

# Simple modelling of disease transmission in the aquatic environment



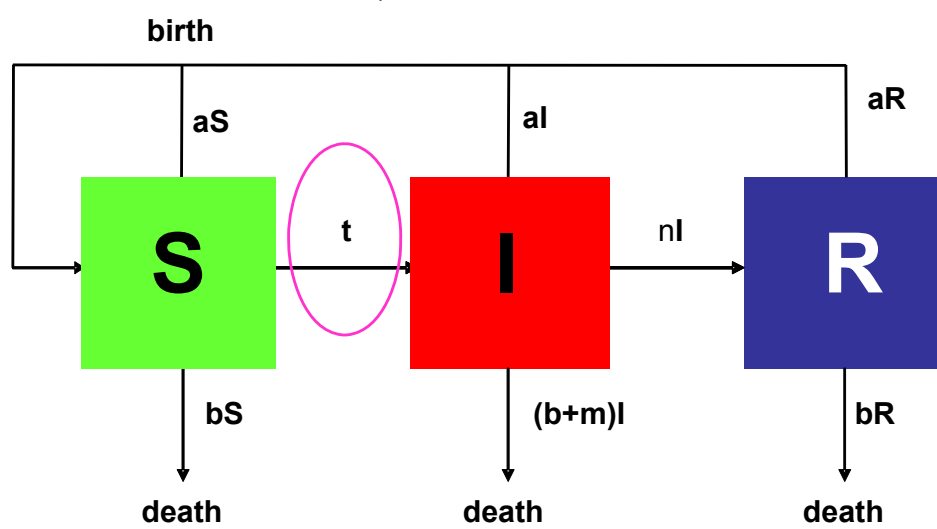
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## Abstract

Disease plays an important role in the functioning of aquatic ecosystems, affecting organisms from bacteria to marine mammals. However the applicability to aquatic environments of models derived from study of diseases in terrestrial systems has been questioned. Here simple Susceptible Infected-Removed models with four different transmission functions are used to analyse the effects of different forms of disease transmission.

The models have been used to analyse conditions when disease outbreaks may occur, pathogens can be eradicated, hosts can become extinct and if stable endemic infection can result. The results have been used to identify effects of different management activities on disease impacts and the models are also useful as a tool for analysing wild-farm interactions.

Figure 1. SIR model structure,  $t$  = transmission term which differs between models  
S = susceptible; I = infected; R = recovered



### Model equations

$$\begin{aligned} dS/dt &= aN/(N+k) - bS - t & (1) \\ dI/dt &= t - rI & (2) \\ dR/dt &= nI - bR & (3) \end{aligned}$$

$$\begin{aligned} N &= S + I + R \\ r &= (b + m + n) \end{aligned}$$

$a$  = birth rate,  
 $b$  = background mortality rate,  
 $m$  = disease mortality rate,  
 $n$  = disease recovery rate

$t$  = transmission, see text below

## Four models of transmission $t$

**Model I.** Transmission increase as numbers of hosts increases  $t = \beta SI$ . This is a Standard epidemiological model for diseases such as influenza.

**Model II.** Transmission depends on proportion of hosts infected and susceptible  $t = \beta SI/N$ . This is a standard model for vector or sexually transmitted diseases, could also apply where separation of fishes is independent of school size.

**Model III.** Transmission requires a minimum dose of infection as a simple model of this I use  $t = \beta I^2 S$ . The non-linear response of this model reflects the rapid increase in spread of infection when threshold dose reached (locally the dose may exceed infection even if not globally, hence some transmission occurs at low I)

**Model IV.** Pathogens are transported for relatively long distances and hence infectious pressure is independent of local infected population  $t = kS$ .

$\beta$  = transmission coefficient;  $k$  = constant

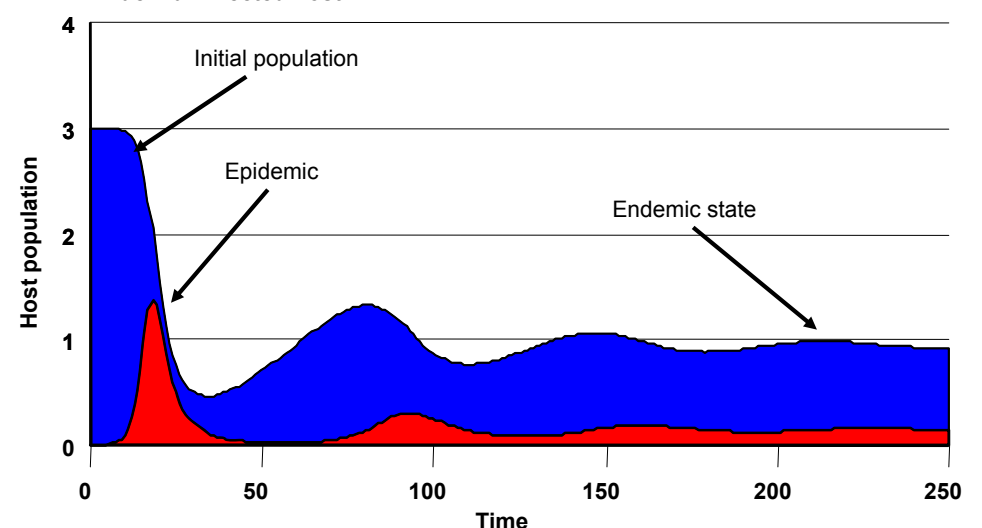
Table 1. Four models of transmission  $t$ . Disease spreads when  $t = rI$  (equation 2). From this we find the threshold  $S$  required for an epidemic under the models

Transmission	Situation	Form	Threshold
Model I	Randomly mixing population	$\beta SI$	$S_t = r/\beta$
Model II	Schooling population, vectors	$\beta SI/N$	$S_t = rN/\beta$
Model III	Transmission dependent on local pathogen concentration in water	$\beta I^2 S$	$S_t = r/\beta Y$
Model IV	Persistent pathogen transported from different source population	$cS$	$S_t = 0$

Table 2. Stable end points of epidemics under the different models

Model	Pathogen may be driven to extinction	Pathogen may become endemic	Host may be driven to extinction
Model I	Yes	Stable	No
Model II	Yes	Stable	Yes
Model III	Yes	Unstable	No
Model IV	No	Stable and unstable	Yes

Evolution of infection (red) from epidemic to endemic state (Model I). Blue = uninfected host.



Management actions can increase (▲), decrease (▼) or have no effect (●) on impact of disease on farmed fish – impact depends on both action and nature of pathogen transmission

Management action	Model I	Model II	Model III	Model IV
Farm density	▲	●	●	▼
Farm size	●	●	▲	▼
Confinement	●	●	▲	●
Biosecurity	▼	▼	▼	▼
Fish inputs	▲	▲	▲	●
Culling: pre-emptive	▼	●	●	▲
Culling: targeted	▼	▼	▼	▲
Vaccination	▼	▼	▼	▼

## Conclusions

Simple models are used to give a flavour of how different modes of Pathogen transmission can result in qualitatively different behaviours of epidemics. Analysis is straightforward, but is rather lengthy and so for reasons of space is not detailed here. However, differences in impacts on host population and optimal management are substantial.