Lesson 1: Basic concepts of marine diseases and theoretical basis for pathogen transmission models

Learning objective: The learning goal of this lesson is to build a theoretical basis for modeling the transmission of marine infectious diseases.

Self-learning steps: The learner will go through the basic concepts about marine disease dynamics and pathogen transsmission modelling necessary to build background knowledge and key points for formulating disease models. First, main processes and mechanisms involved in pathogen transmission to be incorporated into disease models will be explained, including the processes influencing disease spread or which are of potential consequence inhibiting epizootics. Second, the student will understand the differences between contact-based diseases and environmental pathogen-based diseases. Finally, the theoretical basis of models to be studied in this course will be described. The student will need to spend 3 hours to gain a substanttial content knowledge necessary for the following lessons and modelling exercises.

Additional resources for the lecture: Additional learning resources for methods and theoretical concepts of this lecture can be found on links 1, 2, 3 and 4 in section "Readings and other resources".

1. 1. Modelling processes and mechanisms involved in pathogen transmission

Marine disease models represent the dynamics of a variety of host-pathogen systems including those unique to marine systems where transmission of disease is by contact with waterborne pathogens both directly and through filter–feeding processes. Overall, the analysis of the epizootiological models focused on the most relevant processes that interact to drive the initiation and termination of epizootics. *A priori*, systems with multi-step disease infections (e.g., infection-death-particle release-filtration-transmission) reduced dependence on individual parameters resulting in inherently slower transmissions rates. This is demonstrably not the case; thus, these alternative transmission pathways must also considerably increase the rates of processes involved in transmission. Scavengers removing dead infected animals may inhibit disease spread in both contact-based and waterborne pathogen-based diseases. The capacity of highly infected animals, both alive and dead, to release a substantial number of infective elements into the water column, making them available to suspension feeders results in such diseases being highly infective with a very small 'low-abundance refuge'. In these systems, the body burden of pathogens and the relative importance between the release and the removal rate of pathogens in the host tissue or water column becomes paramount. Two processes are of potential consequence inhibiting epizootics. First, large water volumes above the benthic susceptible populations can function as a sink for pathogens. Second, unlike contact-based disease models in which an increase in the number of susceptible individuals in the population increases the likelihood of transmission and epizootic development, large populations of filter feeders can reduce this likelihood through the overfiltration of infective particles.

1.2. Contact-based diseases

Proliferation of marine infectious diseases (MID) substantially impacts the structure and function of diverse ecosystems by causing significant mortalities in ecologically relevant populations of a wide range of marine organisms.How epizootics are initiated and terminated in terrestrial organisms has been described and modeled extensively. Typically, the contact–based and vector-borne infectious diseases of terrestrial vertebrates and their epidemiology are modeled considering that the initiation of an epidemic event begins with one or a few infected individuals and a large number of susceptible neighbors with whom contact is possible. Thus, it is assumed that relatively close contact between the infected individual, or the vector, and the host is required for transmission.

Contact-based diseases also exist in the marine environment, most frequently in fishes , being common in the case of the transmission of multicellular parasites such as trematodes or cestodes. In MIDs, in addition to live infected animals, dead infected animals are an important source of pathogens. For instance, the pathogen body burden in dead oysters infected by Dermo disease and the potential release rate upon death is much higher than those of infected live animals. Similarly, fish that died of disease can be a source of infection by releasing pathogen particles to the surrounding water . In the terrestrial environment, this transmission route is less well represented, although one well known example is the microparasite *Bacillus anthracis*, which infects both humans and animals and where the infectious agent is spores that enter the environment soon after the death of a host .

1.3. Waterborne pathogens, filter-feeders and disease spread

One of the most distinctive features of MIDs, particularly for marine invertebrates, is the importance of spatial factors in determining the spread of disease. The differences in physical properties between seawater and air, such as density, result in greater buoyancy, longer life spans, and longer-distance dispersion for aquatic organisms including pathogens. This, in turn, can result in important pathogen dispersion, concentration, and availability issues for some invertebrates such as sessile filter and suspension feeders (e.g., bivalves and corals). Such species can accumulate pathogens from a dilute solution that may have been released nearby or from many kilometers away, thus the number of neighboring infected individuals may be relatively unimportant in comparison with the number of infective pathogens being supplied by water transport. This suspension– or filter–feeder life style, highly vulnerable to disease transmission and widespread, is a rare condition in terrestrial animals, and apart from swallows who snag insects on the wings when flying, the nearest approach to this condition are the web-spinning spiders. No mechanism has evolved for concentrating particles from the atmosphere in sufficient quantity to provide an adequate food supply for a terrestrial filter feeder.

Disease transmission in filter-feeders probably occurs via an infective dose rather than by unique contact between pathogen and host. The phenomenon of the infective dose may be particularly important for filter-feeders because overfiltration (i.e., the water is filtered more than once as it passes through the population) can occur when the density of animals is high enough; thereby reducing the pathogen concentration available sufficiently to permit the competition for pathogens and the internal inactivation mechanisms to limit body burden below the infective dose level.

Model adaptations to long–distance infection often assume that infected individu-

als cross distance barriers at some rate to make contact with susceptible hosts or define contact. Notwithstanding that the development of an airborne disease in, for instance, a plant metapopulation involves a process of dispersion as well as local dynamics, the transmission process itself can be modeled as a contact–based and point–source process . The effect of pathogen dilution on non–point–source marine diseases transmission common in suspension–feeders , has not yet been investigated theoretically.

The distinctive characteristics of MIDs together with the limited barriers to dispersal potentially makes oceans a much more favorable medium than land for non–point–source processes to control the transmission process and the generation of epizootics. These characteristics are a primary reason why adaptation of terrestrial epidemiological models to marine diseases remains one of the poorly addressed problems in MIDs. In contrast, proliferation–based disease models have received considerable attention since understanding of proliferation of infection was sufficient to describe the disease impact in populations characterized by rapid non–point–source transmission.

This course focuses on the formulation of a series of models exemplifying the dynamics of a variety of MIDs representative of a diversity of host, pathogen, and transmission processes present in marine ecosystems. In this course, disease transmission is caused by either direct contact between susceptible and infective animals, by contact with waterborne pathogens released by live or dead infected animals through passive impingement of infective particles via water currents or through active filtration of infective particles during filter feeding. The formulation and description of each model is presented together with examples of marine host-pathogen systems which might be appropriate examples of the given transmission model. For each modeled MID system, we analyze the basic reproduction number R_0 and consider how changes in model parameters vary the outcome of the transmission process relative to the threshold condition of R_0 =1.

1.4. Theoretical basis for the models

In this course a series of models adapting to a greater or lesser extent the mathematical theory of epidemics are formulated to represent infectious disease transmission processes and dynamics in marine systems. For this purpose, the more complex sessile invertebrate disease models, including contact with or filtration of waterborne pathogens and particle diffusion processes, are built up from those simpler contact-based SI models applied to fish and mammal diseases. The models presented here do not cover facultative bacterial parasites or complex life cycles of protozoan or metazoan parasites requiring intermediate hosts.

The lessons are restricted to compartmental models, the most frequently used class of models in epidemiology. The dynamics of the host-pathogen association is described by a system of ordinary differential equations (ODEs) which reproduce the change with time (in days) deterministically for all subpopulation components. More specifically, models assume a constant area (in m^{-2}) or volume (in m^{-3}), in order to describe the population in terms of density of individuals or concentration of pathogens instead of simply the number of individuals or particles. Absence of migration or recruitment is assumed and non-disease mortality is ignored. In addition, infected individuals always die from disease; individuals do not recover from the disease and, hence, also do not become immune to the disease. This is routinely the case for marine infectious diseases in invertebrates because invertebrates do not have adaptive immune systems excepting some postepizootic coral populations with adaptive immunological resistance in surviving individuals and recuperation of aquaculture species after antibiotic treatment.