

# Epidemic Modelling for a Changing Industry



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## Introduction

In Scotland, there are restrictions on the siting of salmon farms, for reasons of minimising both spread of disease and environmental impact. Between 1999 to 2007, Atlantic salmon production in Scotland varied around 130,000 tonnes/year; however, the number of sites involved reduced from 189 to 135 over the same period <sup>1</sup>.

Fewer sites implies greater separation of sites, which would be expected to act as a 'firebreak', reducing the chance of spread of disease between sites through the water column. But increasing density of susceptible population in a focal area might be expected to increase the risk of local epidemics. I constructed a mathematical model to investigate which effect dominates, and when.

## The approach

The model has the following features:

- **Compartmental** – models infected and susceptible individuals.
- **Metapopulation** – models individual fish within sites, and sites within a region.
- **Stochastic** – gives a range of potential outcomes for a given scenario.

We would like to know from it:

- Will disease spread? (Epidemic when  $R_0 > 1$ )
- How fast will it spread?
- How big an epidemic might be obtained?
- How does this differ for different diseases?

## Analytical solution

$R_0$  is obtained as with the SIR model, at equilibrium.  $R_0 > 1$  means a possible epidemic.

$$R_0 = \frac{\beta A(1 + \Xi)}{n\mu(\gamma + \mu)} \quad \Xi = \sum_{u=1}^n \xi_{u,1}$$

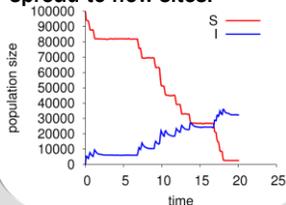
Setting both differential equations ( $s$  and  $i$ ) to zero, equilibrium prevalence is obtained ( $I = \Sigma i$ ).

$$I_\infty = \frac{A}{\gamma + \mu} - \frac{\mu n}{\beta(1 + \Xi)}$$

Above is the solution for density dependence.

## Stochastic spread

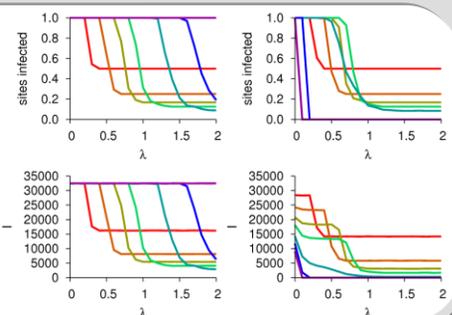
Simulations (Gillespie algorithm) model within-site spread, punctuated by spread to new sites.



## Parameter sensitivity analysis

We can now model for a range of possible diseases: Which parameters are important to know?

I ran the model until time  $t = 50$  and recorded numbers of infected sites (upper panels) and individuals (lower panels) for different  $\lambda$  and  $n$  ( $n$  varied from high – low), for both frequency (left panels) and density dependence (right panels). Farm count is more important with density-dependence, low  $\lambda$  worse for facilitating epidemics. These parameters can be estimated from disease data for specific diseases where available.



## Single-site model

Sites are stocked ( $a$ ) and harvested ( $\mu$ ), susceptible fish ( $s$ ) infected ( $\beta$ ), and infected fish ( $i$ ) are culled ( $\gamma$ ).

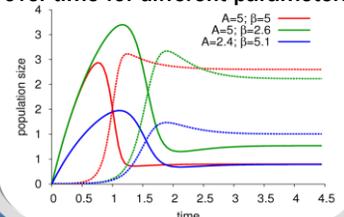
I used density dependence and frequency dependence to model intra-site infectious contact.

density-dependent: 
$$\begin{aligned} i &= \beta s i - \gamma i - \mu i \\ s &= -\beta s i - \mu s + \alpha \end{aligned}$$

frequency-dependent: 
$$\begin{aligned} i &= \beta' \frac{s i}{s + i} - \gamma i - \mu i \\ s &= -\beta' \frac{s i}{s + i} - \mu s + \alpha \end{aligned}$$

## Deterministic simulation

Populations of  $s$  and  $i$  (dotted) over time for different parameters.



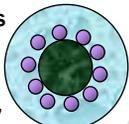
## Multi-site model

Consider an "island" of circumference  $D$  with a ring of  $n$  farms, with total stocking of all farms equal to  $A$ .

Sites  $u$  and  $v$  are distance  $d_{uv}$  apart with force of infection  $\beta \xi_{uv}$  between them.

Intra-site spread is handled separately from within-site

( $\xi_{uu} = 0$ ) and logically,  $\xi_{uv} < 1$  between sites.



## Discussion points

- Density- or frequency-dependence are appropriate for different pathogens. Modelling density in a shoaling animal is difficult.
- Diseases may be subject to a threshold infectious dose effect.

- Particle-tracking models could be useful in parameterising  $\xi$ .
- Similarly, network models incorporate contact information.
- Further work should consider synchronised following periods.

References: <sup>1</sup><http://www.frs-scotland.gov.uk/FRS.Web/Uploads/Documents/surveytext2007final.pdf>

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