The effects of a coral disease on the reproductive output of *Montastraea faveolata* (Scleractinia: Faviidae)

Jill L. Borger[1,2] & Susan Colley[3]

1. Institute for Tropical Marine Ecology, P.O. Box 944, Roseau, Commonwealth of Dominica; jborger@earthlink.net
2. Rosenstiel School for Marine and Atmospheric Science, 1301 Memorial Drive, Cox Science Building 182, Coral Gables, Florida 33124.
3. PBS&J, One Galleria Blvd., Suite 1516, Metairie, Louisiana 70001; sctheodosiou@pbsj.com

Received 01-VIII-2009. Corrected 07-II-2010. Accepted 12-VII-2010.

Abstract: The direct impacts of coral diseases on coral populations have been assessed by quantifying coral tissue loss and colony mortality, but the determination of the indirect effects of diseases, such as disruptions in life history functions (e.g. reproduction, growth and maintenance), are more difficult to ascertain and have been scant. This study involved a comparison of various measures of reproductive output from histological slides of healthy tissue samples of *Montastraea faveolata* and tissue samples from colonies with white plague (WP) infections in Dominica (West Indies). Although the variability in the reproductive data was high, WP had significant negative impacts on the percentage of reproductive polyps per cm^2, the percentage of reproductive mesenteries within a polyp, oocyte quantity per polyp, mean oocyte volume (mm^3), and fecundity (oocyte volume per cm^2 of tissue). However, these effects were only observed in the tissue directly impacted by the WP disease "band" and were not observed in tissue samples taken 20 cm away from the lesion. Therefore, the effects of a coral disease (WP) on reproductive output are localized and not expressed colony-wide. Rev. Biol. Trop. 58 (Suppl. 3): 99-110. Epub 2010 October 01.

Key words: coral disease, white plague, coral reproduction, *Montastraea faveolata*, Dominica.

There is a growing consensus that coral diseases are increasing in frequency and host species and geographic range (Santavy & Peters 1997, Goreau *et al*. 1998, Hayes & Goreau 1998, Richardson 1998, Harvell *et al*. 1999, Williams & Bunkley-Williams 2000, Porter & Tougas 2001, Porter *et al*. 2001, Rosenberg & Ben-Haim 2002). It has been demonstrated that coral diseases have the potential to cause shifts in reef community structure, coral diversity, and coral abundance (Dustan 1977, Gladfelter 1982, Hughes 1994, Holden 1996, Santavy & Peters 1997, Aronson *et al*. 1998, Goreau *et al*. 1998, Greenstein *et al*. 1998, McClanahan & Muthiga 1998, Richardson 1998, Richardson *et al*. 1998a,b, Harvell *et al*. 1999, Porter & Tougas 2001, Porter *et al*. 2001, Aronson & Precht 1997, 2001). The short-term, or immediate, effects of coral diseases can be quantified by colony and/or tissue loss *in situ* (Borger 2003, Borger 2005, Williams & Miller 2005, Kim *et al*. 2006). However, some of the potential indirect effects of coral diseases are not as apparent and are more difficult to ascertain. For example, coral diseases may cause a depression in one or more life history functions, such as reproduction, growth, and maintenance. Reproduction is considered one of the least stress-tolerant life history functions and has been utilized as an indirect measure of sublethal coral stress (Kojis & Quinn 1984).

The general paradigm of coral bio-energetics is that various biological functions (reproduction, growth, maintenance, etc.) are

dependent on the same pool of resources. Thus, there must be a trade-off in the allocation of those resources (Rinkevich & Loya 1989, Harrison & Wallace 1990). Harrison & Wallace (1990) suggested that stressed corals express a hierarchy in resource and energy allocation, with regeneration and growth taking precedence over sexual reproduction. There is an extensive amount of literature documenting decreases, or derangements, in coral reproductive activity in response to pollution (Rinkevich & Loya 1979a, Peters *et al*. 1981, Guzman & Holst 1993), injury/colony disruption (Kojis & Quinn 1981, Kojis & Quinn 1985, Szmant-Froelich 1985, Rinkevich & Loya 1989, Van Veghel & Bak 1994, Ward 1995, Hall 1997, Smith & Hughes 1999), sedimentation (Kojis & Quinn 1984), bleaching (Szmant & Gassman 1990), localized regions of growth (Rinkevich & Loya 1989, Heyward & Collins 1985, Oliver 1985, Wallace 1985, Kojis 1986, Harrison & Wallace 1990), and competitive interactions (Rinkevich & Loya 1985, Chadwick 1991).

This study encompassed a comparative examination of the fecundity of apparently healthy *Montastraea faveolata* colonies and colonies exhibiting white plague (WP) infections. The members of the *Montastraea* species complex are hermaphroditic (oocytes and spermaries developing within the same mesenteries), with an annual cycle of gametogenesis and a split-cycle of broadcast spawning occurring in the late summer season (Szmant-Froelich 1985, Szmant 1986, Szmant 1991, Van Veghel 1994). This was the first attempt to quantify the effects of white plague on coral reproductive activity.

MATERIALS AND METHODS

Sampling Protocol: Coral tissue samples were extracted from *Montastraea faveolata* colonies in Dominica, West Indies in 2000, 2001 and 2002. In 2000, samples were taken from two reefs, Tarou Point (7.5m water depth) and Floral Gardens (18m water depth) (Fig. 1). In 2001 and 2002, samples were collected from only one reef site each year, Floral Gardens in 2001 and Cachacrou (18m water depth) in 2002. According to coral spawning predictions for the southern Caribbean region (Szmant, pers. comm.), samples were extracted prior to the first spawning event of the *Montastraea* species complex. However, since there are no records of coral spawning events in Dominica, data on the exact timing of sampling in reference to spawning date are lacking. The number of sampling days was minimized in order to reduce variability in gametogenesis between colonies. In 2000, all samples were extracted within 3 days (18-20 August), and in 2001 and 2002, all tissue samples were collected on the same day (2001: 28 August, 2002: 8 September).

White plague (WP) infections were identified macroscopically, utilizing published descriptions (Dustan 1977, Richardson *et al*. 1998a,b) and government issued (National Oceanic and Atmospheric Administration, NOAA) disease identification cards (Bruckner & Brucker 1998a,b). There are three types of WP (I, II and III), but the identification of each

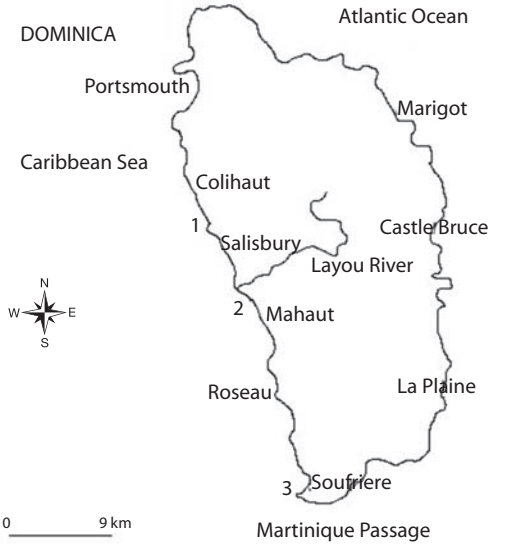

Fig. 1. Dominica, West Indies. Numbers indicate sampling site locations. 1. Floral Gardens (sampled in 2000 and 2001), 2. Tarou Point (sampled in 2000) and 3. Cachacrou (sampled in 2002).

include differences in disease progression rates. Therefore the distinction between types was not made in this study. Tissue samples were taken from large (>3000cm^2, maximum height x maximum diameter) colonies and were not extracted from colony edges in order to avoid intra-colony edge effects and inter-colony size effects in reproductive output (Rinkevich & Loya 1979b, Kojis & Quinn 1981, Babcock 1984, Szmant 1985,1991, VanVeghel & Kahmann 1994, Hall & Hughes 1996). A hand-held wood-borer (diameter≈3.5cm) and a hammer were utilized to extract the tissue samples, which enabled the specific point sampling of the interface between the active disease (exhibited by visible bare, white skeleton, indicating the recent death of coral tissue) and the adjacent, apparently healthy tissue. The wood-borer was driven approximately 1.5cm into the coral calcium carbonate skeleton in order to maintain tissue integrity upon sample extraction. When a diseased colony was sampled, tissue from an additional, apparently healthy colony (H) (devoid of visible abrasions and/or lesions) was removed (from same relative location on the colony surface as that of the diseased sample) as a reference sample. In 2000 and 2001, one sample was removed per colony (2000: n_{WP}=7 and n_H=6; 2001: n_{WP}=3 and n_H=5). In 2002, two samples were taken per colony (n_{WP}=5 and n_H=5). One sample was extracted from the diseased-healthy tissue interface ("WP$^+$"), and the second sample was taken 20 linear cm ("WP$^-$") from the interface into the apparently unaffected tissue. In this case, additional, apparently healthy reference colonies were also sampled twice in the same relative colony locations as the samples removed from diseased colonies. The samples were immediately immersed in a Helly's fixative. They were left in the fixative for 18-24 hours, rinsed in tap water for an additional 18-24 hours and then stored in 70% ethanol until further processing.

Histology and Measurements of Reproductive Activity: Tissue processing followed the techniques outlined by Glynn *et al*. (1991) with the following modifications. Tissue samples were oriented in Paraplast blocks so that both cross sections (n=8-20 polyps section^{-1}) and longitudinal sections (n=2-6 polyps section^{-1}) could be assessed. Four serial sections were made from the mid-polyp region (general location of the reproductive structures of *Montastraea faveolata*) using a microtome.

Stages of oocyte and spermary development were classified according to Szmant-Froelich *et al*. (1985). Late Stage III and Stage IV oocytes were utilized in all calculations. Although fecundity measurements are related to oocytes, the presence of spermaries and their developmental stages were also noted in all samples.

The following information was recorded from one slide (serial section) per tissue sample: number of polyps per cm^2, proportion of reproductive polyps vs. non-reproductive polyps, proportion of mesenteries with either oocytes and/or spermaries (within reproductive polyps), number of oocytes in cross section and longitudinal section and volume of oocytes. The number of polyps per cm^2 was measured using a light box and lupe by overlaying a slide with a 1cm^2 outline. Oocyte volume (mm^3) was extrapolated from a two-dimensional plane by measuring the maximum and minimum oocyte diameters (mm^2) and utilizing the formula for the volume of a prolate spheroid (i.e. one for which the polar radius c is greater than the equatorial radius a: $V=4/3\prod a^2c$). This formula was utilized in place of the formula for the volume of a sphere because of the "squashed" appearance of the oocytes within the mesenteries. If only a few oocytes were present in a polyp, then all late Stage III and Stage IV oocytes were measured (excluding those exhibiting twisted/non-spheroidal shapes). If many oocytes were present in a polyp, then a list of random numbers was used to select which oocytes to measure. Total fecundity was calculated by multiplying the mean number of late Stage III and Stage IV oocytes in cross section by the mean number in longitudinal section by the mean volume of oocytes. This was standardized to 1cm^2 (using the other calculated values) to give a value for the volume of oocyte production (mm^3) per cm^2.

Statistical Analyses: A one-way analysis of variance (or Kruskal-Wallis one-way analysis of variance on ranks, H) was performed to compare the proportion of reproductive polyps to non-reproductive polyps, the proportion of reproductive mesenteries, oocyte volume and fecundity between healthy and WP samples in 2000, 2001 and 2002. Comparisons of diseased vs. healthy corals were made individually by year using a t-test. Non-parametric tests (Mann-Whitney rank sum test) were employed when data transformations were not adequate to satisfy the parametric requirements. When analyzing fecundity, comparisons were made individually by year and using the pooled data (all healthy samples and all WP^- infected samples) with a t-test or Mann-Whitney rank sum test.

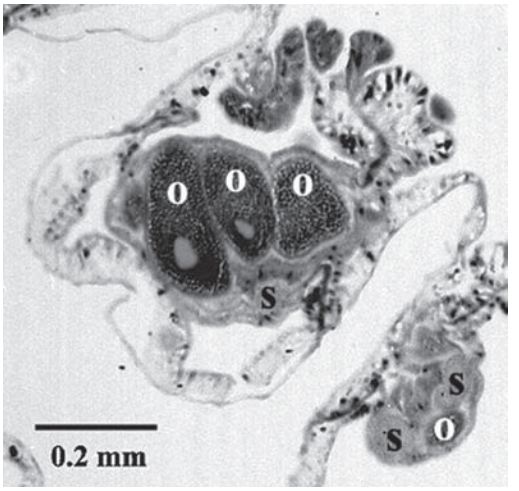

Fig. 2. *Montastraea faveolata* (healthy) reproductive structures (mesenteries): oocytes (o) and spermaries (s) in a healthy tissue sample. Scale bar = 0.2 mm.

RESULTS

General Polyp and Reproductive Features: The mean number of polyps per cm^2 was 11.6 (±2.2, SD), with 12 septal pouches (each with 2 mesenteries) and a total of 24 mesenteries per polyp. The primary mesentery was more prominent (appeared relatively larger and longer in cross-section). However, both sets of mesenteries were capable of producing gametes. Single mesenteries typically contained both oocytes and spermaries (Fig. 2), but there were some mesenteries, and in some cases entire polyps, with only male or female reproductive products. On rare occasions, polyps with as many as 15 septal pouches were observed (mesenteries=30). These polyps were likely in the processes of budding.

The majority of oocytes observed in this study were late Stage III or Stage IV. However, Stages I and II oocytes were also observed on occasion in the tissue samples from each year. In 2001, only 2 polyps of all of the WP-infected tissue samples exhibited oocytes (n_1=1 and n_2=2). In 2000, the highest stage of spermary development observed was Stage III. However, in 2001 and 2002, Stages III, IV and V spermaries were present in the mesenteries.

Proportion of Reproductive Polyps: In the 2001 samples, no reproductive polyps were observed within the haphazardly selected 1 cm^2 areas of the slides. Diseased colonies had a significantly lower proportion of reproductive polyps when compared to healthy samples in each year (2000: t-test, $t=2.76$, $d.f.=45$, $p<0.01$; 2001: Mann-Whitney: $T=21$, $p<0.01$; 2002: t-test, $t=3.35$, $d.f.=30$, $p<0.01$). In 2002, there was a significant difference between the mean proportion of reproductive polyps in the healthy tissue, the WP^+ sample (taken at the interface of the disease) and the WP^- sample (taken 20 cm away from the disease lesion) (One-Way ANOVA: $F=15.81$, $d.f.=31$, $p<0.001$). An *a posteriori* Tukey Test determined that the proportion of reproductive polyps was reduced in the WP^+ tissue when compared to healthy tissue ($q=7.6$, $p<0.001$) and WP^- tissue ($q=5.5$, $p<0.01$). The difference between the healthy tissue and the WP^- tissue was not significant.

Proportion of Reproductive Mesenteries: The mean proportion of reproductive mesenteries was significantly lower in diseased corals versus healthy corals (2000: Mann-Whitney, $T=6326$, $p<0.01$; 2001: Mann-Whitney,

T=21, p<0.001; 2002: t-test, t=2.33, d.f.=77, p<0.05). In the 2002 samples, there was a significant difference between healthy, WP^+, and WP^- tissues (Kruskal-Wallis, H=15.58, d.f.=2, p<0.001). An *a posteriori* Dunn's method test indicated that the WP^+ tissue samples had a significantly lower mean proportion of reproductive polyps than healthy (Q=3.86, p<0.05) and WP^- (Q=3.29, p<0.05) tissue samples. There were no significant differences between healthy and WP^- samples.

Oocyte Volume: The oocyte volume of diseased corals was significantly lower than in healthy corals in 2000 (Mann-Whitney, T=55397, p<0.01) but not in 2001 or 2002 (all WP samples pooled). However, when evaluating the 2002 data by sampling category (i.e. healthy samples, WP^+ samples and WP^- samples), a significant difference between groups was detected (Kruskal-Wallis, H=11.69, d.f.=2, p<0.01). The *a posteriori* test indicated that the oocyte volume of WP^- samples (Fig. 3) was significantly higher than in WP^+ samples (Dunn's Method, Q=3.32, p<0.05). When comparing the groups separately, the oocyte volume of WP^- samples (Fig. 3) was also significantly larger than that of healthy tissue samples (Mann-Whitney, T=17353, p<0.05).

Oocyte Quantity: The oocyte quantity (OQ) was significantly higher in healthy vs. diseased tissue samples in 2001 (Mann-Whiney: T=80, p<0.001) and in 2002 (Mann-Whitney: T=771.5, p<0.05) but not in 2000. The paucity of reproductive products at this interface was the result of both disease-related necrosis (Fig. 4) and a decreased allocation of resources to the affected area (intact, non-necrotic tissue also manifested decreases in reproductive output) (Fig. 5). In 2002, the differences between healthy, WP^+ and WP^- samples were non-significant.

Fecundity: There were no significant differences in the fecundity of healthy colonies in 2000, 2001 and 2002. The same was true for WP-infected colonies between sampling years (Table 1). Thus, the data were pooled (all healthy and all WP fecundity values), and the fecundity of diseased colonies was significantly lower than that of healthy colonies (Mann-Whitney, T=153, p<0.001). However, when the comparisons were made separately by year, the differences in fecundity measurements between diseased and WP colonies were significant in 2001 (Mann-Whitney, T=6, p<0.05) and 2002 (Mann-Whitney, T=38, p<0.05) but not

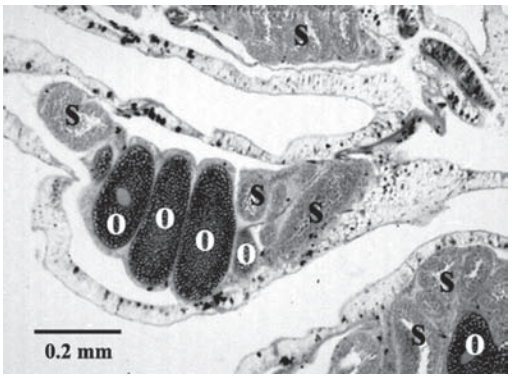

Fig. 3. *Montastraea faveolata* (WP) reproductive structures (mesenteries): oocytes (o) and spermaries (s) in a WP tissue sample extracted from 20cm away from the disease band. Scale bar=0.2mm.

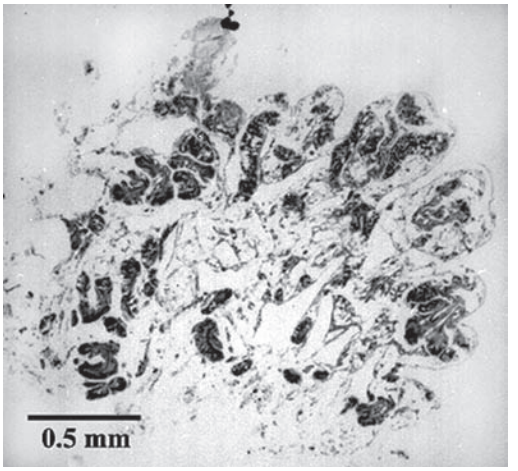

Fig. 4. *Montastraea faveolata* (WP): a cross-section of a single, necrotic polyp taken from the mid-polyp region of a WP infected tissue sample. Tissue sample was extracted from the disease band interface. Scale bar=0.5mm.

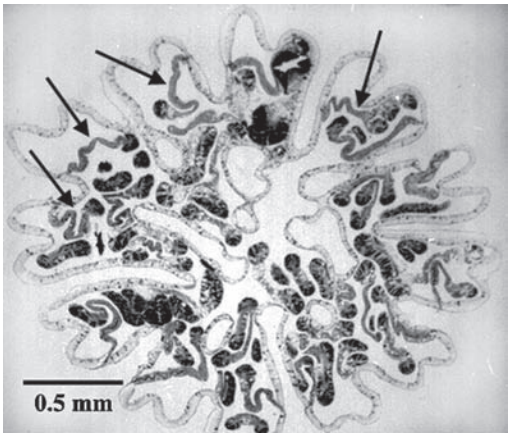

Fig. 5. *Montastraea faveolata* (WP): a cross-section of a single polyp taken from the mid-polyp region of a WP infected tissue sample. Tissue sample was extracted from the disease band interface. Arrows indicate mesenteries that are devoid of reproductive products. Scale bar=0.5mm.

in 2000. There was a significant difference between the fecundity of healthy, WP^+ and WP^- samples (Kruskal-Wallis, $H=7.82$, d.f.=2, $p<0.05$). The *a posteriori* test indicated that the fecundity of healthy tissue samples was significantly higher than in the WP^+ samples (Dunn's Method, $Q=2.69$, $p<0.05$). There was no significant difference in the fecundity of the healthy colonies and the WP^- samples.

DISCUSSION

In this study, the mean number of polyps per cm^2 in *Montastraea faveolata* was very similar to that calculated by Szmant (1986). However, Szmant (1986,1991) reported 12 mesenteries per polyp, while this study documented 24. The corals sampled in this study had many stages of oocyte development present simultaneously within one mesentery, as highlighted by Szmant (1986). Thus, the less developed Stage I and II oocytes observed in the samples were likely allocated towards the second event of the split-spawning of *M. faveolata*. The majority of eggs in all reproductive mesenteries were late Stage III eggs. There were many mesenteries with Stage V spermaries in 2001 and 2002. According to VanVeghel (1994), mature spermaries do not develop in *Montastrea annularis* until one week prior to spawning. This then suggests that the corals sampled in 2001 and 2002 were within one week of their first spawning event.

White plague had significant, deleterious effects on the reproductive output of *Montastraea faveolata* in tissue that was in direct contact with the disease band. The proportion of reproductive polyps and the proportion of reproductive mesenteries within a polyp can be considered a measure of coral fertility (VanVeghel & Kahmann 1994). The proportion of reproductive polyps in healthy samples was similar to that described by Van Veghel & Kahmann (1994) for *Montastrea annularis*, but WP infected colonies had significantly lower fertility than the healthy reference colonies. This effect of WP on fertility was not observed in tissue samples taken 20cm away from the disease-healthy tissue interface. This suggests that although the disease state induces a decrease in fertility, it is not a colony-wide phenomenon.

The values for mean oocyte volume recorded in this study were lower than those described for *Montastrea annularis* by Szmant (1986) and Van Veghel & Kahmann (1994). However, the oocytes measured in this study were predominantly late Stage III eggs, and therefore, presumably had not yet attained their maximum size. In addition, VanVeghel & Kahmann (1994) obtained volume measurements from dissected oocytes (3-dimensional), and this study utilized histology (2-dimensional) to obtain measurements. Thus, histological processing not only results in a 20-30% overall reduction in the size of tissue structures, but the use of a 2-dimensional structure to estimate volume has inherent limitations.

The volume of oocytes in WP^- infected colonies were significantly lower in 2000 but not in 2001 or 2002. The lack of significance in 2001 could be related to the overall paucity of ooctyes in WP^- infected tissue samples during that year. Only 3 oocytes were present in the WP (2001) samples, thus making the

TABLE 1

Reproductive output values (±SD) for diseased (WP) and healthy (H) Montastraea faveolata *colonies in Dominica, West Indies*

Sample	mean # polyps cm^{-2}	mean prop. of RP	mean prop. of RM $polyp^{-1}$	oocyte V (mm^3x 10^{-3})	mean # oocytes $polyp^{-1}$	fecundity (mm^3 cm^{-2})
2000 H	11.7 (±2.54) n_1=10	0.64 (±0.24) n_1=20	0.31 (±0.17) n_2=74	1.20 (±0.62) n_3=259	53.7 (±40.7) n_2=67	0.34 (±0.33) n_4=6
2001 H	12.8 (±2.22) n_1=9	0.87 (±0.21) n_1=10	0.39 (±0.14) n_2=46	1.63 (±1.15) n_3=191	55.3 (±32.9) n_2=43	1.06 (±0.80) n_4=5
2002 H	10.2 (±1.24) n_1=16	0.55 (±0.25) n_1=12	0.43 (±0.30) n_2=41	2.21 (±1.19) n_3=172	91.2 (±63.8) n_2=35	1.21 (±1.19) n_4=5
2000 WP	--	0.41 (±0.32) n_1=27	0.23 (±0.17) n_2=76	1.06 (±0.58) n_3=241	53.4 (±47.8) n_2=58	0.26 (±0.42) n_4=7
2001 WP	--[2]	0 n_1=6	0 n_2=6	1.20 (±2.04) n_3=3	0 n_2=31	0 n_4=3
2002 WP[1]	--	0.24 (±0.26) n_1=20	0.29 (±0.23) n_2=38	2.31 (±1.31) n_3=156	58.8 (±59.3) n_2=29	0.12 (±0.22) n_4=5
2002 WP+	--	0.09 (±0.19) n_1=12	0.12 (±0.18) n_2=14	1.79 (±0.73) n_3=44	32.2 (±27.6) n_2=6	0.09 (±0.20) n_4=5
2002 WP-	--	0.47 (±0.18) n_1=8	0.38 (±0.20) n_2=25	2.51 (±1.43) n_3=112	65.7 (±67.8) n_2=23	0.83 (±1.03) n_4=5

Measurements: mean number of polyps per cm^2 (n_1=1 cm^2), mean proportion of reproductive polyps (RP), mean proportion of reproductive mesenteries (RM) per polyp (n_2=polyps), mean oocyte volume (mm^3) (n_3=oocytes), mean number of oocytes per polyp, and mean fecundity (mm^3 cm^{-2}, n_4=colonies).

[1] represents the combined WP^+ and WP^- values.

[2] tissues not included in analyses.

oocyte sample size very low. However, the results indicate that despite this low density of oocytes, the size of the few oocytes remaining in the tissue was not compromised by the disease state. In 2002, the volume of oocytes in the WP^+ samples was significantly greater than of the WP^- samples, indicating again that the effects of coral diseases are not manifested throughout the colony. Similarly, Hall (1997) reported a localized decrease in total oocyte volume per polyp in injured corals close to the site of injury that was not manifested in tissue farther away. The mean oocyte volume was significantly higher in the WP^- sample versus the healthy tissue samples. Thus, it is possible that diseased corals are compensating for the losses induced at the disease interface by increasing

energy allocation to the remaining oocytes located farther away from the disease lesion.

The mean values of oocyte number per polyp in the healthy colonies sampled in this study corresponded to the ranges calculated by Szmant (1986, 1991), Soong (1991) and VanVeghel & Kahmann (1994) for healthy *Montastraea faveolata* in other Caribbean locations. The average number of oocytes per polyp was significantly higher in healthy vs. diseased coral tissue in 2001 and 2002, the latter localized to the tissue that was in close proximity to the advancing disease line. Thus, in 2000 although the proportion of reproductive polyps and reproductive mesenteries was lower in diseased colonies, the polyps that were reproductive had similar oocyte densities when compared with the healthy colonies.

The fecundity of all healthy vs. all WP samples (calculated only after determining that the variation between years was not significant in either the healthy or the diseased samples) was significantly different. Therefore, WP has a significant negative affect on the fecundity of *M. faveolata*. When comparing each year separately, fecundity was significantly lower in WP colonies in 2001 and 2002 but not in 2000. The relatively high variability in the 2000 data could be a result of utilizing two different sampling locations and depths. Kojis and Quinn (1984) and Rinkevich & Loya (1987) found that reproductive output varied greatly between reefs and within reef sites at different depths. Once again, in 2002 the differences in fecundity were localized to the lesion interface. The paucity of reproductive products at this interface is likely the result of both disease-related necrosis and a decreased allocation of resources to the affected area (intact, non-necrotic tissue also manifested decreases in reproductive output).

There has been a growing concern about some of the non-apparent, detrimental effects of coral diseases on colony fitness and life history functions (Borger 2003). There is a vast amount of literature documenting a decrease in the reproductive output of corals exposed to various external stressors (e.g. oil, injury, sedimentation, etc.) and this is typically related to a presumed divergence of a limited supply of energetic resources. *Montastraea annularis* allocates a large supply of energy to gametogenesis (Szmant 1991), so one would expect to observe a decrease in diseased colonies in which the coral would presumably be allocating resources towards tissue defense and/or regeneration. This study highlights the possibility that some coral diseases may not exert colony-wide effects, because the reproductive output of WP^- samples was not diminished in response to disease infections. VanVeghel & Bak (1994) and Hall (1997) also reported a localized decrease in reproductive output that was not evidenced in other tissue areas of fragmented and injured colonies, respectively. This suggests that the coral is not inducing a significant, colony-wide translocation of energy to the affected area, and thereby supports the localized regeneration hypothesis in which resources are thought to be derived only from polyps directly bordering a lesion (Bak *et al*. 1977, Bak & Steward-Van Es 1980, Bak 1983, Meesters *et al*. 1994, 1997). Oren *et al*. (1997a,b, 1998, 2001) determined that both decreases in reproductive output and translocation of photosynthetic products in response to injury were manifested in tissues up to only 10-15cm away from the affected area. Due to the fact that non-disease interface tissue samples were taken at 20cm away from the lesion, it is possible that more extensive disruptions in reproductive output were overlooked.

Despite the lack of a colony-wide effect, Hughes *et al*. (2000) concluded that even small, sublethal decreases in coral reproductive output have the potential to cause large, negative impacts on recruitment. Thus, the changes in fecundity induced at the disease lesion interface could presumably be sufficient to cause significant negative effects on the recruitment dynamics of *M. faveolata*, which is an important reef framework species in the Caribbean (Goreau 1959, Glynn 1973, Endean & Cameron 1990, Ginsburg *et al*. 1996) that is comparatively slow-growing, hurricane resistant and exhibits characteristically low recruitment rates (Dustan

1975, Stearn *et al*. 1977, Bak & Engel 1979, Bak & Luckhurst 1980, Woodley *et al*. 1981, Porter *et al*. 1981, Rylaarsdam 1983, Rogers *et al*. 1984, Hughes & Jackson 1985, Szmant 1986, Hughes 1988, Guzmán *et al*. 1991, Porter & Meier 1992, Bythell *et al*. 1993).

ACKNOWLEDGMENTS

The Institute for Tropical Marine Ecology and Sascha Steiner offered logistical and financial support during this project.

REFERENCES

Aronson, R.B. & W.F. Precht. 1997. Stasis, biological disturbance, and community structure of a Holocene coral reef. Paleobiol. 23: 326-346.

Aronson, R.B., W.F. Precht & I.G. Macintyre. 1998. Extrinsic control of species replacement on a Holocene reef in Belize: the role of coral disease. Coral Reefs 17: 223-230.

Aronson R.B. & W.F. Precht. 2001. Evolutionary paleoecology of Caribbean coral reefs, p. 171-233. *In* W.D. Allmon & D.J. Bottjer (eds.). Evolutionary paleoecology: The ecological context of macroevolutionary change. Columbia University, New York, USA.

Babcock, R.C. 1984. Reproduction and distribution of two species of *Goniastrea* (Scleractinia) from the Great Barrier Reef Province. Coral Reefs 2: 187-195.

Bak, R.P.M. 1983. Neoplasia, regeneration and growth in the reef-building coral *Acropora palmata*. Mar. Biol. 77: 221-227.

Bak, R.P.M. & J.S. Engel. 1979. Distribution, abundance and survival of juvenile hermatypic corals (Scleractinia) and the importance of life history strategies in the parent coral community. Mar. Biol. 54: 341-352.

Bak, R.P.M. & B.E. Luckhurst. 1980. Constancy and chance in coral reef habitats along depth gradients at Curacao. Oecologia 47: 145-155.

Bak, R.P.M. & Y. Steward-Van Es. 1980. Regeneration of superficial damage in the Scleractinian corals *Agaricia agaricites f. purpurea* and *Porites astreoides*. Bull. Mar. Sci. 30: 883-887.

Bak, R.P.M., J.J.W.M. Brouns & F.M.L. Heys. 1977. Regeneration and aspects of spatial competition in the slceractinian corals *Agaricia agarcites* and *Montasrea annularis*. Proc. 3rd Int. Coral Reef Symp. 1: 143-149.

Borger, J.L. 2003. Three scleractinian coral diseases in Dominica, West Indies: Distribution, infection patterns and contribution to coral tissue mortality. Rev. Biol. Trop. 51: 25-38.

Bruckner, A.W. & R.J. Bruckner. 1998a. Disease and predation on scleractinian corals. Disease identification card, National Oceanic and Atmospheric Administration (NOAA), U.S. Dept of Commerce.

Bruckner, A.W. & R.J. Bruckner. 1998b. Western Atlantic coral disease and predation: Star coral (*Montastraea annularis*, *M. faveolata*, *M. franksi*). Disease identification card, National Oceanic and Atmospheric Administration (NOAA), U.S. Dept of Commerce, USA.

Bythell, J.C., M. Bythell & E.H. Gladfelter. 1993. Initial results of a long-term coral reef monitoring program: Impact of Hurricane Hugo at Buck Island National Monument, St. Croix, U.S. Virgin Islands. J. Exp. Mar. Biol. Ecol. 172: 171-183.

Chadwick, N.E. 1991. Spatial distribution and the effects of competition on some temperate Scleractinia and Corallimorpharia. Mar. Ecol. Prog. Ser. 70: 39-48.

Dustan, P. 1975. Growth and form in the reef-building coral *Montastraea annularis*. Mar. Biol. 33: 101-107.

Dustan, P. 1977. Vitality of reef coral populations off Key Largo, Florida: recruitment and mortality. Env. Geol. 2: 51-58.

Endean, R. & A.M. Cameron. 1990. Trends and new perspectives in coral-reef ecology, p. 469-492. *In* Z. Dubinsky (ed.). Coral Reefs: Ecosystems of the World, Vol. 25. Elsevier, Amsterdam.

Ginsburg, R.N., R.P.M. Bak, W.E. Keine, E. Gischler & V. Kosmynin. 1996. Rapid assessment of reef condition using coral vitality. Reef Enc. 19: 12-14.

Gladfelter, W.B. 1982. White-band disease in *Acropora palmata*: implications for the structure and growth of shallow reefs. Bull. Mar. Sci. 32: 639-643.

Glynn, P.W. 1973. Aspects of the ecology of coral reefs in the Western Atlantic region, p. 271-324. *In* O.A. Jones & R. Endean (eds.). Biology and geology of coral reefs, Vol. 2. Academic Press, New York, USA.

Glynn, P.W., N.J. Gassman, C.M. Eakin, J. Cortes, D.B. Smith & H.M. Guzman. 1991. Reef coral reproduction in the eastern Pacific: Costa Rica, Panama, and

Galapagos Islands (Ecuador). I. Pocilloporidae. Mar. Biol. 109: 355-368.

Goreau, T.J. 1959. The ecology of Jamaican coral reefs. Ecology 40: 67-90.

Goreau, T.J., J. Cervino, M. Goreau, R. Hayes, M. Hayes, L. Richardson, G. Smith, K. DeMeyer, I. Nagelkerken, J. Garzón-Ferreira, D. Gil, G. Garrison, E.H. Williams, L. Bunkley-Williams, C. Quirolo, K. Patterson, J.W. Porter & K. Porter. 1998. Rapid spread of diseases in Caribbean coral reefs. Rev. Biol. Trop. 46: 157-171.

Greenstein, B.J., H.A. Curran & J.M. Pandolfi. 1998. Shifting ecological baselines and the demise of *Acropora cervicornis* in the western Atlantic and Caribbean province: a Pleistocene perspective. Coral Reefs 17: 249-261.

Guzmán, H.M., J.B.C. Jackson & E. Weil. 1991. Short-term ecological consequences of a major oil spill on Panamanian subtidal reef corals. Coral Reefs 10: 1-12.

Guzmán, H.M. & I. Holst. 1993. Effects of chronic oil-sediment pollution on the reproduction of the Caribbean reef coral *Siderastrea siderea*. Mar. Poll. Bull. 26: 276-282.

Hall, V.R. 1997. Effects of injury on growth, reproduction and survivorship for common reef-crest corals. Proc. 8th Int. Coral Reef Symp. 1: 571-574.

Harrison, P.L. & C.C. Wallace. 1990. Reproduction, dispersal and recruitment of scleractinian corals, p. 133-207. *In* Z. Dubinsky (ed.). Coral Reefs: Ecosystems of the World, Vol. 25. Elsevier, Amsterdam.

Harvell, C.D., K. Kim, J.M. Burkholder, R.R. Colwell, P.R. Epstein, D.J. Grimes, E.E. Hofmann, E.K. Lipp, A.D.M.E. Osterhaus, R.M. Overstreet, J.W. Porter, G.W. Smith & G.R. Vasta. 1999. Emerging marine diseases: climate links and anthropogenic factors. Science 285: 1505-1510.

Hayes, R.L. & N.I. Goreau. 1998. The significance of emerging diseases in the tropical coral reef ecosystem. Rev. Biol. Trop. 46: 173-185.

Heyward, A.J. & J.D. Collins. 1985. Growth and sexual reproduction in the scleractinian coral *Montipora digitata* (Dana). Aust. J. Mar. Freshwat. Res. 36: 441-446.

Holden, C. 1996. Coral disease hot spot in the Florida Keys. Science 274: 2017.

Hughes, T.P. 1984. Population dynamics based on individual size rather than age: a general model with a reef coral example. Amer. Nat. 123: 778-795.

Hughes, T.P. 1988. Long-term dynamics of coral populations: contrasting reproductive modes. Proc. 6th Int. Coral Reef Symp. 2: 721-725.

Hughes, T.P. & J.B.C. Jackson. 1985. Population dynamics and life histories of foliaceous corals. Ecol. Monogr. 55: 141-166.

Hughes, T.P., A.H. Baird, E.A. Dinsdale, N.A. Moltschaniwsky, M.S. Pratchett, J.E. Tanner & B.L. Willis. 2000. Supple-side ecology works both ways: the link between benthic adults, fecundity, and larval recruits. Ecology 81: 2241-2249.

Kim, K., A.P. Akler, K. Shuster, C. Quirolo & C.D. Harvell. 2006. Longitudinal study of aspergillosis in sea fan corals. Dis. Aquat. Org. 69: 95-99.

Kojis, B.L. 1986. Sexual reproduction in Acropora (Isopora) species (Coelenterata, Scleractinia). 1. *Acropora cuneata* and *Acropora palifera* on Heron Island Reef, Great Barrier Reef. Mar. Biol. 91: 291-309.

Kojis, B.L. & N.J. Quinn. 1981. Aspects of sexual reproduction and larval development in the shallow water hermatypic coral, *Goniastrea australensis* (Edwards and Haeim, 1857). Bull. Mar. Sci. 31: 558-573.

Kojis, B.L. & N.J. Quinn. 1984. Seasonal and depth variation in fecundity of *Acropora palifera* at two reefs in Papua New Guinea. Coral Reefs 3: 165-172.

Kojis, B.L. & N.J. Quinn. 1985. Puberty in *Goniastrea favulus*: age or size limited? Proc. 5th Int. Coral Reef Congr., 4: 289-293.

McClanahan, T.R. & N.A. Muthiga. 1998. An ecological shift in a remote coral atoll of Belize over 25 years. Environ. Conserv. 25: 122-130.

Meesters, E.H., M. Noordeloos & R.P.M. Bak. 1994. Damage and regeneration: links to growth in the reef-building coral *Montastrea annularis*. Mar. Ecol. Prog. Ser. 112: 119-128.

Meesters, E.H., W. Pauchli & R.P.M. Bak. 1997. Predicting regeneration of physical damage on a reef building coral by regeneration capacity and lesion shape. Mar. Ecol. Prog. Ser. 146: 91-99.

Oliver, J. 1985. Recurrent seasonal bleaching and mortality of corals on the Great Barrier Reef. Proc. 5th Int. Coral Reef Congr. 4: 201-206.

Oren, U., Y. Benayahu & Y. Loya. 1997a. Effect of lesion size and shape on regeneration of the Red Sea coral *Favia favus*. Mar. Ecol. Prog. Ser. 146: 101-107.

Oren, U., B. Rinkevich & Y. Loya. 1997b. Oriented intracolonial transport of ^{14}C labeled materials during regeneration in scleractinian corals. Mar. Ecol. Prog. Ser. 161: 117-121.

Oren, U., I. Brickner & Y. Loya. 1998. Prudent sessile feeding by the corallivore snail *Coralliophila violacea* on coral energy sinks. Proc. Royal Soc. Lond. 265: 2043-2050.

Oren, U., Y. Behayahu, H. Lubinevsky & Y. Loya. 2001. Colony integration during regeneration in the stony coral *Favia favus*. Ecology 82: 802-813.

Peters, E.C., P.A. Meyers, P.P. Yevich & N.J. Blake. 1981. Bioaccumulation and histopathological effects of oil on a stony coral. Mar. Poll. Bull. 12: 333-339.

Porter, J.W., J.D. Woodley, G.L. Smith, J.E. Neigel, J.F. Battey & D.G. Dallmeyer. 1981. Population trends among Jamaican reef corals. Nature 294: 249-250.

Porter J.W. & O.W. Meier. 1992. Quantification of loss and change in Floridian reef coral populations. Am. Zool. 32: 625-640.

Porter, J.W., P. Dustan, W.C. Jaap, K.L. Patterson, V. Kosmynin, O.W. Meier, M.E. Patterson & M. Parsons. 2001. Patterns of spread of coral disease in the Florida Keys. Hydrobiologia 460: 1-24.

Porter, J.W. & J.I. Tougas. 2001. Reef ecosystems: threats to their biodiversity. Encycl. of Biodiv. 5: 73-95.

Richardson, L.L. 1998. Coral diseases: what is really known? TREE 14: 438-443.

Richardson, L.L., W.M. Goldberg, K.G. Kuta, R.B. Aronson, G.W. Smith, K.B. Ritchie, J.C. Halas, J.S. Feingold & S.L. Miller. 1998a. Florida's mystery killer identified. Nature 392: 557-558.

Richardson, L.L., W.M. Goldberg, R.G. Carlton & J.C. Halas. 1998b. Coral disease outbreak in the Florida Keys: Plague type II. Rev. Biol. Trop. 46: 187-198.

Rinkevich, B. 1996. Do reproduction and regeneration in damaged corals compete for energy allocation? Mar. Ecol. Prog. Ser. 143: 297-302.

Rinkevich, B. & Y. Loya. 1979a. Laboratory experiments on the effects of crude oil on the Red Sea coral *Stylophora pistillata*. Mar. Poll. Bull. 10: 328-330.

Rinkevich, B. & Y. Loya. 1979b. The reproduction of the Red Sea coral *Stylophora pistillata*. 1. Gonads and planulae. Mar. Ecol. Prog. Ser. 1: 133-144.

Rinkevich, B. & Y. Loya. 1985. Intraspecific competition in a reef coral – effects on growth and reproduction. Oecology 66: 100-105.

Rinkevich, B. & Y. Loya. 1987. Variability in the patterns of sexual reproduction of the coral *Stylophora pistillata* at Eilat, Red Sea: A long-term study. Biol. Bull. Mar. Biol. Lab. Woods Hole 173: 335-344.

Rinkevich, B. & Y. Loya. 1989. Reproduction in the regenerating colonies of *Stylophora pistillata*, p. 259-265. *In* E. Spanier, Y. Steinberger & M. Luria (eds.). Environmental quality and ecosystem stability. Vol. IVB: Environmental Quality. ISEEQS, Jerusalem.

Rogers, C.S., H.C. Fitz III., M. Gilnack & J. Hardin. 1984. Scleractinian coral recruitment patterns at Salt River Submarine Canyon, St. Croix, U.S. Virgin Islands. Coral Reefs 3: 69-76.

Rosenberg, E. & Y. Ben-Haim. 2002. Microbial diseases of corals and global warming. Env. Microb. 4: 318-326.

Rylaarsdam, K.W. 1983. Life histories and abundance patterns of colonial corals on Jamaican reefs. Mar. Ecol. Prog. Ser. 13: 249-260.

Smith, L.D. & T.P. Hughes. 1999. An experimental assessment of survival, re-attachment and fecundity of coral fragments. J. Exp. Mar. Biol. Ecol. 235: 147-164.

Soong, K. 1991. Sexual reproductive patterns of shallow-water reef corals in Panama. Bull. Mar. Sci. 49: 832-846.

Stearn, C.W., T.P. Scoffin & W. Martindale. 1977. Calcium carbonate budget of a fringing reef on the west coast of Barbados. Bull. Mar. Sci. 27: 479-510.

Szmant-Froelich, A. 1985. The effect of colony size on the reproductive ability of the Caribbean coral *Montastrea annularis* (Ellis and Solander). Proc. 5th Int. Coral Reef Congr., 4: 295-300.

Szmant, A.M. 1986. Reproductive ecology of Caribbean reef corals. Coral Reefs 5: 43-53.

Szmant, A.M. 1991. Sexual reproduction by the Caribbean reef corals *Montastrea annularis* and *M. cavernosa*. Mar. Ecol. Prog. Ser. 74: 13-25.

Szmant-Froelich, A., P. Yevich & M.E.Q. Pilson. 1980. Gametogenesis and early development of the temperate coral *Astrangia danae* (Anthozoa: Scleractinia). Biol. Bull. 158: 257-269.

Szmant-Froelich, A., M. Ruetter & L. Riggs. 1985. Sexual reproduction of *Favia fragum* (Esper): lunar patterns of gametogenesis, embryogenesis, and planulation in Puerto Rico. Bull. Mar. Sci. 37: 880-892.

Szmant, A.M. & N.J. Gassman. 1990. The effects of prolonged "bleaching" on the tissue biomass and reproduction of the reef coral *Montastrea annularis*. Coral Reefs 8: 217-224.

Van Veghel, M.L.J. 1994. Reproductive characteristics of the polymorphic Caribbean reef building coral *Montastrea annularis*. I. Gametogenesis and spawning behavior. Mar. Ecol. Prog. Ser. 109: 209-219.

Van Veghel, M.L.J. & R.P.M. Bak. 1994. Reproductive characteristics of the polymorphic Caribbean reef building coral *Montastrea annularis*. III. Reproduction in damaged and regenerating colonies. Mar. Ecol. Prog. Ser. 109: 229-233.

Van Veghel M.L.J. & M.E.H. Kahmann. 1994. Reproductive characteristics of the polymorphic Caribbean reef building coral *Montastrea annularis*. II. Fecundity and colony structure. Mar. Ecol. Prog. Ser. 109: 221-227.

Wahle, C.M. 1983. The roles of sex, size and injury in sexual reproduction among Jamaican gorgoniasn. Am. Zool. 24: 961.

Wallace, C.C. 1985. Reproduction, recruitment and fragmentation in nine sympatric species of the coral genus *Acropora*. Mar. Biol. 88: 217-233.

Williams, D.E. & M.W. Miller. 2005. Coral disease outbreak: pattern, prevalence and transmission in *Acropora cervicornis*. Mar. Ecol. Prog. Ser. 301: 119-128.

Williams, E.H. & L. Bunkley-Williams Jr. 2000. Major marine ecological disturbances of the Caribbean. Infect. Dis. Rev. 2: 110-127

Woodley, J.D. & 19 co-authors.1981. Hurricane Allen's impact on Jamaican coral reefs. Science 214: 749-755.

Yamashiro, H., O. Hirosuke, K. Onaga, H. Iwasaki & T. Kensaku. 2001. Coral tumors store reduced level of lipids. J. Exp. Mar. Biol. Ecol. 265: 171-179.