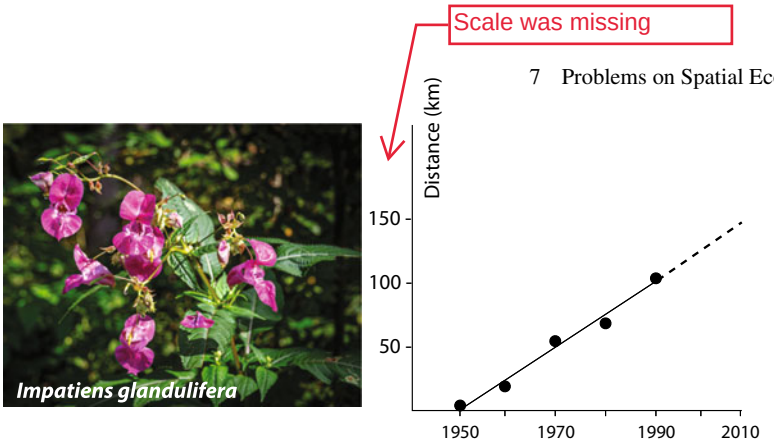



**Problem SE4** The distance travelled by the Himalayan balsam (*Impatiens glandulifera*) in Czechoslovakia as a function of years. Modified after Pyšek and Prach (1995)

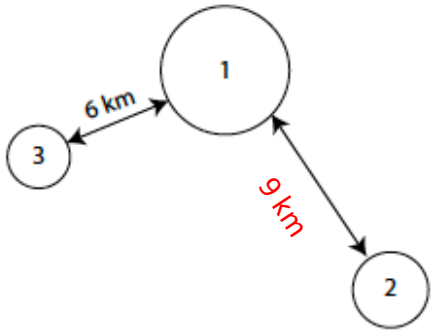
## Problem SE5

The American mink (*Neovison vison*) is a small carnivore of the mustelid family. It is an alien species to Europe where it was imported for fur farming. In Scotland fur farms were first established in 1938 and mink were first recorded breeding in the wild in 1962. Then the carnivore expanded its range.

Fraser et al. (2015) report the following information on mink expansion. In west Scotland, linear expansion rate was in the average 13.8 km year$^{-1}$, while on the coast in the far north-east of Scotland, linear expansion rate was in the average 2.6 km year$^{-1}$. The demography of mink is as follows: 0.6 female offspring per adult female per year, juvenile survival up to adulthood approximately equal to 0.9, adult survival between years approximately equal to 0.75. From these data estimate the diffusion coefficients of mink in the west and the north-east of Scotland.

**Problem SE5** The American mink

**Problem SE17** Scheme of the 3-patch metapopulation

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m a b

the same way, the fraction 1-$M$ of susceptible mosquitoes can be infected by the protozoans in the blood of the fraction $U$ of infectious humans. Typical values for $\gamma$ and $\xi$ are $1.0 \cdot 10^{-2}$ [day$^{-1}$] and $1.0 \cdot 10^{-1}$ [day$^{-1}$], that is the average recovery time of infected humans is about 100 days and the average lifetime of infected mosquitoes is about 10 days. Instead $\beta$ and $\psi$ are quite variable. Actually, $\beta$ can be considered the product of three factors: $\beta = a \cdot b \cdot e$, where $m$ is the number of female mosquitoes per human host, $a$ is the number of bites per mosquito per unit time, $b$ is the probability of transmission of infection from infectious mosquitoes to humans per bite; $\psi$ as the product ($\psi = ac$) of $a$ and $c$, the probability of transmission of infection from infectious humans to mosquitoes per bite.

Model (10.12) has two steady states solutions, namely the disease-free equilibrium (DFE), with $U = 0$ and $M = 0$, and the endemic equilibrium (EE), with

$$\bar{U} = \frac{\beta\psi - \gamma\xi}{\psi(\beta + \gamma)}, \qquad \bar{M} = \frac{\beta\psi - \gamma\xi}{\beta(\psi + \xi)}.$$

The EE is feasible if $\beta\psi - \gamma\xi > 0$. Equivalently, the feasibility of the EE is determined by the basic reproduction number

$$R_0 = \frac{\beta\psi}{\gamma\xi.}$$

Specifically, the DFE is stable for $R_0 < 1$, while the EE is feasible and stable for $R_0 > 1$. $R_0$ can be interpreted as follows. Suppose one infected human is introduced in healthy populations of humans and mosquitoes. Then $\psi$ mosquitoes are infected per unit time. As humans remain infectious for $1/\gamma$ days, this results in $\psi/\gamma$ mosquitoes being infected. In turn each of these mosquitoes will infect $\beta$ healthy humans per unit time. As infected mosquitoes stay alive for $1/\xi$ days, each mosquito, before dying, will have infected $\beta/\xi$ humans. Therefore, one initial infectious human (primary infection) will produce $\frac{\beta\psi}{\gamma\xi}$ secondary infections. Values of $R_0$ can range from around one to more than 3,000 as estimated by Smith et al. (2007) for 121 African populations.

## 10.5 Dynamics of Diseases Caused by Macroparasites

In diseases caused by macroparasites, the dynamics of parasites cannot be neglected because their average lifetime is comparable to that of the hosting organisms. Macroparasites grow inside the host but reproduce by releasing infective stages (typically eggs and larvae) into the surrounding external environment, for example through host defecation. The disease transmission occurs then through ingestion by a new host of these infective stages. Many herbivores are for example infected by intestinal worms whose eggs or larvae are ingested during grazing. Here we consider only macroparasites with a simple life cycle, i.e. without intermediate hosts.

settle in animal populations. It is to be remarked that $R_0$ does not depend on the clumping parameter $k$. Such a parameter is, however, important in determining the parasite average load: in fact, from Eq. (10.17) we can deduce that $H^*$ decreases with increasing $k$ and from Eq. (10.16) that the average parasite load $P^*/H^*$ decreases with $H^*$; thus, ultimately, it increases with $k$. Therefore greater clumping ($k$ small) corresponds to a lower average parasite load.

Until now, we have assumed that the influence of macroparasites on their hosts basically consists of increasing the mortality rate proportionally to the parasite load. However, in many cases, e.g. the already mentioned red grouse population, *Lagopus lagopus scoticus*, hosting the nematode worm *Trichostrongylus tenuis* (Hudson et al. 1992; Dobson and Hudson 1992), the macroparasite exerts its action by decreasing the fertility. We can easily analyse the consequences of this phenomenon by reformulating model (10.13) as follows

$$\begin{aligned} \dot{H} &= \left(\nu - \sum_{i=0}^{\infty} \varepsilon i p_i - \mu\right) H \\ \dot{P} &= \beta L H - mP - \sum_{i=0}^{\infty} \mu i p_i H \end{aligned} \tag{10.18}$$

where we assume that the birth rate decreases proportionally to the parasite load $i$ (with a proportionality constant equal to $\varepsilon$). By making the same assumptions about demography and density of juvenile stages of the parasites that led to formulate model (10.15), we get

$$\begin{aligned} \dot{H} &= rH\left(1 - \tfrac{H}{K}\right) - \varepsilon P \\ \dot{P} &= \tfrac{\lambda P H}{H_0 + H} - (m + \mu) P. \end{aligned} \tag{10.19}$$

The analysis of model (10.19) is simple and interesting. In addition to the trivial equilibrium $\bar{X}_0$ and the equilibrium corresponding to the healthy population $\bar{X}_K$, a third equilibrium $\bar{X}_+ = \left[\bar{H} = H^*, \bar{P} = P^*\right]^T$ can exist, at which

$$H^* = \frac{m + \mu}{\lambda - m - \mu} H_0 \qquad P^* = \frac{r}{\varepsilon} H^* \left(1 - \frac{H^*}{K}\right).$$

It is easy to see that this equilibrium is feasible ($H^* > 0$, $P^* > 0$) if the condition holds that the basic reproduction number of the disease $R_0$ is greater than unity, where

$$R_0 = \frac{\lambda K}{H_0 + K} \frac{1}{m + \mu}.$$

Even if feasible, however, this equilibrium is not always stable. In particular, it can be shown that it is unstable if $H^* < K/2$. The two situations ($H^* > K/2$ and $H^* < K/2$) are illustrated in Fig. 10.16 through the graph of isoclines and a sketch of some trajectories. In the case in which $H^* < K/2$ there is no stable equilibrium and the host and parasite populations converge toward a stable limit cycle (periodic solution) of appropriate period. Note that this situation can establish when the parasite

- $\mu$ = death rate of susceptible people = 0.015 year$^{-1}$;
- $\alpha$ = additional mortality rate due to the disease = 0.005 year$^{-1}$;
- $\beta$ = coefficient of disease transmission = 0.0001 (number of people)$^{-1}$ year$^{-1}$;
- recovery time = 0.5 months.

Assume that susceptible individuals are vaccinated at a rate $V$, where $V$ is expressed as year$^{-1}$. Then

(a) Write down the $SI$ model that governs the epidemiological dynamics;
(b) Calculate the basic reproduction number with no vaccination;
(c) Calculate how big the vaccination rate $V$ should be in order to avoid that the disease establishes in the population;
(d) Calculate the number of susceptible people at equilibrium in the case of successful vaccination.

## Problem PD7

*Cholera* is a microparasitic disease transmitted through exposure to water contaminated by the bacterium *Vibrio cholerae*. In the standard SIB model the rate at which one susceptible becomes infected is assumed to linearly increase with the bacterial concentration. A more realistic assumption is that it increases and saturates with the concentration in the following way:

$$\text{infection rate } = \beta B/(1+B)$$

where $B$ is the normalized concentration of bacteria in the water (namely the concentration of bacteria divided by their carrying capacity). It is thus possible to better analyze the dynamics of *cholera* in a human community in which the recruitment of new susceptible individuals (due to either newborns or migration from nearby communities) is a constant flow $w$ and the susceptible individuals have a mortality rate $\mu$. For the modified $SIB$ model assume that recovered people are permanently immune and parameters have the following values

- average life time of susceptibles: 70 years;
- constant flow $w = 5$ individuals day$^{-1}$;
- infection rate = $\beta B/(1+B)$ with $\beta = 1$;
- recovery rate $\rho = 0.2$ day$^{-1}$;
- contamination rate $\theta = 10^{-6}$ (No.infected)$^{-1}$ day$^{-1}$;
- death rate of *V. cholerae* $\delta = 0.2$ day$^{-1}$.

Based on these data, write down the modified $SIB$ model for the cholera dynamics.

Then, consider a population that at the endemic equilibrium is characterized by 10,000 infected individuals. For this population

(a) Compute the equilibrium prevalence of the disease;
(b) Determine the disease-related death rate $\alpha$.