Session 13

Diseases on the reef: presence, persistence and responses

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Demographics of the Caribbean brown tube sponge *Agelas tubulata* on Conch Reef, Florida Keys, and a description of *Agelas* Wasting Syndrome (AWS)

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Abstract

The brown tube sponge, *Agelas tubulata* (cf. *conifera*) is an abundant and long-lived sponge on Caribbean reefs. Populations of *A. tubulata* were monitored inside 8 circular plots (16 m diameter) on Conch Reef, Florida Keys, USA, from 2010-2015. Within each plot, we recorded and photographed every individual of *A. tubulata*, and monitored for sponge recruitment and mortality. Over the 6-year study period, the sponge population remained fairly stable, only showing an increase between 2010 and 2011, and recruitment and mortality were low. However, monitoring revealed the presence of a previously undescribed pathogenic condition, *Agelas* Wasting Syndrome (AWS), which increased in the sponge population from 7% in 2010 to 35% in 2015. AWS is characterized by sponge tissue necrosis that results in an area of exposed spicule-imbedded organic fibers. Once this portion of the skeleton is shed, a light brown scar is left behind that either slowly returns to normal coloration or continues to erode in the same manner. Tissue loss from AWS was previously interpreted as evidence of predation by fishes or turtles. AWS on *A. tubulata* appears to progress slowly, is usually localized on the sponge surface, and some sponges appear to recover. This study highlights the importance of long-term monitoring as a tool for understanding sponge ecology.

Key words: sponge ecology, pathogenesis, disease, coral reefs

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Introduction

Sponges are important components of Caribbean coral reef ecosystems, where they can be the dominant benthic organisms (Loh and Pawlik 2014). Particularly on reefs that have experienced extensive losses in coral cover, sponges provide habitat complexity and structure (Duffy 1992; Henkel and Pawlik 2005;

McMurray et al. 2008). Sponges serve as a food source for vertebrate predators on the reef, including angelfish, parrotfish, and sea turtles (Meylan 1988; Dunlap and Pawlik 1996; Pawlik 2011), as well as some invertebrates (Pawlik 1983; Birenheide et al. 1993; Wulff 1995; Pawlik and Deignan 2015). Sponges may also play an important role in carbon and nutrient cycling on reefs (de Goeji et al. 2013; McMurray et al. 2016; Pawlik et al. 2016), an environment that is generally considered to be nutrient poor. However, the pumping activity of sponges may also increase exposure of the sponge to environmental pathogens (Hill 2004; Maldonado et al. 2010). Because sponges have historically been understudied, many aspects of their biology and ecology remain unknown (Diaz and Rutzler 2001).

Long-term monitoring of sessile organisms is an important tool in benthic ecology. Here we report the results of a 6-year demographic study of the Caribbean brown tube sponge *Agelas tubulata* (cf. *A. conifera*) from Conch Reef in the Florida Keys, USA. This monitoring program was conducted using the same methods as for the Caribbean barrel sponge *Xestospongia muta* from the same location (McMurray et al. 2010, 2015). *Agelas tubulata* is abundant on Caribbean reefs (Loh and Pawlik 2014) and is generally considered to be a slow-growing sponge, because of its investment in the production of brominated pyrrole alkaloids for chemical defense from predatory fishes (Pawlik et al. 1995, Assmann et al. 2000, Loh and Pawlik 2014). While *A. tubulata* is thought to be a long-lived sponge on Caribbean reefs, no previous demographic studies of this species have been conducted.

While monitoring the brown tube sponge, we observed individuals affected by a syndrome we are calling *Agelas* Wasting Syndrome (AWS). Prior to this monitoring, the tissue loss observed for this species was thought to result from predation; however, consecutive annual photographs and careful field observations provided strong evidence for reporting these lesions as a new sponge syndrome. To date, there have been four previously named diseases associated with Caribbean reef sponges, and AWS is the fifth (Table 1).

Methods

Eight 16m-diameter circular plots were established on Conch Reef (24º56’59”N; 80º27’13”W) off Key Largo, Florida, to monitor mortality, recruitment, and growth of sponges, including *A. tubulata*. Three plots were located at each depth of 15m and 20m, and two plots were located at 30m. Monitoring took

Table 1 Named sponge diseases reported for Caribbean sponges.

Disease Name	Species affected	Reference
Mangrove sponge disease (MSD)	*Geodia papyracea*	Rützler (1988)
Aplysina red band syndrome (ARBS)	*Aplysina cauliformis*	Olson et al. (2006)
Sponge orange band (SOB)	*Xestospongia muta*	Cowart et al. (2006)
Sponge white patch (SWP)	*Amphimedon compressa*	Angermeier et al. (2012)
Agelas wasting syndrome (AWS)	*Agelas tubulata*	Described here

place annually from 2010-2015 over a 2-week period, with the exception of 2012. Due to time limitations in the field, no 30m plots were monitored in 2013. Within each plot, every individual of *A. tubulata* was identified with a distinct alphanumeric tag, and a digital photograph was taken of each sponge with a scale bar from above and in profile, with extra photographs taken as needed to ensure that every sponge tube could be seen in the photographs. A repeated measures ANOVA was used to assess differences in population density among years and post hoc tests of pairwise comparisons were used to determine the years with any significant change in density. The assumption of sphericity was tested with Mauchly's test. Year 2013 was omitted from this analysis, because no plots at 30m were monitored that year.

The volume of sponges were calculated using detailed field measurements of 27 individuals of *A. tubulata* from Conch Reef, as well as an additional 12 individuals from reefs off of Carrie Bow Cay, Belize. Each sponge tube was modeled as a cylinder with subtraction of a cylindrical spongocoel (atrium) volume. A regression analysis between the log of the total tube length of the sponge and the log of the sponge volume was performed ($\log Y = 1.304 \log X + 0.951$, $R^2 = 0.781$). Using the established relationship, the volume of each sponge from the monitoring photographs was calculated using digital image analysis to measure the total tube length of each sponge (UTHSCA Image Tool).

Sponges were separated into size classes I-V based on the twentieth percentiles of the total size range in which the sponges appear on the reef. Percentiles were calculated annually and averaged over the five years. The size classes were defined as follows: size class I: 0-147 cm^3; size class II: >147-370 cm^3; size class III: >370-730 cm^3; size class IV: >730-1561 cm^3; and size class V: >1561 cm^3.

In addition to sponge volume, the digital photographs were used to characterize the progression of AWS. For each photograph, the percentage of sponge tissue affected by AWS was characterized using the following scale: 0 represented ≤1% affected tissue, 1 represented >1-5% affected tissue, 2 represented 6-25% affected tissue, 3 represented 26-50% affected tissue, and 4 represented 51-100%

affected tissue. Percentage affected tissue was based on the total sponge volume of the unaffected sponge (*i.e*. if 20% of the sponge tissue had been eroded at the site of a lesion, then 20% of the sponge tissue was considered to have been affected). A repeated measures ANOVA was used to determine if the prevalence of AWS significantly varied over the study period and post hoc tests of pairwise comparisons were conducted to determine the years with any significant difference in disease prevalence. The assumption of sphericity was tested with Mauchly's test. Year 2013 was not used in this analysis, because no plots at 30m were monitored that year.

The severity of the lesions was monitored through time, and each year the proportion of newly affected sponges and the progression of the lesions (decreasing, increasing, or unchanged) was calculated. The proportion of sponges that healed completely with no remaining tissue damage after showing signs of AWS in the previous year was also calculated. Sponge death was attributed to AWS only if the sponge displayed lesions in the previous year of its reported mortality; however, we acknowledge that sponge death could be attributed to other factors. A sponge was considered dead if there was no visible sponge tissue left on the reef. Finally, log-linear models were used to test for the effect of depth, size class, and time on the occurrence of disease. Analyses were based on a four-way contingency table with the response variable disease, *D*, and explanatory variables plot depth, *P* (15m, 20m, 30m), size class, *S* (I-V), and year, *Y* (2010, 2011, 2014, 2015).

Results

Density of the sponge *Agelas tubulata* in monitored plots on Conch Reef was lowest at 15m, and highest at 30m (Table 2). Mean sponge density for all sites was not calculated for 2013 because data at 30m was not collected that year. Overall, there was a significant increase in sponge density over the study period ($F_{3,21} = 14.041$, $p < 0.001$). Post hoc tests revealed that a significant increase only occurred between 2010 and each subsequent year; there was no difference in density between years 2011-2015. Annual recruitment and mortality were low (0.017 ± 0.015 m^{-2} and 0.006 ± 0.007 m^{-2} year^{-1}, respectively). Analysis of the annual digital photographs for sponges unaffected by AWS revealed the sponge volume did not increase substantially each year, indicating a slow growth rate for this species. However, estimates of sponge growth were affected by the number of sponges exhibiting tissue loss from AWS, and the presence of the lesions precluded reasonable estimates of growth from which age could be extrapolated.

Table 2 Number of *A. tubulata* monitored annually at each depth (top) and mean (±SD) density m^{-2} (bottom) at each depth on Conch Reef

	2010	2011	2013	2014	2015	Mean
15m	73 0.12 (0.06)	80 0.13 (0.06)	80 0.13 (0.06)	84 0.14 (0.06)	85 0.14 (0.06)	- 0.13 (0.05)
20m	201 0.33 (0.09)	218 0.36 (0.09)	221 0.37 (0.10)	218 0.36 (0.09)	223 0.37 (0.10)	- 0.36 (0.08)
30m	150 0.37 (0.06)	166 0.41 (0.07)	n/a	177 0.44 (0.05)	174 0.43 (0.03)	- 0.41 (0.05)
Conch Reef	424 0.26 (0.13)	464 0.29 (0.14)	n/a	479 0.30 (0.15)	482 0.30 (0.15)	

AWS affected discrete portions of the sponge and did not result in short-term mortality. AWS progressed as follows: first sponge tissue became discolored to a lighter shade of brown and sloughed away, leaving behind the skeleton of spicule-imbedded organic fibers (Fig. 1a). The skeleton was then shed, leaving a scarred area with a noticeably lighter color that could return to normal coloration with tissue regrowth over time (Fig. 1b). However, lost tissue was not rapidly regenerated for this slow-growing sponge.

The percentage of diseased sponges increased rapidly during the six-year period (Fig. 2; $F_{3,18} = 32.163$, $p < 0.001$). Post hoc comparisons showed a significant increase in the proportion of diseased sponges across all years, except 2014-2015. Log-linear models showed that AWS did not significantly differ between sponges of different size classes or depths (Table 3). Consistent with the repeated measures ANOVA results, the log-linear models also showed a significant effect of time on the occurrence of AWS.

The number of new sponges with signs of AWS was highest in 2013, and 2014 had the highest number of sponges with lesions becoming more severe than the previous year (Table 4). The proportion of sponges with lesions that remained the same, particularly in 2015, was likely an overestimation, given that sponges classified within the highest level of tissue affected (level 4, >50% of sponge tissue affected) could experience lesions increasing in size but remain classified as level 4. Overall, the majority of sponges with AWS did not recover within a year, and during the study period, 20 sponges died that had been affected with AWS (Table 4).

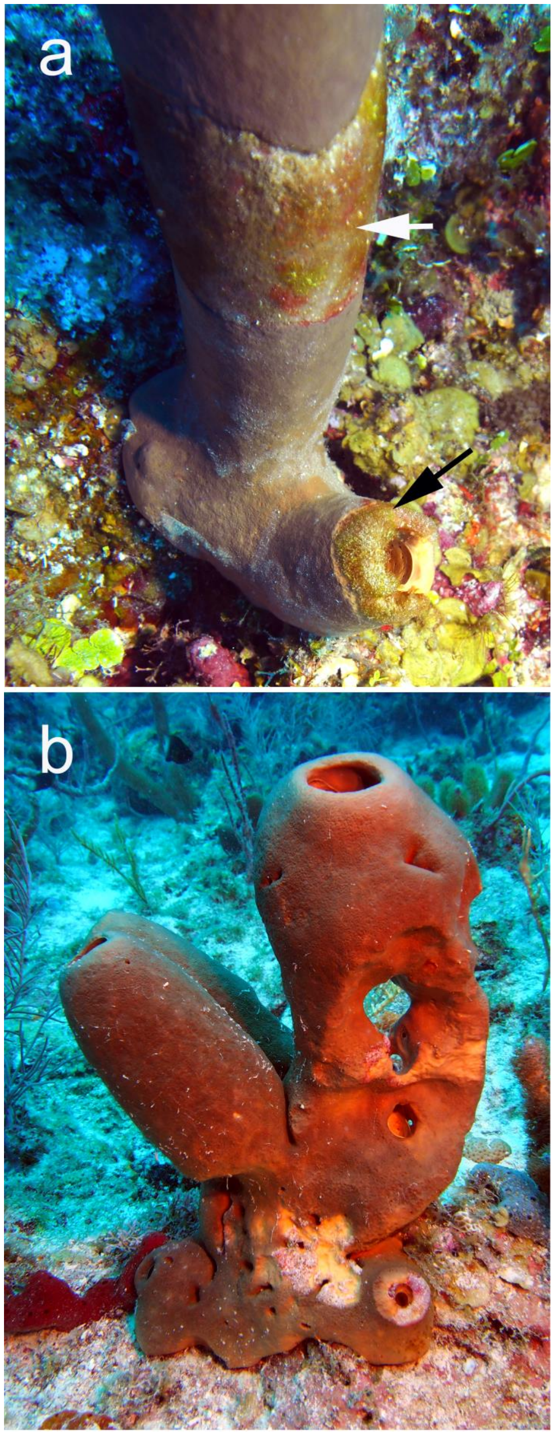

Fig. 1 Photographs of AWS affecting *Agelas tubulata*. **a** Patch of necrotic tissue (white arrow) and skeleton after sloughing (black arrow)(sponge tube width ~5cm; Carrie Bow Cay, Belize) **b** Recent (yellowish) and older patches affected by AWS (widest sponge tube ~ 10 cm; Conch Reef, Florida Keys)

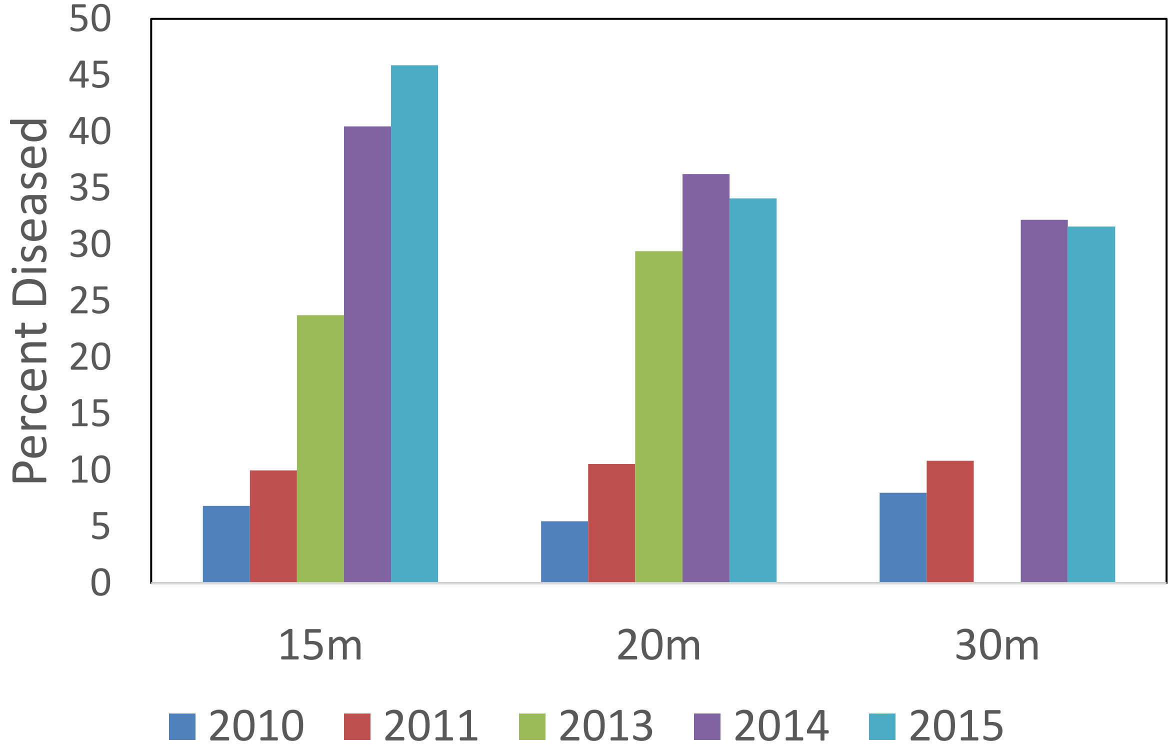

Fig. 2 Annual percentage of diseased sponges at each depth for all plots. No monitoring was conducted in 2012, and the 30 m site was not monitored in 2013

Table 3 Log-linear analysis of disease-frequency data for AWS on *Agelas tubulata* at Conch Reef. Analyses were based on a four-way contingency table with the response variable, disease, *D*, and explanatory variables, plot depth, *P* (15m, 20m, 30m), size class, *S* (I-V), and year, *Y* (2010, 2011, 2014, 2015).

Model	G^2	df	*P*	Contrast	Effect	ΔG^2	Δdf	*P*
M_1 = D, YSP	230.168	59	<0.001	M_1 vs. M_2	P	3.004	2	0.2227
M_2 = DP, YSP	227.164	57	<0.001	M_1 vs. M_3	S	5.689	4	0.2236
M_3 = DS, YSP	224.479	55	<0.001	M_2 vs. M_3	P × S	2.685	2	0.2612
M_4 = TY, YSP	51.932	56	0.630	M_1 vs. M_4	Y	178.236	3	<0.001

Table 4 Patterns of AWS on *A. tubulata* on Conch Reef, 2010-2015. The first row indicates the percentage of the total population of *A. tubulata* with AWS. Subsequent rows indicate the yearly percentage of sponges with AWS that were newly affected with lesions, that had lesions that decreased in size, that increased in size, that remained unchanged, or that healed completely

	2010	2011	2013	2014	2015
Percent of total population with AWS	6.6	10.6	27.9	35.5	35.3
Newly Affected	-	63.2	73.8	51.2	35.3
Lesions decreased in size	-	4.1	0	1.2	17.6
Lesions increased in size	-	16.3	15.5	29.4	11.2
Lesions remained unchanged	-	16.3	10.7	18.2	35.9
Healed completely	-	28.6	4.1	9.5	29.4

Discussion

While the population of *Agelas tubulata* in monitored plots on Conch Reef remained fairly stable over the study period, there was a rise in the occurrence and severity of AWS. For years prior to the analysis of yearly sponge photographs, the scarring associated with AWS was thought to result from predation by turtles or fishes, despite past demonstration that organic extracts of *A. tubulata* were chemically defended from predatory fishes (Pawlik et al. 1995, Assmann et al. 2000, Loh and Pawlik 2014). The progressive nature of the syndrome observed from the annual photographs and field observations revealed that the syndrome is the result of a pathogenic condition and not predation as previously thought. This conclusion would have been difficult to reach without the long-term photographic monitoring of the species. While no standardized surveys were conducted there, AWS has also been observed on *A. tubulata* on the reefs off Carrie Bow Cay, Belize (Fig. 1a).

Reports of sponges displaying disease signs worldwide have recently increased (Rützler 1988; Cowart et al. 2006; Olson et al. 2006; Webster 2007; Maldonado et al. 2010; Angermeier et al. 2012; Sweet et al. 2015), but it is unclear whether these reports indicate an increase in the incidence of pathologies, or reflect a sampling bias because research on sponges has increased. The earliest sponge diseases, dating as far back as the late 1800s, were reported primarily in relation to disruptions in commercial sponge fisheries (Brice 1869; Galstoff et al. 1939; Smith 1941; Galstoff 1942). However, by the mid-1980s, diseases of non-commercial sponge species were being described (Rützler 1988; Wulff 2006a; Wulff 2006b). With the inclusion of AWS, there are now five named diseases associated with Caribbean sponges (Table 1).

AWS differs from the other syndromes previously described to affect Caribbean sponge species in both appearance and virulence (Table 1). AWS cannot be characterized by the appearance of a color spot or band on the leading edge of the lesion, unlike sponge white patch (SWP), *Aplysina* red band syndrome (ARBS), and sponge orange band (SOB) (Cowart et al. 2006; Olson et al. 2006; Angermeier et al. 2012). While AWS can progress and remain visible on a sponge for years without resulting in mortality, SOB progresses rapidly and is often fatal (Cowart et al. 2006). SWP and ARBS both affect sponges with relatively fast-growing, branching morphologies (Olson et al. 2006; Angermeier et al. 2012); the growth form of these species allows them to recover from tissue damage more rapidly than the slow-growing *A. tubulata*. The only other described Caribbean sponge disease, mangrove sponge disease (MSD), occurs from exponential growth of the symbiotic cyanobacteria found in the sponge tissue of *Geodia papyracea* (Rützler 1988). *Geodia papyracea* has been described to isolate overgrowing cyanobacteria into 'pseudogemmules' that are released once the cortex has begun to disintegrate (Rützler 1988).

While the annual monitoring of sponge populations in plots on Conch Reef allowed us to assess the long-term effects of AWS on *A. tubulata* over 6 years, the frequency of monitoring did not permit an analysis of the short-term rate of spread of AWS. Most individuals had signs of AWS for more than one year, but it is unclear whether AWS progresses linearly, or if lesions stop or get worse over shorter periods of time. Histology and microbial studies of healthy and affected sponge individuals should be conducted to better understand the cause, development, and transmission of AWS.

While studies of Caribbean sponge taxonomy and ecology have advanced rapidly, our understanding of the source and spread of sponge diseases remain limited (Olson et al. 2006; Angermeier et al. 2011; Easson et al. 2013). However, the first step in studying new sponge diseases is confirming that the observed tissue damage is the result of disease, and not the result of predation or another abiotic factor. For example, the yellow tube sponge *Aplysina fistularis* was thought to suffer from disease-related lesions, but these were subsequently found to result from superficial grazing scars on the sponge surface caused by sponge-eating molluscs (Pawlik and Deignan 2015). In this paper, we provide evidence for the opposite situation for the brown tube sponge *A. tubulata*, in that tissue loss for this species was thought to result from fish and turtle predation, but has now been shown to result from a disease-related condition (AWS) based on careful monitoring of individual sponges over time.

Several pathogenic conditions have been described for Caribbean sponge species (Table 1), but there is currently no confirmed etiological agent for any Caribbean sponge disease. Complicating matters, other biotic or abiotic stressors of sponges, including storm damage and predation, may be mistaken for sponge disease. Therefore, any potential sponge diseases need to be studied carefully and on a species-by-species basis, with particular attention paid to sponge ecology and temporal monitoring. In this case, long-term monitoring of the Caribbean brown tube sponge resulted in the discovery of a new Caribbean sponge syndrome, AWS.

References

Angermeier H, Kamke J, Abdelmohsen UR, Krohne G, Pawlik JR, Lindquist NL, Hentschel U (2011) The pathology of sponge orange band disease affecting the Caribbean barrel sponge *Xestospongia muta*. FEMS Microbiol Ecol 75:218–230

Assmann M, Lichte E, Pawlik JR, Köck M (2000) Chemical defenses of the Caribbean sponges *Agelas wiedenmayeri* and *Agelas conifer*, Mar Ecol Pro Ser 207:255-262

Blanquer A, Uriz MJ, Cebrian E, Galand PE (2016) Snapshot of a Bacterial Microbiome Shift during the Early Symptoms of a Massive Sponge Die-Off in the Western Mediterranean. Front Microbiol 7:1–10

Birenheide R, Amemiya S, Motokawa T (1993) Penetration and storage of sponge spicules in tissues and coelom of spongivorous echinoids. Mar Biol 115:677–683

Brice JJ (1896) The fish and fisheries of the coastal waters of Florida. US Bur Fish, Rept Comm Fish 22:263-342

Brock TD (1961) Milestones in microbiology. Englewood Cliffs, NJ, USA, Prentice Hall

Cebrian E, Uriz MJ, Garrabou J, Ballesteros E (2011) Sponge mass mortalities in a warming mediterranean sea: Are cyanobacteria-harboring species worse off? PLoS ONE, 6:e20211

Cebrian E, Uriz MJ, Garrabou J, Ballesteros E (2011) Sponge mass mortalities in a warming mediterranean sea: Are cyanobacteria-harboring species worse off? PLoS ONE 6 [doi.org/10.1371/journal.pone.0020211]

Cowart JD, Henkel TP, McMurray SE, Pawlik JR (2006) Sponge orange band (SOB): a pathogenic-like condition of the giant barrel sponge, Xestospongia muta. Coral Reefs 25:513-513

de Goeij JM, Oevelen, DVan, Vermeij MJA, Osinga R, Middelburg JJ, Goeij AFPM De, Admiraal W (2013) Surviving in the marine desert: the sponge loop retains resources within coral reefs. Science 342:108–110

Deignan LK, Pawlik JR (2015) Perilous proximity: Does the Janzen-Connell hypothesis explain the distribution of giant barrel sponges on a Florida coral reef?. Coral Reefs 34:561-567

Diaz MC, Rutzler K (2001) Sponges: an essential component of Caribbean coral reefs. Bull Mar Science, 69:535–546

Dunlap M, Pawlik JR (1996) Video-monitored predation by Caribbean reef fishes on an array of mangrove and reef sponges. Mar Biol 126:117-123

Duffy J (1992) Host use patterns and demography in a guild of tropical sponge-dwelling shrimps. Mar Ecol Prog Ser 90:127-138

Easson CG, Slattery M, Momm HG, Olson JB, Thacker RW, Gochfeld DJ (2013) Exploring Individual- to Population-Level Impacts of Disease on Coral Reef Sponges: Using Spatial Analysis to Assess the Fate, Dynamics, and Transmission of Aplysina Red Band Syndrome (ARBS). PLOSone, doi.org/10.1371/journal.pone.0079976

Erwin PM, Coma R, Lopez-Sendino P, Serrano E, Ribes M (2016) Stable symbionts across the HMA-LMA dichotomy: Low seasonal and inter-annual variation in sponge-associated bacteria from taxonomically diverse hosts. FEMS Microbiol Ecol 91(10):fiv115

Galstoff PS (1942) Wasting disease causing mortality of sponges in the West Indies and Gulf of Mexico. Proc VIII American Science Congress 3:411-421

Galstoff PS, Brown HH, Smith CL, Walton Smith FG (1939) Sponge mortality in the Bahamas. Nature 143:807-808

Gloeckner V, Wehrl M, Moitinho-Silva L, Gernert C, Schupp P, Pawlik JR, Lindquist NL, Erpenbeck D, Wörheide G, Hentschel U (2014) The HMA-LMA dichotomy revisited: an electron microscopical survey of 56 sponge species. Biol Bull 227:78-88

Henkel TP, Pawlik JR (2005) Habitat use by sponge-dwelling brittlestars. Mar Biol 146:301-313

Hill RT (2004) Microbes from marine sponges: a treasure trove of biodiversity for natural products discovery, p.177–190. In A.T. Bull (ed.), Microbial Diversity and Bioprospecting. ASM Press, Washington, DC

Loh T-L, Pawlik JR (2014) Chemical defenses and resource trade-offs structure sponge communities on Caribbean coral reefs. Proc Nat Acad Sci USA 111:4151-4156

López-Legentil S, Erwin PM, Pawlik JR, Song BK (2010) Effects of sponge bleaching on ammonia oxidizing Archaea: Distribution and relative expression of ammonia monooxygenase genes associated with the barrel sponge Xestospongia muta. Microbial Ecology, 60: 561-571.

Maldonado M, Sanchez-Tocino L, Navarro C (2010) Recurrent disease outbreaks in corneous demosponges of the genus Ircinia: Epidemic incidence and defense mechanisms. Mar Biol 157:1577–1590

McMurray SE, Blum JE, Pawlik JR (2008) Redwood of the reef: growth and age of the giant barrel sponge *Xestospongia muta* in the Florida Keys. Mar Biol 155:159-171

McMurray SE, Henkel TP, Pawlik JR (2010) Demographics of increasing populations of the giant barrel sponge *Xestospongia muta* in the Florida Keys. Ecology 91:560-70

McMurray, S.E., Blum, J.E., Leichter, J.J. and Pawlik, J.R. (2011) Bleaching of the giant barrel sponge *Xestospongia muta* in the Florida Keys. Limnol Oceanogr 56: 2243-2250

McMurray, S.E., Finelli, C.M. and Pawlik, J.R. (2015) Population dynamics of giant barrel sponges on Florida coral reefs. J Exp Mar Biol Ecol 473: 73-80

McMurray SE, Johnson ZI, Hunt DE, Pawlik JR, Finelli CM (2016) Selective feeding by the giant barrel sponge enhances foraging efficiency. Limnol. Oceanogr. doi: 10.1002/lno.10287

Meylan A (1988) Spongivory in hawksbill turtles: a diet of glass. Science 239:393-395

Olson, J. B., Gochfeld, D. J., & Slattery, M. (2006) Aplysina red band syndrome: A new threat to Caribbean sponges. Diseases of Aquatic Organisms, 71:163–168

Pawlik JR (1983) A sponge-eating worm from Bermuda: Branchiosyllis oculata (Polychaeta, Syllidae). P.S.Z.N.I: Mar. Ecol. 4:65-79

Pawlik JR (2011) The chemical ecology of sponges on Caribbean reefs: Natural products shape natural systems. BioScience 61:888-898

Pawlik JR, Deignan LK (2015) Cowries graze verongid sponges on Caribbean reefs. Coral Reefs 34:663

Rützler K (1988) Mangrove sponge disease induced by cyanobacterial symbionts: failure of a primitive immune system? Diseases of Aquatic Organisms, 5:143–149

Smith FGW (1941) Sponge disease in British Honduras, and its transmission by water column. Ecology 22:415-421

Sweet M, Bulling M, Cerrano C (2015) A novel sponge disease caused by a consortium of micro-organisms. Coral Reefs 34:871–883

Thomas T, Moitinho-Silva L, Lurgi M, Björk JR, Easson C, Astudillo-García C, Olson JB, Patrick EM, López-Legentil S, Luter H, Chaves-Fonnegra A, Costa R, Schupp PJ, Steindler L, Erpenbeck D, Gilbert J, Knight R, Ackermann G, Victor Lopez J, Taylor MW, Thacker RW, Montoya JM, Hentschel U, Webster NS (2016) Diversity, structure and convergent evolution of the global sponge microbiome. Nat Commun 7:11870 [DOI: 10.1038/ncomms11870]

Vincente VP (1990) Response of sponges with autotrophic endosymbionts during the coral-bleaching episode in Puerto Rico (West Indies). Coral Reefs 8:199–202

Webster NS, Negri AP, Webb RI, Hill RT (2002) A spongin-boring α-proteobacterium is the etiological agent of disease in the Great Barrier Reef sponge *Rhopaloeides odorabile*. Mar Ecol Prog Ser 232:305–309

Webster NS (2007) Sponge disease: a global threat? Environ Microbiol 9:1363–1375

Wulff JL (1995) Sponge-feeding by the Caribbean starfish *Oreaster reticulatus*. Mar Biol 123:313–325

Wulff JL (2006a) Rapid diversity and abundance decline in a Caribbean coral reef sponge community. Biol Conserv 127:167-176

Wulff JL (2006b) A simple model of growth form-dependent recovery from disease in coral reef sponges, and implications for monitoring. Coral Reefs 25:419-426

Zea S, Henkel TP, Pawlik JR (2014) The Sponge Guide: a picture guide to Caribbean sponges. 3rd Edition [www.spongeguide.org]