

and it also showed that this species tends to be equally vulnerable to WBD and CCI. In addition, we observed high variability in tissue mortality of WBD and CCI in *A. cervicornis*, particularly associated to time of the year and site. Such natural variations might result from differential susceptibility among hosts with different genotypes or with seasonal variations. For instance, Vollmer & Palumbi (2007) reported that *A. cervicornis* genotypes can be more or less resistant to WBD in Panamá. In Los Roques, the rate of tissue mortality of WBD in *A. cervicornis* was 1.5-50-fold lower than reported values in Florida (Williams & Miller 2005; Smith & Thomas 2008). These results support that different populations of *A. cervicornis* might be more vulnerable to WBD than others depending on genetic differences and/or local environmental settings.

Environmental changes may enhance virulence in detriment of the host or promote host resistance (Weil & Cróquer, 2009). Sedimentation, eutrophication, pollution and extreme temperatures have been related with at least ten diseases (Sutherland, Porter & Torres 2004). There is evidence that CCI also responds to environmental changes. For example, Rodríguez (2008) reported that rates of disease progression in *Acropora palmata* were higher in summer (August-December) (0.9±0.5 mm/day) than in winter (January-May) (0.4±0.5 mm/day). Further experimental evidence supports that temperature plays an important role in determining the impacts of *Halofolliculina* infections on corals. For instance,

Rodríguez et al. (2009) demonstrated that rates of ciliate colonization on experimentally injured corals maintained at 30°C (90% of colonies) were significantly higher compared with corals maintained at 26°C (70% of colonies). In our study, mortality rates associated to CCI were also higher in the months with higher temperatures April-May 2012 (with a mean temperature of 27.6°C) than in the months of lower temperatures November 2012-March2013 (with a mean temperature of 27.2°C), although the influence of other seasonal variables cannot be discarded.

Disease progression and tissue mortality associated with aggregations of *Halofolliculina* have been documented for four Indo-Pacific and three Caribbean coral species (Page & Willis 2008; Haapkylä et al. 2009; Rodríguez 2008; Rodríguez et al. 2009; Page et al. 2015). In the Pacific, *Acropora muricata* and *A. pulchra* had rates of disease progression of 2±0.3 mm/day and 5 mm/day, respectively (Haapkylä et al. 2009). These mortality rates were three to seven-fold higher than rates obtained in Caribbean species so far: a) this study (*Acropora cervicornis*: 0.7±0.2 mm/day), b) previous studies of *Acropora* (*Acropora palmata*: 0.51±0.20 mm/day and *Acropora cervicornis*: 0.33±0.18 mm/day, Rodríguez 2008), and c) nearly ten-fold higher than that of *Agaricia tenuifolia* (0.26±0.08 mm/day, Rodríguez et al. 2009). These results support that *Halofolliculina* infection represents an important threat to the survivorship of coral reefs in the Caribbean and in the Indo-Pacific.

Our results also showed that CCI produced tissue mortality at a greater rate than Caribbean Yellow Band Disease (CYBD) and Dark Spot Disease (DSD), diseases that had caused significant loss of coral cover (Cróquer & Weil 2009; Page & Willis 2008). In addition, CCI and WBD produced tissue mortality at least ten times faster than tissue regeneration supporting the potential role that CCI could have in the loss of coral cover in the Caribbean. Tissue regeneration and repairing of wounds are complex processes which demand energy in detriment of other physiological processes such as reproduction and growth (Henry & Hart 2005; Rodríguez 2009; Weil, Cróquer & Urreiztieta 2009). Regeneration of coral tissues, where healthy polyps cooperate with the translocation of photosynthetic products, depends on the characteristic of the lesion (i.e., size, form and position) and the level of integration of the colony (Henry & Hart 2005; Page & Willis 2008; Rodríguez et al. 2009). Usually, branching corals repair faster than massive ones because the modules of the former are more integrated than in the later (Henry & Hart 2005). This notion is supported by our study, as *Acropora cervicornis* repaired their wounds faster than the other two massive species.

In conclusion, CCI produced differential mortality between three colony morphologies. In the branching *Acropora cervicornis*, it produced mortality at least 2.5 times faster than in the two massive species of *Orbicella*. Furthermore, in *O. faveolata* with a massive-boulder type of colony, tissue mortality was up to seven-fold faster than in *O. annularis* that has a columnar type of massive growth. We suggest that colony integration may play a role in this difference between CCI progression rates in *Orbicella* species, but other differences in immune response are also possible. Our study shows that tissue mortality by CCI in the two massive *Orbicella* species was consistent between sites and periods of observation whereas in *Acropora cervicornis* tissue mortality varied considerably among colonies, and between sites and diseases along time. For these three reef builders, mortality rates associated with CCI were as high as those caused by other highly virulent diseases such as WBD, WPD-II and BBD, which are capable of producing extensive losses of coral cover at a basin scale. Fastest regeneration rates were up to 15 times slower than mortality rates, further supporting that CCI is a problem of concern for coral species in the Caribbean that prompts further research and amelioration approaches.

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