Intensive fish farming and the evolution of pathogen virulence: the case of columnaris disease in Finland

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Abstract

Ecological changes affect pathogen epidemiology and evolution and may trigger the emergence of novel diseases. Epidemiological models can be used to examine how different ecological factors affect the population dynamics of the pathogens and thus contribute to disease outbreak (1). Pathogen fitness, R_0, is the number of secondary infections produced by a primary infection in a totally susceptible host population (1). Equation 1 shows the relationship between transmission (β) and host population density (N) which are affected by natural host mortality (μ), parasite-induced mortality (*a*) and recovery rate (*ν*). In the absence of genetic correlations between these parameters, R_0 is largest, when pathogen-induced mortality is at its minimum (2). However, trade-offs between the model parameters may change the situation such that any level of virulence may evolve. For example, the pathogen should be able to transmit efficiently, but too fast exploitation of the host can lead to fast killing and lack of transmission. Therefore, the adaptation of virulence to current situation is important for the survival of the pathogen.

$$R_0 = \frac{\beta(N)}{\mu + a + v}$$

Equation 1. Factors defining parasite fitness R_0.

Aquaculture radically alters the ecology of fish and their pathogens. Due to the cost effective nature of fish farming industry, fish may be cultured in densities more than 1000 times higher than under natural conditions. This means an increase in the number of susceptible hosts available (N) and the probability of a contact between infected and susceptible hosts (β) once a pathogen is introduced in the rearing unit. The factors restricting the availability of susceptible hosts (e.g. μ = annual removal of fish from the tanks or mortality due to other diseases) drive pathogen to increase its transmission rate in order to find hosts to maximize R_0.

Flavobacterium columnare is a bacterial pathogen of freshwater fish causing skin lesions, fin erosion and gill necrosis called as columnaris disease. During the last couple of decades it has become the most serious threat for salmonid smolt production in Fennoscandia (3). As a part of a disease prevention and monitoring programme, fish

diseases have been intensively surveyed at some fish farms in northern Finland since 1984. A significant increase in the occurrence and severity of columnaris disease was detected in salmon fingerlings at a fish farm over 23 years (4). The symptoms of the disease have become more harmful and the amount of antibiotic medication (treatments per tank per year) has increased. Since 1993, also the warm water period has become longer making conditions more favourable for columnaris outbreaks. (see reference number 4 for more details). We hypothesize that this emergence of columnaris disease is because of evolutionary changes in bacterial virulence.

Our hypothesis is supported by several issues present in aquacultural systems discussed in paper by Pulkkinen et al.(4). One of the most important feature of columnaris disease that allows virulence evolve to high levels is saprophytism. *F. columnare* can transmit efficiently from dead fish, and maintain infectivity in sterilized water for months (5), strongly reducing the fitness cost of host death likely experienced by the pathogen in nature.

We suggest that fish farms provide an environment that promotes the circulation of more virulent strains of *F. columnare*. This effect is intensified by the recent increases in summer water temperature. More generally, we predict that intensive fish farming will lead to the evolution of more virulent pathogens.

References

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