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Coral disease distribution at Ras Mohammed and the Gulf of Aqaba, Red Sea, Egypt

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Abstract. *Ammar MSA, Ashour F, Abdelazim H. 2013. Coral disease distribution at Ras Mohammed and the Gulf of Aqaba, Red Sea, Egypt. Nusantara Bioscience 5: 35-43.* Six sites along the Gulf of Aqaba and Ras Mohammed, Red Sea, Egypt were studied for coral disease distribution relative to environmental stress. These sites are (i) South Taba, (ii) South Nuweiba, (iii) Canyon, (iv) Eel Garden (at Dahab), (v) Shark Observatory and (vi) Yolanda. Number of coral diseases ranges from 6 diseases at site 4 (Eeel Garden) to 12 diseases at site 3 (Canyon). The site having the lowest number of coral diseases (site 4) is characterized by the highest percentage cover of coral diseases (24%). The coral disease atramentous necrosis attained the highest percentage cover in all sites (5, 5, 6, 6, 2 and 3%) in sites 1-6 respectively. A total of 16 diseases were reported being distributed in the following order in sites 1-6: 9, 9, 12, 6, 8 and 7 respectively. The coral disease atramentous necrosis is the most widely distributed one being found in all 6 sites followed by dark spots disease and ulcerative white spots being reported in 5 sites. The disease that is least distributed is the white tips being reported in site 5 only. The most commonly distributed disease (atramentous necrosis) infected six corals in site 1, two corals in site 2, nine corals in site 3, two corals in site 4, five corals in site 5 and five corals in site 6. However, the least commonly distributed disease (white tips) infected only two corals (*Acropora humilis* and *Millepora dichotoma*). Site 1, having *Cyphastrea serailia* being infected with highest number of diseases is characterized by the maximum metal concentrations of Zn, Cd, Pb and Ni in water and highest metal concentrations for Cu, Zn, and Pb in sediments. Site 2, having *M. dichotoma* being infected with the highest number of diseases, is characterized by the highest Cu concentration in water. Site 4, having fewer number of coral diseases and highest percentage of disease cover attained the highest levels of Cd and Ni in sediments.

Key words: Coral disease, distribution, Ras Mohammed, Gulf of Aqaba, Red Sea

Abstrak. *Ammar MSA, Ashour F, Abdelazim H. 2013. Distribusi penyakit karang di Ras Mohammed dan Teluk Aqaba, Laut Merah, Mesir. Nusantara Bioscience 5: 35-43.* Enam situs di sepanjang Teluk Aqaba dan Ras Mohammed, Laut Merah, Mesir dipelajari untuk mengetahui distribusi relatif penyakit karang terhadap tekanan lingkungan. Lokasi yang diteliti adalah (i) South Taba, (ii) South Nuweiba, (iii) Canyon, (iv) Eel Garden (di Dahab), (v) Shark Observatory dan (vi) Yolanda. Jumlah penyakit karang berkisar dari 6 penyakit di lokasi 4 (Eeel Garden) hingga 12 penyakit di lokasi 3 (Canyon). Lokasi yang memiliki jumlah penyakit karang terendah (lokasi 4) ditandai dengan persentase penutupan penyakit karang tertinggi (24%). Penyakit karang nekrosis atramentous mencapai persentase penutupan tertinggi di semua lokasi (5, 5, 6, 6, 2 dan 3%) secara berturut-turut dari lokasi 1-6. Sebanyak 16 penyakit dilaporkan terdistribusi dengan urutan dari lokasi 1-6 secara berturut-turut sebagai berikut: 9, 9, 12, 6, 8 dan 7. Penyakit karang nekrosis atramentous merupakan penyakit yang paling luas distribusinya yang ditemukan di semua ke-6 lokasi, diikuti oleh penyakit bintik-bintik gelap dan bintik-bintik putih ulseratif yang dilaporkan pada 5 lokasi. Penyakit yang paling sempit distribusinya adalah pucuk putih yang dilaporkan dalam 5 lokasi. Penyakit yang paling luas distribusinya (nekrosis atramentous) menginfeksi enam terumbu karang di lokasi 1, dua karang di lokasi 2, sembilan karang di lokasi 3, dua karang di lokasi 4, lima karang di lokasi 5 dan lima karang di lokasi 6. Namun, penyakit yang paling sempit distribusinya (pucuk putih) hanya menginfeksi dua karang (*Acropora humilis* dan *Millepora dichotoma*). Lokasi 1, terdapat *Cyphastrea serailia* yang terinfeksi penyakit dengan jumlah paling tinggi ditandai dengan konsentrasi maksimum logam Zn, Cd, Pb dan Ni dalam air dan konsentrasi logam tertinggi untuk Cu, Zn dan Pb dalam sedimen. Lokasi 2, terdapat *M. dichotoma* yang terinfeksi penyakit dengan jumlah tertinggi, ditandai dengan konsentrasi Cu tertinggi dalam air. Lokasi 4, memiliki lebih sedikit jumlah penyakit karang dan persentase penutup penyakit tertinggi mencapai tingkat tertinggi Cd dan Ni dalam sedimen.

Kata kunci: Penyakit karang, distribusi, Ras Mohammed, Teluk Aqaba, Laut Merah

INTRODUCTION

Coral disease is defined as an abnormal condition of an organism that impairs organism functions, associated with specific symptoms and signs (ICRI/UNEP-WCMC 2010). It may be caused by external factors, such as infectious disease, or it may be caused by internal dysfunctions. Coral disease outbreaks are having a significant, negative impact on the structure and appearance of coral reefs, and have contributed to unprecedented declines in live coral cover and productivity of coral reef ecosystems upon which many millions of people depend (Galloway et al. 2009). The same authors concluded that several diseases are playing an increasingly important role in controlling coral population size, diversity and demographic characteristics. Large scale disease outbreaks have already fundamentally altered the

structure of reef communities in the Caribbean (Harvell et al. 2004). Research on the causes of coral disease has increased in recent years, especially in terms of identifying the pathogens involved (Harvell et al. 2007). Most biotic coral diseases are believed to be related to infection by one or a group of pathogens (Sokolow 2009). A host of contributing microorganisms (Richardson and Aronson 2000) and macroparasites such as ciliates (Cróquer et al. 2006) have been identified as possible causal agents; however, little is currently known about the involvement of viruses (Sokolow 2009). Research on the causes of coral disease has increased in recent years, especially in terms of identifying the pathogens involved (Harvell et al. 2007). Knowledge of organisms that transmit pathogens from a reservoir to a host (vectors), the mechanisms by which coral disease is transmitted between organisms (vector pathways), and natural reservoirs of coral disease is far from complete (ICRI/UNEP-WCMC 2010). Growing evidence suggests that environmental and anthropogenic stressors are linked with coral disease and mortality in complex ways (Harvell et al. 2007). Examples of those stressors are nutrient enrichment (Garren et al. 2008), ocean acidification (Sokolow 2009), algal competition (Aronson and Precht 2006), irradiance (Boyett et al. 2007) and loss of biodiversity (Keesing et al. 2010).

Coral disease identification is often based on visual cues observed in the field or from photographs. Such techniques have been shown to be insufficient for making coral disease because different causes of disease can result in similar obvious manifestations of disease, or progress from showing the signs of one disease to showing those of another (Ainsworth et al. 2007). Ammar (2012) provided a guide to coral diseases in the northern Red Sea, Egypt. Laboratory analyses of samples to identify the microbiological factors accompanying the disease manifestations, such as the presence or absence of certain pathogens, are therefore necessary to support accurate disease diagnosis and accurate disease identifications (Ainsworth et al. 2007).

The purpose of the study is to quantify the coral diseases in many areas of the Gulf of Aqaba and Ras Mohammed (South Sinai), Egypt. In addition, the environmental drivers of disease, as well as understanding the coral's ability to resist the disease are studied. A data based on coral diseases in the area will be established, this will help using coral diseases as indicators of environmental impacts and acting to remove or minimize these impacts. Removing or minimizing these impacts will improve the coral reef environment, in turn, help to increasing fish stocks, tourist attraction, improving the national income, the economic, scientific and medical values and conserving the marine biodiversity.

MATERIALS AND METHODS

Six sites along the Gulf of Aqaba and Ras Mohammed, Red Sea, Egypt (Figure 1, Table 1) were studied for coral diseases.

Table 1. Latitudes and longitudes of the study sites

Sites	Latitudes	Longitudes
1. South Fanar village (South Taba)	29°20.170` N	034°45.767` E
2. South Nuweiba (2 km south of Nuweiba harbor)	28° 57.521` N,	034° 38.516` E
3. Canyon (north Dahab)	28° 33.277` N,	034° 31.235` E
4. Eel Garden (at Dahab)	28° 30.297` N,	034° 31.171` E
5. Shark Observatory (at Ras Mohammed)	27° 43.921` N,	034° 15.560` E
6. Yolanda (at Ras Mohammed)	27° 43.715` N,	034° 15.383` E

Coral diseases were quantified as percentage cover relative to the bottom cover. SCUBA diving and the camera frame (as a quadrate) were used for surveying the coral diseases. Ten frames, one-meter intervals and one meter from the object were surveyed along a transect fixed horizontally along the reef contour at the depths reef flat, 1 m, 5 m, 10 m, 15 m, 20 m or till the end limit of coral growth at each of the studied sites. A FinePix F50, 12 Mega Pixels Digital Camera, was used for taking a series of underwater photos to help identification of species and coral diseases. The computer software Photogrid 1.0 beta Acad was used for ecological analysis of digital photographs for coral diseases.

Coral disease pathogen identification was achieved using ICRI/UNEP-WCMC (2010), Raymundo et al. (2008), Rosenberg et al. (2007).

Disease definition and disease types

Only clear and unequivocal signs of disease were recorded. Coral disease was also carefully distinguished from coral bleaching (Brown 1997), which superficially can look like disease. To make a disease determination, observers looked for active tissue necrosis. Often this was accompanied by bared skeleton, mucus production and partial disintegration of polyps. Blemishes, slight discolorations and small, cryptic examples of disease were not scored. We chose characteristics that were as pathogenomics possible for underwater determinations. Anchor scrapes, parrot fish bites, predatory snail wounds, etc. were not scored as diseases but as causative agents.

Quality assurance/quality control

After the first survey of sites, the underwater survey lines were taken up, and then reset and surveyed once again. In addition, a videotape of lines were done.

RESULTS AND DISCUSSION

Number and percentage cover of coral diseases, healthy corals and associated biota in each of the studied sites are shown in Table 2, while the infected coral species by different diseases are found in Tables 3. Number of coral diseases ranges from 6 diseases at site 4 (Eeel Garden) to 12 diseases at site 3 (Canyon). However, the site having the lowest number of coral diseases (site 4-6 diseases)

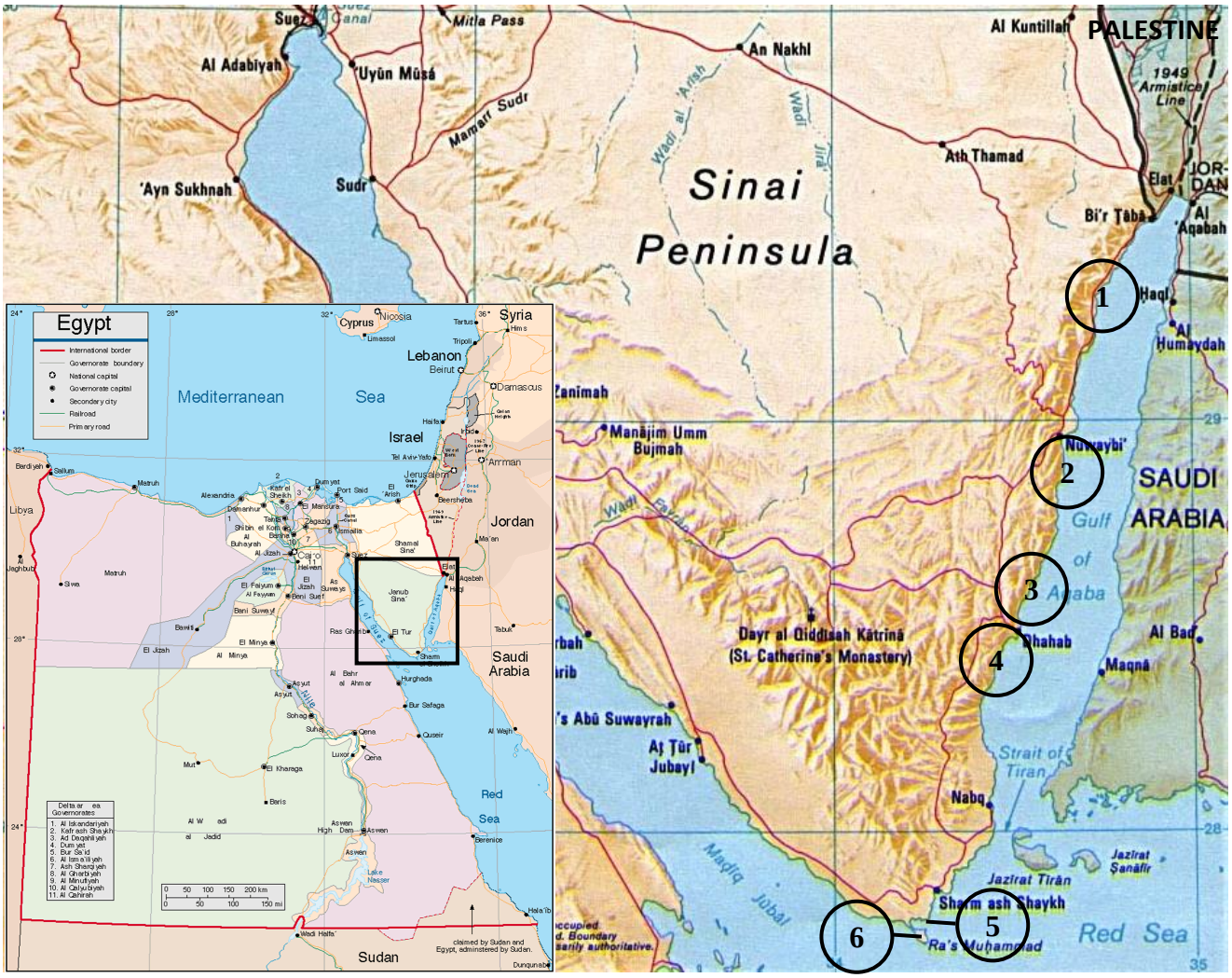

Figure 1. Map of the studied sites. 1. South Fanar village (South Taba), 2. South Nuweiba (2 km south of Nuweiba harbor), 3. Canyon (north Dahab), 4. Eel Garden (at Dahab), 5. Shark Observatory (at Ras Mohammed), 6. Yolanda (at Ras Mohammed)

is characterized by the highest percentage cover of coral diseases (24%) Those diseases are sediment damage, dark spots, coral neoplasia, ulcerative white spots, coral hyperplasia, and atramentous necrosis. Sites 5 (Shark Observatory) and 6 (Yolanda) are characterized by the highest amount of percentage healthy corals (85% and 83% respectively) while the lowest value is found in site 4 (16%). The highest percentage cover of algae/seagrasses (22%) is found in site 4 while the lowest percentage cover of each of algae/seagrasses, macroborers, and sediments is found in site 5.

The coral disease atramentous necrosis attained the highest percentage cover in all sites (5, 5, 6, 6, 2 and 3%) in sites 1-6 respectively. Diseases having lowest percentage cover are black band disease (site 1), white band disease (site 2), pigmentation response (site 3), coral hyperplasia (sites 4, 5) and black band (site 6). A total of 16 diseases were reported being distributed in the following order in sites 1-6: 9, 9, 12, 6, 8 and 7 respectively. The coral disease atramentous necrosis is the most widely distributed one being found in all 6 sites followed by dark spots and ulcerative white spots being reported in 5 sites. The disease that is least distributed is the white tips being reported in site 5 only. However, each of the black band, white spots, white band, pigmentation response, sediment damage, and rapid wasting is reported in 2 sites only. The most commonly distributed disease (atramentous necrosis) infected six corals in site 1, two corals in site 2, nine corals in site 3, two corals in site 4, five corals in site 5 and five corals in site 6. However, the least commonly distributed disease (white tips) infected only two corals (*Acropora humilis* and *Millepora dichotoma*).

It is observed that the coral disease ulcerative white spots is always associated with vermetidae predation in both earlier and later stages of the disease, and in many cases with *Tridacna* boring in later stages of the disease. However, vermetidae predation is also associated with tissue discoloration (non-white pigmentation response) while *Drupella* predation is associated with skeletal eroding band. The coral disease tissue coral neoplasia is found only in site 4 having 4 percentage cover and infecting the two coral species *Leptoseris incrustans* and *Favia speciosa*.

Table 2. Percentage of coral diseases and other habitats in the studied sites

Sites	No. of coral diseases	% coral diseases	% healthy corals	% dead corals	% algae/ seagrasses	% macro-borers	% sediments	% others
1. South Fanar village (South Taba)	9	15	30	20	10	8	12	5
2. South Nuweiba (2 km south of Nuweiba harbor)	9	20.5	23	19.5	13	9	13	2
3. Canyon (north Dahab)	12	22	35	16	7	6	9	3
4. Eel Garden (at Dahab)	6	24	16	15	22	2	20	1
5. Shark Observatory (at Ras Mohammed)	8	7.75	85	2.5	0.5	1.25	0	3
6. Yolanda (at Ras Mohammed)	7	8	83	3	1	2	1	2

Table 3. Coral diseases at Ras Mohammed and **t**he Gulf of Aqaba, Red Sea, Egypt (site 1-6).

Sites	Coral disease	Infected coral species	% disease cover	Remarks
1. South Fanar village	1. Atramentous necrosis	*Goniastrea retiformis* *Goniastrea pectinata* *Cyphastrea serailia* *Platygyra lamellina* *Porites solida* *Millepora platyphylla*	5	Mechanical breaking
	2. Black band disease	*Goniastrea retiformis*	0.4	
	3. Brown band disease	*Acropora nasuta* *Favites flexuosa*	0.5	
	4. Ulcerative white spots	*Porites solida* *Favia speciosa* *Goniastrea retiformis*	2.6	Vermetidae predation *Tridacna* boring
	5. White spots disease	*Echinopora gemmacea*	0.5	
	6. Dark spots disease	*Cyphastrea serailia* *Hydnophora exesa* *Porites solida* *Favites flexuosa*	2.5	
	7. White patches	*Favites flexuosa*	1	
	8. Skeleton eroding band	*Cyphastrea serailia* *Siderastrea savignyana* *Platygyra lamellina*	2	*Drupella* predation *Drupella* predation *Drupella* predation
	9. White plague	*Cyphastrea serailia* *Goniastrea retiformis*	0.5	
	Total % disease cover		15	
2. South Nuweiba	1. White plague	*Millepora dichotoma* *Millepora platyphylla*	3.5	Overgrowth by *Padina*, Red filamentous algae, coralline algae
	2. Atramentous necrosis	*Cyphastrea serailia* *Millepora dichotoma*	5	*Tridacna* boring Mechanical breaking
	3. Brown band disease	*Millepora dichotoma*	2	
	4. White patches	*Cyphastrea serailia*	2	Surface cyanobacteria
	5. Dark spots disease	*Pavona cactus* *Montipora verrucosa*	2	Gastropod boring
	6. White band disease	*Stylophora pistillata*	1	
	7. Ulcerative white spots	*Porites solida*	1.5	
	8. Coral hyperplasia	*Millepora dichotoma*	1.5	Mechanical breaking
	9. Partial bleaching	*Favia favus* *Goniastrea retiformis*	2	
	Total % disease cover		20.5	
3. Canyon	1. White plague	*Porites solida*	2	Aggressive coralline algal overgrowth Aggressive filamentous algal overgrowth Aggressive sponge overgrowth
	2. Atramentous necrosis	*Goniastrea pectinata* *Goniastrea retiformis* *Platygyra daedalea* *Lobophyllia corymbosa* *Favites flexuosa* *Acropora tenuis* *Millepora platyphylla* *Millepora dichotoma* *Porites lutea*	6	Mechanical breaking *Tridacna* boring Mechanical breaking Mechanical breaking Mechanical breaking Mechanical breaking Mechanical breaking Mechanical breaking *Tridacna* boring
	3. Brown band	*Montipora informis*	2	Gastropod drilling

			Montipora verrucosa		Vermetidae predation
			Porites lutea		
		4. White patches	*Acropora valida*	3	
			Acropora hemprichii		*Drupella* predation
			Acropora tenuis		
		5. Pigmentation response	*Stylophora pistillata*	0.4	Ciliate infection
		6. White spots disease	*Porites rus*	1.6	
			Astreopora myriophthalma		Gastropod boring
			Porites solida		
		7. Coral hyperplasia	*Montipora verrucosa*	0.5	
		8. White band disease	*Porites lutea*	0.5	
			Montipora verrucosa		
		9. Partial bleaching	*Goniastrea retiformis*	1	
		10. Sediment damage	*Favites flexuosa*	0.5	
		11. Skeletal eroding band	*Platygyra daedalea*	1.5	
			Montipora informis		*Drupella* predation
			Porites lutea		
		12. Rapid wasting	*Montipora tuberculosa*	3	Parrot fish predation
			Porites lutea		Parrot fish predation
			Porites solida		Aggressive coralline algal overgrowth
		Total % disease cover		22	
4.	Eel Garden	1. Sediment damage	*Leptoseris incrustans*	4	Aggressive red filamentous algal overgrowth
			Favia speciosa		
		2. Dark spots disease	*Leptoseris incrustans*	3	
		3. Coral neoplasia	*Leptoseris incrustans*	4	High sediment load
			Favia speciosa		
		4. Atramentous necrosis	*Favia favus*	6	Aggressive red filamentous algal overgrowth
			Goniastrea retiformis		
		5. Coral hyperplasia	*Psammocora haimeana*	2	High sediment load
		6. Ulcerative white spots	*Goniastrea retiformis*	5	
		Total % disease cover		24	
5.	Shark Observatory	1. Atramentous necrosis	*Stylophora pistillata*	2	Mechanical breaking
			Goniastrea retiformis		Vermetidae predation
			Porites lutea		Mechanical breaking
			Acropora humilis		
			Favia stelligera		
		2. Pigmentation response	*Stylophora pistillata*	1	Gastropod boring
			Porites solida		Vermetidae boring
		3. Dark spots disease	*Porites solida*	0.5	
			Stylophora pistillata		
		4. Ulcerative white spots	*Goniastrea retiformis*	1	
			Favites flexuosa		
		5. Coral hyperplasia	*Stylophora pistillata*	0.25	Mechanical breaking
		6. Partial bleaching	*Favites flexuosa*	1	
			Favia stelligera		
		7. White tips	*Acropora humilis*	1	Vermetidae boring, mechanical breaking
			Millepora dichotoma		
		8. Rapid wasting	*Pavona explanulata*	1	Aggressive coralline algal overgrowth
			Echinopora gemmacea		
		Total % disease cover		7.75	
6.	Yolanda	1. Dark spots disease	*Stylophora pistillata*	1	
			Stylophora mamillata		
			Goniastrea retiformis		
		2. Atramentous necrosis	*Stylophora mamillata*	3	Mechanical breaking
			Stylophora pistillata		Vermetidae predation
			Goniastrea retiformis		Vermetidae predation
			Porites rus		Vermetidae predation
			Pocillopora damicornis		Mechanical breaking
		3. Ulcerative white spots	*Goniastrea retiformis*	1	Vermetidae predation
			Porites solida		
		4. Black band disease	*Porites solida*	0.25	
		5. Skeletal eroding band	*Porites lutea*	0.5	*Drupella* predation
		6. White plague	*Porites lutea*	1	Vermetidae
			Porites solida		Vermetidae
			Stylophora pistillata		Aggressive coralline algal overgrowth
		7. White patches	*Favites abdita*	1.75	Parrot fish predation
			Porites rus		
			Porites solida		
			Stylophora mamillata		
		Total % disease cover		8	

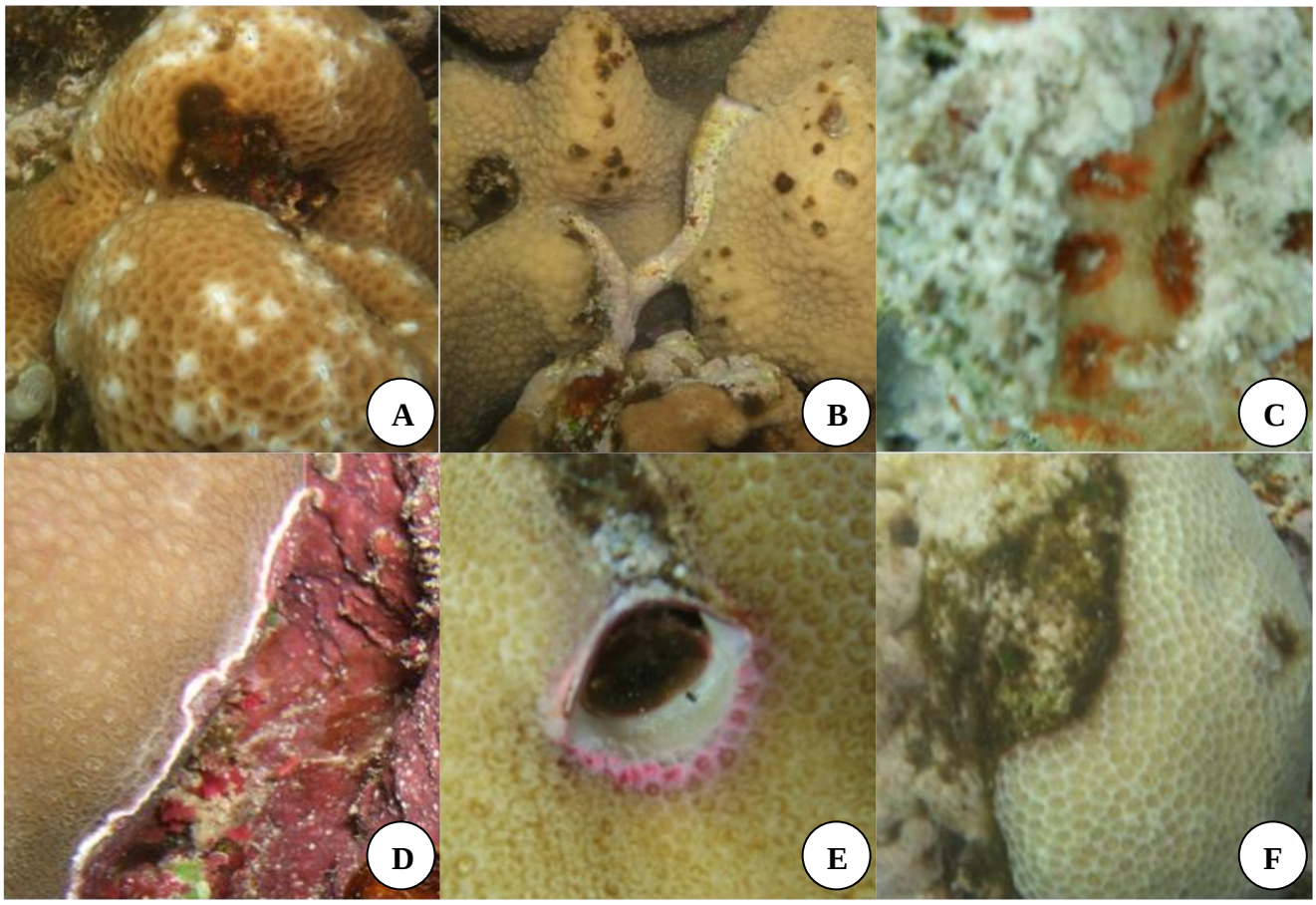

Figure 2. Several coral **d**iseases at Ras Mohammed and **t**he Gulf of Aqaba, Red Sea, Egypt. A. Ulcerative white spots, B. Dark spots disease, C. Coral tissue tumor, D. White band disease, E. Pigmentation response, F. Black band disease

Trace metals in water and sediments

Total metal concentrations in seawater varied between 0.1 ppb for Cu and 2.51 ppb for Zn. Site 1, in the north, recorded the maximum metal concentrations for Zn, Cd, Pb, and Ni. This may be due to the high pollution load from large cities and harbors like Aqaba and Elat. Site 2, south to Nuweiba, recorded highest Cu concentration, this may be due to pollution coming from Nuweiba harbor city. While, metal concentrations in sediments varied between 1.5 ppm for Cd and 20.79 ppm for Ni. Site 1 recorded the maximum metal concentrations for Cu, Zn, and Pb. Site 4, in Dahab recorded the highest levels of Cd and Ni (Table 4).

Table 4. Trace elements in surface water and sediments (ppb)

Element	Site 1	Site 2	Site 3	Site 4	Site 5	Site 6
Water						
Cu	0.26	0.28 +	0.22	0.13	0.11	0.09
Zn	2.51+	1.98	1.03	0.96	0.72	0.68
Cd	1.56 +	1.11	0.73	0.72	0.66	0.61
Pb	1.11 +	0.87	0.71	0,77	0.61	0.57
Ni	0.87 +	0.66	0.57	0.49	0.39	0.41
Sediment						
Cu	4.67 +	3.97	3.72	3.63	2.92	*
Zn	11.33 +	7.27	6.25	6.71	5.28	*
Cd	3.57	3.27	3.43	3.89 +	1.49	*
Pb	17.74 +	17.55	16.76	16.91	12.46	*
Ni	17.23	16.49	19.25	20.79 +	13.58	*

Note: *Rocky bottom with no sediments

Discussion

As it is obvious from the present study, the site has the lowest number of coral diseases (site 4) is characterized by the highest percentage cover of coral diseases indicating space monopolization and outbreak of those diseases. The same site is characterized by the highest percent cover of sediments, suggesting that sedimentation may increase the percent coral diseases at the expense of disease number. This could be due to decreased coral mortality with increased sedimentation, decreasing the available substrate or space for diverse diseases. Decreased resistance of the host coral caused by adverse environmental conditions may increase opportunistic diseases (Harvell et al. 1999) leading to increased coral mortality (Haapkyla et al. 2009). Changes in the population size (e.g. percentage cover), growth and reproduction of a community's primary producers (e.g. algae) and major framework builders will have impacts on the community. These changes are especially relevant given the longevous age structure of corals and, as compared to macroalgae, their relatively slow coral recruitment (Tougas and Porter 2002). This agrees with the results of the present study in which the highest percentage cover of algae/seagrasses is associated with the highest percentage cover of coral diseases (site 4), but the lowest percentage cover of each of algae/seagrasses, macroborers, and sediments is associated with the lowest percentage cover of coral diseases (site 5).

Jones et al. (2004) indicated that Fluorescence in situ hybridization (FISH) techniques and cloning, and analysis of the 16S rRNA genes from diseased coral tissue infected with atramentous necrosis, identified a mixed microbial assemblage in the diseased tissues particularly within the Alphaproteobacteria, Firmicutes, and Bacteroidetes. In the present study, the presence of the coral disease atramentous necrosis attaining the highest percentage cover and infecting the highest number of corals in all sites, is associated with vermetidae predation in site 1, *Tridacna* boring, vermetidae in site 2, mechanical breaking, vermetidae in site 3, filamentous algal overgrowth in site 4, mechanical breaking, vermetidae in sites 5 and 6. This is evidence that vermetidae predation, *Tridacna* boring and mechanical breaking may evoke growth of Alphaproteobacteria, Firmicutes, and Bacteroidetes, promoting the growth of filamentous algae. Outbreak of the coral disease atramentous necrosis may be greatly attributed to the terrestrial runoff caused by higher rainfall and in turn, decreased salinity (Harvell et al. 1999). This may have lead to increased stress on corals that may have reduced their immune responses, and/or increased virulence of pathogen (s) causing the disease. Decreased resistance of the host coral caused by adverse environmental conditions may also increase opportunistic diseases (Harvell et al. 1999). Results of the present study showed that the coral disease atramentous necrosis attained the highest percentage cover in all sites. This result, together with the fact that the present sites lie all in wadis being liable to terrestrial runoff , make the present results in agreement with that of Harvell et al. (1999).

Lesions with signs that are similar to ulcerative white spots (UWS) can be caused by fish bites. Parrotfish lesions can be distinguished by the presence of skeletal damage, while the tubelip wrasse, *Labrichthys unilineatus* will remove tissue without damaging the skeleton. Arboleda and Reichardt (2010) stated that the causative agent of the Indo-Pacific coral disease, *Porites* ulcerative white spot syndrome (PUWS), that affects *Porites* spp. and a few other coral genera has so far remained unidentified. In the present study, the association of the coral disease ulcerative white spots with vermetidae predation in both earlier and later stages of the disease, and in many cases with *Tridacna* boring in later stages of the disease is evidence that vermetidae is a causative agent of the disease but *Tridacna* spp. could take the disease as a suitable substrate. In the present study, this disease infected *Porites solida*, *Goniastrea retiformis*, *Favites flexuosa* and *Favia speciosa*.

Wooldridge (2010) indicated that coral's failure to prevent the division of zooxanthellae leads to ever-greater amounts of the photosynthesis-derived carbon to be diverted into the algae rather than the coral. This makes the energy balance required for the coral to continue sustaining its algae more fragile, and hence the coral loses the ability to maintain its parasitic control on its zooxanthellae leading to bleaching. In the present study, the white tips, which is some kind of bleaching, and *infecting only the two species Acropora humilis* and *Millepora dichotoma*, indicates that those two species are the most sensitive species that loses the ability to maintain its parasitic control on its zooxanthellae. Lesions of tissue discoloration (non-white pigmentation response) may be caused by borers, competitors, algal abrasion, fish bites, breakages, etc (Beeden et al. 2008). In the present study, the non-white pigmentation response was associated with vermetidae predation) while skeletal eroding band was associated with *Drupella* predation.

Yamashiro et al. (2000) found coral neoplasia to be associated with the global coral bleaching event (1998). In the present study, the coral disease tissue coral neoplasia, being found only in site 4, was associated with high sediment load, low salinity due to fresh water coming from the adjacent tourist showers which are just close to the shore.

Sites 5 and 6, having the most healthy, rich, and nice reef slopes in the Red Sea, have their diseases restricted only on the reef flat. Those diseases of the reef flat are associated with mechanical breaking (due to trampling on the reef flat), vermetidae predation, gastropod boring, aggressive coralline algal overgrowth, *Drupella* predation, and Parrot fish predation.

Diseases having lowest percentage cover are black band disease (site 1), white band disease (site 2), pigmentation response (site 3), coral hyperplasia (sites 4, 5), black band disease (site 6). However, Richardson (1998) indicated that the incidence and prevalence of black band disease may also increase when corals are stressed by sedimentation, nutrients, toxic chemicals, and warmer-than-normal temperatures. Histopathological examinations of diseased tissue of white band disease (WBD) revealed basophilic ovoid bodies up to 40 μm (Peters et al. 1983). Electron microscopy of thin sections of the ovoid bodies revealed that they were composed of Gram-negative bacteria, suggesting that these bacteria may be the causative agent of the disease. Pigmentation response is considered response of the coral host to a variety of stressors (e.g., unidentified pathogens, competition, predation, boring fauna, abrasion, etc.), suggesting that organism health is compromised (Raymundo et al. 2008).

White plague was reported in sites 3, 6. Richardson (1998) succeeded in isolating from diseased corals with white plague, a new species of *Sphingomonas* that infected healthy corals in laboratory experiments. Although the mechanism triggering coral neoplasia or tissue coral neoplasia is still unknown and thought to be a genetic mutation that may be the result of environmental conditions (Yamashiro et al. 2000), tissue coral neoplasia disease in the present study, being reported only in site 4, was associated with high sediment load, low salinity due to fresh water coming from the adjacent tourist showers which are just close to the shore. The disease in the present study was recognized as slightly hemispherical protuberances with fewer numbers of polyps per surface area, fewer zooxanthellae per polyp, finer skeletal structures than normal and reduced fecundity in coral neoplasia areas.

Infected corals relative to water and sediment quality

As human populations continue to increase, nutrients, terrigenous silt, pollutants and even pathogens themselves can be released into nearshore benthic communities (Raymundo et al. 2008). It was further discussed in that

book that while the link between anthropogenic stress and disease susceptibility is currently poorly understood, one hypothesis is that coral disease is facilitated by a decrease in water quality, particularly due to eutrophication and sedimentation. Growing evidence suggests that environmental and anthropogenic stressors are linked with coral disease and mortality in complex ways (Harvell et al. 2007). Like, many benthic filter feeders, corals assimilate differentially certain amounts of solid metals, mainly through ingestion of contaminated particles (Madkour 2011). Ammar et al. (2005) concluded that the toxic effect of a certain metal on a coral may have the growth rate, in turn skeletal density, decreased with increasing metal concentration. This may foster the infection of the coral with a certain disease as well. Site 1 has 9 diseases infecting 12 coral species, of which *Cyphastrea serailia* is infected with highest number of diseases (atramentous necrosis, dark spots disease, skeletal eroding band, and white plague). Site1 is characterized by the maximum metal concentrations of Zn, Cd, Pb and Ni in water and highest metal concentrations for Cu, Zn and Pb in sediments due to the high pollution load from large cities and harbors like Aqaba and Eilat. Site 2 has 9 diseases infecting 10 corals, of which *Millepora dichotoma* is infected with the highest number of diseases (white plague, atramentous necrosis, brown band disease, and coral hyperplasia). Site 2 is characterized by the highest Cu concentration in water due to pollution coming from Nuweiba harbor. Site 3 has 12 diseases infecting 19 species (highest number of all sites), of which *Porites lutea* is infected with the highest number of diseases (atramentous necrosis, brown band disease, white band disease, and skeletal eroding band). Site 4 has 6 diseases infecting 11 species, each of which is infected with only one disease except *Favia speciosa* and *Goniastrea retiformis* which are infected with 2 diseases for each one. Those fewer number of coral diseases in site 4 attained the highest percentage disease cover indicating space monopolization of those six diseases. This site attains the highest levels of Cd and Ni in sediments and highest percentage sediments. Site 5 has 8 diseases infecting 10 species, of which *Stylophora pistillata* is infected with the highest number of diseases (atramentous necrosis, pigmentation response, dark spots disease, and coral hyperplasia). Site 6 has 7 diseases infecting 8 species, of which *Porites solida* is infected with the highest number of diseases (ulcerative white spots, black band disease, white plague and white patches). Sites 5 and 6, having low number and lowest percentage cover of coral diseases, are characterized by lowest levels of trace elements in both water and sediments. Nice and ideal slopes also characterize them. Their coral diseases were reported only on the reef flat, probably because the reef flat is sheltered, lying below high mountains, a condition that may promote bacterial growth on the reef flat.

CONCLUSIONS AND RECOMMENDATIONS

Sedimentation may increase the percent of coral diseases at the expense of disease number**.** However, the highest percentage cover of algae/seagrasses is associated with the highest percentage cover of coral diseases. The coral disease atramentous necrosis attains the highest percentage cover in all sites which lie all in wadis being liable to terrestrial runoff. There is evidence that vermetidae predation could be causative agents of ulcerative white spots but *Tridacna* spp. could take the disease as a suitable substrate. The coral disease tissue coral neoplasia, being found only in site 4, was associated with high sediment load, low salinity due to fresh water coming from the adjacent tourist showers which are just close to the shore. Site 1 has 9 diseases infecting 12 coral species, of which *Cyphastrea serailia* is infected with highest number of diseases (atramentous necrosis, dark spots disease, skeletal eroding band, and white plague). This site is characterized by the maximum metal concentrations of Zn, Cd, Pb and Ni in water and highest metal concentrations for Cu, Zn, and Pb in sediments. Site 2 has 9 diseases infecting 10 corals, of which *Millepora dichotoma* is infected with the highest number of diseases (white plague, atramentous necrosis, brown band disease and coral hyperplasia). The highest Cu concentration in water characterizes this site. The fewer number of coral diseases in site 4 attained the highest percentage disease cover indicating space monopolization of those diseases. This site attained the highest levels of Cd and Ni in sediments and highest percentage sediments. Sites 5 and 6, having low number and lowest percentage cover of coral diseases, are characterized by the lowest levels of trace elements in both water and sediments.

Identifying knowledge gaps that impede understanding coral disease mechanisms, and limiting elucidation of causes, significance or control of coral disease. Recommending directed research and education to fill these knowledge gaps. Standardizing methods for investigating coral disease outbreaks considering both biotic and abiotic etiologies. Addressing issues relative to the management of coral reef resources; and fosters collaboration among partners, stakeholders, key marine resource management agencies, and regional networks. Developing guidance for the proper handling and containment of corals in infectious disease experiments. Fostering the development of a cohesive coral disease research community.

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