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Primary Research Paper

Spatial epidemiology of Caribbean yellow band syndrome in *Montastrea* spp. coral in the eastern Yucatan, Mexico

Janet E. Foley[1,2,*], Susanne H. Sokolow[2], Evan Girvetz[3], Colin W. Foley[4] & Patrick Foley[5]
[1]*School of Veterinary Medicine, Department of Medicine and Epidemiology, University of California, Davis, CA, 95616, USA*
[2]*School of Veterinary Medicine, Center for Vectorborne Disease, University of California, Davis, CA, 95616, USA*
[3]*Department of Environmental Design, University of California, Davis, CA, 95616, USA*
[4]*Davis, 95616, California,*
[5]*Department of Biological Sciences, California State University, Sacramento, CA, 95819, USA*
*(*Author for correspondence: Tel.: 530-752-9387; Fax 530-752-7701; E-mail: jefoley@ucdavis.edu)*

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Abstract

Caribbean yellow band syndrome (YBS) is a poorly understood, progressively fatal disease primarily affecting *Montastraea* spp. coral. This disease has exhibited rapid spread throughout the entire Caribbean over the last few decades. In this study, geographical information systems (GIS) and spatial statistics were used to analyze the distribution of YBS in Akumal Bay, Mexico, and host and environmental risk factors for YBS were evaluated epidemiologically. In this Bay, there are hundreds of colonies of *Montastraea annularis* from 1 m depths inside the fringing reefs to reef crests and beyond. Of 63 corals that were evaluated, the overall prevalence of YBS in Akumal Bay was 28.6%, with 35.7% in large colonies, 23.8% in medium-sized colonies, and 23.8% in small colonies, where small colonies were <200 cm diameter, medium-sized were 200–500 cm, and large were >500 cm. Lesions covered 3.8% (±1.3 s.e.) of the surface of colonies assessed, compared with a mean percentage of dead colony cover of 54.4% (±4.2 s.e.). Analysis for spatial clustering documented that *M. annularis* colonies (well and sick) were highly spatially clustered, compared to expected complete spatial randomness. However, compared with all *M. annularis* corals, colonies with YBS tended to be less spatially clustered (i.e. within the overall clustered spatial distribution of *M. annularis* colonies, YBS-affected colonies' distribution was more regular). These findings are consistent with several hypotheses for the etiology of YBS, including near-shore pathogens or toxins either directly inducing disease or indirectly leading to disease by increasing host susceptibility. Ongoing investigations into the management and cause of YBS can use this information to develop management strategies and more efficiently target future sampling.

Introduction

Caribbean coral reefs are experiencing devastating morbidity and mortality. Numerous environmental factors have been suggested to contribute, including infectious diseases, climatic factors, direct human damage, overharvesting, poisoning, eutrophication (commonly from human and terrestrial animal sewage), and global warming (Stone et al., 1999). Distinct coral 'diseases' include white plague, white plague type II, black band disease (BBD), Caribbean yellow band syndrome

(YBS), and others. Very little is known about the causes of coral disease, with primary pathogens identified only in five disease syndromes (BBD, aspergillosis, white pox, white plague type II, and bacterial bleaching), and fulfillment of Koch's postulates for disease causation only in four (Carlton & Richardson, 1995; Kushmaro et al., 1996; Richardson et al., 1997; Geiser et al., 1998; Richardson et al., 1998; Banin et al., 2000; Patterson et al., 2002; Denner et al., 2003). In the majority of coral diseases, mechanisms of disease and roles of pathogens are completely unknown, and prospects for intervention elusive.

Diseases emerge due to synergistic interactions of pathogen, host, and environmental factors. Many 'infectious' diseases are attributable not to a single highly virulent primary pathogen, but rather to infection with opportunistic pathogens which take advantage of hosts with impaired defenses. It has been hypothesized that susceptibility to opportunistic pathogens is significantly increased in coral that are debilitated from climatic and physical stresses (Richardson, 1998). Thus understanding environmental and host factors in disease may be key to better understanding syndromes of coral disease and decline.

The Mexican Yucatan area of the Caribbean Sea is widely appreciated for its diverse, healthy coral reef ecosystems and crystal-clear water, yet natural forces and human development and recreation have negatively impacted local reefs. A die-off of a critical herbivore, *Diadema antillarum*, occurred in the 1980s (Lessios 1988) which contributed to severe algal overgrowth observed throughout the Caribbean. Increases in dissolved water nitrogen and other pollutants add additional stress on coral and may also contribute to the observed increases in coral mortality and algal overgrowth in recent times. Water quality testing performed by the Centro Ecológico Akumal demonstrated that fecal contamination within Akumal Bay in June through September, 2002, ranged from 2 to 128 fecal coliforms (*E. coli*)/ 100 ml water. For reference, both the US and Mexican standards for drinking water allow no more than 2 col/100 ml and for bathing waters or waters in contact with human skin, levels must be less than 200 col/100 ml (Brown & Shaw, 2002). Other factors which might have increased coral stress and disease susceptibility include global warming and natural disasters such as hurricanes Gilbert in 1988 and Roxanne in 1995, and human impacts such as overharvesting and physical damage by SCUBA divers and boat anchors.

One of the coral diseases which is particularly poorly understood is YBS. YBS is most commonly found on large scleractinian coral such as *Montastraea annularis, M. faveolata*, or *M. cavernosa* (Goreau et al., 1998; Cervino et al., 2001) and presents initially as a yellow-discolored irregularly-shaped patch on the surface which progresses in diameter while the inner portion of the lesion dies and then fills with sediment and algae. This disease has exhibited a rapid spread throughout the entire Caribbean since 1997 (Weil et al., 2000) and is one of the most prevalent of coral diseases, affecting in some sites 18–90% of *Montastraea* spp. corals (Weil et al., 2000; Cervino et al., 2001). It is not known what the cause of the syndrome is, but possible etiologies could include an infectious agent or agents (Cervino et al., 2004), a response to a toxin or other environment problem, or a combination of multiple factors. Patterns of disease with respect to environmental parameters might be useful for inferring causation. Therefore, the objectives of this study were to understand the distribution of YBS using geographical information systems (GIS) and spatial statistics. The research was intended to expand opportunities for understanding coral disease by introducing a novel methodology by which to study an important emerging cause of reef-building coral mortality.

Materials and methods

Study site

Akumal Bay, Quintana Roo, is located on the eastern Yucatan Peninsula in the Mexican Caribbean, in the Caletas subregion at UTM coordinates 467000 easting and 2255000 northing in Zone 16, about 104 km south of Cancun. The small town of Akumal has been a low-key resort since the 1960s and has experienced significant growth, particularly since the 1980s. The bay extends approximately 1 km × 300 m on a marine-originated flat platform formed mostly of Eocene–Holocene calcareous rocks, and consists of a semicircular lagoon bounded by a sandy shore and

a fringing coral reef. Most groundwater drainage through the Karstic limestone is subterranean. Coral reefs of Akumal have been heavily impacted by tropical storms, heavy fishing, coliform-rich groundwater which seeps out of limestone substrate throughout the coastal region, and possibly recreational physical damage, resulting in marked decline of live coral cover and algal overgrowth over the last several decades. Sampling associated with this study was performed in the lagoon, along the back reef, and on the reef crest to depths of 2.0 m in the summer of 2002.

Caribbean yellow band syndrome case definition

Any *M. annularis* coral with a demarcated pale or yellow area on the surface greater than 1 cm was considered suspect for YBS. If the lesion was bright yellow or contained a necrotic or sediment-filled central region, the case was considered definitive.

Sampling and study design

All *M. annularis* colonies within Akumal Bay were mapped along four haphazardly chosen 6×60 m transects running from the shore to the reef crest, to a maximum depth of 2 m. Snorkellers swam underwater and visually identified the center of all colonies and communicated with a partner on the surface who acquired the position in Universal Transverse Mercator (UTM) coordinates by a global positioning system (GPS). All colony positions were obtained by GPS five times and the mean and variance evaluated, in order to detect error in location. Variance was very low with 95% of the repeated acquisitions having no variability among repeated position estimates (data not shown). In addition, when colonies were re-located using GPS on repeated visits, colonies with the same depth and size characteristics were exactly where indicated with previous GPS points, supporting the reliability of GPS for positioning.

Sampling to evaluate risk factors for YBS in *M. annularis* coral was performed along the four transects as described above. The colonies along the transects were examined visually for signs of YBS and the case status recorded. Severity of lesions on each coral boulder was graded on a scale, where 0 indicated no visible lesion, 1 + indicated a 1–25% effacement of the corallum with disease, 2 + indicated 25–50% effacement, and 3 + indicated > 50% effacement. Subcolonies were defined as distinct lobes of aggregated clonal corallites that form a recognizable unit of a main corallum ('boulder'). Surface bleaching, algal cover on colony, and coral death were recorded as percentages per boulder. Colonies and subcolonies were measured using hand-held cloth measuring tape including height and maximum diameter. The temperature of water and depth were determined *in situ* using a handheld dive computer.

Data were maintained in Excel 2002 (Microsoft, Redmond, WA) and analyzed in 'R' (The R-Development Core Team, http://www.r-project.org). Individual locations were displayed as XY coordinates using a geographic information system (ArcMap Version 8.0, ESRI, Redlands, CA). Second-order analysis consisted of assaying for deviation from complete spatial randomness (Diggle, 1983), that is, the presence of clusters of well and diseased coral, by calculating the 'inhomogeneous K' function (Baddeley et al., 2000), a generalization of the Ripley *K*-function (Ripley, 1976) to allow for inhomogeneous point processes, using the function 'Kinhom' in the 'R' library 'Spatstat'. The function used by the 'R' program for 'Kinhom' is simply $Kinhom\ (r) = \pi^* r^2$, with $1 < r < 100$ (m) for our data. Possible bias caused by unequal sample collection was assessed by comparing results of the 'Kinhom' function of affected colonies with that of unaffected colonies by examining the function $D(r)$, which compares *K*-function results from the two populations (Diggle & Chetwynd, 1991). If $D(r) > 0$, then affected coral were more clustered than would be expected as the result of random sample collection of the population; that is, there was spatial aggregation not accounted for by environmental inhomogeneity or the underlying clustering of coral in general. If $D(r) < 0$, then the affected coral were less clustered than would be expected due to the underlying clustering of coral in general.

Results

Environmental parameters, presence of susceptible coral hosts, and presence of YBS were assessed in Akumal Bay in the Yucatan Peninsula in the

summer of 2002. Sixty three subcolonies from 19 *M. annularis* boulders (with 1–8 subcolonies per boulder) were examined. Boulders ranged in size from 34 to 200 cm (mean 103.5 cm) in diameter, and 29–80 cm (mean 53.4 cm) in height, while mean water temperatures ranged from 27 to 31 °C (mean 293 °C) (Table 1).

Many *M. annularis* colonies showed evidence of YBS to varying degrees, with extensive coral death and algal replacement yet little to no active bleaching at the times of sampling. Along the transects, the overall prevalence of YBS was 28.6% (18.4–41.5% 95% CI) among *M. annularis* colonies. The prevalence was 35.7% (14.0–64.4% 95% CI) in large colonies, 23.8% (9.1–47.5% 95% CI) in medium-sized colonies, and 23.8% (9.1–47.5% 95% CI) in small colonies, where small colonies were <200 cm diameter, medium-sized were 200–500 cm, and large were >500 cm. Of all subcolonies assessed, 12.1% (9.2–15.6% 95% CI) contained lesions of YBS. Lesions covered 3.8% (± 1.3 s.e.) of the surface of colonies assessed, compared with a mean percentage of dead colony cover of 54.4% (± 4.2 s.e.). No significant relationships were detected between disease and host or environmental factors.

Table 1. Average, range, and standard deviation for environmental and host parameters assessted

	Average	Range	Standard deviation
Temperature (°C)	29.73	27–31	0.933
Depth (m)	1.32	0.6–2.7	0.447
Colony max diameter (cm)	103.57	34–200	52.380
Colony max height (cm)	53.39	29–80	17.523

The distribution of healthy and YBS-affected coral within the sampling transects is shown in Figure 1. Analysis for spatial clustering documented that *M. annularis* subcolonies (well and sick) were highly spatially clustered, compared to expected complete spatial randomness. However, compared with all well *M. annularis* coral subcolonies, subcolonies with YBS tended to be less spatially clustered at a distance of analysis between 10 and 30 m (i.e. within the overall clustered spatial distribution of *M. annularis* hosts, YBS-affected distribution was less clustered than expected, see Figure 2). At a scale below 10 m and above 30 m no difference in well and YBS-affected subcolonies was detected.

Figure 1. Spatial plot of YBS-affected and healthy *Montastraea* spp. coral along experimental transects in Akumal Bay, Mexico.

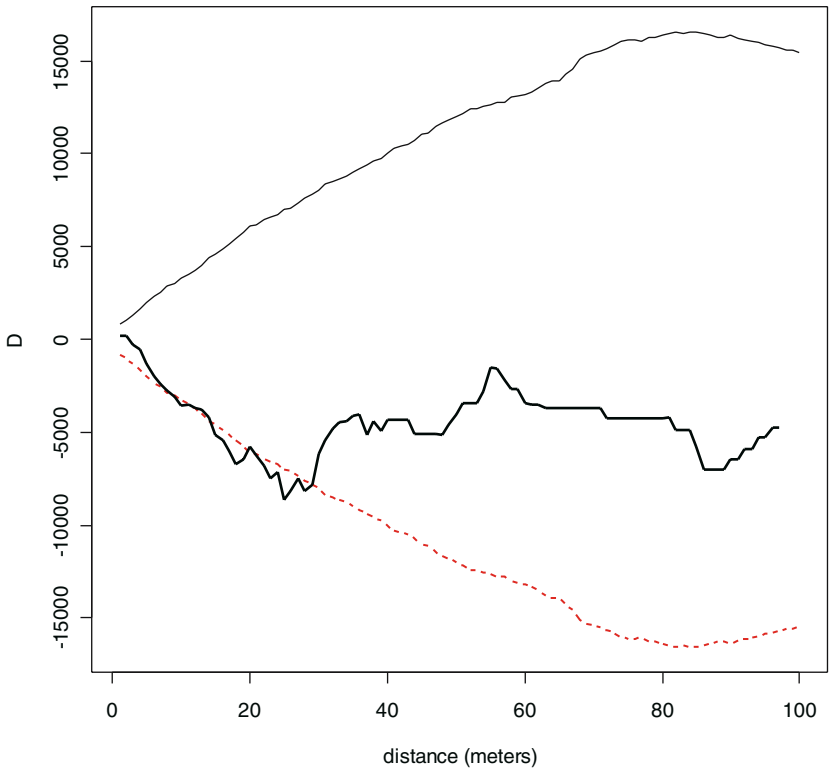

Figure 2. Estimate of the *D* statistic at various distances for *Montastraea* spp. coral with and without gross signs of YBS in Akumal Bay, Mexico. Solid thick line: *D* statistic for sick minus healthy coral; solid thin line: upper boundary for standard error of *D*; dashed line: lower boundary for standard error of *D*.

Discussion

Caribbean yellow band syndrome is a potentially devastating emerging problem in Caribbean coral that, to date, has not been definitively associated with any specific infectious pathogen. In this study, we document spatial distribution and contributing environmental and host factors which could represent opportunities for further surveillance, investigation, and potential management of this problem.

Caribbean yellow band syndrome has emerged only in the last few decades. In Akumal Bay, there was approximately 30% prevalence, including greater prevalence in larger corals as has been reported previously (Santavy et al., 1999; Cervino et al., 2001). As a comparison, a study in San Blas Panama documented 5–10% prevalence in waters deeper than those evaluated in Akumal (>4.5 m) (Santavy et al., 1999). The prevalence exceeded 90% in Bonaire, 55% in Turks and Caicos, and 18% in Grenada (Cervino et al., 2001). Unfortunately, the moderately high prevalence in Akumal was coupled with approximately 50% loss of live coral cover in *Montastraea* spp. Good quality historical data for Akumal coral are not available, so it is not known how rapidly YBS has emerged in this site and to what extent YBS is responsible for the extensive *Montastraea* spp. surface death observed throughout the study area. The disease has been reported to progress over a period of years at rates less than 1 cm(month until, in some cases, the entire colony is killed (Cervino et al., 2001). The data in this study were suggestive (although not statistically significant) of an increased risk in larger colonies. This could represent some underlying host developmental factor or could represent the fact that the higher surface area of large colonies provides a statistically greater target for disease.

Many environmental stressors, disease hosts, pathogens and vectors are nonrandomly distributed

in space and it was hypothesized that evidence of clustering in YBS could help implicate spatially variable risk factors and help define the appropriate spatial scale for further ecological, epidemiological, and microbiological studies of YBS. This hypothesis is supported by recently reported findings that nutrient enrichment was associated with increased tissue loss and advancement of the front of disease in *Montastraea* spp. coral with YBS in a field experiment (Bruno et al., 2003). The spatial components of host-disease interactions for coral are especially critical, in large part because the hosts are sessile and reflect an increasing emphasis in ecology generally to account explicitly for space in ecological processes (Kareiva & Tilman, 1997). Spatial investigation of coral disease has been performed previously, primarily to evaluate for patterns consistent with pathogen spread by direct contact, vectors (e.g. fish), or currents (e.g. (Nagelkerken et al., 1997)). Mapping of *Montastraea* spp. in Akumal Bay revealed underlying clustering of the colonies and subcolonies in general. Superimposed on this highly clustered distribution was *less* clustering (i.e. more regular) in the diseased coral than expected by chance. Competition and interference are considered important forces promoting regularity in distribution; the pattern of YBS on *M. annularis* colonies, characterized by less clustering than expected, implies that close proximity to other *M. annularis* colonies may offer protection from disease, possibly in the form of barriers to disease agents or toxins, or genetic resistance which is shared by clonal subsets of coral boulders which co-occur in space. The data do *not* support a model of simple contagion, or direct spread, of some disease-causing agent in YBS, although this possibility could not be ruled out completely.

Issues of spatial scale are important in ecology and epidemiology. This study examines the distribution of YBS on coral hosts at the local scale, or the scale of individual and subindividual colonies in one bay. Although there is limited data regarding the YBS distribution and the *Montastrea* spp. host distribution on a Caribbean-wide scale, it is likely that host and disease are aggregated at some larger scale than that examined in this study. It is not uncommon for patterns in ecology to change and even reverse when observed at multiple scales. For example, ecological studies of productivity–diversity relationships in ponds have documented that the productivity–diversity relationship is consistently 'hump shaped' at local (pond) scales and linear at regional (watershed) scales (Chase & Leibold, 2002). In our study, a pattern of lower clustering (more dispersal) among YBS affected corals than expected was observed when spatial analysis was restricted to between 10 and 30 m, but at scales below 10 m and above 30 m, the pattern changed such that clustering among well and affected *M. annularis* corals was not substantially different. The phenomenon that patterns can change with scale of observation only enhances the importance of examining the spatial patterns of ecological systems and disease systems at various scales. With the advent and availability of remote sensing and GIS, large scale studies of coral and coral disease distribution may be possible. Future studies investigating the spatial pattern of Caribbean yellow band and other coral diseases at local, regional, and global spatial scales are warranted.

The specific etiology of YBS is not known, but histological examination of affected tissue revealed degenerative changes and gastric cavity crystalline inclusions (Santavy & Peters, 1997) as well as reduced zooxanthellae numbers and zooxanthellae which were vacuolated with depleted organelles and pigment content (Cervino et al., 2001). Cervino et al. (2004) suggest that a consortium of four *Vibrio* spp. bacteria may be involved in YBS pathogenesis. Because the exact etiology of YBS remains unknown, it is important to continue to consider possible pathogen (or toxin), host, and environmental factors that may be contributory. The analysis in this study did not directly suggest an association of disease with environmental parameters, although the data did show temperatures high enough to induce bleaching and increased disease severity in some reports (Davies et al., 1997; Cervino et al., 2004). The lack of an association between YBS and the measured environmental and host parameters can be explained either by a real lack of an association or due to a statistical inability to detect differences secondary to a small sample size. Previous studies have suggested an effect in which coral hosts are increasingly susceptible to disease due to risk factors such as nutrient enrichment (Bruno et al., 2003) and larger colony size (Nugues, 2002). Environmental stressors have been extensively documented to

induce coral bleaching and to compromise innate disease resistance in coral (Hoegh-Guldberg & Smith, 1989; Lesser, 1996; Brown, 1997). An excellent example of environment–host-pathogen interactions promoting coral disease is mortality in *Platigyra* sp. coral (Mitchell & Chet, 1975) in which overgrowth of *Desulfvibrio* sp. and *Beggiatoa* sp. bacteria was observed during periods of coral mucus overproduction due to experimental challenge with crude oil. In this disease model, treatment of the water with broad spectrum antibiotics allowed the coral to survive the crude oil challenge. Further investigation of the association of environmental and host risk factors with YBS, as well as the spatial extent, mechanisms of spread, and response of the coral during this disease process, are crucial to understanding the disease and to developing management strategies for protecting Caribbean coral.

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