Exploring disease dynamics on coral reef ecosystems through system dynamics models

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Abstract

Coral reefs in the Caribbean are under a growing threat from climate change and local environmental pressures. In 2014-2015, the reefs are under an additional threat from the spread of a coral virus starting from the Florida region. There is a lack of knowledge about coral viruses in general and the way they spread specifically. This paper introduces a model which includes two of the main hypotheses about the way a coral disease could spread. Through simulating the effects of both hypotheses, the results help researchers and policy makers to understand implications for environmental policy to limit further devastation by the virus.

Keywords: Coral reefs, epidemics, sustainability, Caribbean

1. Introduction

Coral reefs around the world are under great threat from a combination of local environmental pressures and changes in global climate conditions. In one coral reef area in the world, the Caribbean, large areas of live coral cover have been displaced by growing populations of macro-algae. Additionally, warming events of the water, especially in the northeastern Caribbean and the Florida Keys have led to coral bleaching and even greater declines in live coral cover (Jackson et al. 2014). As one of the most diverse ecosystems in the world (comparable to the rainforest), coral reefs are of utmost importance in terms of ecosystem services. The value of benefits derived from coral reefs has been estimated at nearly US$ 30 billion each year worldwide, mainly from tourism, fisheries, and coastal protection (Cesar et al. 2003). Especially in the Caribbean, many of the local communities are heavily dependent on the coral reef for their food intake, income from tourism and protection against hurricanes.

Amid the growing global and local pressures on the coral reef, the reefs in the Caribbean are now facing an additional threat: coral disease. A coral disease is defined as "*an abnormal condition of an organism that impacts organism functions, associated with specific symptoms and signs"*(ICRI/UNEP 2010, p.3). The last three years have seen a large outbreak of coral disease starting from the Virginia Key in Florida[2]. There is growing evidence that the 'the White Plague' epidemic is not the cause of bacterial infection but originates from a virus (Soffer et al. 2014). The White Plague outbreak starting from the Virginia Key in the end of 2014 has been shown to follow a contagion-based model (Precht et al. 2016). While coral disease in general has been a relatively understudied

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[2] While consulting coral experts in the region it has been argued that this particular location for the outbreak might be related to the dumping of debris from the dredging project at the Port of Miami.

topic, the role of coral viruses has received even less attention. There are still major doubts about the origins of the virus and the way they behave within the coral colony.

This paper is an attempt to explore the differences between the existing theories about the spread of coral viruses. It does so by developing two simulation models, based on hypothesis about the origins and diffusion dynamics of the virus, to increase understanding about the possible behavior of the virus in the future. Additionally, the simulation model provides a framework to prioritize areas for further research on coral viruses.

2. Dynamic Hypotheses for the spread of coral reef viruses

Now that there is increasing evidence that a virus might be responsible for white plague diseases in the Caribbean region (Soffer et al. 2014), other important questions arise. The most fundamental question is whether the virus originates from an external source or that it develops within the coral colony itself. Soffer et al. (2014) have provided evidence that 'herpes-like' particles are produced within the coral colony and that the viruses might already be present within coral colonies and only become fatal when the resistance of the coral organisms to the virus decreases below a certain point. The question of virus origin is also important with regards to the spread of the virus. There is still little knowledge about the existence and mechanisms of transmission of coral diseases from one colony to another (Precht et al. 2016). Precht et al. (2016) produce evidence of a white-plague disease spreading both north and south of Port Miami with the outbreak following a clearly 'contagion-based model'. However, it might also be possible that both regions north and south of the outbreak faced similar environmental conditions which led to an outbreak which was produced by the same virus which was already present within the coral colony. In that case, the outbreak of the disease does not mimic the behavior of an epidemic in which a disease is rapidly transmitted from one organism to another.

The next chapter postulates two hypotheses about the herpes-like virus, or white plague disease. For these different hypotheses it describes the structure and behavior of the spread of the disease within an individual coral colony. When the spread of the coral disease is transmissible, it means that other coral colonies further away from the infected colonies potentially also get an increased chance of contact with the virus, for example through spawning events on neighboring (infected) coral colonies.

2.1 The spread of coral disease through an externally-produced virus

Figure 1 explores a disease diffusion model based on the hypothesis that the virus originates from outside of the coral colony. The virus entering the coral reef could, for example, be the result of the disposal of untreated human sewage in the waters surrounding the coral reef. The structure of the model focused around four stocks:

1) Susceptible coral population;
2) Infectious coral population;
3) Recovered coral population; and
4) White skeleton (e.g. where the live coral tissue has died).

The model follows the basic diffusion model of infectious diseases as described by Sterman (2000, p.300) and elaborated by Wheat (2014).

The virus entering the coral colony has been modeled using a one-time shock in which ten hectares of the coral colony becomes infected by the virus. Once part of the coral colony is infected, it interacts with other parts of the coral colony which have not been infected yet but are assumed to be susceptible for the virus through transmission. It is hypothesized that there are a variety of ways in which the coral organisms, which do not 'commute' themselves[3], can get in contact with other parts of the coral colony. This is for example possible through the transmission of the disease by other small organisms which live within the coral reef ecosystem. Because of a lack of data, the contact rate (e.g. monthly contacts per infected coral) has been assumed to be five, meaning that it interacts with other parts of the coral colony at least once a week. The infection rate of the susceptible coral is based on the total contact between infected and susceptible coral and the infection probability which has been assumed to be 50%. The total contact between infected and susceptible coral increases when the infectious coral population increases, but decreases when there are not much susceptible corals left. After having been infected, the infected corals either recover (recovery rate) or die (death rate). When the coral organism die, the coral reef substrate once again becomes available (white skeleton) for occupancy either by new coral recruits, macro-algae or sediment. Research by Precht et al. (2016) show that white-plague infection led to total coral colony mortality. In this model, a death fraction of 80% has been assumed. Furthermore it has been assumed that, on average, an infected coral organism dies within one month. Although it might be possible that coral viruses 'establish long-term non-fatal infections in corals' (Soffer et al. 2014), the model assumes that an coral organism which survives the disease is able to recover from the virus within 10 months. The model also assumed that the recovered coral organism has antibodies to protect it from renewed infection by the same virus.

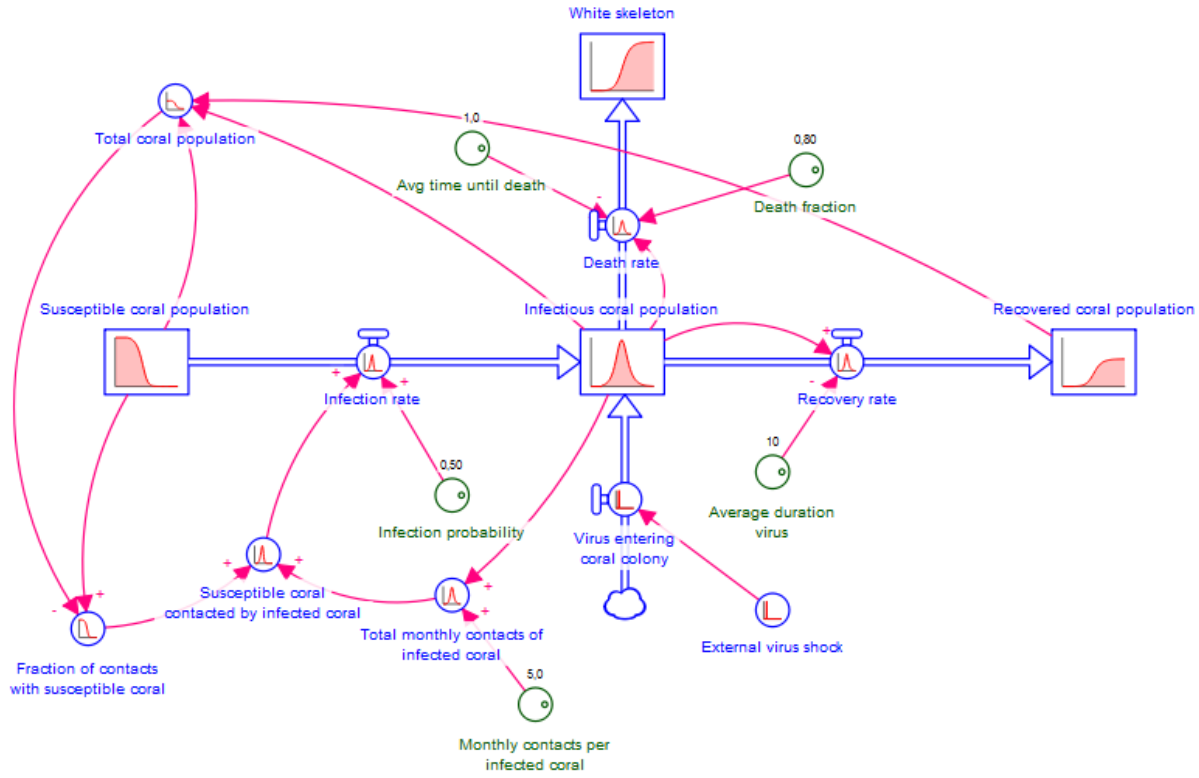

Figure 1. Dynamic structure of a coral disease epidemic (external)

[3] After a spawning event, a coral polyps (recruit) attaches itself to part of the coral reef substrate for the rest of its lifetime

2.1 The spread of coral disease through an internally-produced virus

Figure 2 explores an epidemic diffusion model based on the hypothesis that the virus originates from within the coral colony. The model is almost identical to the external coral disease model with the exception that it distinguishes between coral organisms which are passively or actively carrying the virus. This follows the hypothesis by Soffer et al. (2014) that viruses might be present within the coral colony but the health of the coral colony is strong enough to resist the activation of the virus. This structure is similar to the occurrence of many viruses within humans and animals, which are often non-lethal but have been linked to the development of cancer by the American Cancer Society (2017) under conditions where the human body is too weak to resist the virus from becoming active.

Within the model, the coral population with the passive virus can still recover within ten months. However, through the virus activation rate, the virus becomes active and the coral organism will not be able to recover (e.g. death fraction '1'). Through the structure of this model, it becomes clear that it might be possible that infection of coral colonies by viruses is nothing new and has happened in the past. The main difference would be that the current health conditions of the coral colony is so bad that viruses have taken their chance to become active, leading to massive coral mortality within the colony. It is beyond this model to explain the major causes of the decrease in coral health which have led to activation of the coral virus. However, increased coral disease is potentially related to coral bleaching events (caused by thermal heating) and deteriorating environmental conditions (e.g. sedimentation and water pollution). Most likely there are multiple stressors and it is not sure if there one 'trigger' which has made the coral reefs more susceptible to disease. For model simulation, it is assumed that the virus becomes active, through a combination of the factors mentioned; in 50 hectares of the coral colony each month starting with the most heavily affected parts of the reef. The most heavily affected parts of the reef are for example the regions which are closest to human disturbances such as water pollution and sedimentation.

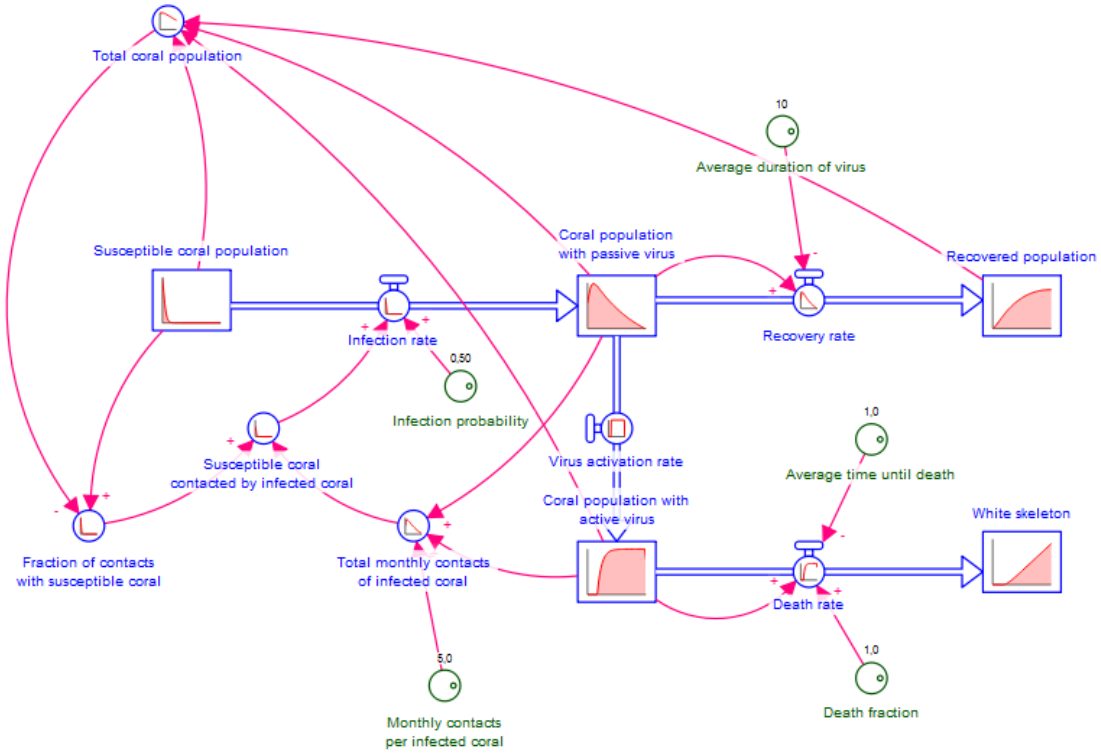

Figure 2. Dynamic structure of a coral disease epidemic (internal)

3. Simulation results and feedback analysis

Figure three and five compare the simulation results of the model hypotheses as described in the previous chapter. The hypothesis in which the coral disease is caused by an external virus clearly shows a rapidly increasing part of the coral colony which becomes infected. The increase slows once a larger part of the colony has become infected and the number of susceptible corals declines. The cumulative number of coral deaths follows a s-shaped growth pattern, which is typical for epidemics of infectious diseases (Sterman 2000, p.300). The behavior of the epidemic can be explained by the feedback structure as shown in Figure 4.

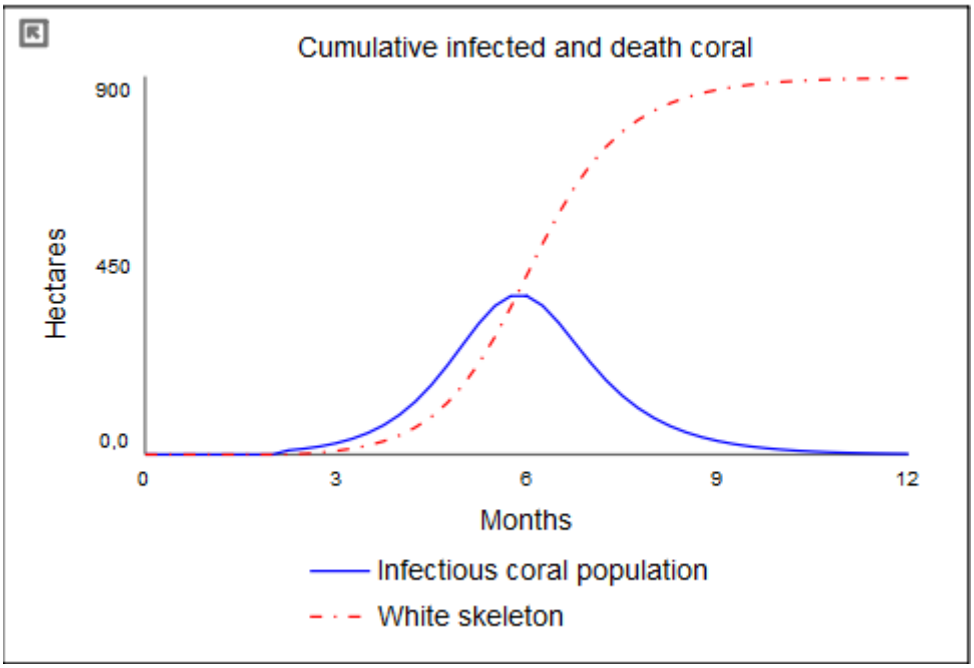

Figure 3. Simulation results external coral virus

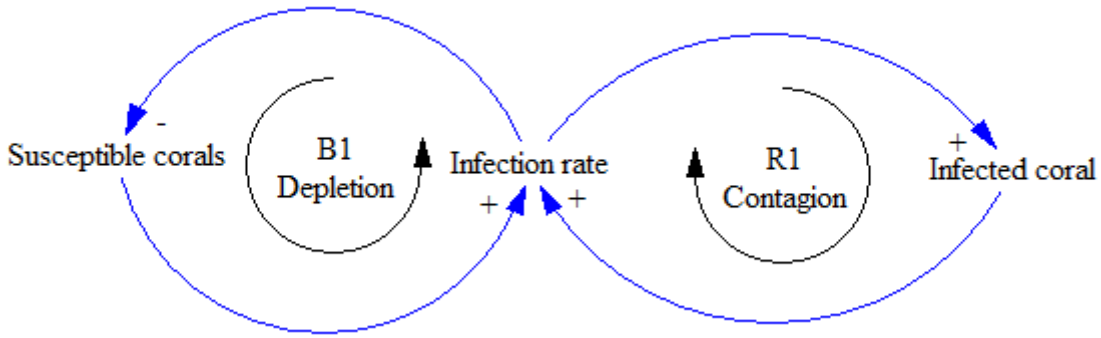

Figure 4. Causal-loop diagram virus epidemic

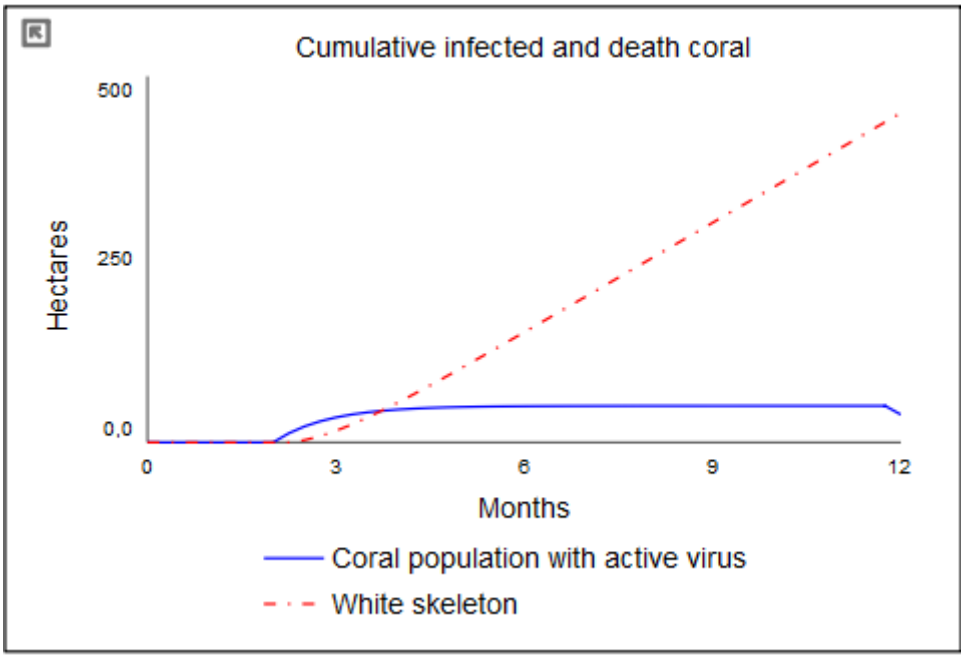

Figure 5. Simulation results internal coral virus

The hypothesis in which the coral disease is caused by an internal virus shows very different behavior in terms of the growth and spread of the virus. The main difference is caused by the assumption that only the coral population with an active virus will die from the disease. This means that both the coral population with the active and passive virus spread the epidemic in the same way as in the other model since it has the same feedback structure. However, only the coral population with the active virus is at risk to die, and the virus activation rate is assumed to be constant. In terms of the number of deaths, this will lead to a linear increase in the number of deaths (white skeleton), as opposed to the s-shaped growth in the first model.

4. Environmental policy based on model structure and simulation results

In terms of decision-making on environmental policy it is important to understand and know the differences in the structure behind the disease dynamics. In the case where the epidemic is caused by an external virus, it will be necessary to remove the origin of the virus as soon as possible. Additionally, it might be necessary to explore possibilities to prevent the coral virus to spread from one coral colony to another. Especially when a virus has a high infection probability, the epidemic could rapidly spread from Florida to infect the rest of the Caribbean coral reefs. Such policies could, for example, threat the coral reefs in the uninfected areas with antibodies to resist the virus once it enters the coral colony. However, the precautionary principle must be taken into account while intervening in the complex coral reef environment.

In the case where the epidemic is caused by an internal virus, it will be necessary to increase understanding about the major factors which are impacting the virus activation rate of the coral colony. Such policies most likely include the improvement of the local environmental conditions on the reef as to increase its resilience against the virus.

5. Future research based on model structure

The models and simulation results described in this paper are an initial exploration about the possible structure which could be responsible for the disease epidemics on coral reefs in the Pacific. As such, it might help to provide a framework which can be used to evaluate and prioritize future research areas. Questions which can be raised after exploring the models as presented in this paper are:

- What is influencing the infection probability?
- Have the initial infected coral colonies recovered after the epidemic?
- If particular coral species or colonies survived the epidemic, which factors were responsible for their resilience?
- How often and through which mechanisms do coral polyps within a colony interact with each other?
- What are the sources of externally-induced coral viruses?
- What is affecting the activation rate of coral viruses?
- Are there any feedback effects in the activation rate of the virus? For example, when parts of the colony become sick, the whole coral colony might become more susceptible for activation.

Many of these questions will be difficult to answer by studying the real life coral reefs, as there are many unknowns and it will be hard to factor out specific causal relationships. It might be advised to study some of these questions in laboratory setting. Most likely, there will be multiple stressors and cumulative impacts which make the coral reefs susceptible for coral disease.

6. Discussion

This objective of this paper was to develop a better understanding about how the structure underlying the spread of a coral disease affects coral ecosystems and to explore possibly environmental policies to reduce the consequences of coral disease. Because this is a relatively new topic for which both detailed studies and data are absent, the model is by definition very hypothetical and can provide very limited validation. Most of the parameter values have not been based on scientific studies but are based on the author's assumptions.

However, the model might be useful for the purpose of increasing attention for the problem of coral disease and showing how such epidemics could have devastating impacts in terms of future coral mortality and potential recovery, especially when the spread dynamics of the disease are not understood.

7. References

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