10. Summary and Conclusion:

Coral reefs are renowned for their spectacular diversity and have significant aesthetic and commercial values, particularly in relation to tourism and fisheries. Coral reefs are being degraded on a global scale by a wide range of impacts, including mass bleaching events, diseases, pollution and several human activities. Diseases can be divided into biotic and abiotic diseases. The parasitic relationship between a host and its symbionts eventually leads to the biotic disease of host. The biotic disease can be recognised when the presence of a pathogen results in a distinct deviation from normality as displayed by functional or structural deficit.

During the surveys in the Lakshadweep islands, extensive partial mortality was observed in the dominant scleractinian coral species *Porites lutea*. Most of these corals showing partial mortality were clearly distinguished by a pink line around the dead patch which was colonized by the cyanobacterium *Phormidium valderianum* and the width of the line ranged from a few mm to a cm. The pink coloration is of the tissue rather than that of the skeleton or from any associated organisms. The disease incidence is higher in the southern lagoon than the northern lagoon. This is attributed to the slower current velocity that is prevailing in the southern lagoon that facilitates relatively calm water for the settlement of any pathogen on the coral colonies.

No dense bacterial populations were found in association with the PLS-affected polyps. A hyaline non-sporulating fungus and the dark melanized *Curvularia lunata* were frequently isolated from the PLS-affected tissue. Their associations with the PLS-affected polyps were confirmed by the
immunofluorescence detection method. Verification by Koch's postulate showed that these fungi were not the disease-causing agents. On the other hand, the cyanobacterium *Phormidium valderianum* which was isolated from the pink line successfully induced the disease syndrome (PLS) in *P. lutea*. The abiotic factors, which are detrimental to the host when the cyanobacterium interacts with the polyps, are the CO₂ produced by the cyanobacterium and the resulting acidic microenvironment.

The histological studies showed that the PLS-affected polyps underwent extensive tissue mortality with a variety of cellular events that lead to the death of the polyps. Some of the features of the cellular events are destruction of ectodermal and calicoblastic cells by a phenomenon similar to apoptosis, leaving behind cellular debris in these granular layers. The gastrodermal cells were ruptured, vacuolated and mostly free from their zooxanthellae. The expelled zooxanthellae were found on the surface of the coral colony and in the coelenteron. Most of the gastrodermal cells were found detached from the wall. Calcium accumulation was also found to be common in the gastrodermal cells.

Anomaly of the zooxanthellae population in the PLS-affected polyps was triggered by the cyanobacterial carbon concentration mechanism (CCM) found in *P. valderianum*. The cyanobacterial production of the CO₂ through its CCM, supplies extra CO₂ for the carbon-limited zooxanthellae. Thus, they overcome the host control on their division. Satisfying the inorganic demand of the zooxanthellae minimises its photosynthesate translocation to the host. This causes stress that finally leads to death of the host cells.
There was very little change in the protein content and in the RNA/DNA ratio of between the PLS-affected and the healthy polyps. The protein profile showed that there was a new production of 29 kDA protein in the PLS-affected polyps. This might be due to the recycling of the non-essential proteins available in the cytoplasm to produce essential proteins as the PLS-affected polyps were depleted in energy by the low zooxanthellae density and an increased growth rate of the zooxanthellae. Alternatively, it needs to be confirmed whether this new protein is a heat shock protein synthesized in response to the stress.

10.1. Conclusion:

It is suggested in the present study that a foreign organism may induce disease symptoms not necessarily through parasitic interactions, but by the generation of abiotic factors such as acidic microenvironment in the vicinity of the host. Similar symptoms can be generated through the induction of the particular abiotic factors such as pCO₂ even in the absence of a foreign organism. In a disease where a pathogen is involved, the response can be immunological. On the other hand, disease symptoms that are produced by abiotic factors generated by a foreign organism, such a response may not be elicited from the host. As a result physiological disorders are produced in the host. The present study describes a novel approach to research in coral pathology. The sequence of events that is proposed to be the cause for the formation of the pink line in the coral is as follows.

1. The cyanobacterium P. valderianum settles on the coral colony.
2. The cyanobacterial carbon concentration mechanism (CCM) produces CO$_2$ (see Chapter 7), surplus amounts of which leaks out from their cell.

3. This becomes the source of excess C$_1$ required for the carbon limited zooxanthellae in the host cell.

4. Zooxanthellae grow and overcome the host control.

5. Host looses its share of organic carbon from the zooxanthellae as this is used by the dividing zooxanthellae.

6. Calcification and growth of the host polyps is retarded.

7. The acidic environment created by the release of CO$_2$ may cause mortality of coral polyps and skeletal erosion.

8. The synergistic effects of CO$_2$ influx and acidic environment around the polyps might form the pink coloration of the host.