Solution to Exercise 3: Description of SIPF and SIPDF models for marine disease transmission through filtration of environmental pathogens

Some marine microparasitic pathogens can survive several months in the water column to be absorved by filter-feeders (e.g. bivalves). Once inside, the pathogen can invade the host if they find suitable conditions for reproduction. The host can also fight agains the pathogen (e.g. phagocytosis). This transmission from the environment occurs via pathogens released from infected and dead infected animals. In this SIPF model, pathogens are released by infected animals. Examples of host-pathogen systems associated to this model and the alternative model SIPDF for which pathogens are released by infected dead animals are shown in Table 1.

Model	Transmission	Applicable systems
SIPF	Filtration of infective particles released by infected individuals; dose dependence	OsHV1 in pacific oysters; MSX and Dermo diseases in oysters; Perkinsosis in clams.
SIPDF	Filtration of infective particles released by dead infected individuals; dose dependence	Oysters infected by Dermo disease (*Perkinsus marinus*) release pathogens into the water by natural decomposition or the action of scavengers, then to be filtered by the population. This is a likely route for many other molluscan diseases.

Table 1: Models, model characteristics, and example disease potentially applicable. The disease list is not meant to be comprehensive, nor does a unique mention of a disease imply restriction of the disease to that particular model

As in the previous lesson, R_0 is formulated for each model and details for getting R_0 of these models by applying the next-generation matrix (NGM) method is described

in lesson 5.

SIPF model

SIPF model incorporates the filtration of infectious particles by, for example, bivalve filter-feeders. In this model the waterborne pathogens are filtered by susceptible and infected individuals at a rate f (Equation **??**). Noteworthy in this case is the fact that infected individuals also filter out infective particles; this activity represents a debit to the waterborne infective particle pool without initiating any new infections. The specific particularities of the host and pathogens will determine if f is the same or not for S and I; that is, S and I individuals may filter at different rates.

At any point, some particles will have been filtered out by the susceptible population, but these particles may not be sufficient to initiate an infection. Thus, the SIPF model also incorporates the concept of an infective dose which is considered to be important in the bivalve transmission process. F is the total number of particles inside the S population (Equation **??**). Considering that f is the portion of the local volume filtered per individual and time, the number of pathogens removed from the local volume by a susceptible individual per time is $f\,P$ and the number of pathogens filtered by the population from the local volume is represented by $f\,P\,S$ (see parameters description in Table 2).

The internal pool of pathogens in the susceptible population is a balance between the rate of uptake by filtration and the rate a of inactivation by or loss from the animal, which might be due to pseudo-fecal rejection, defecation, digestion, deactivation by the immune system, or diapedesis. As the total number of filtered particles (F) increases, the average body burden in the susceptible population increases, which in turn increases the rate of infection. F/S represents the average pathogen body bur-

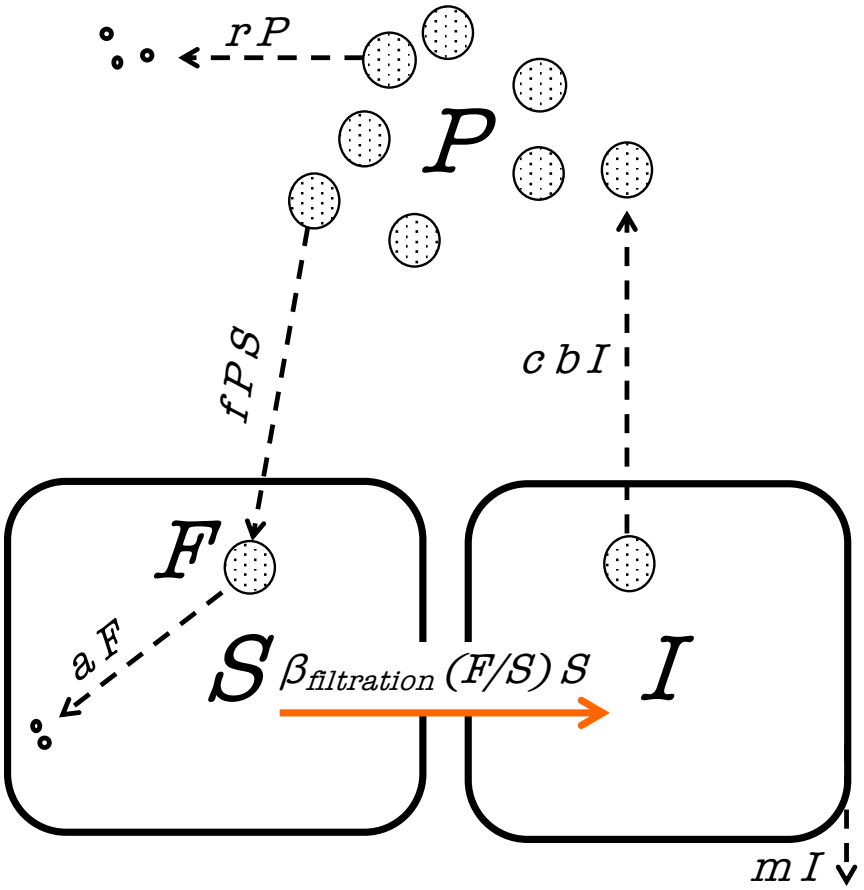

Figure 1: Flow diagram for the SIPF model. The variables (compartments) for each model are represented by upper letters: susceptible animals S, infected animals I, infected dead animals D, infective free-living particles P, and internal pool of particles in the susceptible population F. The model parameters are represented by lower letters described in Table 2. Orange solid arrows represent the transmission processes and dashed black arrows represent the other main processes.

den of the susceptibles. Consequently, disease transmission is linearly proportional to the average body burden per individual times the number of individuals; that is, $\beta_{filtration}(F/S)\,S$ (Equation **??**).

This model diverges from previous models in three important ways: (1) S no longer is present as a discrete variable in equations **??** and **??**: the variable F acts as a surrogate; (2) infective particles are lost due to mortality or dilution r, but also

by filtration f: that is, the population is an active contributor to particle loss; and (3) the dose-response relationship is described by the new Equation **??** that relates filtration f to particle loss a. The governing equations are:

$$\frac{d\,S}{dt} = -\beta_{filtration}\,\frac{F}{S}\,S; \tag{1}$$

$$\frac{d\,I}{dt} = \beta_{filtration}\,F - m\,I; \tag{2}$$

$$\frac{d\,P}{dt} = c\,b\,I - (r + f\,(S+I))\,P; \tag{3}$$

$$\frac{d\,F}{dt} = f\,P\,S - a\,F. \tag{4}$$

The basic reproduction number for this model is

$$R_0 = \sqrt[3]{\frac{\beta_{filtration}}{m}\,\frac{c\,b}{a}(\frac{f\,N}{r+f\,N}}). \tag{5}$$

In this model, R_0 increases nonlinearly with increasing N (Figure **??**). The generation of an epizootic is regulated by the same parameters as in the SIP model (Equation **??**), but also by the filtration rate f and the inactivation rate of pathogens inside the animal a. Large populations with a relatively high filtration rate are less vulnerable to epizootic development in conditions of relatively high disease mortality m and relatively high inactivation of pathogens inside the animal a with respect to the release of pathogens c.

The initial population N and the removal of pathogens from the water, by filtration f or by dilution or loss r, have varying influences on R_0. R_0 is less sensitive to changes in N particularly when the inactivation of pathogens in the environment r is slow or filtration rate is relatively high. Similarly, the model is relatively less sensitive to changes in filtration rate beyond a certain f, and more drastically for low r.

The impact of the particle loss rate from the waterborne particle pool is determined by the ratio between the loss due to filtration of particles by the population and the total loss rate ($fN/(fN+r)$). When fN is relatively much smaller than r, due

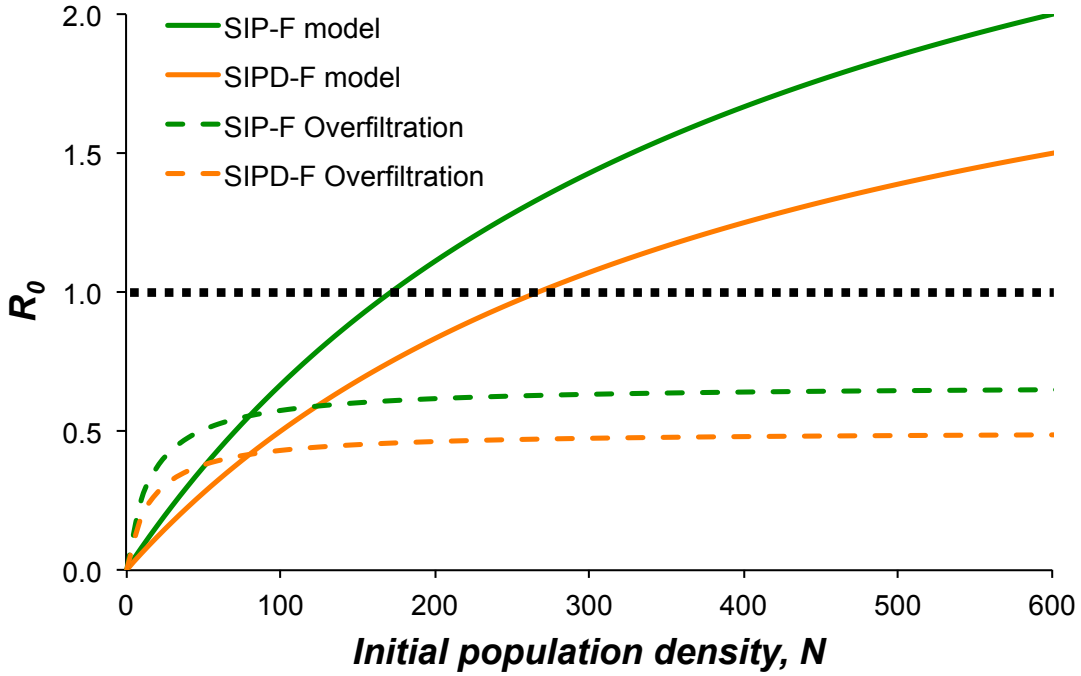

Figure 2: Theoretical estimations of R_0 for SIPF and SIPDF (Table **??**) were obtained using the following values of the parameters $m = d = 1{\times}10^{-1}$), $c = 1{\times}10^{-3}$, $b = 1{\times}10^{4}$, $r = 8{\times}10^{-1}$), $\beta_{filtration} = 1{\times}10^{-5}$, $f = 2{\times}10^{-3}$, $a = 1{\times}10^{-3}$. Parameters are described and units are presented in Table **??**. The orange dotted line at $R_0 = 1$ represents the critical value for the epizootic to occur.

to the fact that filtration rate is very low or the initial population is small, the pathogen inactivation rate in the waterborne pool is an important limiter on epizootic development. In contrast, when filtration rate is high, or the initial population N is large, both leading to high fN with respect to r, then $fN/(fN + r) \approx 1$ and R_0 becomes highly insensitive to changes in r. This situation is the overfiltration scenario, wherein the population is filtering all the pathogens that are released. In this case, $P \approx 0$, so that once the population rises above a certain initial population, R_0 remains constant (Figure **??**, dashed green line). Whether the epizootic develops depends on the balance between the *in vivo* inactivation of pathogens a and the rate of particle acquisition through filtering that determines whether the body burden of infective particles will exceed the infective dose.

SIPDF model

SIPDF model is very similar to the SIPF model, but, in this case, dead infected animals (D) are responsible of releasing particles into the water (Equation **??**) instead of live infected animals. We suspect that this transmission process is common to many proliferative marine diseases that are accompanied by high mortality rates (see Table **??**), but inadequate confirmatory data exist. This model consists of a system of 5 equations:

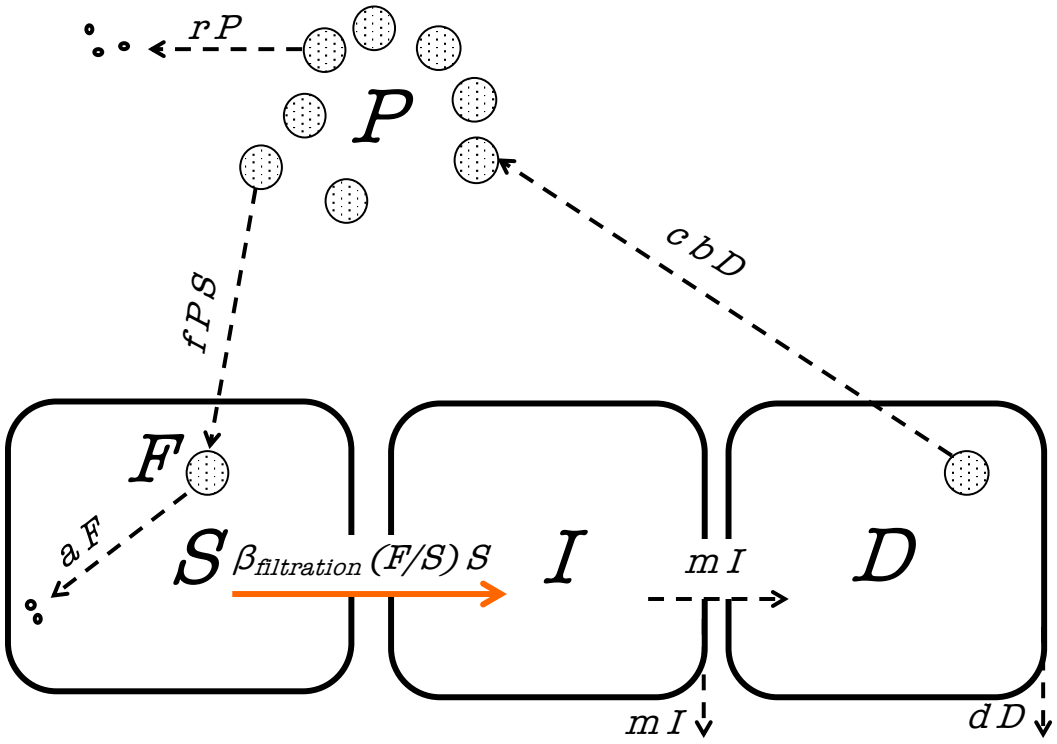

Figure 3: Flow diagram for the SIPF model. The variables (compartments) for each model are represented by upper letters: susceptible animals S, infected animals I, infected dead animals D, infective free-living particles P, and internal pool of particles in the susceptible population F. The model parameters are represented by lower letters described in Table 2. Orange solid arrows represent the transmission processes and dashed black arrows represent the other main processes.

$$\frac{d\,S}{dt} = -\beta_{filtration}\,\frac{F}{S}\,S; \tag{6}$$

$$\frac{d\,I}{dt} = \beta_{filtration}\,\frac{F}{S}\,S - m\,I; \tag{7}$$

$$\frac{d\,D}{dt} = m\,I - d\,D; \tag{8}$$

$$\frac{d\,P}{dt} = c\,b\,D - (r + f\,(S + I))\,P; \tag{9}$$

$$\frac{d\,F}{dt} = f\,S\,P - a\,F; \tag{10}$$

The basic reproduction number is:

$$R_0 = \sqrt[4]{\frac{\beta_{filtration}}{d}\,\frac{c\,b}{a}(\frac{f\,N}{r + f\,N}).} \tag{11}$$

R_0 is controlled by the removal of dead animals by the action of scavengers or natural decomposition d (Equation **??**), instead of the mortality of infected individuals m (Equation **??**; Figure **??**). As in the SID model, the nature of the process of organic matter destruction is decisive as it controls the release rate of pathogens to the water.

The sensitivity of R_0 to the parameters is lower than for the SIPF model due to the additional process involved. Although, the transmission process is inherently slower than that in the SIPF model (Figure **??**, the rate of infection is likely to be increased considerably by higher rates of some of the parameters, such as c, as in the SIPD model. The overfiltration scenario in this model also has a similar pattern to the SIPF model (Figure **??**, dashed orange line).

Variables, Parameters	Definition	Units
S	Susceptible hosts in the population	Number of individuals
I	Infected hosts in the population	Number of individuals
D	Dead infected hosts in the population	Number of individuals
P	Waterborne pathogens in the enviornmet (i.e. local pool)	Number of particles
F	Total number of pathogens absorbed or filtered by the population	Number of particles
Γ	Waterborne pathogens in a remote pool	Number of particles
N	Susceptible hosts in the initial population	Number of individuals
R_0	Basic reproduction number	Nondimensio nal
$\beta_{filtration}$	Disease transmission rate by filtration of waterborne pathogens by susceptibles.	Particle $(internal)^{-1}$ day^{-1}
m	Disease mortality rate	day^{-1}
d	Removal rate of dead individuals by scavengers or bacteria (decay)	day^{-1}
c	Release rate of pathogens from infected or dead animals	day^{-1}
b	Average body burden of pathogens in infected or dead animals	Number of particles
r	Loss rate of waterborne pathogens from the local pool	day^{-1}
f	Filtration or absorption rate of infective particles by hosts	$Individual^{-1}$ day^{-1}
a	Reduction rate of pathogens inside hosts by diapedesis, phagocytosis, apoptosis, etc.	day^{-1}

Table 2: Description of variables and parameters. The last column identifies the models in which the variable or parameter is used. An asterisk identifies the use of the variable in the R_0 formulation for that model. Note that all models have an implicit surface area (m^{-2}) or volume (m^{-3}) for individuals and waterborne pathogens respectively.