Modeling of Epizootics on Four Genera of Arabian Gulf Corals

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Modeling of Epizootics on Four Genera of Arabian Gulf Corals

By

John Alexander Kluge

Submitted to the Faculty of
Nova Southeastern University Oceanographic Center
in partial fulfillment of the requirements for
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Sam Purkis, Ph.D.
Abstract:

Coral colonies, from a reef near Abu Dhabi, United Arab Emirates (UAE), were counted and assessed for condition using photo-transects. An epidemic model, used to track how a communicable disease moves through a population, was constructed to help predict the future condition of this coral reef. *In situ* data from a disease outbreak that occurred in September 2011 provided a baseline for the model. Coral Populations of *Porites*, *Platgyra*, *Acropora* and *Dipsastrea* were modelled using condition categories that included Healthy, Black Band Disease Infected, Cyanobacteria Infected, Recovered, Recruits or Dead. Results from the modelling indicate that populations of *Platgyra* and *Dipsastrea* are healthy and growing, even with continued presence of diseases, due to the high rates of recovery (chance for host colony to overcome infection; high recovery rate = high chance of colony recovering from the infection) and low mortality rates (chance of dying from an infection; low mortality rate = low chance of a infected colony dying from the infection) in the genus. *Porites* showed no signs of population growth, but stabilized near its initial population size, despite having a high infection rate because population growth (recruitment) and recovery rate were canceled by a high mortality rate. *Acropora* showed a loss in population numbers over time, losing 25% of its population before the disease was eliminated. Diseases may have been eliminated from the *Acropora* population because population density was low and coral died quickly after becoming infected with a disease, due to the high mortality rate of this genus, before infecting other colonies. *Acropora* was the only genus to display what seems to be a density dependent infection rate, since chance of infection was reduced and then eliminated by the rapid mortality of infected colonies, if the population was higher disease spread may have been higher. In addition to results obtained using *in situ* data, higher modified infection rates were used to assess how they might impact these coral populations. Results suggest that all four genera seem to be resilient, shown by *in situ* modeling and parameters extracted from the phototransects, and able to withstand acute (rapid increase of infection rate which was then again quickly brought back to normal infection rate, an infection “spike”) increases of disease infection, which is shown by either a high recovery rate (*Dipsastrea* and *Platgyra*), a high recruitment/low mortality rate (*Porites*), or a high mortality rate (*Acropora*) that may not allow for the diseases to spread. However, all four genera would be slowly driven to extinction by a sustained (chronic) increase of disease infection rate brought on by growing stressors such as an increase in average water temperature or pollutants within the Gulf. These results demonstrate fragility of Gulf coral genera when exposed to chronic episodes of disease, which over time causes total collapse of the coral populations.

**Key words:** Epidemic Modeling; Arabian Gulf; Coral Disease; Coral; SIR; Temperature Rise
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Introduction:

1.1 Global Coral Reef Decline

Coral decline is widespread, with some level of degradation observed on almost every reef. For example, coral cover on the Great Barrier Reef dropped from 28.0% to 13.8% from 1985 to 2012 (De’ath et al. 2012) and in the Caribbean from about 70-50% to 25-10% from 1970 through 2012 (Gardner et al 2003, Jackson et al. 2014). Among many other factors, like bleaching due to increased ocean heat (Baker et al., 2008), coral diseases have been implicated as causative agents of this decline. In the Caribbean, White Band Disease (WBD) reduced cover of A. cervicornis and A. palmata by nearly 95 percent and impacted reef dynamics throughout the region (Bruckner 2002, Aronson and Precht 2001, Riegl et al 2012). In the Arabian Gulf, multiple disease outbreaks reduced coral cover significantly (Riegl et al. 2012), which also caused shifts in reef community structure (Riegl and Purkis 2009). In 1996, a mass coral bleaching event coupled with an outbreak of several different coral diseases caused Acropora to nearly be removed from the southern Arabian Gulf (Riegl and Purkis 2009). In 2011 the Gulf had another disease outbreak, documented in this study, which primarily consisted of Black Band Disease and Red Mat Cyanobacteria. The diseases were modeled and show probable outbreak scenarios for in situ data. Little doubt remains therefore, that coral diseases are a key factor in global coral reef decline (Selig et al., 2010) and that they therefore merit investigation.

It appears that peripheral seas, like the Caribbean and the Arabian Gulf, have suffered disproportionately much from coral diseases. The Arabian Gulf is home to many areas of dense coral growth, despite its extreme climate (Riegl and Purkis 2012). Coral condition is, as anywhere, controlled by water quality and temperature (Bruno et al. 2003, De’ath and Fabricius 2010). Changes in thermal properties, pollution and nutrient enrichment introduced to the marine environment through runoff, effluent, or improper waste disposal can increase coral bleaching as well as disease outbreaks (De’ath and Fabricius 2010, Haapkyla et al. 2011, Bruno et al. 2003, Harvell et al. 2007).
This thesis examines corals from the Arabian/Persian Gulf, in the United Arab Emirates (UAE), and examines the effects of a coral disease outbreak on some important coral taxa. Questions asked relate primarily to the effects of diseases on coral population dynamics. But it is also important to contemplate why corals in the Gulf should be subjected to these severe disease outbreaks. Stress on coral from anthropogenic influences are increasing due to major economic expansion, including coastal construction, sewage and hydrocarbon-based effluent (Alyahya et al 2011), and heavy metals such as copper, lead and zinc that are dumped into the Gulf from the expanding industrial base. Chronically oil polluted areas have shown to (1) damage the reproductive systems of corals; (2) decreased viability of coral larvae; and (3) changes to the physical properties of reefs area due to loss of rugosity caused by dying or dead coral colonies (Loya and Rinkevich 1979). Heavy metals can build up in coral communities and lead to toxic conditions (Dubinsky and Stambler 1996). Sewage effluent has caused a variety of effect ranging from increased harmful algal blooms (Gilbert et al 2005) to increased water turbidity (Starkey & Karr 1984). All of these factors, combined with an increasingly strong footprint of global climate change, could act to create the severe disease conditions as treated in this thesis.

Figure 1: Map of the Arabian Gulf and the study sight off of Abu Dhabi, UAE. (Riegl 2011)
1.2 Study site and Parameters

This thesis examines corals in a site in eastern Abu Dhabi, UAE. Corals in the Arabian/Persian Gulf region occur in one of the most severe thermal environmental settings anywhere, with an annual temperature range of 24 deg. C. (Purkis and Riegl 2005). With increasing average temperatures, these extremes will be even more accentuated and resultant stress is obvious (Yonge et al. 1931, Brown 1977, Lirman et al. 2011). Despite the wide temperature range so far experienced by corals found in the Arabian Gulf, corals in the region thrived until recently (Riegl et al. 2012, Yao 2008). Many corals now go through a bleached-to-recovery cycle caused by increases in the summer thermal maximum far above the long-term mean. Such events occurred in 1996 and 1998, and then again in 2002, 2010, 2011, and 2012, when the temperature spiked above 34 degrees Celsius (Riegl et al 2012). The 1996 and 1998 events led to extensive mortality of some key genera. For example, Acropora, which comprised a majority of the reef assemblage, is now relatively rare in the region (Riegl 2002). As a result, the current reef community is primarily comprised of the hardier in respect to bleaching and disease genera such as Dipsastrea, Platygyra, and Porites with Acropora in low abundance (Riegl 2011).

![Figure 2: Sea Surface Temperatures over four seasons in the Arabian Gulf (Yao 2008).](image)
1.3 Disease

Coral disease appears to be correlated with environmental stressors, and could be a driving force that controls reef coral community structure within the Arabian Gulf (Riegl 2002, Sutherland 2004). Two prominent diseases, Black Band Disease (BBD) and Red Mat Cyanobacteria, occurred as major outbreak events in autumn of 2011 in the southern Arabian Gulf and form the material for the present thesis.

Black Band Disease (BBD) was first reported in the early 1970s (Antonius 1973, recorded in Belize). The disease can migrate across a coral colony, leaving behind dead tissue (Figure 3), and, depending on the size of the colony, can cause complete mortality after a few months (Richardson 2004, Richardson 2008, Sekar et al. 2008). Studies have shown that BBD has a wide distribution both spatially and temporally, but that infections show variability in prevalence and dispersion (Peters 1993, Fenner 1998, Bruckner 2002). Black Band disease in the Arabian Gulf affects 8 species of coral (Riegl 2012) but the main genera affect are Acropora, Dipsastrea and Platygyra. Porites was primarily unaffected (4 out of the 2739 Porites colonies within this study were effected), where the only infections seem to be through direct contact with an infected colony (Riegl 2012). Acropora was formerly abundant and its decline was probably caused by BBD (Riegl et al. 2012), in accompaniment to local stressors (Brown 1977, Lirman et al. 2011, Goldenberg et al. 2014). With the recent loss of Acropora, the remaining abundant genera are Dipsastrea, Porites, and Platygyra.

Figure 3: Black Band Disease moving horizontally across a coral colony.
Red Mat Cyanobacteria (RMC) is an infection caused by a Lynbya-like alga that is red to pink in coloration and grows on the coral colony. The disease (syndrome) has previously not been recorded or scientifically documented. The description for RMC comes from photographs and first hand recounts of the infection, however further studies will be needed. The infection spreads quickly across the reef community primarily targeting *Porites* colonies but can spread to neighboring non-*Porites* colonies. It appears to spread quickly across an infected colony, due to the limited cases of minor infections (small portion of colony infected) throughout this study, but it is still unknown how quickly.

![Figure 4: Porites Colony Infected with Red Mat Cyanobacteria](image)

RMC disease was documented for the first time in the Arabian Gulf reefs and appears highly infectious with nearly half of the *Porites* colonies infected in some way (personal observations). RMC appears to infect new hosts through direct contact or at least close proximity, but again more research is needed to find the exact mechanism for infection. RMC did not seem to actively move across the coral colony, such as BBD or WBD where a clear line of infection is present (Bruckner 2002, Aronson and Precht 2001, Sekar et al. 2008), but instead grew in random interconnected plots across the colony. The mechanism that kills the coral tissue is still unknown. However, RMC appears to
suffocate the coral tissue until nothing remains but dead skeletal tissue (Figure 4). To reiterate, besides from data collected in this study, data relating to RMC was not found in previous research or known coral disease. To fully investigate RMC future research needs to be conducted.

1.4 Modeling Epidemics:

To understand disease dynamics, a rich mathematical theory and many models have been developed to predict or follow disease outbreaks for plants, animals, and even coral (Segarra et al. 2001, Wearing et al. 2005, Riegl 2012). The basic epidemic model (SIR model; S= Susceptible, I=Infected, R=Recovered) going back to Kermack and McKendrick (1927) is commonly used to predict the outcomes of disease outbreaks. The SIR model is a three equation model where each group (Susceptible, Infected, and Recovered) feeds into the next (S $\rightarrow$I$\rightarrow$R) until the population gains immunity (if allowed in the model), dies, or overcomes the disease. The SIR model is also used as a “building block” for more complex models that take account of other variables to recreate natural population dynamics. While not perfect in recreating natural dynamics, models can help to understand how diseases move through populations and also predict the chance that individuals within a population survive.

Epidemic models have been used previously within the Arabian Gulf to track and predict disease outbreak scenarios for Arabian Yellow Band Disease (AYBD), the White Syndromes (WS) and Black Band Disease (BBD) (Riegl et al. 2012). The study explains how diseases are one of the most important regulatory factors for animal populations (Anderson and May 1979, Riegl et al. 2012), and followed the SIR model with modified equations to include recruitment, mortality and natural death, in order to model more closely a “real world” environment and help to make the results more accurate. The model differences were based off exponential reproduction, natural disease induced mortality, or re-acquired susceptibility to the diseases. The model outcomes showed a variety of results ranging from stability in the population to a crash to near extinction levels. Disease outbreaks, at least for some diseases such as BBD and WS, seem to be
seasonal and primarily infect hosts during times of increased thermal stress and seem to disappear during the cooler seasons (Riegl et al., 2012).

My thesis evaluates a disease outbreak of Black Band Disease and Red Mat Cyanobacteria that occurred in 2011 in the Arabian Gulf, in the aftermath of a coral bleaching event. Using data from photo-transects, a mathematical model was parameterized to describe the observed dynamics and predict the impact these disease can have on four different coral genera. The dynamics of coral populations are subject to infection by these diseases was investigated using a modified SI (S= Susceptible, I=Infected) model (Segarra 2001, Earn 2000, Henry 2009) that was formed and explored using R as the computational environment.

Methods:

2.1 Phototransects

Randomly positioned photo-transects around a central GPS point were used to collect 636 5m x 1m photographs. Over 8,700 individual coral colonies were captured in the photographs. Transects were taken to include 5 to 8 photos per transect using SCUBA in September 2011, off the coast of Abu Dhabi, United Arab Emirates (24.8502N, 54.7201E). The corals were then counted and categorized in the lab. The photographs included the number of colonies in the dominant coral genera at the site, specifically Acropora, Porites, Dipsastrea and Platygyra in order to find the variables and rates to input into the modified epidemic model.

2.2 Coral Genera Sampled

The southern Arabian/Persian Gulf is home to about 40 species of scleractinian corals (Coles 2004; Riegl et al. 2012), but only relatively few are very common and are known to be keystone species, with regards to their importance in the community. Four of these were chosen for further study in this thesis.

Acropora is a genus in the order Scleractinia and is the largest genus of hermatypic corals and the most widespread (Wallace 1999). Worldwide there are over 149 species
with 8 species residing within the Arabian Gulf (Riegl 1999, Cibahy 2012). The common “branch like” structure of *Acropora* allows for rapid growth and unique water flow dynamics around the colonies (Harriott 1998). The “branches” of this genus also allow for asexual reproduction, through fragmentation (Wallace 1985), forming dense thickets which are ecologically important for reef fish and invertebrates (Wallace 1999). However, the dense thickets make *Acropora* highly susceptible to disease outbreaks, due to the individual colonies close proximity to each other in the thicket, that may remove entire populations from a reef (Aronson and Prechet 2001, Bruckner 2002) cause the associated fauna to disappear as well (Wallace 1985). *Acropora* normally grow in shallow waters on a hard substrate. They can be found in a range of habitats from an oceanic surge zone to a protected lagoon habitat (Harriott 1998). Within this study *Acropora* was found growing in shallow water (>5 meters) on a hard substrate in a flattened fan shape. *Acropora* was once common and abundant throughout the Gulf but were nearly driven from the reef population after the 1996 and 1998 disease outbreaks that primarily targeted this genus (Riegl and Purkis 2009). The species most commonly occurring in the study area are *A. downingi*.

*Porites* is a hermatypic genus of coral in the family Poritidae. These corals are defined by their “finger-like” morphology. Massive colonies of *Porites* are among the most important reef builders (Lough 2014), due to the high amount of calcium carbonate (skeleton) deposited onto the reef. Worldwide there are over 65 species of *Porites* with 6 species residing within the Arabian Gulf (Riegl 1999, Cibahy 2012). *Porites* are normally found in turbid coastal waters and form large aggregations (Huang 2012). *Porites* within the Gulf study site were normally found growing on a hard substrate growing in a cylindrical shape. The most common species, and the one with greatest relevance to this study, was *P. harrisoni*.

*Dipsastrea* and *Platygyra* are hermatypic genera of scleractinian corals in the Merulinidae family. Worldwide there are over 25 species of *Dipsastrea* with 4 species represented in the Gulf (*D. pallida* the most common). *Platygyra* has roughly 12 species worldwide with 2 (*P. dadalea* and *P. lamellina*) being commonly found within the Gulf (Riegl 1999, Cibahy 2012). These corals have mound/dome shaped growth form and
Platygyra are referred to as “brain corals.” Both genera grow in shallow water habitats and, for this study, were seen growing or encrusting onto the hard substrate. Dipsastrea colonies were normally smaller in size than Platygyra and were sometimes found growing closer to sandy areas. They are known to indicate sandy habitats within the Gulf (Riegl 1999). These corals were shown to be resilient to environmental disturbances allowing for high population densities on reefs (Huang 2012) after the Acropora die off in 1996 and 1998 within the Gulf (Riegl and Purkis 2009).

2.3 Model Formation and Stability Evaluation

The basic epidemic model is comprised of three groups: Susceptible, Infected, and Recovered, which constantly flow into one another as follows:

![Basic epidemic model stages showing how each group, Susceptible (S), Infected (I) and Recovered (R) feed into one another.](Towers 2013)

Such models have had wide distribution in the theoretical ecological literature and have been applied to a variety of organisms (Edelstein-Keshet 1986). The equations for how an individual moves from one group to another are explained below:

\[ S' = \frac{dS}{dt} = -\frac{BSI}{N} \]

\[ I' = \frac{dI}{dt} = \frac{BSI}{N} - \nu I \]

\[ R' = \frac{dR}{dt} = \nu I \]

\[ N = S + I + R \]

**Equation 1:** The formula components for the basic epidemic model. (Kermack and McKendrick 1927). \( S' \) denotes the derivative of \( S \) and could also be expressed as \((dS/dt)\), same for the other variables.
The change in number of *Susceptible* individuals (S) at each time step is computed by taking the disease prevalence (B), multiplying it by the change in number of *Susceptible* (S) and by the total change number of *Infected* (I) and dividing by the total population (N). This allows the number of total *Infected* colonies throughout the population to cause further infection in the *Susceptible*. BSI/N is subtracted from the total number of *Susceptibles* at this time step and added to the *Infected* population. Then, the *Infected* that have *Recovered* (v) from the disease and moved into the *Recovered* group (R) are subtracted (Munz et al 2009). Below is a basic example of the outcome of a SIR model, which clearly shows the flow among the model groups:

In a “normal” disease outbreak the population starts off with nearly all of its population within the *Susceptible* group (S) indicated by the blue line in Figure 6. The *Susceptible* population is reduced when individuals move to the *Infected* population (I) represented by the red line in Figure 6. When the Infected population grows the *Recovered* population also increases because some “faster” individuals recover and get moved into the *Recovered* group (R). In this model the *Recovered* group is presumed to gain immunity from the disease and the outbreak will slowly disappear from the population (Munz et al 2009). This, however, is not realistic for the examined corals due to the models base assumptions on immunity gained by recovered group and the lack of morality caused by the infection(s), because of this the model had to be modified.

![Figure 6](image_url)

**Figure 6:** Epidemic model showing the basic relationship among the three groups with no mortality. S= Susceptible, I= Infected, R=Recovered
Instead, an SI model (S= _Susceptible_, I=_Infected_) model (Segarra 2001, Earn 2000, Henry 2009) was developed to include factors that would normally be seen in the real world environment, such as mortality, recruitment and removes the assumption (from the SIR Model) of immunity gained by all corals after the infection, for which there is no indication. The SI model was then explored in R as the computational environment. The SI model used is shown below:

\[
S' = -\frac{BSI}{N} + vI + Nr
\]

\[
I' = \frac{BSI}{N} - MI - vI
\]

\[
N=S+I
\]

**Equation 2:** Modified epidemic model down to a SI model.

The first equation for the *Susceptible* population consists of the following: newly Infected individuals leave the *Susceptible* population via the term (-BSI/N) where B is the disease prevalence, S is the size of the *Susceptible* population, I is the size of the *Infected* population and N is the total population (S+I). The “Susceptible” population’s loss is balanced by *Recovered* individuals entering from the *Infected* population (vI, where v is a recovery constant) as well as by recruitment (Nr, where r is a recruitment constant). Recruitment is from *Susceptible* as well as *Infected* individuals since coral diseases are not known to decline a colony’s overall fertility, except in the part that has been killed (Mydlarz et al. 2009).

The *Infected* population grows by receiving individuals that have just been *Infected* via the same term as the loss is imparted to the *Susceptible* population (BSI/N). It declines by the number of *Recovered* individuals leaving via term (vI) as well as by loss from the population by mortality (MI, where M is a mortality constant) at each time step. The Recovered individuals re-enter the *Susceptible* population (+vI) since recovery has not shown to give corals immunity to disease (Sutherland et al 2004).
Thus, using only two equations, the SI model allows predictions for how a disease moves through a population and can potentially be used to help manage the coral populations by identifying at risk genera early, which could allow conservations to focus on the at risk genera within the Gulf. These equations will interact with each other as outlined in Figure 7:

![Figure 7](image-url)

**Figure 7:** The diagram depicts how the model for this project behaves and what factors control each step throughout the model. N=Total Population, S=Susceptible, I=Infected, M=Mortality Rate, B=Infection rate, \( v \)= recovery rate.

### 2.4 Stability criteria

Models consisting of more than one function are traditionally explored for their stability properties. The steps involved are solutions of the nullclines (zero growth isoclines), search for the intersection of both (in the case of a 2-equation system, as employed here). The intersection gives the equilibrium, where the two lines of zero growth meet at a point where neither component will increase) and evaluation of the trajectories around the intersection point (which will show whether a trajectory begun in the vicinity of the equilibrium point will be attracted towards that point, or will be reflected away from it. The former will define a stable equilibrium, the later an unstable equilibrium). In the following, the theoretical stability properties are explored to provide a framework for the expectations for the model.

A nullcline is a set of points in a phase plane so that \( \frac{dS}{dt} = 0 \) and \( \frac{dI}{dt} = 0 \), which means that along these points (or lines), no growth occurs. The nullclines thus separate
domains of growth (usually to the left of the nullcline) from domains of shrinkage (mostly to the right of the nullcline, where the direction is determined by the size of I or S). Thus, the nullclines were used to find the equilibrium where $\frac{ds}{dt} = 0$, $\frac{dl}{dt} = 0$. Each equilibrium was derived from the models equations below:

$$S' = -\frac{BSI}{N} + vl + Nr$$

$$l' = \frac{BSI}{N} - MI - vl$$

The Nullcline for the *Infected* equation solves as:

$$0 = \frac{BSI}{N} - MI - vl$$

$$0 = l\left(\frac{BS}{N} - M - v\right)$$

$$I_1 = 0$$

Now, since N (total population) was equal to the total number of *Infected* (I) plus the total number of *Susceptible* (S):

$$N = I + S$$

$$N = \emptyset + S$$

$$S_1 = N$$

The equation of I can then be solved for S:

$$0 = \frac{BS}{N} - M - v$$

$$\frac{BS}{N} = M + v$$

$$S_2 = \frac{N(M + v)}{B}$$
Then, the nullcline was solved for the \textit{Susceptible} equation as \( S' \) in order to find \( I_2 \):

\[
0 = -\frac{BSI}{N} + vI + Nr
\]

\[
\frac{BSI}{N} = vI + Nr
\]

\[
BSI = Nvl + NNr
\]

\[
BSI - Nvl = NNr
\]

\[
I \left( BS - Nv \right) = NNr
\]

\[
I = \frac{NNr}{(BS - Nv)}
\]

\[
I = \frac{NNr}{N \left( \frac{BS}{N} - v \right)}
\]

\[
I = \frac{Nr}{BS - Nv}
\]

\( S_2 \) was inserted into \( S' \) for equation to simplify \( I_2 \):

\[
I = \frac{Nr}{B \left( \frac{N(M + v)}{N} \right)}
\]

\[
I_2 = \frac{Nr}{M + v}
\]

The equilibria resulting from these operations are:

Equilibrium 1: \([\bar{I}_1, \bar{S}_1]\) = [0,N]

This is a trivial equilibrium, since if no infection exists, all individuals have to be per definition \textit{Susceptible}.

Equilibrium 2: \([\bar{I}_2, \bar{S}_2]\) = \( \left[ \frac{Nr}{M + v}, \frac{N(M + v)}{B} \right] \)
Dynamical systems, such as the one illustrated here, have theoretical stability criteria. Often used are the Routh-Hurwitz criteria (Edelstein-Keshet 2008) that can be reduced essentially to requiring for stability that the trace (i.e, the sum of all elements in the diagonal) of the Jacobian matrix (a matrix of all partial derivatives of the system of equations) must be smaller than 0, and its determinant > 0. The Jacobian matrix, which is a matrix of all partial derivatives, for the model equations was created below:

\[
\begin{bmatrix}
S \frac{d}{dS} & S \frac{d}{dl} \\
I \frac{d}{dS} & I \frac{d}{dl}
\end{bmatrix} = \begin{bmatrix}
-\frac{B I}{N} & -\frac{B S}{N} + v \\
\frac{B I}{N} & \frac{B S}{N} - M - v
\end{bmatrix}
\]

The equations of the equilibrium can then be applied to this matrix to test against the Routh-Hurwitz Criteria:

Equilibrium 1: \([I_{1}, S_{1}] = [0, N]\)

\[J = \begin{bmatrix} 0 & -B + v \\ 0 & -M - v \end{bmatrix}\]

\[J_{11} + J_{22} = -M - v < 0 \rightarrow \text{Stable}\]

\[J_{11} \cdot J_{22} - J_{21} \cdot J_{12} = 0 - 0 = 0 \rightarrow \text{Stable}\]

Equilibrium 2: \([I_{2}, S_{2}] = \left[\frac{N r}{M + v}, \frac{N(M + v)}{B}\right]\) is applied to the Jacobian matrix:

\[J = \begin{bmatrix} -\frac{B r}{M} & -M \\
\frac{B r}{M} & 0 \end{bmatrix}\]

\[J_{11} + J_{22} = -\frac{B r}{M} < 0 \rightarrow \text{Stable}\]

\[J_{11} \cdot J_{22} - J_{21} \cdot J_{12} = Br > 0 \rightarrow \text{Stable}\]

Stability of the model can be shown graphically through a phase plane. In such a graph, two “phases”, in the present case the *Infected* phase and the *Susceptible* phase are plotted.
against each other, allowing to visualize the flow of the populations in mutual dependence.

**Figure 8:** Phase plane example from Cyanobacteria Infection on *Porites*. The inward spiral towards a central stable point shows stability. Multiple starting “Infected” and “Susceptible” populations show common stable point for this disease-host interaction.

The phase plane shows the flow of two equations over time represented by the curved lines in Figure 8. Data points represent the two populations, which vary over time producing the curved line. Phase planes show stability or instability in oscillatory systems. An inward spiral of the curved line, in a phase plane, represents stability as the oscillations reach the equilibrium point and an outward spiral towards infinity represents instability. Stability appears in scenarios where the infected and susceptible population co-exists within the epidemic model. Figure 8 shows an inward spiral, representing stability, meaning the population is stable at the attractor defines by a specific level of *Infected* and *Susceptible*, and should not experience growth away from this equilibrium, once reached.

A different way of expressing this dynamics is by showing the trend of the entire population. Therefore, the time-derivative of the same population (where N=S+I) represented by Figure 8 in the phase plane is shown below.
The Porites population represented in Figure 8 and Figure 9 show an example of an oscillatory system that reaches a stable equilibrium (represented by the inwardly oriented spiral in the phase plane). With these analyses we would expect the modified epidemic model to be stable around the equilibrium points. Thus, in the following simulations we should expect the S-I system to settle down at an equilibrium. However, some dependence on parameter values can be expected with regards to the position of the equilibrium (the trivial equilibrium will only change with regards to N, but does not merit further attention).

2.5 Rates, In Situ data, and Modified Disease Prevalence

To use the previously described model for exploration of disease dynamics in the Arabian Gulf, it was necessary to first extract the required model parameters from the field data. To achieve this, data from the photographic records were used. Acropora, Porites, Dipsastrea and Platygyra colonies were assigned to several condition categories.
“Healthy corals”: included colonies that did not have any tissue loss due to disease. Completely bleached corals that did not suffer from any disease were also included. Justification for ignoring bleaching was based on the single focus of the model – disease.

“Diseased corals”: included two categories of colonies with the diseases Black Band Disease (BBD) or Red Mat Cyanobacteria (RMC). Since no time series was available, infection rate was considered the same as prevalence observed at the time of sampling. This assumption is justified by the rapid outbreak and spread of these diseases in the Gulf (Riegl 2001). The disease(s) only remain active in the population(s) from September to November (Riegl 2001). Therefore, the single snapshot of disease prevalence taken at about half-time of the outbreak’s duration was considered valid as a proxy for the calculation of infection rate. The proxy rate at the half-life of the outbreak(s) was further used for the remaining conditional categories, which allowed for the further rates to be obtained.

“Recovered corals” were a third category including colonies that showed evidence of disease recovery. Coral diseases leave distinctive evidence that remains visible long after the disease is gone, such as a circular dead patch or band on a colony indicating a BBD infection while RMC leaves behind random blotches of exposed skeletal tissue that mimics how the infection presents (Figure 4). However, since it was not possible to be certain which disease had infected colonies in the past, both were lumped, which resulted in a static recovery rate for the respective coral genus for both diseases. However, some direct recovery rates (specific to one infection) may be inferred due to high specificity for some diseases on certain coral genera, such as RMCs primary host Porites. Colonies were identified as Recovered if any portion of the colony was still alive after the disease infection had stopped.
Figure 10: Sample photo transect with examples of class groupings.

“Dead corals” which included colonies when they had complete and recent tissue loss (not covered in turf algae) or complete infection over all remaining live tissue.

“Coral recruits” were also identified and classified by size or polyp count (recruits were generally golf ball size and lower) but not included in any other grouping, since all recruits found within this study were found to be healthy. This category was needed to provide an accurate assessment of recruitment rate, to insure recruits were not being added on multiple occasions.

Since available data were from a single visit during a single disease event, estimation of parameters is necessarily incomplete. Images during the event captured dieback at a single moment in time. While no repeat measurements were available that allowed derivation of mortality and recovery rates, speed of disease progression across a colony surface could be estimated by the amount of bare skeleton that had not yet been colonized by turf algae. Any white bar (no algal colonization) surface could be expected to be less than a few days old, and was thus indicative of rapid disease spread over the colony. Thus, a recently affected coral could, with high confidence, be assigned to disease as cause of mortality. Equally, colonies that exhibited parts of eroded skeleton not covered by tissues must have suffered die-back, most likely due to diseases, in the past. Therefore, any colonies that were observed to exhibit large parts of ‘old-dead” skeleton (covered by turf algae) tissue could be considered to have originated from a previous
disease infection. It is understood that these estimations could be improved with a better time-series of data. However, these data were not available. Each conditional category represented a variable in the modified epidemic model with the method of computation shown in Table 1.

<table>
<thead>
<tr>
<th>Conditional Categories</th>
<th>Method</th>
</tr>
</thead>
<tbody>
<tr>
<td>Susceptible (S)</td>
<td>Count of all colonies without visible active infections (Including recovered coral colonies)</td>
</tr>
<tr>
<td>Infected (I)</td>
<td>Count of all colonies with visible active infection of either kind (RMC, BBD)</td>
</tr>
<tr>
<td>Recovery Rate (v)</td>
<td>Any colony with old tissue loss likely due to disease, as judged by shape of living tissue divided by Total Population</td>
</tr>
<tr>
<td>Disease Prevalence (B)</td>
<td>Total number of all Infected divided by Total number of all colonies (live and recently dead)</td>
</tr>
<tr>
<td>Recruitment Rate (r)</td>
<td>Total Number of Recruits divided by Total number of all colonies (live and recently dead)</td>
</tr>
<tr>
<td>Mortality Rate (M)</td>
<td>Total Number of Recently Dead divided by Total number of all colonies (live and recently dead)</td>
</tr>
<tr>
<td>Total Population (N)</td>
<td>Total number of Susceptible, Infected, Recruits, and recently dead</td>
</tr>
</tbody>
</table>

Table 1: The table expresses how each conditional group’s values were computed.

In situ data were applied to the SI model for each genus and each disease. The models created will show how each of the various Susceptible (S) and Infected (I) populations grow or decline over time. The changes in each coral population cause by disease show how threatened each genera is from each infection. These predictions may assist conservation efforts. If the spread of diseases throughout populations is better understood, then remedial action, if required, may be considered. Therefore, my models will investigate to what level the different genera are threatened by the diseases. Based on this, remedial action or special protection may be required.

Additional model runs were conducted using in situ data that was modified by increasing disease prevalence. Disease prevalence increases were modelled in several ways, including chronic (long lasting), acute (sharp peak), loss of recovery rate, and high base infected populations. The models built from the modified in situ data document how populations change under variable disease and population conditions.
Results:

3.1 Overall combined rates and totals:

The total coral population (“N”) totaled at 8,714 individual coral colonies, of which 3,956 were healthy. “Healthy” was defined by the total number of corals not currently or previously infected with a disease (i.e., no visible blemishes or areas of tissue retreat). The next group was the number of infected coral colonies, which numbered at 1,087 individuals (Black Band Disease (BBD) and Cyanobacteria infections were combined to reach the total). The diseased population was synonymous with the infected population or “I” and this also allowed for some infection rates “B” to be estimated (see pg. 24) for each disease. The fourth grouping was the Recovered “R” population for all species totaled at 2,489 individual corals when *Acropora*, *Porites*, *Dipsastrea* and *Platygyra* was combined. The Recovered and Healthy populations together comprise the Susceptible population “S” (total number of corals susceptible to disease infection). The fifth group was the total number of dead colonies combined from the four genera, however a mortality rate for each genus was calculated separately and when combined with recruits from each genera (the final grouping), allowed for a population growth or decline estimate. These last two groups were the only variables that directly affected the total population (N).

![Figure 11: Overall counts by combining totals. (Total Healthy Coral=Susceptible).](image-url)
3.2 Healthy vs Infected Individuals:

Each coral genus was evaluated for *Susceptible* (healthy) and *Infected* groups (Figure 12, “A” group *Platgyra*, “B” group *Dipsastrea*, “C” group *Porites* and “D” group *Acropora*). Thus, Healthy corals (*Susceptible, S*) could be compared to the total number of corals infected with one or both of the diseases. The total number of “Healthy” coral results from the combination of the previously uninfected and the recovered corals. In this study the assumption was made that the coral genera do not gain immunity after recovery, since in only a minority of cases do corals gain immunity to a disease after being infected.

![Figure 12: Genera break down of Healthy and Infected Populations](image)

*Platgyra* (Figure 12A) had an even distribution of disease prevalence with the total number of Black Band Disease (BBD) infections at 195 individuals and Cyanobacteria infections of 113 individuals. The number of *Susceptible*, i.e. healthy, coral (S) for *Platgyra* was 1934 individuals composed of 1,116 previously uninfected corals and 774 recovered corals. The disease prevalence proxy (see pg. 24) gave BBD an
infection rate (B-BBD) of 10% and cyanobacteria an approximate infection rate (B-Cyano) of 6%.

*Dipsastrea* (Fig. 12 B), the most common corals in the Arabian Gulf, showed the lowest rate of disease infection of the four genera. The number of BBD-infected *Dipsastrea* totaled at 29 individuals and the number of Cyanobacteria infections was 117. When compared to the total number of healthy coral at 3,256 individuals (2,149 previously uninfected and 1,107 recovered individuals) the disease infection rate for BBD was 0.09% and 3.5% infection rate for Cyanobacteria. When comparing *Dipsastrea* to the *Porites* or *Acropora* the infection rates show the genera’s resilience to disease and environmental changes within the Arabian Gulf.

*Porites* (Fig. 12 C) had the highest level of infection, particularly from Red Mat Cyanobacteria (RMC). The number of BBD-infected individuals was negligibly small, with 4 infected colonies. However, the number of Cyanobacteria infections totaled at 627 compared to the total number of Susceptible (healthy) individuals, which numbered 1,174 (630 previously uninfected and 544 recovered), infection rate for BBD was 0.0035% and an infection rate for Cyanobacteria at 53.4%.

*Acropora* (Fig. 12D) was the scarcest genus in the study, the total population (N) was 83 individual colonies. Within the small population only one infection for BBD and Cyanobacteria was found, which gave an infection rate of 1.2% for each disease. The total number of recovered was more than triple the amount of previously uninfected corals with 17 previously uninfected and 64 recovered colonies, meaning the susceptible population was comprised primarily of previously infected individuals.

### 3.3 Total Recruits, Mortality, Recovery Rates

Recruitment, mortality and recovery rates are shown in Figure 11 and show the proportion of coral recruits compared to the number of recovered and the amount of dead colonies. This gives total counts for each group as well as recruitment rates (N * r), recovery rates (v = inferred from percentages) and the mortality rates (M) of each
species. This will be combined with the disease prevalence (proxy infection rate) from the previous section to give inferred death rates for each disease on each genus of coral.

Recruited, Recovered and Dead *Platgyra* sum to a total of 38 recruits, 774 recovered and 114 dead individual colonies (Fig. 13A). The recruited population when compared to the total population of *Platgyra* (Healthy + Infected), which numbers at 2,242 individuals gives us a recruitment rate (number of recruited/*Platgyra* population) in the study site of 1.6%. This low recruitment rate is compared to total mortality rate (dead/*Platgyra* population), which totals at 4.8%. It shows that the death rate is approximately four times the rate of new additions to the population. However the recovery rate from disease throughout this population is higher than in any other genus with a rate of 64.7% when compared with the infected population (Infected + Recovered + Dead). This suggests a high chance of survival for *Platgyra* throughout the Arabian Gulf. However, a mortality rate four times that of recruitment, may not bode well for the population.

![Figure 13: Genera break down of Recruits vs Recovered vs Dead](image)

*Figure 13: Genera break down of Recruits vs Recovered vs Dead A) Genus *Platgyra* B) Genus *Dipsastrea* C) Genus *Porites* D) Genus *Acropora*
Figure 13B shows the total amount of Recruited, Recovered and Dead *Dipsastrea* which totaled 120 recruits, 1,107 recovered and 447 dead. The recruited population when compared to the total population of *Dipsastrea* (120/5,076 individuals) gave a recruitment rate of ~2.4%. Mortality rate (percentage of population that died from infection) was ~8.8%. *Dipsastrea* had a recovery rate at 65.1%. *Dipsastrea* had a recruitment rate that was a quarter of the mortality rate. However, recruitment rate was influenced by total population (N) while Mortality rate (M) only affects the Infected population.

Recruited, Recovered and Dead *Porites* (Fig. 13C) was at 41 recruits, 544 recovered and 349 dead individuals. The recruited population, compared to the total *Porites* population (41/2,739) gave the recruitment rate of ~1.5%. When this was compared to the mortality rate of ~12.7%, calculated in the same manner as the recruitment rate, it showed a more extreme difference then the previous two genera with approximately an eight times higher rate of mortality then recruitment. *Porites* also had the lowest recovery rate of ~35.7%, which is almost half of the previous two genera. The low rate of recovery for *Porites* may be a sign of this genus being “at risk” or heavily influenced by disease in the Gulf.

The final group in Figure 13D showed the total amount of Recovered and dead *Acropora*. There were no recruits and healthy individual were rare, with only 17 throughout the population. The recruitment rate of 0% and a mortality rate of 46.8%, measured by taking the total dead *Acropora* (73) and dividing the total population (156 individuals) suggested problems for the population. When comparing mortality and recovery rates, which is ~ 46%, this suggests that there is a ~50% chance of the individual dying from the disease. This analysis suggests that *Acropora* is at the highest risk of extinction within the Gulf due to the low population, 0% recruitment and 46% chance of mortality from each disease.
3.4 Rates and Trends

<table>
<thead>
<tr>
<th>Genus</th>
<th>BBD inf. Rate</th>
<th>Cyanobacteria Inf. Rate</th>
<th>Recruitment Rate</th>
<th>Recovery Rate</th>
<th>Mortality Rate</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Platygyra</em></td>
<td>0.100827</td>
<td>0.058428</td>
<td>0.016129</td>
<td>0.647157</td>
<td>0.048387</td>
</tr>
<tr>
<td><em>Dipsastrea</em></td>
<td>0.008906</td>
<td>0.035933</td>
<td>0.02364</td>
<td>0.651176</td>
<td>0.088061</td>
</tr>
<tr>
<td><em>Porites</em></td>
<td>0.003407</td>
<td>0.534071</td>
<td>0.014968</td>
<td>0.356955</td>
<td>0.127418</td>
</tr>
<tr>
<td><em>Acropora</em></td>
<td>0.012345</td>
<td>0.012345</td>
<td>0.0</td>
<td>0.460431</td>
<td>0.467948</td>
</tr>
</tbody>
</table>

Table 2: Values for each conditional category for each coral genus collected from the phototransects.

Black Band disease had a ten times higher infection rate on *Platygyra* than on any other genus within the study. This was also true for Cyanobacteria infection on the genus *Porites* where the rate was again ten times the infection rate of the next closest genus. This suggests host specificity and that mortality rates for both *Platygyra* and *Porites* could be attributed to their respective diseases. A hierarchy also seemed to exist between coral genera for adaptation to the climate/epizootics of the study site. *Dipsastrea* was clearly coping best with a small infection rate for both diseases, as well as the highest recovery rate. If a *Dipsastrea* was infected given the high recovery rate and the low overall mortality rate, it was likely that the individual would survive. This was the opposite for *Acropora* with the highest mortality rate and second lowest recovery rate throughout the study. However, since *Acropora* had such a low population, the calculated recovery rate may be inaccurate due to the limited amount of individuals in the study. The last trend is probably the most troubling for this study site: mortality rate was at least four times higher for each genus than the recruitment rate.

With knowledge of infection rates for each disease and each coral genus, Susceptible and Infected population predictions could be modeled as well phase plane models to show the stability (see stability section pg. 24). Future populations were forecast for each disease using the epidemic equations (Vanderplank 1963, Segarra et al. 2001, Earn et al. 2000). In addition to the SI population predictions, genera that have reached stability within the SI model were investigated using phase plane analysis.
confirmed by total population over time graphs, to show stability or instability. Models used of Excel and R as the computational environment. Such predictions can help to understand which genera are in greatest danger of disappearing from the coral reef community. This can give some early warning signs for future conservation efforts within the Arabian Gulf.

3.5 Dipsastrea - Epidemic Model

The first genus modeled was Dipsastrea, where each infected population (I) in the initial infected groups dissipated over time. A low rate of infection caused diseases within the population to be quickly removed by the recovery and mortality rate. This was seen at the start of the model where the initial growth in the susceptible population (S) grew slowly at first and then reached linear growth after ~10 time steps. Since the model included no carrying capacity, the population is here assumed to just keep increasing. This suggests that Dipsastrea was relatively safe from the two diseases and proved to be the least threatened genus within the study. However, if Dipsastrea were to become too prominent on the reef in the future, a disease that targets this genus could spread quickly across the reef community. The models for each disease are shown below.

![Black Band Disease Infection on Dipsastrea Population](image)

**Figure 14:** Black Band disease infection of Dipsastrea susceptible (S) and infected (I) populations. Real population(s) will not grow linearly. The linear model shown above simply shows that the disease will not diminish future population growth.
Figure 15: Cyanobacteria infection of *Dipsastrea* susceptible (S) and infected (I) populations. Real population(s) will not grow linearly. The linear model shown above simply shows that the disease will not diminish future population growth.

### 3.6 Platygyra – Epidemic Model

Similarly to *Dipsastrea*, the infection rates were relatively low when compared to the recovery rates. Both Black Band Disease and Cyanobacteria infections started with a low number of infected, when compared to the population, and dissipated quickly over time. The susceptible population (S) grew slowly as the diseases were removed from the population by recovery and mortality rates. The “S” population reached linear growth after the infected population (I) was removed. However, real population(s) will not grow linearly. The linear model shown above simply shows that the disease will not diminish future population growth. This population was deemed more vulnerable than *Dipsastrea* because of its lower recruitment rate, which led to a lower population at the end of the model at approximately 4,000 individuals where the *Dipsastrea* ended at approximately 5,000 individuals with the same amount of time steps. Nonetheless, *Platygyra* populations still appeared safe, with low level of mortality and infection. However, similar to *Dipsastrea*, if any disease that targets *Platygyra* were introduced into the Arabian Gulf it could be detrimental to the reef community. The models for each disease are shown below:
Figure 16: Black Band disease infection of *Platygrya* susceptible (S) and infected (I) populations. Real population(s) will not grow linearly. The linear model shown above simply shows that the disease will not diminish future population growth.

Figure 17: Cyanobacteria infection of *Platygrya* susceptible (S) and infected (I) populations. Real population(s) will not grow linearly. The linear model shown above simply shows that the disease will not diminish future population growth.
3.7 *Porites* – Epidemic Model

The genus *Porites* was only modeled for the Cyanobacteria infection because of the limited number of Black Band Disease infections on *Porites* colonies. Unlike in the previously shown models for other genera, disease presence is not as diminished by the recovery rate, and was shown to continuously return over time. The population did not obtain a state of steady growth like the previous genera. Instead it would rise and fall with the presence of the disease until an equilibrium point between the *Susceptible* and *Infected* populations formed. The reason for the diseases constant recurrence was due to the high infection rate, which as at least 10 times higher than in the other genera. This means that when the infected population (I) reaches a stable point (approximately 250 individuals) and the recovery rate was not enough to eliminate the disease and the infected population grew again causing more disease-related mortalities. The populations were confirmed and shown to be stabled which can be seen in Figure 19.

![Cyanobacteria Infection on Porites Population](image)

**Figure 18:** Cyanobacteria infection of *Porites* susceptible (S) and infected (I) populations.
Figure 19: A) Phase plane of Porites population influenced by Cyanobacteria. B) Total population of Porites over time while under the influence of Cyanobacteria.

Thus, any other detrimental environmental or anthropogenic force that affects the disease or the coral could drive the population low enough to make recovery impossible. This would lower biodiversity within the Arabian Gulf, which has been shown to be detrimental to the ecosystem (De’ath et al. 2012, Riegl 2012).

3.8 Acropora – Epidemic Model

Acropora is at the highest risk of extinction within the Gulf. It had previously suffered due to temperature anomalies in 1996, 1998, 2002, 2010, 2011, and 2012, (Riegl 2012, Yao 2008). Only 83 susceptible (S) Acropora were found and only 1 individual for each infected an Acropora colony. The mortality rate was higher than recovery rate, suggesting that infection from either disease led to death. The graph for both diseases was combined into one since the rates of the infection used were identical. The population steadily declined over time until the infection was removed. Loss of total population (N) stopped due to the infection being driven out (Figure 20) allowing for Acropora to reach a pseudo stable point. While the disease was found to be quickly removed and Acropora continues to persist within the Gulf every individual lost was problematic due to the absence of recruitment in this genus. This caused Acropora to be the most at risk genus for this study and its eventual extinction within the Gulf is likely.
Figure 20: *Acropora* population after mortality caused by both Black Band Disease and Cyanobacteria

**Discussion:**

4.1 Hypothetical Modeling

With the *in situ* data presented and modeled above, “what if” statements were asked to explore the sensitivity of the reef and what could happen if variables changed in the future. In the present section, variables will be adjusted to show different situations or conditions and their effect on the reef community genera. As an example, the Cyanobacteria infection on *Porites*, which exhibited a stable population with little growth or decline (Figure 18) was explored. The first experiment addressed was a sharp peak and fall of disease infection that could come from acute temperature changes or unspecified anthropogenic stressors that would rapidly increase disease presence on the reef (Patterson et al 2001, Riegl 2002, Bauman 2013).
Figure 21: *Porites* populations over time with one high infection peak and one low infection period.

Figure 18 was obtained with starting and ending infection rates as seen from *in situ* data. Subsequently, two changes were added to the disease infection rate (B), one high and one low, over the length of 6-8 time steps. The first change, “the peak,” occurred due to raised infection rate from 0.534071 to 1.30388. The disease infection rate (B) being approximately doubled by temperature changes documented trends (Harvell et al 2007). Temperature has been shown to be able to triple the infection rate with a temperature shift of 13 degrees Celsius (Cervino et al. 2004), where this study site has shown a 20+ degree Celsius shift making the data applicable. The peak was formed by quickly ramping up B over 3-time-steps instead of an immediate jump, which can be seen by the initial slow rise of the total number of the infected (I). The rise in the B can also be seen through the total susceptible population’s (S) initial drop and the mild repression of the total population (N). Once the model was past the high peak of infection, approximately around time step 20, there was a reduction in the number in the *Infected*. However, the overall population (N) still fell until the disease once again reached *in situ* conditions, from which point onward some growth of the susceptible population can be seen. Once a stable (flat) trajectory was reached, the second change in B, which reduced disease infection rate (which can also be attributed to temperature changes) to 0.175004, caused a significant reduction in mortality and a linear rise in total
population. The infection rate was once again returned to \textit{in situ} conditions after 6 to 8 time steps. This model, in which the overall population receiver and N grows after the shows the effects of changes, in disease prevalence on the population and how they compare to the next model which focused on a chronic increase in infection.

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{Temperature_Dependent_Changes_of_Infection_Force_on_Porites}
\caption{\textit{Porites} populations over time with extended periods above normal disease prevalence levels.}
\end{figure}

Similar to the previous model, the start and end time steps used the \textit{in situ} infection rate of 0.534071, which was increased and decreased slowly over time to a maximum of 0.946139 and a minimum of .403209 with some additional resting points above the normal disease infection rate. As a result, the model (Figure 22) showed an initial rise in population at the base-level disease infection rate (B) until time step 6 which observed a slow increase of B to the maximum (0.946139). This slow increase was then followed by a slow decrease back to the normal value of 0.534071 over 10 time steps. Temperature changes can cause a shift of infection rate as stated above (Cervino et al 2004) as well as pollutants and other anthropogenic stressors potentially impacting the coral holobiont. The impact brought on by the stressors can translate into a loss of host resistance and a potential elevation of disease severity or rate of infection (Harvell et al 2007). This is reflected in the overall decrease in total population (N), caused by the increase in mortality, due to the higher number of the infected population (I). Once B had reached its normal infection rate (0.534071) it continued to decrease over 10 time steps. This resulted in the decrease of the infected population (I) seen in the first portion of the
model (~time step 15). However, even though the number of infected (I) decreased, the total population (N) continued to fall due to the recovery rate slowly moving some infected to the Susceptible population giving time for the mortality rate to continue to remove individuals from the infected population. If this trend of below normal infection rate continued it would follow the previous model (Figure 21), causing a slow increase in total population (N) until the disease was driven from the population. However, in this model that trend was never met and the disease infection rate once again increased slowly over time. This model was created to show how prolonged or permanent change to the ecosystem, such as an increase in average water temperature, can affect the reef ecosystem. These two models (Figure 21 & 22) illustrate the difference between acute and chronic disease along with the effect on the population.

With the examples that came from stable populations (Porites), additional models were constructed for the uninhibited population of Platygyra. This population was predicted to continue growing due to the relatively low infection rate and high recovery rate. The models allowed for the investigation of what would happen if this were changed, by either environmental stressors or temperature change, causing loss of recovery within the genus. The in situ data for Platygyra gave it a recovery rate of ~0.65, but when this was suddenly dropped, assuming unfavorable conditions, it caused a major change to the stability of the populations. In the model (Figure 23) all variables were kept the same as for the Black Band Disease outbreak except for the recovery rate value which was dropped zero. Such a situation could arise from anthropogenic or pollutant stress on the coral reducing/removing the coral colonies ability to fight the disease (Harvell et al 2007). At first the susceptible population (S) grew until the number of infected colonies started to increase, causing this growth trend to stop and slowly decrease. Soon the number of infected surpassed the number of susceptible (S), until around time step 50 where the number of susceptible passed the number of infected (I). This was stabilized after the mortality rates and recruitment rates caused that allowed for the susceptible population to grow larger than the infected population. The two populations (S, I) found an equilibrium, as could be expected from theoretical considerations (Figure 24 and see p.24-28), with little or no growth in either population. This showed that Platygyra is still safe from the two diseases studied, even with the absent recovery rate.
Figure 23: *Platygrya* Populations modeled with a total loss of recovery rates.

Figure 24: A) Phase plane for the genus *Platygrya* influenced by Black Band Disease (BBD) when recovery is reduced to zero. B) *Platygrya* population over time under the influence of BBD when recovery rate is reduced to zero.

In the remaining stable group, *Dipsastrea*, lowering recovery to zero showed little to no effect on the population and growth in N continued due to its high level of recruitment and low level of mortality. With heavy modifications to disease prevalence, only uninhibited growth came to a stop in the *Dipsastrea* population. Thus, no stability could be expected, and for this reason the model was not further examined in the phase-plane.

*Acropora* was modeled with the *in situ* population of 83 healthy and the number of infected colonies at 1. As previously stated, *Acropora* may be density
controlled. High infection and mortality rates may not completely remove *Acropora* from the Gulf due to the potential inability of the disease to infect the next host due to the small and sparse population. This is shown in Figure 17 where the infected were rapidly removed from the population and population decline stopped at approximately 20 individuals. The following model (Figure 21) was changed to increase disease infection rate to be 4 times higher than that of the recovery rate with the starting amount of infected set at 25, up from 1. The model below mimics the *in situ* model, where the numbers of susceptible (S) and infected populations (I) started off at their initial values and quickly dropped to an apparently stable point, due to the high mortality rate and zero recruitment rate. The increases to infected population and infection rate do not fall within expectable natural conditions, but serves to explore potential *Acropora* population dynamics. This model attempts to show that the *Acropora* populations are very limited, but they are also apparently stable, with each model reaching some stable resting point due to disease mortality removing the infected population. What happens to the populations can also be shown in the phase plane. Figure 26 again shows the trajectories of *Acropora*. Disease was shown here to control the *Acropora* population within the Arabian Gulf with population trajectories invariably ending up at a low point. That is dynamics is likely caused by any infection that reaches the *Acropora* colonies, even if not spread from one colony to another, will most likely kill its host by removing the colony from the reef community. Thus, any recovery efforts to help *Acropora* in this area will be heavily effected by disease and disease may one day remove the *Acropora* populations from the Gulf.
Figure 25: *Acropora* populations over time with an increased population of infected as well as an increased disease prevalence.

Figure 26: A) Phase plane of the *Acropora* dynamic influenced by both diseases. B) Population flow of *Acropora*, with increased infected population, over time.

The *in situ* and hypothetical models show the coral genera’s sensitivity to outbreak scenarios coupled with increased stress factors, such as increased average water temperatures or pollutants, which may inhibit the corals’ ability to resist infection.

4.2 Related Work

Modeling epizootics in corals populations has been researched previously by multiple authors using a variety of techniques to investigate the dynamic. Yakob and Mumby (2010) used a variation of the epidemic model (SIR) to investigate possible demographic disease resistance. The authors observed coral genera that showed
populations with high turnover rate were generally resistant to disease. The model used to research their observation was similar to the model for this study, where the recovered group was removed and the variables recruitment and mortality were added to fit the natural dynamic. The two models differ at this point, where Yakob and Mumby (2010) applied a carrying capacity to their recruitment and natural mortality in an attempt to follow the observed system. Williams et al. (2010) also studied coral epizootics but used a different model, the Boosted Regression Trees (BRT) system. The authors used 17 variables, either abiotic or biotic, and were assessed and prioritized by their effect on coral disease and the host genus *Porites*. BRT works by using a weight system, where each variable is balanced at the beginning of the model but assigned its own unique weight based off the observations of the researcher. As the model progresses, variables with the highest total weight are considered to have the greatest impact on the system. It is therefore intrinsically different from the model used in this thesis.

Epidemic modeling (SIR) from this study showed that degradation caused by coral disease was increased under chronic stress conditions (i.e. increasing average water temperature, etc.). Yakob and Mumby (2010) concluded, through observations and modeling, that climate change can increase host susceptibility due to increased stress on coral populations, which was also found within this study. It was also found that epizootics, within the Caribbean, primarily infect populations with slow turnover rates. However, this is completely opposite of what was observed in the Indo-Pacific (as well as the Arabian Gulf) where *Acropora*, normally a quickly reproducing genus, showed the greatest degradation due to disease (Peterson et al. 2001). Williams et al. (2010) presented the strongest predictors given by the BRT model and the expected correlation was found between abiotic stressors (i.e. temperature, turbidity, etc.) and coral disease, which had the greatest effect on epizootics for *Porites*. The BRT model, although different in approach than the SIR models, also showed abiotic factors as the prominent stressors of coral disease. Again, similar to this study and the research done by Yakob and Mumby (2010), abiotic factors are suspected to have the greatest impact on coral epizootics.

Although the cited studies differ with regards to the procedures and findings from the present research, it could aid other authors using the Half-life proxy procedure which
would lower the length of time to gather data. The procedure gives researchers the ability to obtain rates based off a snap-shot study or the rapid onset of disease (p.24). Yakob and Mumby (2010) as well as other studies could use the procedure to model outbreak scenarios without the need of multiple time stepped data. Half-life proxy data may not be as accurate as a multiple year study, but nevertheless allows insight into possible disease dynamics. Thus even the admitted shortcomings of snap-shot-data allow realistic evaluation of potential trajectories, rather than waiting multiple seasons to collect data.

Conclusion

My models and techniques have proven capable of adding significant process information to the available “snap shot” observations during a disease outbreak. It was possible to extract the parameters required for an SI model and to predict potential outcomes based on a variety of environmental assumptions that could influence disease spread. My models suggested that under observed conditions, the diseases had three distinct effects on the coral populations: in the cases of Dipsastrea and Platygyra disease did not hinder population growth, in the case of Porites, it slowed or negated population growth, in the case of Acropora, it led to significant decline. When infectious conditions were changed, as could be expected under scenarios of environmental stress (e.g. climate change, pollution, etc.), both Porites and Acropora were found to severely decline.

The investigated coral communities in the Arabian Gulf currently appear unhealthy with only half of the genera in the study predicted to grow unimpeded by disease. The other half are at risk of loss from the ecosystem due to either limited growth in population or slow decline due to disease (Riegl 2012, Jaap 1993, Santavy & Smith 2001). The research conducted in this study will aid to the limited data currently present from the Arabian Gulf while following up on previously modeled outbreaks (Riegl 2002). The in situ models for Porites show that this genus may be fragile to any additional stress, with the current population at equilibrium with disease. If any factor(s) negatively affect the ability of Porites to maintain equilibrium, it may be severely reduced or removed from the Gulf based on these models. The situation is the same for Acropora.
My models show that *Acropora* populations seem almost fully controlled by disease dynamics. If other stressors (anthropogenic or biological) were placed on *Acropora* it may be removed completely from the study area.

The two models that change disease infection rate (B) show the effect of temporary spikes in water temperature and increased average water temperature on a previously stable genus. The peak disease prevalence model (Figure 18) could arise from a heat wave or abnormally hot summer months, where the increase in water temperature has shown to be able to more than triple disease infection rate (Cervino et al. 2004). The increased infection rate would lead to a high amount of mortality and die back until water temperatures returned to normal. A prolonged increase in average water temperature would have even greater impact. This was shown in the chronic increase in disease infection rate model (Figure 22), where factors such as temperature or pollutants kept infection above normal for the entirety of the model. The chronic stress caused the coral population to decrease slowly over time indicating a possible crash in the future. The conditions that need to be met in order to see this result are currently taking shape with global increases in average water temperature already being recorded (Feary et al. 2011).

The Arabian Gulf corals are at risk of disappearing, including the currently more resilient genera, by increased global water temperatures or anthropogenic influences. If trends continue *Dipsastrea* and *Platygyra* may also reach a “breaking point” and may be removed or diminished similarly to *Porites* and *Acropora*. Hopefully this paper, and others like it, can assist in conservation or relief efforts for corals populations globally.
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Appendix

SI Code Static Disease prevalence

rm(list = ls())

SI <- function(t,y,p) {
{
    S <- y[1]
    I <- y[2]
}
    with(as.list(p), {
    dS.dt <- -B * I * S/N + (g*I) + b*(N) - d*(S)
    dI.dt <- B * I * S/N - (g *I) -(d+M)*I
    return(list(c(dS.dt, dI.dt)))
})
}

N <- 2047
I <- 195
S <- N-I

# B = infection rate
# g = recovery rate
# b = recruitment rate
# M= Mortality rate

parms <- c(B=.058428, g=.647157, d=0, b= .016129, M = .048387)

Time <- seq(0,80, by = 0.01)

require(deSolve)

SI.out <- data.frame(ode(c(S,I), Time,SI,parms))

matplot(Time,SI.out[,1],type ="l",lty = 1:2, col = 1,ylab="")

ylab=""
# specify axis options within plot

title(main="Cyanobacteria Infection on Platygyra Population", sub="", xlab="Time", ylab="Population")

legend("right", c("I", "S"), lty = 2:1, col = 3:1, bty="n")

**SI Code Non-Static Disease prevalence**

# Clear the console

```r
rm(list = ls())
```

# Define your variables initially

```r
N = 2390
I = 627;    # infected pop
S = N - I    # susceptible pop
g = 0.356955 # recovery rate
d = 0      # natural mortality
b = 0.014968 # birth rate
M = 0.127418 # mortality from illness
t = rep(0, 1000) # time
B = .534071  # infection rate at start
```

# Getting my Table in

```r
path = 'C:/Users/PC/Documents' # add in the directory where the file is saved
file = 'DummyB3.txt' # list of fake B
CB = read.table(file = paste(path, file, sep = '/'), header = T, sep = '\t', skip = 0) # changing infection rate
```

# Bring everything into a data frame

```r
dat <- data.frame(t, N, S, I, B, g, d, b, M, CB)
```

for(i in 2:length(dat$t)){
    # Time step
$\text{dat$t[i] = dat$t[i-1] + 1$

# These values are constant at each time step
$\text{dat$g[i] = dat$g[i-1]$
$\text{dat$d[i = dat$d[i-1]$
$\text{dat$b[i] = dat$b[i-1]$
$\text{dat$M[i] = dat$M[i-1]$
$\text{dat$B[i] = dat$B[i-1] # need this starting B$

# These values are changing at each time step
$\text{dat$CB[i] = dat$B.1[i-1]$
$\text{dat$N[i] = dat$N[i-1] - (dat$N[i-1] * dat$d[i]) + (dat$N[i-1] * dat$b[i]) - (dat$I[i-1] * dat$M[i])$
$\text{dat$S[i] = dat$S[i-1] + (dat$b[i] * dat$N[i-1]) - (dat$S[i-1] * dat$d[i]) + (dat$I[i-1] * dat$M[i]) + ((dat$CB[i] * dat$S[i-1] * dat$I[i-1]) / dat$N[i-1])$

$\text{dat$I[i] = dat$I[i-1] - (dat$I[i-1] * dat$g[i]) - (dat$I[i-1] * dat$d[i]) - (dat$I[i-1] * dat$M[i]) + ((dat$CB[i] * dat$S[i-1] * dat$I[i-1]) / dat$N[i-1])$

}$

# Plot up the data

plot(xlim=c(0, 50), ylim=c(0, 3000), x = -1, y = -1, pch = 19, col = 'white', main = "Peaks of Infection Force on Porites", xlab = "Time", ylab = "Population")

points(x = dat$t, y = dat$N, pch = 19, cex = 0.5, col = 'black')

points(x = dat$t, y = dat$S, pch = 19, cex = 0.5, col = 'blue')

points(x = dat$t, y = dat$I, pch = 19, cex = 0.5, col = 'red')

#make it look nice

legend("left", c("N", "I", "S"), lty = 1, col = c("black", "red", "blue"), bty = "n")