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The Status of Elkhorn Coral, *Acropora palmata* in Southwest Tobago

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ABSTRACT: Two reefs in southwest Tobago were surveyed during May-June 2004 to evaluate the present state of elkhorn coral, *Acropora palmata*. Seven shallow (1-3m depth) 100m² areas were surveyed at each site, Buccoo Reef and Mount Irvine Reef. *A. palmata* colonies were significantly larger at Buccoo Reef compared with Mount Irvine, both in terms of maximum diameter and colony height. 51% of the sample population were infected with white pox disease (WPD) and colonies with WPD were found to be significantly aggregated within the population. 4.5% of colonies had white band disease (WBD) and 1% showed evidence of bleaching. Underwater visual census techniques gave an estimation of the species abundances of coral-biting parrotfish and damselfish in each survey area. Parrotfish and damselfish species abundances could not be associated with damage caused by corallivorous fish to *A. palmata* colonies. This study could provide baseline data for future monitoring of the health of ecologically important *A. palmata* colonies in Tobago and in the Caribbean.

KEY WORDS: *Acropora palmata* · White band disease · White pox disease · *Coralliophila abbreviata* · Damselfish · Parrotfish · Tobago ·

INTRODUCTION

Over the past 30 years there has been a massive region-wide decline of coral reefs across the Caribbean, with average hard coral cover being reduced by 80% from about 50% to 10% (Gardner et al. 2003). Caribbean coral reefs that were formerly dominated by scleractinian corals and algal turfs have since become overgrown by macroalgae (Edmunds & Carpenter 2001). The mass mortality of *D. antillarum*, and long-term overfishing of herbivorous fishes such as parrotfish (Scaridae) and surgeonfish (Acanthuridae) in many areas of the Caribbean, is implicated in the proliferation of macroalgae and the loss of live coral from many Caribbean reefs (Miller & Hay 1998). The most significant factors as well as overgrowth by competing algae have been storms, an increase in sedimentation and nutrient run-off, overgrowth by sponges and tunicates, animal predation, bleaching and a host of diseases.

Populations of acroporid corals have experienced catastrophic mortality in the Caribbean over the past two decades (Aronson & Precht 1997, Williams et al. 1999, Miller et al. 2002) attributed to their susceptibility to diseases such as white band disease (WBD) and physical disturbance (Bythell & Sheppard 1993).

Elkhorn coral, *Acropora palmata* (Lamarck) (Cnidaria: Scleractinia: Acroporidae) is an arborescent branching coral that forms colonies on shallow, high-energy reef environments throughout the Caribbean (Lirman 2000). While Indo-Pacific coral reefs contain a plethora of fast-growing acroporid species, *A. palmata* and staghorn coral *Acropora cervicornis* are the only significant ones in the Caribbean. This lack of species richness heightens the ecological importance of *A. palmata* as a primary constructor of Caribbean reef accretion and habitat complexity (Miller et al. 2002). The widespread regional decline of both *A. palmata* and *A. cervicornis* throughout

the Caribbean (Aronson & Precht 2001), has led to their designation as Candidate Species for listing as Threatened or Endangered under the US Endangered Species Act (ESA) in 1999 (Becker & Mueller 2001, Miller et al. 2002). *A. palmata* is one of the most important reef-building corals in the region due to fast growth-rates (5-10cm yr⁻¹), high reef-accretion rates and rapid carbon fixation rates (Lirman 2000). *A. palmata* typically formed dense monospecific stands or 'thickets' on shallow exposed reefs (Bythell & Sheppard 1993, Williams et al. 1999) and serve an important ecological function by providing habitat for numerous reef species (Miller et al. 2002). Although colonies are susceptible to breakage during hurricanes and tropical storms, they are generally adapted to high wave energy through modifications in colony morphology, such as branch orientation and thickness (Lirman & Fong 1997). *A. palmata* is a broadcast spawner, releasing eggs into the water column for fertilisation and development (Knowlton 1992) but exhibits low rates of sexual recruitment and the primary mode of propagation is by colony fragmentation (Bruckner & Bruckner 2001).

A. palmata is affected by WBD, white pox disease (WPD) and neoplasia and disease has been implicated in losses of *A. palmata* in the Florida Keys National Marine Sanctuary averaging 87% or greater (Miller et al. 2002, Patterson et al. 2002).

White Band Disease Type I (WBD I) is a presumed bacterial infection specific to the genus *Acropora* (Wapnick et al. 2004) and has affected Caribbean acroporid corals since the late 1970s (Bythell & Sheppard 1993). It is the only disease to have caused a major change in the composition and structure of reefs and has been the most significant source of mortality to acroporid corals over large areas of the Caribbean. The cause of the disease remains elusive (Bythell et al. 2002) and there is no association of WBD outbreaks with proximity to human influences (Aronson & Precht 2001).

White Band Disease Type II (WBD II) was recorded for the first time in 1997 and can closely resemble WBD Type I with time (Ritchie & Smith 1998). Whilst WBD I affects both *A. palmata* and *A. cervicornis*, WBD II has been reported only from the Bahamas, exclusively affecting *A. cervicornis* (Ritchie & Smith 1998). WBD II is always associated with the bacterium *Vibrio charcharia* but attempts to fulfil Koch's postulates have been unsuccessful (Ritchie & Smith 1998).

White pox disease (WPD) also termed serratoses (Patterson et al. 2002) and patchy necrosis (Rodriguez-Martinez et al. 2001) and was first documented in 1996 in Florida and in the eight years since has been observed on reefs throughout the Caribbean (Patterson et al. 2002). White pox exclusively affects *A. palmata* and colonies affected by the disease are characterised by the presence of irregularly shaped white lesions where tissue has disappeared from the skeleton (Patterson et al. 2002). WPD is caused by the common fecal enterobacterium *Serratia marcescens* (Patterson et al. 2002) found in faeces of humans and other animals and in water and soil however the origin and pathogenic mechanisms of the pathogen are unknown (Patterson et al. 2002).

Neoplasia has been described as local abnormalities in calcification pattern and polyp arrangement (Bak 1983) and uncontrolled cell proliferation (Sutherland et al. 2004). Skeletal anomalies such as these pose a serious threat because the primary defence mechanism provided by mucous secretory cells which remove foreign material from the colony surface, are lost (Sutherland et al. 2004).

Bleaching in corals is a response to stress that causes the coral to lose its symbiotic algae (zooxanthellae) that give the coral its normal yellow, brown or green colour. Coral bleaching is caused by a combination of elevated water temperatures and ultra-violet radiation (Green & Bruckner 2000). *A. palmata* is currently living

close to its thermal maxima and increases in sea temperature of at least 1–2 °C are expected by 2100 in response to enhanced atmospheric greenhouse gas concentrations (Harvell et al. 2001). Bleaching events are expected to increase in frequency and intensity and elevated temperature has been shown to accelerate the growth rate and disease activity pathogens (Harvell et al. 2001).

Invertebrate corallivores are widely recognised as having important effects on reef development and reef coral populations in both the Caribbean and Pacific (Miller & Hay 1998). The coral-eating snail *Coralliophila abbreviata* aggregates on branches of *Acropora* spp. and will consume coral tissue from the base of a branch to its tips. With reported mean *A. palmata* tissue consumption of 1.9cm² snail⁻¹ day⁻¹ (Baums et al. 2003), *C. abbreviata* has the potential to have a major impact on its coral prey and could contribute to further loss of *A. palmata*. There may be a link between coral disease and outbreaks of corallivorous snails but a firm connection has not been established (Aronson & Precht 2001). The marine fireworm *Hermodice carunculata* often feeds on elkhorn coral branches. It is a winter reservoir and spring-summer vector for *Vibrio shiloi*, the causative agent of bleaching of the coral *Oculina pataginica* in the Mediterranean Sea (Sussman et al. 2003). It is possible that other corallivores act as vectors for disease.

Corallivorous fish, which cause extensive damage to the surface of coral colonies, are thought to act as vectors for disease by transporting disease pathogens in the mucus surrounding their mouths (Peters 1983). Parrotfish are primarily herbivorous but have historically grazed live coral and can cause significant tissue loss and bioerosion (Bruckner et al. 2000). In the western Atlantic, stoplight parrotfish *Sparisoma viride* and several other large parrotfish species forage using their jaws to scrape and excavate epilithic and endolithic algae from carbonate substrates (Bruckner et al. 2000). ‘Spot biting’ occurs when parrotfish take random individual bites scattered over the surface of coral creating obvious grazing scars that may be in pairs, formed by the upper and lower jaws. ‘Focused biting’ is extensive removal of tissue and skeleton through repeated, overlapping bites. The threespot damselfish *Stegastes planifrons* repeatedly bites at the same location on the living coral surface of *A. palmata* and causes small lesions. Regeneration of such lesions is continually interrupted by subsequent biting. As a result, the calcifying regeneration lip surrounding the lesion does not grow over the lesion but extends vertically, chimney shaped, enclosing the tufts of algae growing on the lesion (Bak 1983). Yellowtail damselfish *Microspathodon chrysurus* may also create small lesions on elkhorn coral. Bite marks are dispersed over the upper branch surface.

The ability to recover from injuries is important in corals since poor regenerative ability can lead to a reduction in colony fitness. Injuries provide sites for the entry of pathogens and bioeroders and space for the settlement of other organisms such as algae, sponges and other corals. These organisms may later compete with the coral for food and space or cause structural damage to the coral skeleton and injuries also reduce the surface area available for feeding, photosynthesis and reproduction (Hall 1997).

Although *A. palmata* appears to be a superior regenerator among Caribbean coral (Hall 1997) both natural and anthropogenic perturbations have led to the decline of *A. palmata* and its ability to recover may be compromised when natural stressors such as storms act synergistically with anthropogenic stressors. It is widely believed that disease is playing an increasingly important role in regulating the population size and demographic characteristics of reefs (Hayes & Goreau 1998). Increases in frequency, intensity and variety of disease occurrences have been described as unprecedented (Goreau et al. 1998) especially for the tropical west Atlantic and Caribbean regions where the majority of disease studies have been carried out (Green & Bruckner 2000). Despite being a

major problem, the etiology and pathogenesis of coral diseases are poorly understood. A modest increase in nutrient concentrations can significantly increase coral mortality and disease severity (Bruno et al. 2003) and changes in the environment caused by human activities may have impaired host resistance and / or have increased pathogen virulence (Hayes & Goreau 1998, Harvell et al. 2001). The habitat structure provided by *A. palmata* can have a major influence on the abundance and diversity of reef fish populations (Clarke 1996, Lirman 1999) so changes in the population size, growth and reproduction of such a major framework builder will have far reaching impacts on the community (Porter et al. 2001).

This project aims to determine the extent to which two reefs in southwest Tobago are currently affected by disease and predation and set up a baseline for future long-term monitoring. Future research will be necessary to see how disease persists and to correlate incidences of disease with sources of pollution. Data may be of use in transplantation research.

METHODS

Study Sites

The island of Tobago is located at the southwestern end of the Caribbean archipelago. The research was carried out on two reefs in southwest Tobago.

Buccoo Reef

Buccoo Reef (declared a marine park in 1973) is located between 11°08'N to 11°12'N latitude and 60°40'W to 60°51'W longitude. It is the largest of Tobago's numerous coral habitats and one of its most ecologically and economically important. It includes five emergent fringing reefs and a large sheltered lagoon, which together cover an area of approximately 7 km². Buccoo reef is a major visitor attraction and the only protected marine area in Trinidad and Tobago. Areas of the reef flats are dominated by *A. palmata* and the substrate of the shallow forereef is mainly composed of rubble and dead remains of *A. palmata* (Laydoo 1985) resulting primarily from storm and hurricane damage in the past combined with anchor damage and reef-walking activities. Chronic eutrophication from inadequately treated sewage pollution is thought to have led to the loss and degradation of hard corals and the increase in macroalgae on Buccoo Reef and may be affecting numerous other fringing reefs around Tobago. The most damaging anthropogenic impact on this reef complex is from land-based sources of nutrient pollution.

Mount Irvine Reef

Mount Irvine Bay has a fringing reef that extends from the shoreline at a depth of 3 metres until reaching an almost vertical forereef slope, which goes down to 17m, known as Mount Irvine Wall. No coral research has been carried out at Mount Irvine Bay and water quality has not been investigated.

A total of 1400m² was surveyed, seven 100m² areas at each site. 100m² survey areas were haphazardly located within the reef at Mount Irvine Reef and within the elkhorn dominated areas of Buccoo Reef.

Colony Measurements

For the purposes of this study, an elkhorn colony was defined as any autonomous coral skeleton with living tissue hence *A. palmata* fragments and crusts were considered to be individual colonies. A colony divided by partial mortality into separate patches of living tissue, but morphologically still one entity, was considered one colony. For *A. palmata* colonies located within each 100m² the height and widest point were measured and the following was estimated: % dead, % diseased and type of disease, % damselfish chimneys / bites, % snail damage and number of snails, % parrotfish bites, % fireworm damage and number of fireworms.

Lesions caused by disease and predation were distinguished using criteria defined by A. Bruckner in a field guide to Western Atlantic Coral Diseases and other causes of coral mortality (<http://www.unep-wcmc.org/marine/coraldis/cd/intro.htm>) (Table 1).

Table 1. Disease & Predation affecting *Acropora* spp.

Fish Community Structure

The abundances of parrotfish and damselfish were estimated by point count underwater visual census to record all fish entering the 100m² survey area within a 10 minute time period.

Disease Progression

Six diseased colonies (3 WPD, 2 neoplasia, 1 WBD) at Mount Irvine Bay were tagged and lesions sizes were drawn onto sheets of acetate. The growth of lesions was monitored over a five-day period.

DATA ANALYSIS

Colony Measurements

The maximum diameters of colonies were assigned to one of six size classes: 0-20, 21-40, 41-60, 61-80, 81-100 and 101-250cm and the size-frequency distribution between Mount Irvine and Buccoo Reef was compared using the chi-squared (χ^2) analysis. Colony heights were assigned to one of five size classes: 0-10, 11-20, 21-30, 31-40 and 41-60cm and were analysed using the chi-squared test.

Incidence of Disease and Predation

Percent damage caused by different syndromes for each colony was used to calculate the mean occurrence of each syndrome per colony at each survey area.

Aggregation of Disease

The frequency of disease per survey area was standardised to the total number of colonies sampled (n = 200) to give a proportion of the total population affected. The variance / mean ratio was used to test whether the

