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# Distribution and Disease Prevalence of Coral Associated Bacteria at Some Impacted Red Sea Reefs

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## Abstract

Prevalence and distribution of coral disease were surveyed in three impacted sites at Red Sea coast. Prevalence were significant difference between studied reefs (ANOVA; F = 10.777, p < 0.0001). Sites closed to oil pollution (sites 1 and 2) displayed much higher levels of disease prevalence (25.3% ± 8.3 2 and 18.5% ± 3.1, respectively), than site 3 that close to landfilling (12.9% ± 2.1). Totally, 99coral braches, representing 20 species affected by at least 12 diseases. However, site 3 is characterized by the highest diversity (Simpson's Index = 0.081) and highest percentage cover of life coral (35.2% ± 11.4) showed the lowest coral diseases number (3). In opposite, 10 coral diseases were recorded at site 1, and 8 coral diseases at site 2. While, the coral disease atramentous necrosis attained the highest prevalence percentage at sites 1 and 2 (3.2% and 4.5%, respectively), did not recorded at site 3. Sediment damage disease recorded the highest prevalence percentage at site 3 (5.1%), but did not record at sites 1 and 2. Diseases having lowest percentage cover were white band disease (sites 1 and 2) and white tips disease. The most commonly distributed disease (atramentous necrosis) infected five corals in site 1 and six corals in site 2. The least commonly distributed disease (white tips) infected only two corals (*Acropora humilis* and *Acropora formosa*) at site 3. The genus Porites at sites was particularly vulnerable to all diseases. However, *Galaxea fasicularis* at site 3 recorded the highest percent cover of sediment damage disease. Coral disease atramentous necrosis is always associated with Vermetidae predation and Tridacna boring. While, Drupels predation is associated with skeletal eroding band. Aggressive filamentous algal overgrowth is associated with sediment damage disease at site 3. The pathogenic bacterium isolated from the diseased *Stylophora pistillata*, *Porites* sp., and *Acropora* sp., referred to as *Vibrio fischeri*.

**Keywords:** Coral; Disease prevalence; Distribution; Coral bacteria; *Vibrio* sp.; Red sea

## Introduction

Globally, we are witnessing on of coral reef decline. An important factor contributing to coral decline is the widespread infectious diseases, especially coral reef of the Red Sea [1,2]. Coral disease is an abnormal condition of an organism that causes organism dis-functions, associated with strange symptoms and signs ICRI / UNEP-WCMC, 2010. Disease causation may be result from pathogens, environmental stressors, or a combination of biotic and abiotic factors [3]. Biotic diseases are caused by pathogenic microorganisms such as viruses, bacteria, fungi, and protists and are often species-specific [4,5] and infectious [6]. While, abiotic diseases result from natural and anthropogenic stressors associated with exposure to pollutants [1,7]. However, biotic diseases may be associated with environmental threats that trigger the pathogenic process, or increase the rate of disease transmission [8,9]. However, Furby et al. [2] reported a strong correlation between the presence of lesion corals and decreased water quality. Other studies suggested a link between snail predators and disease spread such as outbreak of 'white syndrome' in Red Sea acroporid corals correlated with an outbreak in the coral livorous snail Drupellacornus [10]. Exposure to *Hermodice carunculata* (polychaete) infected with the bleaching pathogen *Vibrio shiloi* has been shown to induce bleaching in the Mediterranean coral *Oculina patagonicain* laboratory aquaria [11].

Threats to biodiversity are considered one of the major predicted impacts of new diseases and those infecting multiple species [12]. A relationship between diversity and disease prevalence, as high prevalence of a disease with multiple hosts can lower diversity [8]. In contrast, a high diversity reef could be predicted to be more resistant to an infectious disease, if host species are less abundant. However, previous reports found that most coral diseases affect only a few species [13]. While, the finding of this study and others, Richardson et al. [14], Green and Bruckner [15] and Weil et al. [16], it was reported that the total number of infected species increased with the several of geographical area, and that host breadth of most diseases is wider than previously thought. Sheridan et al. [17] reported that corals from reefs affected by river discharge and terrestrial sediments were more affected by white syndrome than reefs located far from any source of terrestrial run-off. Terrestrial runoff-affected reefs also displayed a wider diversity of coral species affected by this disease. The objectives of this survey were to identify the susceptibility to diseases among the Red Sea coral reefs species, and to determine the disease prevalence that were previously recorded within the country, to develop a species list of hosts affected by each disease observed and to describe any apparently 'new' syndromes or potential diseases. 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.

## Material and Methods

Coral disease distributions and prevalence were studied in three

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impacted sites at the Red Sea coast (Figure 1 and Table 1). These sites were Ras Gharib, Gble Elzyet and Crescent reef in front of National Institute of Oceanography and Fisheries-Hurghada. Surveys were conducted using SCUBA diving and Line Intercept transects (LIT), each covering an area measuring 20 m × 2 m. Five horizontal transects between 10 m and 12 m were completed at 7-8 dive locations. Transect were run to obtain a detailed description of substrate composition and the following substrate categories were noted: live hard coral, soft coral, recently killed hard coral, weathered rock with turf algae, rubble, sand, silt, canopy-forming algae and other encrusting organisms (such as sponges, ascidians, gorgonians). Additional information on poaching, pollution sources and other human stressors was obtained by personal observation. Species were identified in situ where possible or small pieces were collected for identification in the laboratory when necessary. Identification of specimens was based on [18,19]. Species diversity was determined using Simpson's index of concentration by combining species lists for all transects within a reef. Simpson's index is useful for such sampling as it does not depend on sample size or require a specific type of abundance distribution [20]. All diseased colonies within LIT were noted and the number of diseased vs. healthy colonies per species was counted so that disease prevalence could be calculated.

Disease prevalence was calculated as follows (see explanation above): number of diseased colonies per transect / total number of colonies per transect × 100.

To examine a possible connection between disease prevalence and coral species host abundance, the relative abundance of each species was calculated per reef as the total number of colonies per species as the total number of all colonies and this also allowed a calculation of species abundance relative to all other species. A 2-way ANOVA was used to detect differences in prevalence between sites.

In March 2014, totally 54 colonies from different coral reef species signs disease were haphazardly chosen and collected at 2 m to 3 m depth from the studied sites. Different size branches ranging from 1.2 cm to 3 cm were tagged and sampled using SCUBA and a long nosed bone cutter. Sample was kept in a separate plastic package under water to be, transported to NIOF laboratory. Each sample was washed twice with 0.2 µm filtered and autoclaved sea-water to remove any loosely associated microbes. Branches were crushed using a previously flamed sterilized mortar and pestle and 2 ml of filter sterilized seawater. The resulting mix was homogenized and plated on trypticsoya agar media (Difco) and incubated for no more than 24 h. [21]. The suspected bacterial isolates were isolated, purified using thiosulphate citrate bile salt sucrose agar (TCBS, Difco) and characterized using the standard biochemical method as described by [22]. Commercial miniaturized API 20E galleries (Bio Merieux) were also used according to the manufacturer's instructions.

## Results

A summary of the survey sites is presented in (Table 2). We identified a number of diseases and syndromes present on our surveyed reefs; these are described in Tables 3 and 4. (Figure 2) presents photographs of each disease. Between reef differences in prevalence were significant (ANOVA; $F = 10.777$, $p < 0.0001$). Sites closed to oil pollution (sites 1 and 2) displayed much higher levels of disease prevalence (25.3 ± 8.32 and 18.5 ± 3.1, respectively), than site 3 that close to landfilling (12.9 ± 2.1). The maximum percent of dead corals were also showed at sites 1 and 2 (25.3 ± 8.3 and 18.3 ± 5.7, respectively), while the lowest one was at site 3 (19.5 ± 4.3). However, sites 1 and 2 recorded the highest percent cover of soft coral; they recorded the lowest percent of live hard corals.

A total of 12 coral diseases were detected on transects on the reefs of three studied sites. The maximum number of coral diseases was recorded at site 1 (10 coral diseases), while the minimum one was at site 3 (5 coral diseases). However the site having the lowest number of coral diseases (site 3) is characterized by the highest diversity (Simpson's Index = 0.081) and highest percentage cover of life coral (35.2 ± 11.4). Despite, the coral disease atrementous necrosis were not recorded at

| | Sites | Latitudes | Longitudes | Possible stressors |
|---|---|---|---|---|
| 1 | **Ras Gharib** | 28°21'44.62"N | 33° 5'47.97"E | Oil pollution and sewage |
| 2 | **Gabel El-Zayet** | 27°45'17.85" | 33°33'47.21" | Oil pollution |
| 3 | **Crescent Reef** | 27°17'4.41"N | 33°46'36.79"E | Poaching, Swimming and Landfilling |

**Table 1:** Latitudes and longitudes of the study sites.

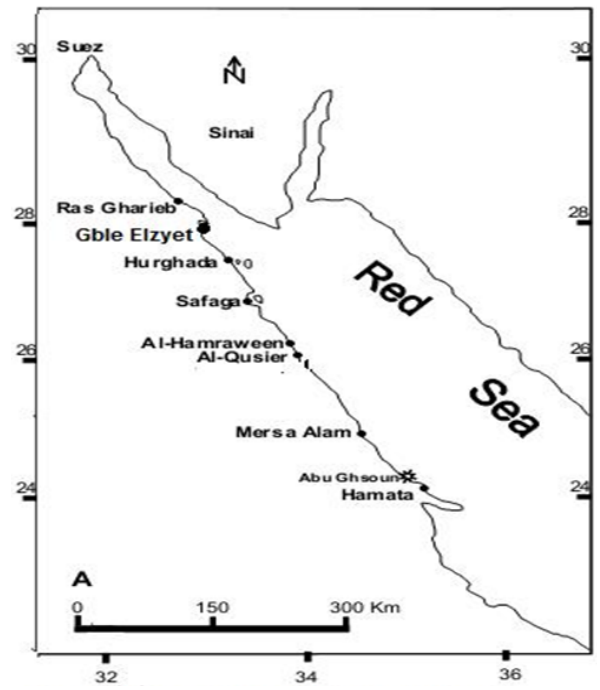


**Figure 1:** Location map of the study sites.

| Sites | Simpson's Ind | NCD | Percent substrate composition | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | | | DP | DCC | LHC | DC | SC | S&R | Algae and Sea grass |
| **1** | 0.17 | 10 | 23.3 ± 3.2 | 16.2 ± 5.2 | 20.1 ± 5.4 | 25.3 ± 8.3 | 21.2 ± 6.4 | 9.7 ± 2.3 | 7.5 ± 1.7 |
| **2** | 0.12 | 8 | 18.5 ± 3.1 | 14.3 ± 4.1 | 27.2 ± 6.3 | 22.8 ± 6.7 | 18.3 ± 5.7 | 9.1 ± 2.2 | 8.3 ± 1.9 |
| **3** | 0.081 | 5 | 12.9 ± 2.1 | 11.2 ± 3.6 | 35.2 ± 11.4 | 19.5 ± 4.3 | 12.7 ± 4.5 | 12.5 ± 3.1 | 8.9 ± 1.8 |

**Table 2:** Site descriptions of reefs surveyed for disease. NCD = Number Coral Disease; DP = Disease Prevalence; DCC = Disease Coral Percent Cover; LHC = Live Hard Coral; DC = Dead Coral; SC = Soft Corals; S and R = Sediments and Rocks. Percent substrate composition values are mean ± SE. Simpson's index values close to 0 signify higher diversity and those approaching 1 signify lower diversity.

| Site 1 | Coral disease | Host species | DP | DCC | Remarks |
|---|---|---|---|---|---|
| | White band disease | *Porites lutea* | 0.2 | 0.3 | Mechanical breaking Aggressive filamentous algal overgrowth |
| | | *Montipora verrucosa* | 0.1 | 0.2 | |
| | | *Stylophora pistillata* | 0.4 | 0.4 | |
| | | *Acropora humilis* | 0.1 | 0.2 | |
| | | *Millepora dichotoma* | * | * | |
| | | **Total** | **0.8** | **1.1** | |
| | White spots disease | *Porites rus* | 0.4 | 0.5 | Gastropod boring Gastropod drilling |
| | | *Astreopora myriophthalma* | 0.1 | 0.3 | |
| | | *Porites solida* | 0.5 | 0.5 | |
| | | **Total** | **1** | **1.3** | |
| | White patches | *Acropora valida* | 0.2 | 0.5 | Parrot fish predation |
| | | *Acropora hemprichi* | 0.3 | 0.3 | |
| | | *Favites flexusa* | 0.8 | 1 | |
| | | *Acropora tenuis* | 0.2 | 0.3 | |
| | | **Total** | **1.5** | **2.3** | |
| | Atramentous necrosis | *Millepora platyphyla* | 0.1 | 0.3 | Mechanical breaking Vermetidae predation Tridacna boring Gastropod drilling |
| | | *Millepora dichotoma* | * | * | |
| | | *Porites lutea* | 1.3 | 1.6 | |
| | | *Lobophyllia corymbosa* | 0.2 | 0.3 | |
| | | *Favites flexusa* | 1.1 | 1.3 | |
| | | *Acropora tenuis* | 0.5 | 0.7 | |
| | | *Goniasatrea pectinata* | * | * | |
| | | **Total** | **3.2** | **4.2** | |
| | Brown band | *Montipora informis* | 0.3 | 0.5 | Gastropod drilling Vermetidae predation |
| | | *Montipora verrucosa* | * | * | |
| | | *Porites lutea* | 0.6 | 0.7 | |
| | | *Favites flexusa* | 0.4 | 0.6 | |
| | | **Total** | **1.3** | **1.8** | |
| | Pigmentation response | *Stylophora pistillata* | 0.3 | 1 | Ciliate infection Mechanical breaking |
| | | *Acropora hemprichi* | * | * | |
| | | *Porites rus* | 0.1 | 0.2 | |
| | | **Total** | **0.4** | **1.2** | |
| | Partial bleaching | *Goniastrea* | * | * | Mechanical breaking Aggressive filamentous algal overgrowth |
| | | *Porites rus* | 1.1 | 1.7 | |
| | | *Stylophora pistillata* | 1 | 1.5 | |
| | | *Acropora tenuis* | 0.3 | 0.5 | |
| | | *Montipora informis* | * | * | |
| | | **Total** | **2.4** | **3.7** | |
| | Skeletal eroding band | *Platygyra daedalea* | 0.8 | 1.2 | Drupella predation Gastropod boring Mechanical breaking |
| | | *Montipora informis* | 0.3 | 0.5 | |
| | | *Porites lutea* | 0.7 | 0.9 | |
| | | *Stylophora pistillata* | 0.8 | 1 | |
| | | *Favites flexusa* | 0.2 | 0.3 | |
| | | **Total** | **2.8** | **3.9** | |
| | Dark spots disease | *Porites solida* | 0.6 | 0.9 | Aggressive coralline algal overgrowth Gastropod boring Mechanical breaking |
| | | *Stylophora pistillata* | 0.5 | 0.8 | |
| | | *Acropora humilis* | 0.1 | 0.2 | |
| | | *Leptoseris incrustans* | 0.1 | 0.2 | |
| | | **Total** | **1.3** | **2.1** | |
| | Rapid wasting | *Pavona explanulata* | * | * | Parrot fish predation Aggressive filamentous algal overgrowth |
| | | *Stylophora pistillata* | 0.6 | 0.8 | |
| | | *Porites solida* | 0.5 | 0.7 | |
| | | *Echinopora gemmacea* | 0.3 | 0.2 | |
| | | **Total** | **1.4** | **1.7** | |
| | **Total** | | **16.2** | **23.3** | |

| Site 2 | Coral disease | Host species | DP | DCC | Remarks |
|---|---|---|---|---|---|
| | White band disease | *Porites lutea* | 0.4 | 0.23 | Mechanical breaking Aggressive filamentous algal overgrowth Gastropod boring |
| | | *Montipora verrucosa* | 0.2 | 0.1 | |
| | | *Stylophora pistillata* | 0.3 | 0.17 | |
| | | *Acropora humilis* | 0.2 | 0.1 | |
| | | **Total** | **1.1** | **0.6** | |
| | Dark spots disease | *Porites rus* | 0.7 | 0.6 | Gastropod drilling, coralline algal overgrowth Gastropod boring Mechanical breaking |
| | | *Astreopora myriophthalma* | 0.3 | 0.2 | |
| | | *Porites solida* | 0.5 | 0.6 | |
| | | **Total** | **1.5** | **1.4** | |
| | White patches | *Acropora valida* | 0.2 | 0.3 | Parrot fish predation Aggressive filamentous algal overgrowth |
| | | *Acropora hemprichi* | 0.5 | 0.65 | |
| | | *Favites flexusa* | 0.6 | 0.7 | |
| | | *Acropora tenuis* | 0.1 | 0.15 | |
| | | **Total** | **1.4** | **1.8** | |
| | Atramentous necrosis | *Millepora platyphyla* | 0.2 | 0.09 | Mechanical breaking Tridacna boring Vermetidae predation Gastropod drilling |
| | | *Porites lutea* | 1.7 | 1.3 | |
| | | *Lobophyllia corymbosa* | 0.5 | 0.31 | |
| | | *Favites flexusa* | 1.4 | 0.4 | |
| | | *Acropora tenuis* | 0.4 | 0.2 | |
| | | *Goniasatrea pectinata* | 0.3 | 0.1 | |
| | | **Total** | **4.5** | **2.4** | |
| | Brown band | *Montipora informis* | 0.2 | 0.3 | Vermetidae predation |
| | | *Montipora verrucosa* | 0.4 | 0.3 | |
| | | *Porites lutea* | 0.7 | 0.4 | |
| | | *Favites flexusa* | 0.3 | 0.2 | |
| | | **Total** | **1.6** | **1.2** | |
| | Pigmentation response | *Stylophora pistillata* | 1.5 | 1.3 | Vermetidae predation Gastropod drilling Mechanical breaking |
| | | *Acropora hemprichi* | 0.8 | 1.1 | |
| | | *Porites rus* | * | * | |
| | | **Total** | **2.3** | **2.4** | |
| | Partial bleaching | *Goniastrea retiformis* | 0.4 | 0.3 | Aggressive filamentous algal overgrowth Mechanical breaking |
| | | *Porites rus* | 1.6 | 0.9 | |
| | | *Stylophora pistillata* | 1.2 | 0.8 | |
| | | *Acropora tenuis* | * | * | |
| | | *Montipora informis* | 0.9 | 0.6 | |
| | | **Total** | **4.1** | **2.6** | |
| | Skeletal eroding band | *Platygyra daedalea* | * | * | Drupella predation Gastropod boring Mechanical breaking |
| | | *Montipora informis* | 0.3 | 0.2 | |
| | | *Porites lutea* | 0.4 | 0.32 | |
| | | *Stylophora pistillata* | 1.1 | 0.97 | |
| | | *Favites flexusa* | 0.2 | 0.11 | |
| | | **Total** | **2** | **1.6** | |
| | **Total** | | **18.5** | **14.3** | |

| | | | | | |
|---|---|---|---|---|---|
| Site 3 | **White tips** | *Acropora humilis* | 0.4 | 0.38 | Aggressive filamentous algal overgrowth |
| | | *Acropora Formosa* | 0.5 | 0.42 | |
| | | **Total** | **0.9** | **0.8** | |
| | **Dark spots disease** | *Porites rus* | 0.7 | 0.46 | coralline algal overgrowth Gastropod boring Mechanical breaking |
| | | *Astreopora myriophthalma* | 0.1 | 0.09 | |
| | | *Montipora informis* | 0.1 | 0.09 | |
| | | *Porites solida* | 0.2 | 0.16 | |
| | | **Total** | **1.1** | **0.8** | |
| | **Atramentous necrosis** | *Porites lutea* | 0.6 | 0.29 | Tridacna boring Drupella predation Vermetidae predation |
| | | *Lobophyllia corymbosa* | 0.8 | 0.35 | |
| | | *Favites flexusa* | 0.7 | 0.28 | |
| | | *Acropora tenuis* | 0.1 | 0.09 | |
| | | *Goniasatrea pectinata* | 0.1 | 0.09 | |
| | | **Total** | **2.3** | **1.1** | |
| | **Sediment damage** | *Stylophora pistillata* | 0.8 | 0.68 | Aggressive filamentous algal overgrowth Mechanical breaking coralline algal overgrowth |
| | | *Acropora hemprichi* | 0.4 | 0.33 | |
| | | *Galaxea fasicularis* | 2.3 | 2.26 | |
| | | *Favites flexusa* | 0.7 | 0.57 | |
| | | *Porites rus* | 0.9 | 0.76 | |
| | | **Total** | **5.1** | **4.6** | |
| | **Partial bleaching** | *Goniastrea retiformis* | * | * | Mechanical breaking Aggressive filamentous algal overgrowth |
| | | *Porites rus* | 0.7 | 0.9 | |
| | | *Stylophora pistillata* | 2.2 | 2.3 | |
| | | *Acropora tenuis* | 0.6 | 0.7 | |
| | | *Montipora informis* | * | * | |
| | | **Total** | **3.5** | **3.9** | |
| | **Total** | | **12.9** | **11.2** | |

**Table 3:** Percent disease prevalence and percent cover of disease affecting coral reef species at the studied sites. DP = Disease Prevalence; DCC = Disease Coral Percent Cover.

| Tests | Result | Tests | Result |
|---|---|---|---|
| Colony shape | Round | Colony colour | White Creamy |
| Gram stain | -Ve rods | Motility | +Ve |
| Cytochrome oxidase | +Ve | Catalase | +Ve |
| Growth in 0% NaCl | - | Growth in 1.5% NaCl | + |
| 3% NaCl | + | 6% NaCl | + |
| **API20E** | | | |
| ONPG | + | Gelatin hydrolysis | - |
| Arginine dihydrolase | - | Glucose | - |
| Lysine decarboxylase | + | Manitol | - |
| Ornithine decarboxylase | - | Inositol | - |
| Citrate | - | Sorbitol | - |
| H2S | - | Rhaminose | - |
| Urea hydrolysis | + | Sucrose | - |
| Tryptophanedeaminase | - | Malonate | - |
| Indole | - | Adonitol | - |
| Voges-Proskauer | - | Arabinose | - |
| Raffinose | - | Salicin | - |
| Xylose | - | Lactose | - |

**Table 4:** Results of the biochemical characterization of the *Vibrio fischeri* isolates.

site 3, it attained the highest prevalence percentage at sites 1 and 2 (3.2 and 4.5, respectively). On the other hand, the Sediment damage disease recorded the highest prevalence percentage at site 3 (5.1), but the same disease did not recorded at sites 1 and 2 Diseases having lowest percentage cover are white band disease (sites 1 and 2) and white tips disease (Figure 3).

We found 99 coral braches, representing 20 species affected by at least 12 diseases. The most commonly distributed disease (atrementous necrosis) infected five corals in site1and six corals in site 2. However, the least commonly distributed disease (white tips) infected only two corals (*Acropora humilis* and *Acropora formosa*) at site 3. The genus Porites at sites was particularly vulnerable to all diseases we observed; indeed, white patches disease was the only disease noted that did not affect the genus. However, *Galaxea fasicularis* at site 3 recorded the highest percent cover of sediment damage disease. Coral disease atramentous necrosis is associated with Vermetidae predation, and in many cases with Tridacna boring. However, Vermetidae predation is also associated with tissue discoloration (non-white pigmentation response) while Drupella predation is associated with skeletal eroding band. Aggressive filamentous algal over growth is associated with Sediment damage disease at site 3.

## Isolation and characterization of the bacterial strains

54 bacterial strains have been isolated from the infected *Stylophora pistillata, Porites* sp., and *Acropora* sp. by different diseases. Of which; 38 isolates were not biochemically similar and identified through their morphology, conventional biochemical tests and API20E system tests as *Vibrio fischeri* isolates (Table 4), and as unknown a typical vibrio species (16 isolates).

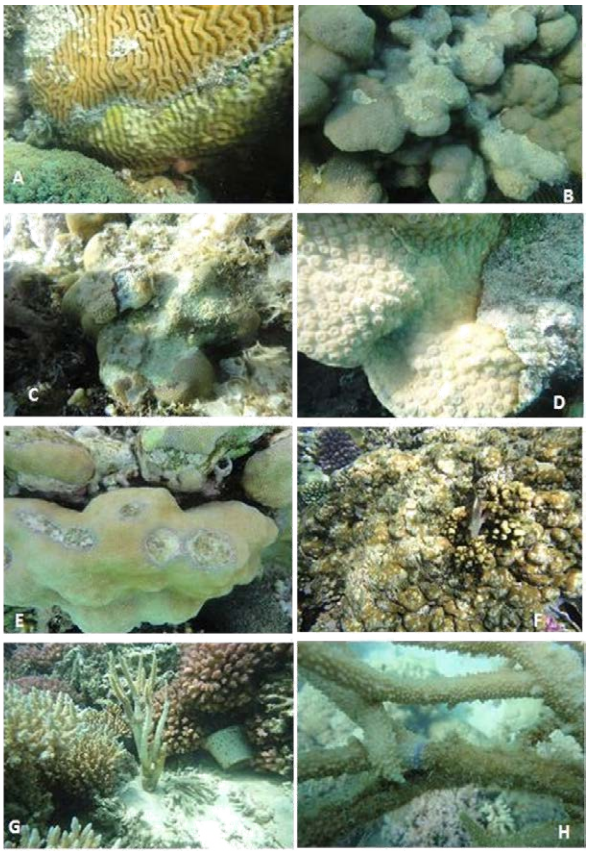


**Figure 2:** Most common diseases and syndromes in Red Sea. Compromised tissue responses affecting massive coral (A) Platygyra and (B) Porites, Black band disease affecting massive coral (C) Montipora and encrusting coral (D) Echinopora, Pigmentation response affecting massive coral (E) Porites, Ulcerative white spots affecting (F) Porites, White syndrome affecting (G) Acropora and White band disease affecting (H) Acropora.

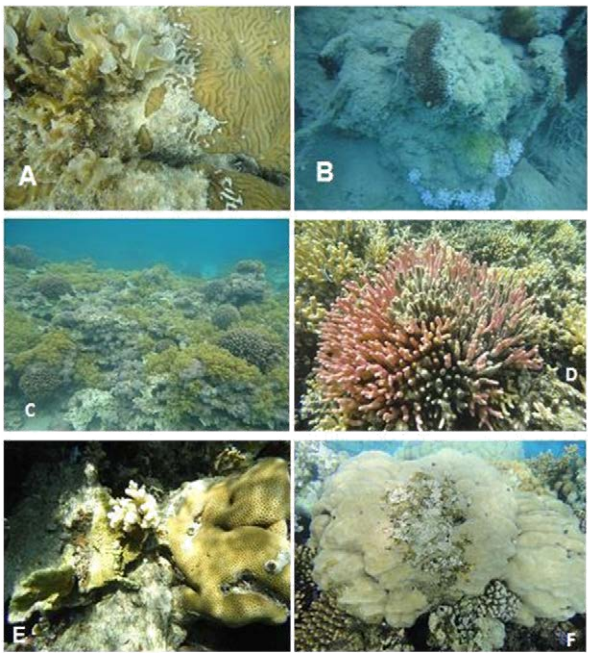


**Figure 3:** Sediment damage on (A) Platygera and (B) Galaxea in site 1. Over growth of (C) soft corals and partial dead colony of (D) Stylophora in site 2. (E) Corallivorous Vermetidae on Favia and (F) Drupella on Porites in site 3.

## Discussion

Our surveys have provided baseline information on the prevalence of coral disease affecting reefs in the Red Sea, a country within the global center of coral reef biodiversity. As it is obvious from the present study, diseases prevalence were significant differences between reefs, of which reefs closed to oil pollution (sites 1 and 2) displayed much higher levels of disease prevalence, % cover of diseases and % cover of dead corals, than reef close to landfilling (site 3). This coincides with the result of Al Hammady [7,23], that oil pollution induced coral disease, moderately and severe bleaching and cause reproduction damage. However, Gong et al. [24] identified a knowledge gaps that needs further research to facilitate sounder assessment of fate and effect of oil pollution on marine ecosystem. Furby et al. [2] reported a strong correlation between the presence of lesioned corals and the elevation of ammonium concentrations, and also reported changes in microbial communities with decreased water quality. Disease causation may due to pathogens, environmental stressors, or a combination of biotic and abiotic factors. Biotic diseases are caused by the infection of pathogenic microorganisms such as viruses, bacteria and fungi [4,6]. Abiotic diseases result from both natural and human induced environmental stressors including change in ambient conditions or exposure to pollutants [1]. Moreover, biotic diseases may be associated with environmental stressors that: (1) hinder the resistance of host organisms, (2) promote growth and virulence of pathogens, (3) trigger the pathogenic process, or (4) increase the rate of disease transmission [8,9]. While, abiotic diseases may be exacerbated by secondary opportunistic infections [4].

The site having the lowest number of coral diseases (site 3) is characterized by the highest percentage cover of coral disease sediment damage, indicating space monopolization and outbreak of this disease. The higher sedimentation at site 3 may increase the percent cover of sediment damage coral disease at the expense of disease number. This could be due to increase coral mortality with increased sedimentation, increasing the available substrate or space for diverse diseases. On the other hand, decreased resistance of the host coral caused by environmental threats could increase opportunistic diseases [25] leading finally to increased coral mortality [26]. 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 macro algae, their relatively slow coral recruitment [27]. This agrees with the present results in which the highest percentage cover of algae / sea grasses is associated with the highest percentage cover of sediment damage coral diseases (site 3), but the lowest percentage cover of each of algae / sea grasses and sediments is associated with the highest percentage cover of coral diseases (sites 1 and 2). In contrast, Ammar et al. [1] found that the highest percentage cover of algae / sea grasses is associated with the highest percentage cover of coral diseases at Eel Garden at Dahab-northern Red Sea. Sheridan et al. [17] reported that corals from reefs affected by river discharge and terrestrial sediments were more affected by white syndrome than reefs located far from any source of terrestrial runoff. Terrestrial runoff-affected reefs also displayed a wider diversity of coral species affected by this disease.

At present, the pathogenic bacteria *Vibrio* sp. was isolated the from atramentous necrosis disease. Ritchie and Smith [28] reported a similar result, that white band disease is always associated with *Vibrio charcharia*, but attempts to fulfill Koch's postulates with this bacterium have been unsuccessful to date [28]. While, Ben-Haim and Rosenberg [3] used 16S rDNA sequence and isolate the pathogenic bacterium *Vibrio coralyticus* as an new Vibrio genus from the diseased tissue of *Pocillopora damicornis*.

A link between snail predators and increased disease prevalence has been suggested by other studies. Antonius and Reigl [10] reported that, an outbreak of white syndrome in the Acroporids Red Sea coral was correlated with an outbreak of the corallivorous snail *Drupella cornus*. Moreover, Miller et al. [29] found that, the snail predator *Drupella cornus* is common on Florida acroporid corals, and many areas of the Caribbean [30]. Exposure to *Hermodice carunculata* (polychaete) infected with the bleaching pathogen *Vibrio shiloi* has been shown to induce bleaching in the Mediterraneancoral *Oculina patagonica* in laboratory aquaria [11]. *H. carunculatais* also a common predator of *Acropora cervicornis* in the Florida, feeding by engulfing a branch tip and digesting the coral tissue, leaving bare skeleton behind [31].

Threats to biodiversity are considered one of the major predicted impacts of new diseases and those infecting multiple species [12]. We expected a relationship between diversity and disease prevalence, as high prevalence of a disease with multiple hosts can lower diversity [8]. In contrast, a high diversity reef could be predicted to be more resistant to an infectious disease, if host species are less abundant. Our diversity calculations vary widely between sites and the reef with the highest diversity, sites 3, had the healthiest reef community, and least coral disease prevalence. However, a previous report stated that most coral diseases affect only a few species [13]. Results from this study and other reports Richardson et al. [14], Green and Bruckner [15] and Weil et al. [16], show that the total number of species diseased is increased according to geographical location, and that host breadth of most diseases is wider than previously have been thought. The lowest diversity of coral reef at sites 1 and 2 could be explained by the fact that, soft corals at these sites, which are mostly abundant, common or frequent in contrast to stony corals which are mostly scarce. This seems

that, soft corals have higher growth rate, and higher competition for space and recovery than stony corals [32]. This agrees with Benayahu and Loya [33] who found that soft corals are the major competitors overgrowing hard corals. Benayahu [34] stated that, soft corals compete for space with true stony corals and cause their death. Although one of our objectives was to examine the effect of reef health on disease prevalence, the rarity of truly healthy reefs within our sampling regions made it impossible to conduct such a comparison. A larger scale survey is needed to elucidate relationships between disease prevalence and reef health, water quality, hard coral cover and hard coral diversity. Thus, there appears to be a low correlation between reef health and disease out breaks and apparently healthy reefs show high prevalence of disease [13,35-37].

## Conclusion and Recommendation

The present study found that impacted Red Sea reefs associated with diseases and syndromes. Moreover, sites closed to oil pollution displayed much higher levels of disease prevalence than site close to landfilling. The most commonly distributed disease was atramentous necrosis and the least one was white tips at all sites. The genus Porites was particularly vulnerable to all diseases. The pathogenic bacterium isolated from the diseased *Stylophora pistillata, Porites* sp., and *Acropora* sp. was *Vibrio fischeri*.

It is clear that research on coral disease pathogenesis requires more laboratory and field studies. Recommending directed research and education to fill these knowledge gaps. Continuous efforts at threated reefs are still needed to understand coral diseases necessary to contribute to the effective maintenance of coral reefs.

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