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2B Coral reefs: is the past the key to the future?

Partial mortality and re-sheeting in fossil corals: a disease proxy

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Abstract. Coral skeletons preserve growth histories over tens to hundreds of years, and therefore growth irregularities in the cross sections of colonies can provide a semi quantitative estimate of disease and other disturbance over significant periods of ecological time. Here I examine the incidence of growth interruptions and re-growth in cross sections of large Pleistocene coral colonies of Curacao. Hundreds of large, mostly in-place, colonies of *Acropora palmata* from three time intervals (216ky, 122ky, 120ky) and three environments (windward reef crest and flat, and leeward reef) were examined. All samples had a low incidence (0-2%) of growth interruption and re-growth as marked by partial horizons of bioerosion and encrustation within the coral colony. No examples of large scale re-sheeting, as is occurring today on skeletons remaining from the White Band Disease die-off of the 1980s, were observed. Large coral domes (>1m in cross section) were similarly undistorted over their life spans suggesting that coral disease and partial mortality were generally rare in the Pleistocene reefs of Curacao. These results provide a pre-human baseline that stands in strong contrast to recent Caribbean reefs that have experienced the large scale die-off of *Acropora*, and the increasing prevalence of coral disease and partial mortality.

Key words: White Band Disease, Fossil, Baseline, Curacao.

Introduction

Worldwide decreases in live coral cover (ex. Gardner et al. 2003; Bruno and Selig 2007) have been attributed to myriad causes including a few, such as coral diseases, that are not the clear direct result of human activities. Increased disease prevalence and the continuing emergence of new coral diseases (ex. Harvell et al. 2004) may be indirectly linked to human activities via overfishing (Sandin et al. 2007; Raymundo et al. 2009) or other effects, but we have little long term understanding of the importance and prevalence of coral disease prior to human impacts.

The most profound example of disease impacting reef structure has been White Band Disease (WBD) in the Caribbean. Beginning in the late 1970s WBD began to kill most *Acropora palmata* and *A. cervicornis*, the two most abundant branching corals in shallow water (Gladfelter 1982). Despite local comebacks (e.g., Zubillaga et al. 2005; MacIntyre and Toscano 2007) WBD is still common in Caribbean *Acropora* (ex. Mayor et al. 2006) and the abundance of Caribbean *Acropora* species remains so far below historic levels that they were designated "threatened species" under the US Endangered Species Act (Federal Register 2006).

In order to determine whether the most recent outbreak of WBD reflects a natural cycle or a unique event, it is necessary to use geological data to reconstruct a pre-human baseline (ex. Pandolfi and Jackson 2007; Lescinsky et al. 2012). Recent work on cores and outcrops suggests that *A. cervicornis* dominated shallow patch reefs of Belize for at least the last 3000 years (Aronson et al. 1998; Aronson and Precht 2001) and Holocene reefs of the Dominican Republic from 6-9,000 years ago (Greer et al. 2009). The record for *A. palmata* is less well known and preliminary reviews of core data suggest at least two millennial long gaps in its record throughout the Caribbean (Hubbard 2009), suggesting possible intervals of disease prevalence in the past.

An alternative approach to investigating disease in the past is to take advantage of the growth history that is preserved within the cross section of coral skeletons. Episodes of partial mortality, due to disease or other factors, will be recorded as growth hiatuses within the coral skeletons. These hiatuses are in turn easily identifiable as horizons of boring and encrustation which have subsequently been overgrown by the recovering coral. Even in cases where the entire coral is killed, conspecifics may resettle on and "re-sheet" the dead skeleton as is now occurring on *A. palmata* skeletons that have remained standing for decades after perishing (Jordan-Dahlgren 1992, Bonito and Grober-Dunsmore 2005). In this study I examine cross sections of Pleistocene coral skeletons from Curacao to examine the incidence of partial mortality and disease in the past.

Methods

The post-mortem signature of *A. palmata* killed by WBD was investigated in still-standing skeletons collected in 2002 at two sites at Buck Island, St. Croix, 20-25 years after they had died (Gladfelter 1982) (Table 1, Fig. 1). Cross sections of 8-10cm pieces were examined for encrustation and bioerosion. Encrustation was recorded as the % of the perimeter of each piece that had a bioaccumulation of encrusters, and its maximum thickness (mm). Surficial bioerosion (and maximum depth) were recorded as the % of the perimeter that had boring in the coral's outer surface (often below the encruster layer).

Fossil *A. palmata* skeletons were observed in the field in the coastal Pleistocene terraces of Curacao (Pandolfi and Jackson 2001, Lescinsky 2008). Large (to 5m) *A. palmata* skeletons are abundant and in place throughout in the Cortalein (216kya) and Hato (123 kya) rocks of the lowest terrace, and on both windward and leeward shores. The Cortalein and Hato units are separated by an unconformity and corals above and below the unconformity were examined. In addition, two samples >2m above the unconformity were included as a distinct sample, representing a period several centuries after the basal Hato samples. A total of 1450 large skeletons (average maximum visible dimension 44cm) were inspected in cross section in outcrop for growth hiatuses as marked by internal horizons of bioerosion and/or encrustation.

Vertical and horizontal cross sections of large (>1m diameter) massive corals from the same outcrops were also examined for partial mortality as marked by internal horizons and altered growth vectors within the colony.

	Modern *A. palmata*	N=
Buck Island Bar (BB)	20+ years, standing dead, 5m deep	40
Buck Island East Shore (BI)	20+ years standing dead, 2m deep	22
	Fossil *A. palmata* (216ka)	
Boca Degu	Windward Reef Crest	110
Boca Degu	Windward Reef Flat	21
Boca Manzanilla	Windward Reef Crest	97
Boca Cortalein	Windward Reef Crest	65
Boca Grande	Windward Reef Crest	84
Boca Labandera	Windward Reef Crest	103
	Fossil *A. palmata* (123ka, < 2m above unconformity)	
Boca Un	Windward Reef Crest	97
Boca Un	WW Reef Flat	86
Boca Degu	WW Crest (< 2m above uncon.)	66
Boca Cortalein	WW Crest (< 2m above uncon.)	109
Boca Grande	WW Crest (< 2m above uncon.)	102
Punta Halvedag	Leeward Reef	109
Porto Mari	Leeward Reef	57
Playa Jeremi	Leeward Reef	108
Playa Lagun	Leeward Reef	110
	Fossil *A. palmata* (<123ka)	
Boca Un	WW Crest (>2m above uncon.)	52
Boca Degu	WW Crest (>2m above uncon.)	74

Table 1: Sampling localities, Buck Island (St. Croix) and Curacao. Detailed locality information in Hubbard et al. 2005, Pandolfi and Jackson 2001.

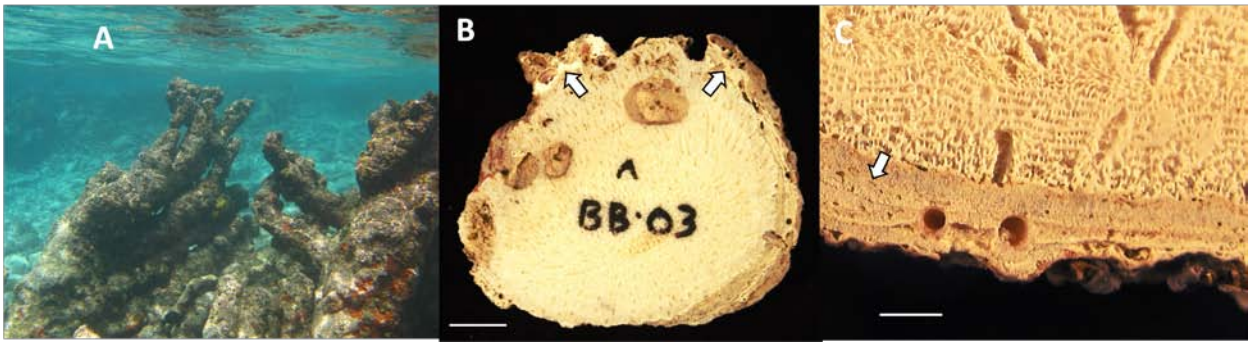

Figure 1. Effects of White Band Disease on *A. palmata*. A. Standing dead *A. palmata* St. John Virgin Island, 2011, approximately 30 years after widespread WBD death. B. Cross section through skeleton collected 2002. Arrows show discontinuous coralline algae crust on top surface. Also visible are larger internal sponge borings and holes of serpulid worms within the crust. Scale bar 1cm. C. Detail of lower surface of *A. palmata* skeleton with *Gypsina* crust (arrow, foraminiferan) and worm tubes. Scale bar 2mm.

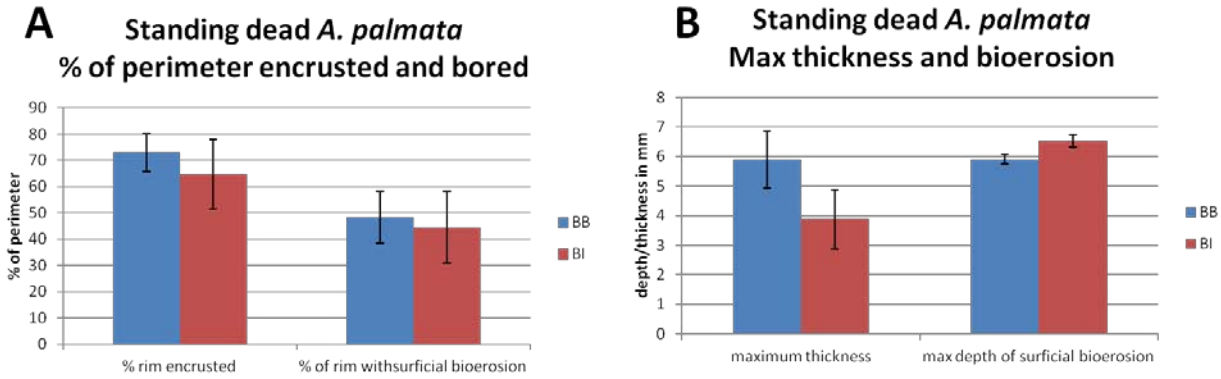

Figure 2. Boring and encrustation on standing dead *A. palmata* approximately 25 years after death due to White Band Disease. A. Widespread algal and foraminiferal crust and bioerosion occur over most of the skeletal surface. B. Maximum thickness of crust was 4-6mm and maximum depth of boring was about 6mm. Error bars are 95% confidence intervals.

Results

Modern "standing dead" *A. palmata* skeletons are intensely encrusted and bored after 20+ years, and probably much earlier. Over 60% of the perimeter of the samples was encrusted, primarily by coralline algae, foraminiferans, serpulids and vermetid gastropods); over 40% of the perimeter was marked by bioerosion horizons in the outer layer of the coral skeleton (Fig. 2) These horizons are easily identifiable in cross-section and would be readily preserved in fossils.

Large Pleistocene *A. palmata* colonies (average visible length in outcrop 44cm) had few growth hiatuses within the cross section of their skeletons (Fig. 3,4). This was true for the 3 paleo reef environments (windward reef crest, windward reef flat, and leeward reef) and three time intervals (216kya, 123kya, < 123kya) that were examined (Fig. 5). Less than 1% of the colonies had growth hiatuses and many of these probably resulted from overgrowth of one branch of the colony by another.

There were no cases of extensive growth hiatus horizons such as those that would have occurred during the re-sheeting or reoccupation of dead skeleton following disease.

Large massive corals similarly were devoid of horizons of encrustation/ bioerosion growth hiatuses. The two examples of growth irregularities that were observed involved *Colpophyllia* and these may be a result of the low density fast growing skeleton of that species.

Discussion

Evidence for Partial Mortality in the Past

One generally overlooked measure of coral health is the incidence of partial mortality in individual coral colonies. To some extent trends in partial mortality are emergent in summative "% live coral cover", but live coral cover is problematic, though not impossible, to measure in past reefs (Lescinsky et al. 2012). Examining partial mortality, as preserved by

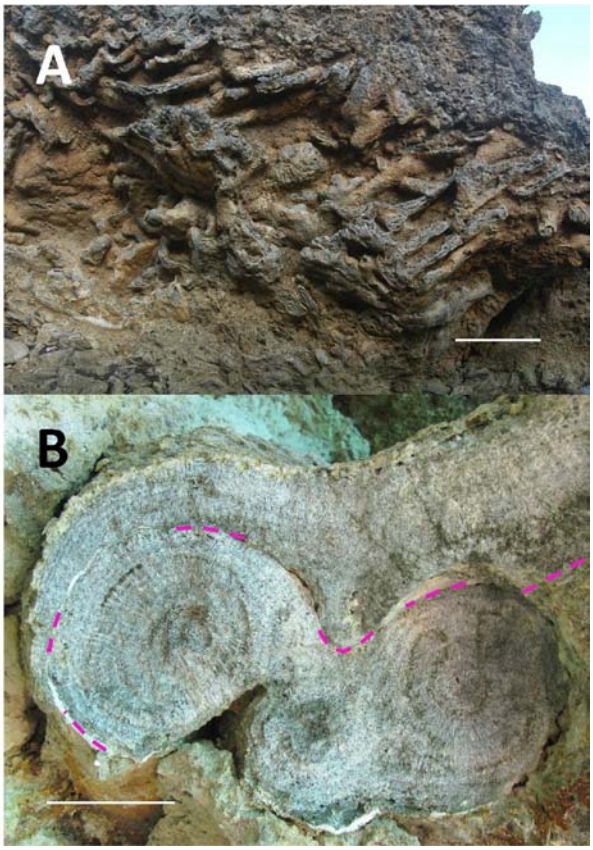

Figure 3. Outcrop view of *A. palmata*, Curacao. A. Large in place colony, scale bar is 1m. B Cross-section illustrating how three adjacent branches were overgrown and joined in the growning colony. Dashed line partially outlines the thin coralline algae growth hiatus. Lack of bioerosion and thinness of encrustation suggest limited post-mortem interval, scale bar is 10cm.

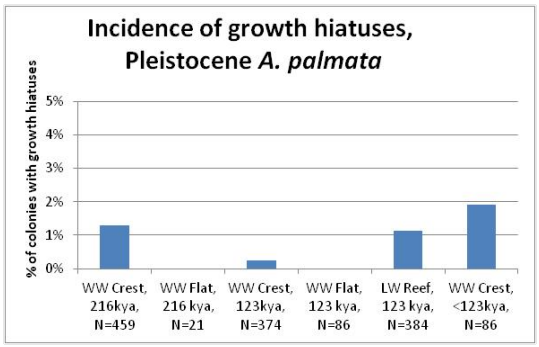

Figure 4. The incidence of boring and encrustation growth horizons within Pleistocene *A. palmata* of Curacao are very low (0-2%) suggesting there was little disease or other forms of partial mortality when these corals were growing.

the growth trajectories and taphonomic horizons within cross sections of coral skeletons provide another means to determine coral and reef health in the past. This study documents that easily identifiable horizons of encrustation and boring form on corals in growth position, and that examples of partial mortality and re-growth can be identified in fossil corals. In some cases, partial mortality probably results from natural growth processes within the colony, such as when adjacent branches are covered and fused together (Fig. 3).

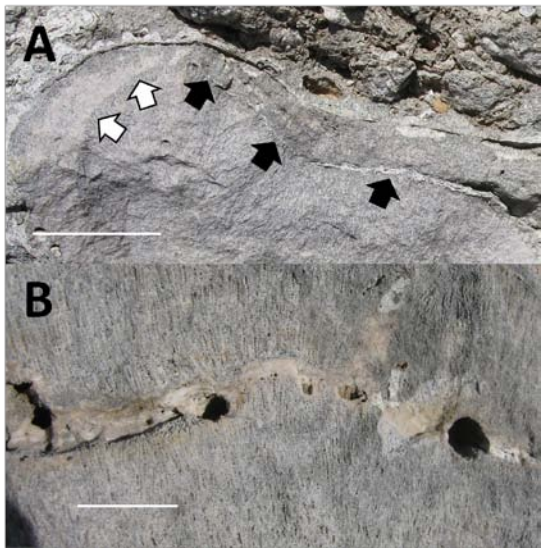

Figure 5: Detail of growth hiatuses in *A. palmata*. A. Coral growth is continuous on left (white arrows), but there is a growth hiatus on right (black arrows). This illustrates re-growth after partial mortality, probably by part of the colony that is now hidden within the outcrop. Scale Bar 10cm. B. Close up of well developed growth hiatus marked by coralline algae and serpulid worm tubes. Scale bar 3cm.

Partial mortality can also result from biological disturbance as disease, as well as physical disturbances such as burial and bleaching. For example, widespread "pancake" like morphologies in massive corals occur in areas of high sedimentation (Hubbard et al. 2008) and represent cycles of partial mortality and regrowth during pulses of burial and exhumation. Partial mortality of massive corals may also occur during thermal bleaching and regeneration over subsequent years will result in a change in corallite trajectory as isolated patches of live polyps expand laterally across the encrusted and bored exterior of the dead colony (ex. see figures in Schuhmacher et al 2005).

	N=	hor	vert	# gr def.
C. natans	6	4	2	2
D. clivosa	2	0	2	0
D. strigosa	13	7	6	0
Montastraea sp.	9	3	6	0
Total domes	30	14	16	2
% growth def.				7%

Table 2. Numbers of massive corals (>1m) examined in outcrop in horizontal and vertical cross sections, Curacao.

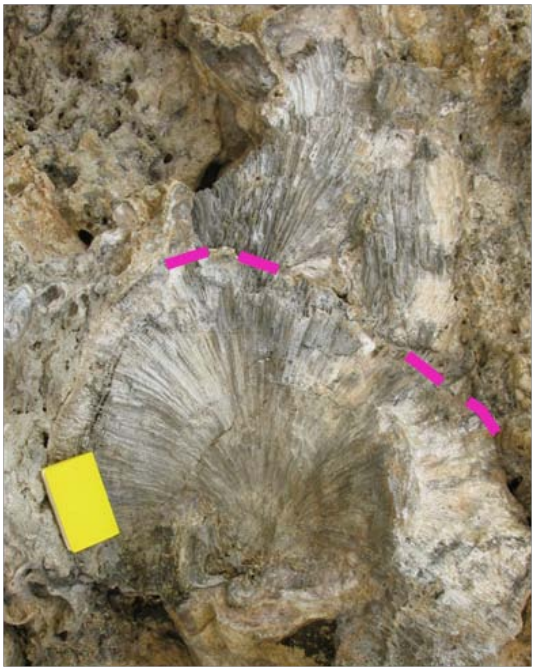

Figure 6. Vertical cross section through large *Colpophyllia*. Dashed line denotes growth hiatus, above which surviving parts of the colony re-grew. Note change in trajectory/lateral spread of corallites above growth horizon. Yellow notebook has maximum length of 19cm.

Proxy for disease?

While examples of partial mortality do not strictly suggest disease, their absence does indicate that disease, bleaching, and other disturbances that don't have obvious sedimentological signatures were rare. Calibrating the proxy for use on modern reefs, however, is difficult because we are unlikely to cross section large numbers of healthy and diseased corals on extant reefs to examine their growth histories. Our anecdotal observations of corals regenerating after bleaching and the re-occupation and re-sheeting of long dead *A. palmata* skeletons suggest that growth hiatuses within coral skeletons are frequent at least on some reefs today.

History of White Band Disease in A. palmata

The sampling of 1450 large in-place *A. palmata* from the Pleistocene of Curacao suggest that for century+ intervals in the past that WBD was not common in the reef zones examined. Similarly large massive corals did not exhibit evidence of disease or growth deformation. This record is in stark contrast to the recent regional die-off *A. palmata* in the Caribbean and the high observed incidence of coral diseases in general (Harvell et al 2004). The pre-human geological baseline presented here, although extremely limited in time coverage, suggests that the incidence of disease on other forms of disturbance (ex. bleaching) is much higher today than it was at these times in the geological past.

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