2. Review of coral diseases:

Disease is defined as any impairment (interruption, cessation, proliferation, or other disorder) of vital body functions, systems or organs (Peters 1997). Importance of pathogens as regulators of coral populations in the tropical marine environments is poorly understood (Peters 1988). A disease not only kills the coral colony, but also exposes a substratum from the diseased corals for a new recruit (Connel and Keough 1985). Some disease causing organisms of corals have been identified and mechanisms of mortality have been studied in some diseases, such as the black band disease. However, many others remain poorly investigated. Infectious diseases in corals are different from genetic diseases found in them, such as unusual growth patterns resembling tumors, neoplasms or galls, which have been analogous to cancers (Goreau et al. 1998). Infectious diseases are not to be confused with overgrowth of other animals (Goreau et al. 1998). Often vectors spread pathogenic agents. For example, parrotfish is believed to spread the pathogens through oral mucus (Antonius 1981a). Diseases can be classified as biotic and abiotic (Peters 1997). In biotic diseases, various biological factors are responsible for the disease, while in abiotic diseases, abnormal features among environmental factors such as salinity, temperature, ultraviolet light, sedimentation or exposures to toxic chemicals may cause disorder. Biotic and abiotic factors are often interrelated. Physiological disorders often result from extreme environmental conditions. For example, corals expel zooxanthellae during times of anomaly in sea surface temperature (Bruno et al. 2001). Many coral diseases are reported from all
over the world in hexacorals and octocorals. These diseases are discussed in
detail below.

2.1. Diseases in hexacorals:

2.1.1. Black band disease (BBD):

Antonius (1973) first reported the black band disease (BBD) on corals
in Caribbean reefs. The BBD is a major factor in decline of coral reefs in
Florida reefs (Porter and Meier 1992; Peters 1993). The corals that are more
susceptible are Montastrea sp., Diploria sp. and Colpophyllia sp. (Antonius
1981a and Rützler et al. 1983). The cyanobacterium Oscillatoria
submembranacea (Antonius 1973) later renamed as Phormidium corallyticum
by Rützler and Santavy in (1983) was suspected to be a pathogen. The black
band disease was named based on the appearance of a black line covering
the coral tissue between the polyp-depleted skeleton and the healthy portion
of the colony (Antonius 1981b; Rützler et al. 1983). This band contains a
microbial consortium consisting mainly Phormidium corallyticum. The
darkness of the band is due to the presence of the dark accessory
photosynthetic pigments, phycoerythrin and phycocyanin (Richardson 1996;
Richardson and Carlton 1993). Thickness of the band is about 1 mm to 10
mm (Richardson 1996). It migrates at the speed of about >1 cm day⁻¹, but
bright sunlight enhances the speed of the cyanobacterial movement (Rützler
and Santavy 1983). As they migrate, the cyanobacterial filaments lyse the
coral tissue underneath, resulting in the exposure of the skeleton (Richardson
1996). In addition to the cyanobacterium, the band consists of numerous
heterotrophic bacteria (Garrett and Ducklow 1975), fungi (Ramos-Flores
1983) and sulphur-reducing bacteria like Desulfovibrio and the sulphur
oxidizer, *Beggiatoa* sp. (Ducklow and Mitchell 1979; Antonius 1981b). The active front end of the band is dominated by *P. corallyticum* and the rear end is dominated by *Beggiatoa* sp. (Rützler and Santavy 1983). The band contains, scavenging bacteria such as *Flexibacter*, *Saprospira* types, *Nitrosomonas* and ciliates such as *Philaster* and *Porpostoma* (Rützler and Santavy 1983). It was proposed that neither *P. corallyticum* nor the *Beggiatoa* sp. was the primary pathogen and their combined effect in the formation of the disease was postulated (Ducklow and Mitchell 1979). The filaments of the cyanobacterium, *P. corallyticum* migrated towards the coral tissue (Rützler et al. 1983). Widening of the band is diurnal in nature. The movement of the cyanobacterial filament in the BBD was observed to be light dependent (Richarson 1996) and proportional to the temperature, the activity being higher in the warmer months (25° C) (Antonius 1985; Rützler et al. 1983). The black band migrates at an average speed of 3.5 cm day⁻¹ (Rützler et al. 1983) and the speed attains the maximum velocity of 10 cm day⁻¹ depending upon the environmental conditions (Antonius 1981b). The cyanobacterial filaments showed forward movement at the front edge of the band and backward movement at the rear end. This movement facilitates more light capturing capacity for the mat (Richardson 1996). The scleractinian corals are more susceptible when the corals are suffering from acute white band disease (WBD) or when bordered by a dense growth of green algae (Antonius 1985). The occurrence of the cyanobacterium *Phormidium* among the epilithic growth of chlorophytes was believed to cause BBD in the corals having white band disease (Antonius 1985). The infection is contagious. The healthy colonies
kept 2 mm apart from the diseased colony were infected with the BBD pathogen (Rützler and Santavy 1983).

Water samples from the field did not show the presence of the cyanobacterial pathogen. Ducklow and Mitchell (1979) tried to induce the disease in corals without stirring the water so that less oxygen or anoxic condition will be created around the coral polyps. However, this experiment did not yield the disease symptom. Instead, it showed hypoxia-related syndromes and proliferation of the anaerobic bacterium, *Beggiatoa sp*. The authors (Ducklow and Mitchell 1979) concluded that the presence of the cyanobacterium *P. corallyticum* is necessary for the formation and maintenance of the black band. Rützler et al. (1983) proved that the presence of the cyanobacterium *P. corallyticum* not only maintains the band but also is the cause for the black coloration of the band and destroys the tissue. Inoculation of the BBD material from diseased corals onto healthy ones resulted in the black band. These workers removed the colored cyanobacterial filaments from heterotrophic bacterial populations in the black band mat, under a microscope. The resulting inoculum, free of the cyanobacterium did not cause the black band. The inoculum that contained few *P. corallyticum* filaments started showing migration towards the coral tissues. The artificial inoculation experiments with *P. corallyticum* proved that the cyanobacterium is the pathogen. Healthy corals in the wild expelled the inoculum of the pathogen within few hours of inoculation by their ciliary action. Stressed specimens maintained in the laboratory were affected by the cyanobacterium within 4-6 hours of inoculation.
The main cause of the coral tissue mortality covered by *P. corallyticum* is due to sulphur accumulation underneath the band where the tissue is undergoing lysis. The bottom of the band is always anoxic or sulphide enriched (Richardson and Carlton 1993). Levels of >800 μM sulphide during the night time were estimated in the band (Richardson 1996). The formation of the sulphide underneath the black band in the BBD is because of the presence of the sulphur-reducing bacteria such as the *Desulphovibrio* in the microbial consortium of the black band disease (Garrett and Ducklow 1975). This sulphide inhibits electron flow in a manner similar to the inhibition of aerobic respiration by cyanide (Richardson and Carlton 1993). Thus the sulphide accumulation inhibits the electron flow in the photosystem II of the cyanobacteria, which then revert to anaerobic photosynthesis, in which the sulphide functions as an electron donor to the photosystem I (Richardson 1996). The anoxia and the higher sulphur concentrations in the range 40-400 μM (Richardson and Carlton 1993) are harmful to the obligatorily aerobic corals, which, like other aerobic marine animals, may have less tolerance to anoxia (Vismann 1991; Llanso 1991).

2.1.2. White band disease (WBD): -

Little work has been done on the WBD (Gladfelter 1982; Peters 1984 and Antonius 1981a &b; Ritchie and Smith 1998). WBD is a sharp line of advance where the distally located brown zooxanthellae bearing coral tissue is cleanly and completely removed from skeleton, leaving a sharp white zone about 1 cm wide that grade proximally into algal successional stages (Gladfelter 1982). Zooxanthellae-bearing coral tissue peels off from the skeleton into little balls, held together with strands of mucus. These tissue
Balls are carried away by the ciliary action of the corals. The white band spreads from the basal region of the colony to the tip. No environmental factors that alter the speed of the white band are known. The white band causes substantial decrease in skeletal deposition. The WBD tissues contain both gram positive and gram-negative bacteria. The disease is not transferable and it does not spread to the adjacent colony even when these colonies are fused naturally. The WBD does not respond to antibiotics as the black band does (Antonius 1985). WBD is found to occur only in the corals Acropora palmata, Diploria strigosa, Montastrea annularis and Mycetophyllia ferox (Antonius 1981a). This disease is considered to be the slower version of the shut down reaction (SDR) (Antonius 1981a) that is characterized by spontaneous disintegration of the coral tissues resulting in the denuded skeleton. An association of a rod shaped gram-negative bacterium was found in histological sections of WBD-affected Acropora palmata (Peters 1984; Santavy and Peters 1997). This bacterium was found in healthy specimens as well. But the diseased specimens had higher abundance of these bacteria than the healthy one. Two types of tissue loss, designated as type I and type II have been demonstrated in association with the WBD (Ritchie and Smith 1998). In type I, tissue loss shows active tissue necrosis along the disease line, whereas in type II, different stages of disease processes that varies from the tissue destruction to the exposure of the coral skeleton were found. Studies on the type II specimens have shown the presence of the gram-negative bacterium, Vibrio charcharii (Ritchie and Smith 1995, 1998) as a conspicuous one among the other bacteria. Koch's postulate experiments
have yet to prove the role of this bacterium associated with the WBD in causing the disease.

2.1.3. White plague: -

The white plague was first reported from Florida Keys (Dustan 1977) and recently also from the Puerto Rico reefs (Bruckner and Bruckner 1998). The terms ‘white plague’ and ‘plague’ are synonymous (Dustan and Halas 1987). It was found later that the disease affects many more species than previously described (Dustan and Halas 1987). The affected colonies had no visible microbial flora on the surface of the colony. Microscopic studies revealed tissue degeneration and remnants of zooxanthellae, giving a bleached effect to the diseased colonies (Richardson et al. 1998a). In north Florida Keys, 17 to 43 species were susceptible to the disease and 38% of the most susceptible species were dead within a period of 11 months (Richardson et al. 1998 a &b). Two variants of the plague epizootics were found based on the rate of tissue destruction. The plague type I spread more slowly compared to the plague type II that spreads almost a cm day$^{-1}$ (Richardson et al. 1998b). The plague II affected a stretch of more than 400 Km of Florida reefs between 1995 and 1997 (Richardson et al.1998b). A single bacterium, *Sphingomonas* sp. was isolated from the diseased corals (Richardson et al. 1998a). The bacterium was later proved to be the pathogen through laboratory studies. This disease is transmissible and occurs seasonally (Richardson et al. 1998a).

2.1.4. Rapid wasting syndrome (RWS): -

The Rapid wasting syndrome is most prevalent in the Caribbean. The disease leaves an eroded skeleton as it spreads laterally on the colony. The
skeletal erosion may be as deep as 2 cm (Goreau et al. 1998). RWS is the synonym for the Rapid Wasting Disease (RWD) (Cervino et al. 1998). The RWS mostly affects colonies of Montastrea annularis and Colpophyllia natans. A fungus and a ciliate were found in the microscopic examination of the affected specimens (Cervino et al. 1998). The fungus is not an endolithic species and it might have been spread by parrotfish bites (Cervino et al. 1998).

2.1.5. White Syndrome: -

White syndrome is the whitening of coral tissues that is thought to be a reaction to toxic chemicals leached from antifouling paintings of marine installations (Antonius and Riegl 1997)

2.1.6. Shut down reaction: -

Stressed corals may die by even relatively mild impacts, such as a simple scratching, which will not kill healthy ones, causing a Shut Down Reaction (Antonius 1977). Shut down reaction (SDR) is a complete, spontaneous disintegration of the coral tissue, starting at the borderlines of the injury. Coenosarcal tissue sloughs off in thick strands or blobs. The disease spreads along the branches in a ramose form, leaving denuded coral skeleton without a trace of tissue. The disease advancement on the affected colony is about 10-cm hour⁻¹. The advancement is a non-intermittent process, which does not stop before killing the entire colony. SDR is transmitted by contact. A piece of sloughed tissue triggers SDR within 5-10 min after a contact with another healthy colony.
2.1.7. Yellow band disease: -

Yellow band disease (YBD) has been found in the coral *Montastrea annularis* while the colony was recovering from the bleaching event (Hayes and Bush 1990). The disease is found to appear continuously thereafter in the Caribbean (Goreau et al. 1998).

2.1.8. Coral bleaching: -

Coral reefs have been projected in the report of the intergovernmental panel on climate change, to be among the most sensitive ecosystem to long-term climate change (IPCC 1998). When physiologically stressed, the critical balance that maintains their symbiotic relationship with algae is lost. The corals may lose some or most of their algae, a major source of nutrition and color. In this condition, corals are referred to as "bleached". Bleaching is defined as the paling of the host due to the loss of zooxanthellae (Hoegh-Guldberg and Smith 1989; Fitt and Warner 1995; Lesser et al. 1990) or loss of photosynthetic pigments from zooxanthellae (Sharp 1995; Fitt and Warner 1995). In-depth investigations on the bleaching mechanisms show that there are several mechanisms involved in the expulsion of zooxanthellae during the bleaching process. The primary mechanism involved in the coral bleaching is the host cell detachment of zooxanthellae (Brown 1997b). The zooxanthellae number in the tissue is reduced by the release of the zooxanthellae into the coelenteron or by the release of the detached gastrodermal cells into the coelenteron (Gates et al. 1992). In addition to the reduction of zooxanthellae in the bleached tissue, chlorophyll content per zooxanthellae has also been observed to be reduced (Brown et al. 1995), reaching near zero levels, although the carotenoid levels did not change (Lee-Shing Fang et al. 1995).
Chlorophyll c content was 35 times lower in the bleached corals than in healthy specimens (Kleppel et al. 1989). Zooxanthellae are degenerated or are released from the damaged gastrodermal cells or the gastrodermal cells themselves are released along with the zooxanthellae. Tissue growth is halted in the affected species and skeletal accretion is stopped (Goreau and Macfarlane 1990, Leder et al. 1991), while sexual reproduction is suspended (Szmant and Grassman 1990). Corals survive if the stress is brief, but will die if it is prolonged (Wilkinson, et al. 1999; Glynn 1996). However, even a sublethal stress may make corals highly susceptible to infection by a variety of opportunistic pathogens. Disease outbreaks (epizootics) may result in significant coral mortality (Hayes and Goreau 1998). Once mortality occurs, the coral’s soft tissue becomes a food source for scavengers, making the increasingly bare skeleton a site of attachment for rapidly growing seaweed and other opportunistic organisms (Hayes and Goreau 1998). Coral bleaching is most often associated with a significant rise in sea surface temperatures (Brown 1997b; Glynn 1996; Goreau et al. 1993; Glynn 1991; Cook et al. 1990; Gates 1990; Jockiel and Coles 1990; Hoegh-Guldberg and Smith, 1989; Jaap 1985; Fankboner and Reid 1981). On site observations and National Oceanic and Atmospheric Administration (NOAA) satellite-derived sea surface temperature records from North Atlantic and Caribbean reef locations show a significant correlation between all large scale bleaching events and high sea surface temperatures (Strong et al. 1998; Gleeson and Strong 1995; Goreau et al. 1993). Water temperatures of even one degree Celsius above normal summer maxima lasting for at least two or three days appear to provide a potentially useful predictor of consequent bleaching (Goreau and Hayes
While there are differences in response among species and populations, most corals are likely to bleach but survive if temperature anomalies persist for less than a month, enabling corals to recover. However, the chronic stress of sustained high temperatures can cause physiological damage that may be irreversible (Wilkinson et al. 1999). Stress related bleaching may also be induced if corals are subjected to a reduction of salinity, intense solar radiation (especially ultraviolet wavelengths), exposure to the air (by low tides or low sea level), sedimentation, or xenobiotics such as copper, herbicides, and oil (Brown 1997a; Glynn 1996). Often, these conditions are at least an indirect consequence of extremes in weather (such as hurricanes and typhoons) which may be produced by or occur concurrently with elevated sea surface temperatures. As a consequence, multiple factors may act in concert to cause bleaching. High solar irradiance (particularly ultraviolet wavelengths) is thought to be especially stressful to corals when coupled with elevated sea surface temperatures (Glynn 1996). The recent 1997-98 El-Nino warming event is considered the strongest on record by some measures (Chavez et al. 1999; Mc Phaden and Xuri Yu 1999; Enfield 2001). The El-Nino of 1997-98 was estimated to have caused $33 billion damage and 23,000 deaths world wide (Kerr 1999). The mass coral bleaching of 1997-98, coincident with the 1997-98 El-Nino, is believed to be the most severe on record of International Society for Reef Studies (ISRS 1998). Coral reefs in the Indian Ocean were severely affected with shallow reef coral mortalities of up to 90% (Wilkinson et al. 1999) It has been suggested that large colonies have more energy available for regeneration (Loya 1976; Bak 1983). This is due to the nutrient translocation from healthy tissues adjoining
the bleached ones (Mascarelli and Bunkley-Williams 1999). It was recently reported that bleaching of the coral *Occulina patagonica* from the Mediterranean Sea is the result of a bacterial infection (Kushmaro et al. 1996, 1997, 1998; Rosenberg et al. 1998). The causative agent, *Vibrio shiloi* was shown to infect the host at elevated temperatures (Kushmaro et al. 1998). Temperature affects the adhesion of the bacteria to the coral to a β-galactoside containing receptor (Warner et al. 1999). These bacteria were shown to multiply in the coral tissue but were unculturable in the normal routinely used media (Banin et al. 2000).

2.2. Diseases of Octocorals: -

The octocorals, similar to the scleractinian hexacorals, serve as hosts for numerous commensals, symbionts and parasites and also provide refuge for reef fish (Bayer 1961). Among the octocorals, the diseases affected the gorgonians. Causes that result in the loss of tissue in gorgonians are detachment, fracture of the skeleton and overgrowth by fouling organisms (Yoshioka & Yoshioka 1991). There are a few reports of disease-related mortality in gorgonians and other octocorals. Some of the diseases of gorgonians are described below.

2.2.1. Black band disease (BBD): -

BBD is known in the scleractinian corals caused by the cyanobacterium *Phormidium corallyticum* (Rützler and Santavy 1983). The same pathogen, *P. corallyticum* is also causing black band disease in the gorgonia *Pseudopterogorgia acerosa* and *P. americana* (Feingold 1988) and the mode of tissue loss in the gorgonians are similar to the BBD in the scleractinians corals.
2.2.2. Red band disease (RBD): -

The RBD was reported to affect the octocoral *Gorgonia ventalina* in Belize (Rützler and Santavy 1983). RBD contains a cyanobacterium from the genus *Oscillatoria* (Richardson 1992) in addition to other cyanobacteria. There is no particular cyanobacterium observed to be associated with the diseased corals, and different species have been thought to be responsible in different locations (Santavy and Peters 1996). RBD is similar to the BBD in its development of a microbial consortium in the mat, containing other cyanobacteria, the sulphur-oxidizing bacterium (*Beggiatoa*), heterotrophic bacteria and the nematode, *Araeolaimus* (Santavy and Peters 1996).

2.2.3. Aspergillosis: -

The fungal disease in a gorgonian is the first coral disease in which the complete processes such as entry and spread of the pathogen in the coral reef ecosystem and the role of global change in the disease propagation have been studied. There has been a correlation between the decline in the Caribbean coral reef and sharp increase in the transport of the African dust over the western Atlantic (Shinn et al. 2000). It is hypothesised that the prolonged drought in the highly grazed grasslands of the Sahel in Africa and the desiccation of the water bodies resulted in abundant fungal spores that are transported through the wind to the western Atlantic Ocean (Shinn et al. 2000). This finding was further supported by the study that shows that there are no spores in the clear air (Weir et al. 2000) and, therefore, the African wind was established as an effective carrier of fungal spores from African deserts to the western Atlantic region. *Gorgonia ventalina* and *G. flabellam* in the Caribbean suffer by the recession of rind tissues called coenenchyme,
which is the outer organic rich matrix containing the living polyps (Smith et al. 1996). Only one species of fungus was found common to all the affected colonies. The fungus was identified to be Aspergillus sp. The 18S ribosomal RNA analysis showed that the fungus may be A. fumigatus (Smith et al. 1996). The fungus was later identified as Aspergillus sydowii (Geiser et al. 1998). Weir et al. (2000) successfully established Koch’s postulates by inoculating the A. sydowii cultured from the spores collected from the African dust.

2.3. Summary of the diseased states of the corals:

<table>
<thead>
<tr>
<th>Name of the diseased states</th>
<th>Hexa/Octocoral</th>
<th>Etiological agent</th>
<th>References</th>
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<td>The cyanobacterium <em>Phormidium corallyticum</em></td>
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<td>The bacterium <em>Sphingomonas</em> sp.</td>
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<td>Cervino et al. 1998</td>
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<td>Hexacorals</td>
<td>Toxic chemicals</td>
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