Potential effects of outside host community diversity on population dynamics of *Flavobacterium columnare* – a modeling study

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Abstract

The epidemiological dynamics of *Flavobacterium columnare*, the causative agent of columnaris disease in fish, have not been explored by the use of mathematical modeling. Additionally, the classical epidemiological models are mainly ill-suited for this task because epidemiology of columnaris disease does not meet the underlying assumptions of these models. *F. columnare*, unlike strictly obligate pathogens, may grow actively in the environment and as such survive indefinitely outside a host. This enables transmission via the environment, and makes the disease persistent; an outbreak may occur even without a single infected individual in the initial host population. Classical models do not allow this, and as such may not be suitable for facultative, or opportunistic, pathogens.

Another special aspect that is characteristic of *F. columnaris* is that it is saprotrophic, meaning that a large part of its multiplication in the infection cycle results from consumption of a dead host body [1]. Classically it is seen that virulence of a disease is traded off with transmissibility because obligate pathogens require a living and moving host for transmission. With the ability to be transmitted via the environment and consume the dead host body it would only benefit the pathogen to kill its host. Environmentally transmitted diseases can thus in general be highly lethal, which is indeed seen both in practice and in theory [2]. The dynamics of saprotrophism have only been treated in a model by Godfray et al. [3].

It is completely overlooked also in recent epidemiological theory that the pathogens may be affected by ecological interactions in the environment in between infections. Many interactions with other organisms affect the bacterial densities negatively, with the exception of the bacteria feeding on some organism, as e.g. in a disease. In the environment, bacteria face competitive pressure from other bacteria, infective pressure from bacteriophages, and predatory pressures from many larger organisms. In the end the environment determines largely how well the pathogenic bacteria fare and how many there are when a suitable host becomes available. The probability of infection, as well as the severity of an infection, is known to be dose dependent [4]. Therefore, if the environment supports only a low pathogen density the initial infection is less likely to occur, and vice versa. With a saprotrophic pathogen the first few host deaths are likely to result in a massive increase of pathogen density in the environment possibly causing an infection cascade when the remaining hosts are in contact with an increasing amount of pathogens. Unfavourable environmental conditions may, however, decrease the chances for successive infections.

Mathematical modeling is an excellent tool for exploring possible epidemiological dynamics and producing hypotheses for various areas of interest. Concerning the columnaris disease specific dynamical models have not been published. Mathematical modeling tools could, however, provide insight into the epidemiology and perhaps more importantly generate and explore hypotheses concerning the control and management of *F. columnare* epidemics. With the inclusion of ecological interactions in the model it would be natural to consider mainly

‘soft’ or ‘biological’ control methods as such and in combination with more straightforward removal and growth limiting methods.

Our idea was to construct a dynamical model that would be in accordance with the epidemiologically important properties of *F. columnare*, i.e. environmental growth and saprotrophy. The ecological interaction we have chosen to implement in this model is competition for nutrients, which is perhaps the most prevalent of all ecological interactions. Bacteria compete with each other in various ways and therefore a good starting point for modeling was the most general one, the well-known Lotka-Volterra competition model. In the model one bacterial strain was set to be facultatively pathogenic, reacting with a susceptible host compartment to generate infected hosts. The infected hosts could either recover with no immunity or die from the disease. With the death of an infected host a number of pathogenic bacteria would emerge to the environment. In other words, the constructed model combined a Lotka-Volterra type competition system for bacteria in the environment, and a classical SI-type epidemiological model for hosts. In the model we assume that there is one pathogenic species and a certain number of non-pathogenic competitors.

Analysis of the model shows that there are three possible outcomes: a) competition in the outside host environment prohibits the pathogen from reaching high enough densities for infections to occur, b) long-term cyclic outbreaks of infection, and c) the pathogen causes a rapid cascade of infections leading to host extinction. The reduction in pathogen densities because of competition is dependent on two things: the trade-off, or price, in competitive ability the pathogen has to pay for being able to infect, and the number of competing species. These together form the intensity of competition and are interchangeable in their effect.

In columnaris disease cases in fisheries the only favourable outcome is that the pathogen densities remain too low for infection cascades, whether cyclic or stable, to occur be it from competition in the environment or any other reason. What the model shows is that competition may help to prevent infections. The competitive ability of *F. columnare* is, of course, fixed in a given environment, but the intensity of competition depends on the competitive abilities other micro-organisms present. This can be increased or decreased e.g. by inoculating with certain species or using disinfectants.

Any action to remove competition, such as the use of unspecific antibiotics, disinfectants etc. may inadvertently result in the pathogen momentarily reaching high enough densities to cause a few infections, which in turn may cause several more. In fact, any disturbances in a complex microbial community might cause transient fluctuations in bacterial densities with a risk that the pathogen density could reach a high enough density to efficiently cause infections.

According to the model competition is an important factor in epidemiological dynamics of environmentally transmitted pathogens, and should not be overlooked. To utilise competition in biological control of epidemics it would be necessary to test this in practise. Based on the model it is possible to propose a hypothesis that introducing a few efficient competitors against *F. columnare* may prevent the initial infections and thus the infection cascade in fisheries. Relatively low cost is a clear advantage of this management procedure as compared to many others.

We suggest that the modeling approach could be used to gain a better understanding of all disease management possibilities in fisheries. The effects of using e.g. bacterial predators or

bacteriophages, as well as antibiotics and disinfectants, could be modeled and simulated before turning into expensive field trials and tests.

References

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