V Modelling Week UCM, Madrid
Problem 4: Biological Control of Rabbits

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Chapter 1

Introduction

1.1 Biological control of rabbits

Rabbits were first introduced in Australia in 1859 with the release of twelve rabbits for hunting purposes.

For 50 years they have became a great problem because they have reached plague proportions throughout the country. This results in environment damage like erosion or outcompeting of native species for scarce food resources.

In 1901 a Royal Commission was held to investigate the situation. Once the problem was understood, various control methods were tried to reduce or limit the population of rabbits in Australia, like shooting rabbits or poisoning them.

These methods had limited success until the introduction of biological control methods in the latter half of the 20th Century.

The first bacterial rabbit disease they introduced was Myxomatosis, which initially affects the skin and eyes. It also weakens rabbits' immunity to other diseases. Almost all infected rabbits die, usually within two weeks. However, although it was very effective in decreasing the rabbit population, their numbers have been recovering since then as resistance to myxomatosis developed. This is the reason why they then introduced a calicivirus that spreads rabbit haemorrhagic disease (RHD), but the situation seems to be the same as the resistance is again developing.

In order to understand what makes a biological agent effective we are interested in using mathematical models of epidemics.

1.2 General view of the models

We are going to consider two mathematical models of epidemics:

- Mathematical models in which the rabbits´disease is transmitted by rabbits´contact (SIR Model and variants).
- Mathematical models in which the rabbits´disease is transmitted by fleas (vector-driven dynamics model).

For each model we consider two different options:

- temporal (ODE models)
- spatiotemporal (PDE models)

1.2.1 Rabbit to rabbit transmission

The easiest model considers evolution of the following populations:

- Susceptible rabbits, which are healthy rabbits that could get the disease.
- Infected rabbits, which are ill rabbits.
- Recovered rabbits, which are immune and dead rabbits (in this case, practically all will be dead).

subject to the following rules:

- When an infected rabbit encounters a susceptible rabbit, the infected rabbit population grows and the susceptible rabbit population decreases.
- Infected rabbit population decays at a constant rate into recovered rabbits.

▶ It is called the SIR Model.

We can also add/leave out some factors or populations depending on the purpose of our model. Firstly,

- Recovered rabbits altogether are omitted, because they don't have any influence in other populations dynamics.
- Natural growth of susceptible rabbits' population, which is exponential, is added.

▶ We get the Lotka-Volterra Model.

The next step could be change exponential growth into logistic growth, which is more realistic.

▶ We get a model where the rabbit populations are bounded.

1.2.2 Disease transmission by fleas

As we have done before, we start our study with the easiest model, introducing a new population

- Infected fleas

and considering the following evolution possibilities:

- Infected rabbit population increases as a result of contact between susceptible rabbits and fleas.
- Infected rabbit population decreases exponentially.
- Infected flea population grows in proportion to the number of infected rabbits and decreases exponentially.

▶ If we do these, we get the vector-driven SIR model.

1.2.3 Other considerations

Furthermore, We can consider more complicated factors for each model we have studied. As an example, we have done some research in the following aspects:

Introducing spatial variation of one or more populations into the model

- by adding a diffusion-like term representing random movement of rabbits and/or fleas.

▶ We get a SIR-diffusion model.

Introducing Time-varying rate of infection

- λ is chosen periodically time variating.

▶ We get a time-varying SIR model.

A complete model could be the result of a combination of all those aspects.

Chapter 2

First attempt

2.1 SIR model

SIR Model consists on dividing rabbit population into three populations that are mutually exclusive: susceptible rabbits, infected rabbits and recovered ones. Considering that infected and recovered rabbits depend linearly on the contact between an infected and a susceptible ones, and that infected rabbits´ evolution and recovered rabbits evolution depend linearly on infected rabbit population, we can write:

$$\begin{cases} \dot{S} = -\alpha IS \\ \dot{I} = \alpha IS - \beta I \\ \dot{R} = \beta I \end{cases}$$

where $\frac{1}{\beta}$ means the period of time that a rabbit is infected. During that period of time, $\frac{\alpha S}{\beta}$ shows the susceptible rabbits which could be infected.

By no-dimensionalising, we can write the system as function of only one parameter, so we could study it´s behavior and the importance of each different term. Choosing $[t] = \frac{1}{\alpha N}$ y $\lambda = \frac{\beta}{\alpha N}$, we get

$$\begin{cases} \dot{S} = -IS \\ \dot{I} = IS - \lambda I \\ \dot{R} = \lambda I \end{cases}$$

where the parameters are noted as
λ, rate of recovery

$R_0 = \frac{1}{\lambda}$, basic reproductive ratio (for $S \sim N$, where N is the total number of rabbits)[1]

2.2 Observations and discussion

The first observation we make is that, because we have divided rabbit population into three populations that are mutually exclusive ($N = S + I + R$), the number of rabbits is constant during the whole process. In fact, replacing the first and the third equations into the second one, we conclude that:

$$\frac{d}{dt}I = \frac{d}{dt}(-S) + \frac{d}{dt}(-R) \quad \Leftrightarrow \quad \frac{d}{dt}(I + S + R) = 0 \quad \Leftrightarrow \quad N \equiv constant$$

From the different values of λ we can understand the system behavior.

For example, we can wonder how the disease is going to finish. This means that we have to study what happens when $I = 0$. Because $N = S + I + R$, $I = 0 \quad \Rightarrow \quad N = R + S$. From the first and the third equations we can write:

$$\frac{dS}{dR} = -\frac{IS}{\lambda I} \quad \Rightarrow \quad S = S_0 e^{\frac{-R}{\lambda}} \quad \Rightarrow \quad N = R_\infty + S_0 e^{\frac{-R_\infty}{\lambda}}$$

Therefore,
$\lambda \ll 1 \quad \Rightarrow \quad R_\infty = N$, that means that all the rabbits are recovered.
Whereas
$\lambda \gg 1 \quad \Rightarrow \quad R_\infty = N - S_0 = R_0$, that means there isn´t illness evolution.

Solving the system numerically, we could find out that in the case of $S_0 - \lambda > 0$, we have an epidemic situation, in which the rabbits population tends to become all recovered for sufficiently long period of time (figure 2.1).

In the case of $S_0 - \lambda > 0$ and considering a sufficiently long period of time, infected rabbit population tends to almost zero, which means the disease dies away (figure 2.2).

[1]For example, for SARS, $R_0 \sim 3 - 5$, or for measles, $R_0 \sim 12 - 14$

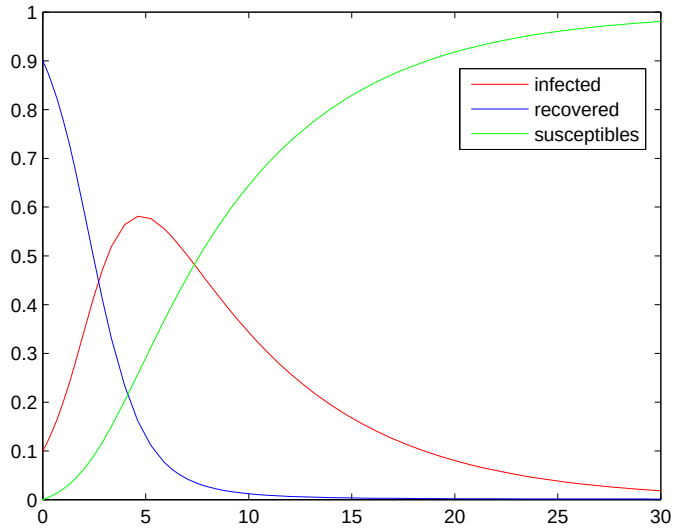


Figure 2.1: SIR, $\lambda = 0.1, T = 30$ days

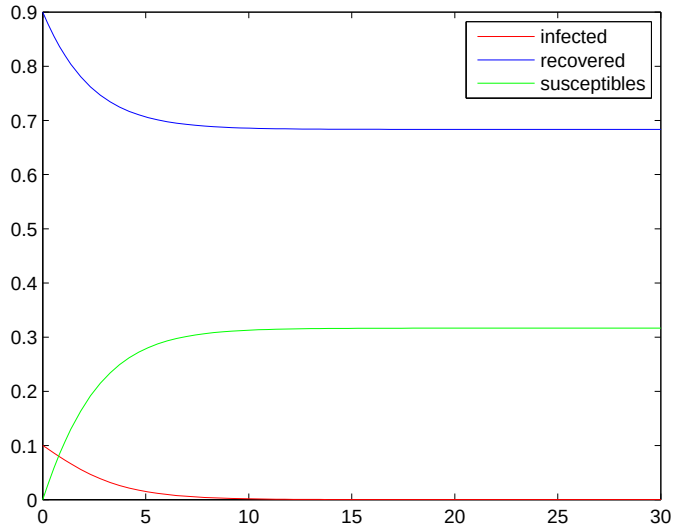


Figure 2.2: SIR, $\lambda = 1.1, T = 30$ days

Chapter 3

More realistic SIR models

The SIR model is the first step towards a sensible model of the spread of the disease. We now look to add two features: firstly growth, and then a spatial dependence.

3.1 Including Grwoth

The next step would be adding an exponential growth of the susceptible populations due to new births. Under this assumption, the population of rabbits in the absence of a disease grows exponentially. This is known as Malthus' Law, proposed in 1798. The system of equations becomes

$$\begin{cases} \dot{S} = -\alpha IS + \gamma S \\ \dot{I} = \alpha IS - \beta I \end{cases}$$

Like in the SIR model, we will proceed to nondimensionalise the system of equations.

$$\begin{cases} \frac{dS}{dt} = \frac{\#}{[t]} = -\alpha \#^2 + \gamma \# \\ \frac{dI}{dt} = \alpha \#^2 - \beta \# \end{cases}$$

stands for the unit 'number of rabbits' and $[t]$ is time. Doing the appropriate algebra we arrive to

$$\begin{cases} \hat{\dot{S}} = -\hat{I}\hat{S} + \kappa S \\ \hat{\dot{I}} = \hat{I}\hat{S} - \lambda I \end{cases}$$

Is is important to remark the meaning of the nondimensional parameters: κ is the birth rate relative to the infection rate. If $\kappa < 1$, the disease spreads

quicker than the birth rate, whereas λ is just the death rate relative to the infection rate. The higher λ, the faster a individual dies.

One would expect that for a effective population control a very aggressive disease should be introduced among the rabbit population. That is, one with a high λ. However our results show that for any $\lambda > 1$—even in the case where $\kappa < 1$—the disease kills so quickly that there is no time for spreading as shown in the following figure.

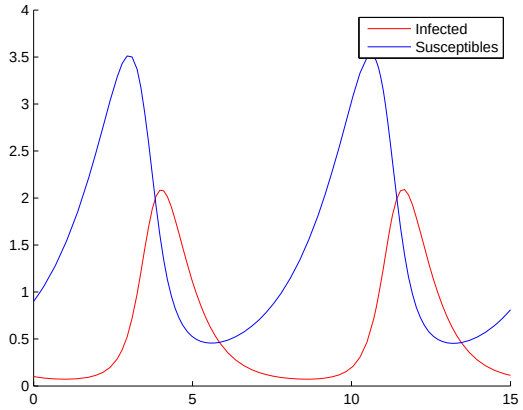


Figure 3.1: Evolution of the infected and the susceptible subjects, for $\kappa = 0.6$ and $\lambda = 1.5$

Although the behavior of the disease seems periodic, and the infected grow again when the susceptible grow enough, in reality this point is never reached, due to the so-called 'atto-fox' effect. When the population of a species reaches a value below one, then it is effectively extinct, On the other hand if the disease has a small λ the disease progressively affects the whole population.

Again the atto-fox effect is present but in the population of susceptible rabbits.

The phase portraits for both cases confirm what we have shown.

One of the main drawbacks of the model Malthusian model is that the population of rabbits grow to infinity in the case that no exiting diseases. This is not a right assumption, for two obvious reasons: Food is limited and space is limited. Therefore the growth might be exponential, but is should stop at a certain point. This effect is achieved with the following model:

$$\begin{cases} \dot{S} = -\alpha IS + \delta S\left(1 - \frac{S}{K}\right) \\ \dot{I} = \alpha IS - \beta I \end{cases}$$

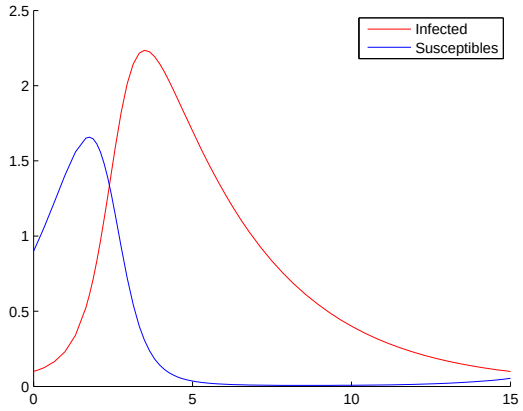


Figure 3.2: Evolution of the infected and the susceptible subjects, for $\kappa = 0.6$ and $\lambda = 0.3$

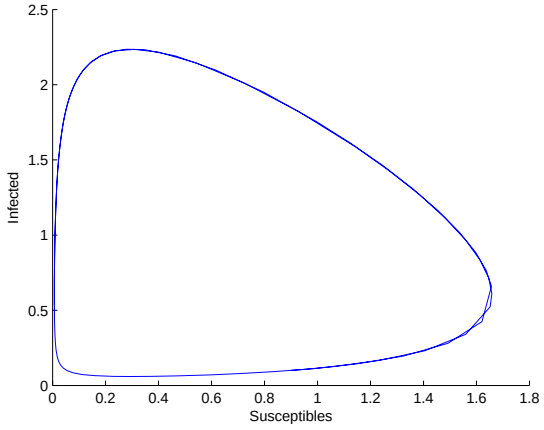


Figure 3.3: Phase portrait for $\kappa < 1$ and $\lambda < 1$

Where ι is the carrying capacity. When $S = K$ growth stops and reaches a steady state. Now the growth without diseases follow this plot:

Performing the nondimensionalisation of the equations like before, we get:

$$\begin{cases} \dot{\hat{S}} = -\hat{I}\hat{S} + \kappa\hat{S}(1 - \hat{S}) \\ \dot{\hat{I}} = \hat{I}\hat{S} - \lambda\hat{I} \end{cases}$$

where $\kappa = \frac{\gamma}{\alpha\iota}$ and $\lambda = \frac{\beta}{\alpha\iota}$. The 'physical' meaning of the nondimensional parameters is equivalent, namely κ is the birth rate relative to the infection

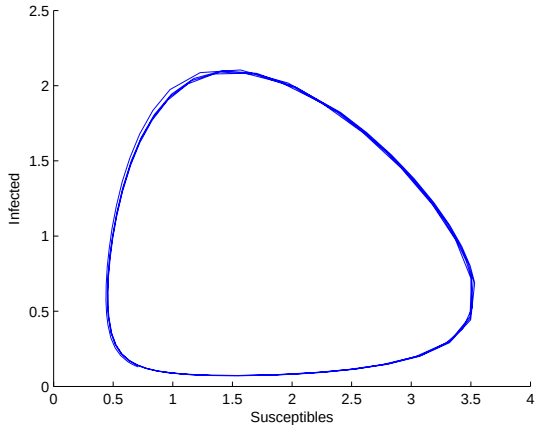


Figure 3.4: Phase portrait for $\kappa < 1$ and $\lambda > 1$

rate. If $\kappa < 1$, the disease spreads quicker than the birth rate, whereas λ is just the death rate relative to the infection rate. The higher λ, the faster a individual dies.

In figures 3.5 to 3.8 we show the temporal evolution of the species, and the phase portrait in the two cases of interest: $\lambda > 1$ and $\lambda < 1$.

3.2 Spatio-Temporal Dynamics

Our main goal is to accurately model population dynamics in the face of a disease spreading through Australia. All of our models so far have considered a population made up of susceptible, infected, and recovered rabbits. Individual rabbits move between these groups at prescribed rates that depend only on the sizes of the groups, and some scalar parameters.

However, Australia is an expansive country, and it is clear that spatial distance plays a huge role in real epidemics.

Rabbit families typically live in underground *burrows*. Such underground systems can be expansive, and many burrows can be linked together. Such a system is called *warren*, and the rabbits that inhabit it are collectively known as a *herd*. These are close-knit communities, and infection spreads easily within them. In contrast, infection between herds is rather rarer.

We suppose that we are modelling such a population on a large scale, Hence, we can approximate a 'continuum' of warrens occupying $x \in [0, 1]$.[1]

[1]This is non-dimensional, the size of the domain is captured in the nondimensional parameter D given momentarily.

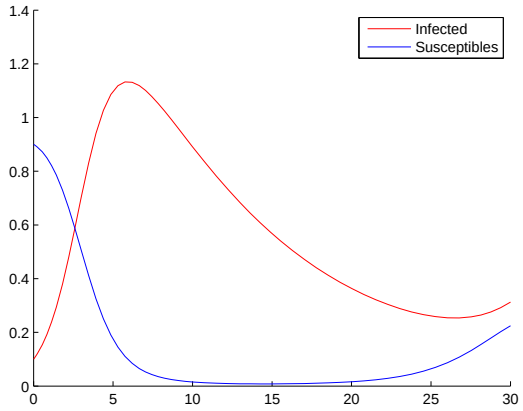


Figure 3.5: Evolution of the infected and the susceptible subjects for $\kappa < 1$ and $\lambda < 1$

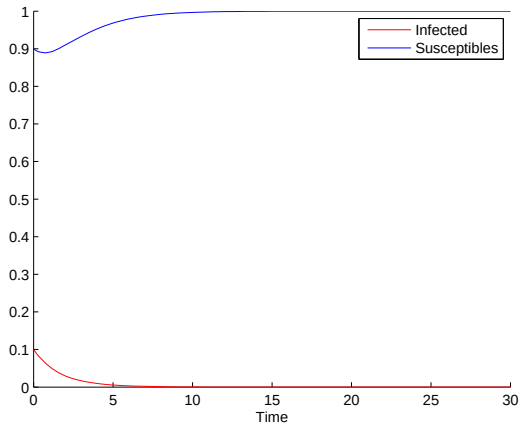


Figure 3.6: Evolution of the infected and the susceptible subjects for $\kappa < 1$ and $\lambda > 1$

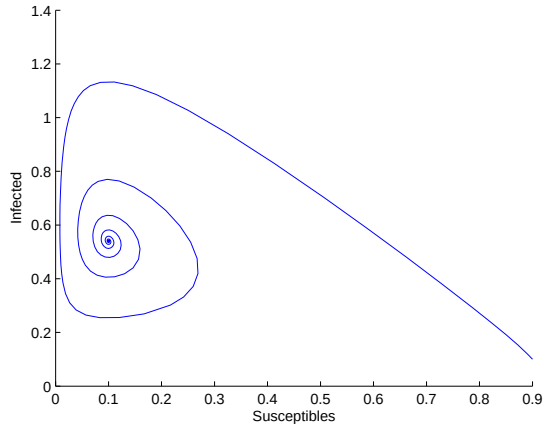


Figure 3.7: Phase portrait for $\kappa < 1$ and $\lambda < 1$

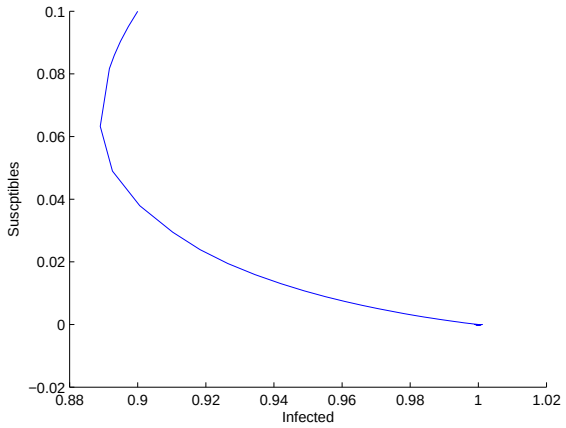


Figure 3.8: Phase portrait for $\kappa < 1$ and $\lambda > 1$

Infections move between warrens only by the movement of rabbits. To model this movement, we simply added a diffusion of susceptibles and infecteds.

$$\frac{\partial S}{\partial t}(x,t) = -S(x,t)I(x,t) + D\frac{\partial^2 S}{\partial x^2}(x,t), \qquad x \in [0,1],\, t > 0 \quad (3.1)$$

$$\frac{\partial I}{\partial t}(x,t) = S(x,t)I(x,t) - \lambda I(x,t) + D\frac{\partial^2 I}{\partial x^2}(x,t), \quad x \in [0,1],\, t > 0 \quad (3.2)$$

where D is a diffusion parameter.[2] This can be interpreted as rabbits hopping a normally-distributed distance; a *random motility* of rabbits!

We would like our rabbit-populated area to be finite. At the boundaries $x = 0, 1$, we impose homogenous Neumann conditions. This choice represent rabbits reaching a boundary, turning around, and hopping straight back.[3]

$$\frac{\partial S}{\partial x} = 0 \qquad x = 0, 1,\, t > 0 \quad (3.3)$$

$$\frac{\partial I}{\partial x} = 0 \qquad x = 0, 1,\, t > 0 \quad (3.4)$$

Of course, our initial conditions are as before, albeit with a spatial dependence.

The resulting system of PDEs can be solved numerically in Matlab using the `pdepe` routine. Our results are shown in Figure 3.9 and 3.10, where we have added exponential and logistic growth respectively.

We choose the initial population S of susceptibles to be evenly distributed in x, and the initial population of I to be non-zero only in a small portion at the left boundary. This is intended to represent an 'injection' of diseased rabbits at $x = 0$.

For our non-dimensional parameter κ we choose a realistic value based on the gestation period of a doe, their average litter size, and the time to sexual maturity of rabbit kittens. This we calculated to be around 0.2228. We choose the diffusion parameter (effectively, the length of the spatial region) so that the spatial effects are best displayed, and the λ value so that the oscillations of the system are contained below one. Ideally λ would be replaced with experimental values.

For both exponential and logistic growth we see an expected initial diffusion of the initial conditions. Both solutions then exhibit a travelling 'wavefront' of infected and susceptible rabbits, which sweeps through from left to right. This is a pleasing result, representing an initial wave in the epidemic.

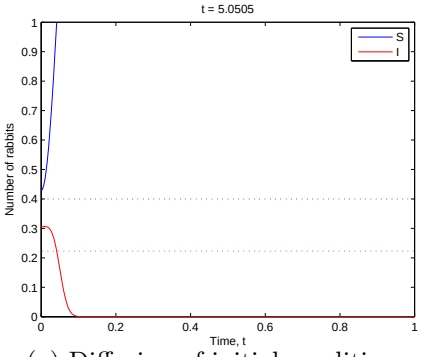
[2] D is non-dimensional, so we may happily add it straight into our non-dimensional model. It effectively controls the size of the spatial domain.

[3] At least, hopping back according to diffusion.

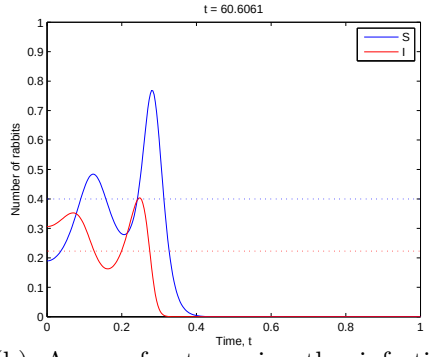
As this wavefront reaches the right boundary, the PDE system attempts to satisfy the boundary conditions. In the case of exponential growth, this results in large oscillations as the system jumps onto larger orbits in the phase plane. Eventually the system settles, with the solution at each x oscillating around the equilibrium, as predicted by the spatially independent model.

For the case of logistic growth, the system again jumps onto different paths in the phase plane as the wavefront reaches the right-hand side. However, since each path is a stable spiral, the oscillations are rather more contained. This seems far more realistic. Again, each point spirals into equilibrium.

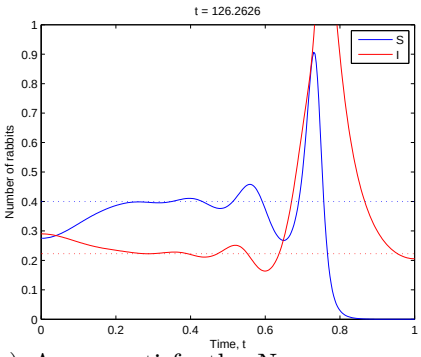
Although it is not straightforward to implement in Matlab, a natural first step would be to move to a two-dimensional model. λ could be further modified to include a spatial dependence, with lower values representing areas of Australia where it is more difficult for the infection to travel through.



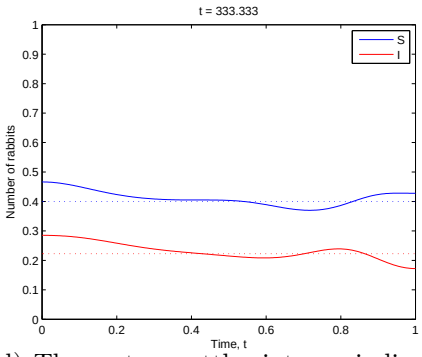
(a) Diffusion of initial conditions.



(b) A wavefront carries the infection through the warrens.

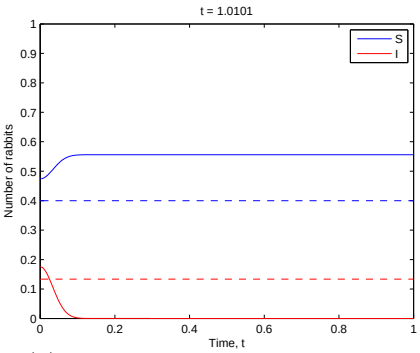


(c) As we satisfy the Neumann conditions at the right boundary, we move onto larger orbits.



(d) The system settles into periodic orbits at each x.

Figure 3.9: SIR model with spatial diffusion and exponential growth. The equilibria for S and I are given as dashed lines.



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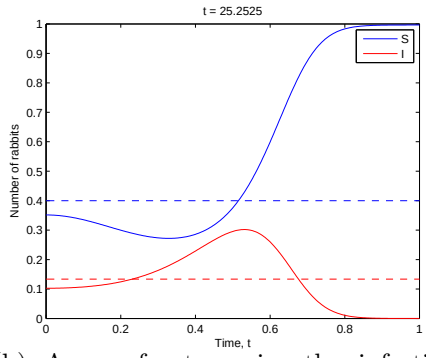
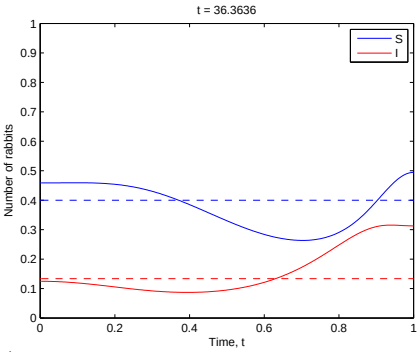
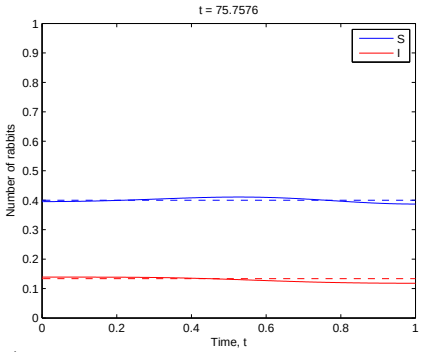


(b) A wavefront carries the infection through the warrens.



(c) Satisfying the right-boundary condition is rather less wild than the exponential growth case.



(d) Each x eventually spirals to equilibrium.

Figure 3.10: SIR model with spatial diffusion and logistic growth. λ is chosen so that the equilibrium point is a stable spiral. The equilibria for S and I are given as dashed lines.

Chapter 4

Vector-driven dynamics

4.1 SIR with infected fleas population:

Considering that fleas are the main vector of the disease we can neglect other transmission factories. We assume susceptible population, S varies in accordance with

$$\dot{S} = -\alpha SF + \delta S(1 - \frac{S}{K}), \tag{4.1}$$

where α represents the probability of infection in case of meeting between susceptible rabbits and infected fleas, F, δ is rabbits birth rate and K is the carrying capacity. Consequently the infected rabbits population, I,has to be

$$\dot{I} = \alpha SF - \beta I, \tag{4.2}$$

where β is the steady death rate. The last equation of our dynamic system represents the infected fleas variation and reads

$$\dot{F} = \gamma I - \psi F, \tag{4.3}$$

where we are assuming γ as the number of infected fleas for infected rabbits in time, while ψ is the fleas steady death rate. After the nondimensionalisation the dynamic system is:

$$\begin{cases} \dot{S} = -SF + \kappa S(1 - S) \\ \dot{I} = SF - \lambda I \\ \dot{F} = \frac{1}{\varepsilon}(I - F) \end{cases} \tag{4.4}$$

with

$$\lambda = \frac{\beta\psi}{\alpha\gamma S_0}, \frac{1}{\varepsilon} = \frac{\psi^2}{\alpha S_0 \gamma}, \kappa = \frac{\delta}{\alpha K}. \tag{4.5}$$

The equilibrium states of the dynamical system can be obtained, as before, equating to 0 the right hand side of (4.8). Two equilibrium solutions that exist for all parameter values are $A(0,0,0)$ and $B(1,0,0)$. Additional equilibrium point $C(\lambda, \kappa(1-\lambda), \kappa(1-\lambda))$ exists only if $\lambda < 1$. The Jacobian matrix related to the system (4.8) is

$$J = \begin{bmatrix} -F + \kappa - 2\kappa S & 0 & -S \\ F & -\lambda & S \\ 0 & \frac{1}{\varepsilon} & -\frac{1}{\varepsilon} \end{bmatrix} \tag{4.6}$$

A is always a saddle point while B is asymptotically stable (the three eigenvalues of the Jacobian matrix are negative) for $\lambda > 1$.

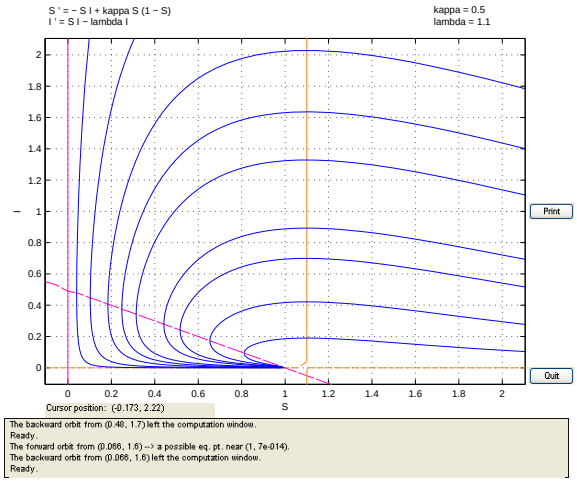


Figure 4.1: Phase portrait with $\lambda > 1$

When the point C exists it is asymptotically stable [1] while B becomes unstable (see figure (4.2)).

We can observe that the dynamic system (4.8) is equivalent to a SIR model with logistic growth when F is in a steady state. Choosing a small ε we find this behavior because F and I go to infinity in the same way. In the case $\lambda < 1$ we have an endemic disease where infected and susceptible

[1]The equation for the calculation of Jacobian eigenvalues is

$$\mu^3 + \mu^2(\frac{1}{\varepsilon} + \lambda + \kappa\lambda) + \mu(\frac{\kappa\lambda}{\varepsilon} + \kappa\lambda^2) + \frac{\kappa(1-\lambda)}{\varepsilon} = 0. \tag{4.7}$$

Using Cartesio's signs rule for cubics equations we can say that no positive solutions exist because there aren't sign's changes.

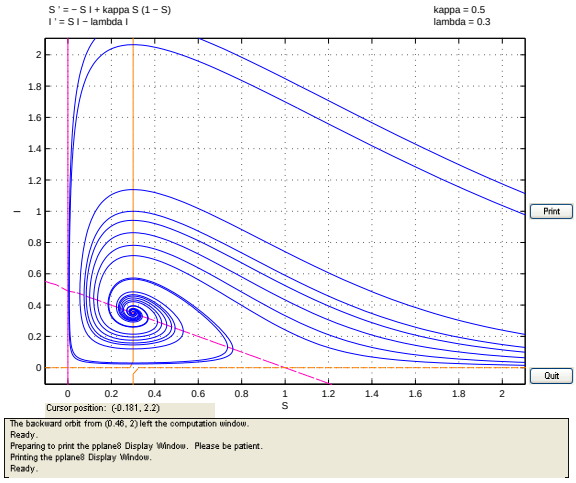


Figure 4.2: Phase portrait with $\lambda < 1$

rabbits live together. When $\lambda > 1$ the equilibrium point suggests that the situation will returns to the initial condition when time goes to infinity and this confirm the choice of a small value of λ.

4.2 Diffusion of fleas

As we have done before we introduce diffusion terms for fleas and rabbits

$$\begin{cases} \frac{\partial S}{\partial t} = -SF + \kappa S(1-S) + D_r \frac{\partial^2 S}{\partial t^2} & t > 0, x \in [0,1] \\ \frac{\partial I}{\partial t} = SF - \lambda I + D_r \frac{\partial^2 I}{\partial t^2} & t > 0, x \in [0,1] \\ \frac{\partial F}{\partial t} = \frac{1}{\varepsilon}(I - F) + D_f \frac{\partial^2 F}{\partial t^2} & t > 0, x \in [0,1] \\ S(x,0) = S_0(x) & x \in [0,1] \\ I(x,0) = I_0(x) & x \in [0,1] \\ F(x,0) = F_0(x) & x \in [0,1] \\ \frac{\partial S}{\partial x}|_{x=0,1} = 0 & t > 0 \\ \frac{\partial I}{\partial x}|_{x=0,1} = 0 & t > 0 \\ \frac{\partial F}{\partial x}|_{x=0,1} = 0 & t > 0 \end{cases} \tag{4.8}$$

where D_r represents the diffusion term for rabbits, susceptible and infected too, and D_f the diffusion term for infected fleas. We are assuming that fleas spread faster than rabbits. Results from a numerical simulation are shown in Figure (4.3).

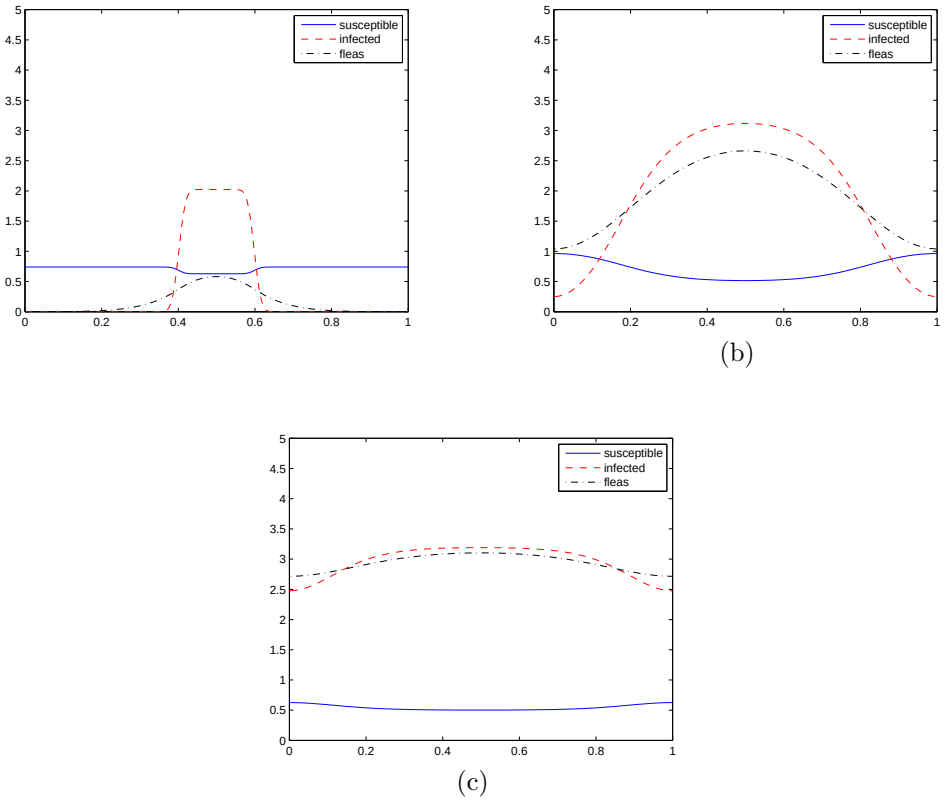


Figure 4.3: Spread of infection at different times

4.3 Time-varying rate of infection

We now assume, according with the viruses used to reduce rabbits, to have a seasonal disease. In effect this means a time-varying rate of infection. Our dynamic system is now:

$$\begin{cases} \dot{S} = -\alpha(t)SI \\ \dot{I} = -\alpha(t)SI - \beta I \end{cases} \tag{4.9}$$

where $\alpha(t)$ has to be a periodic function as:

$$Asin(\omega t) + \alpha_0 \tag{4.10}$$

with α_0 the medium value assumed by the function, ω the frequency [2] and A the amplitude of the function. It must be $\alpha_0 > A$ in order to have $\alpha(t)$ always positive. After nondimensionalisation our dynamic system becomes

$$\begin{cases} \dot{S} = -\alpha(t)\mu SI \\ \dot{I} = -\alpha(t)\mu SI - \eta I \\ \alpha(t) = 1 + \Gamma sin(t) \end{cases} \tag{4.11}$$

where

$$\mu = \frac{\alpha_0 S_0}{\omega}, \eta = \frac{\beta}{\omega}, \Gamma = \frac{A}{\alpha_0}. \tag{4.12}$$

First of all the average value of basic reproductive ratio R_0 is equal to $\frac{\mu}{\eta}$ just like it was in the SIR model while for each time t we can obtain $R_0 = \frac{\mu\alpha(t)}{\eta}$. We proceeded by numerical simulations and we can see in the next images how the seasonality of disease makes the populations trend periodic (see figure (4.4)).

To be more realistic we have considered a disease that has effect only in a short period of the year (few months) defining

$$\alpha(t) = \begin{cases} \sigma & for\ \ t \in [a, b] \\ 0 & otherwise \end{cases} \tag{4.13}$$

in which σ is a real positive number depending on the considered virus, while $[a, b]$ is the period of disease activity (see figure (4.5)).

Of course we need a disease death time for infected rabbits shorter than the period T but is mathematically interesting to notice that when R_0 is big enough the period of population is larger then the disease one (see figure (4.6)).

[2] $\omega = \frac{1}{T}$, with T is assumed to be one year.

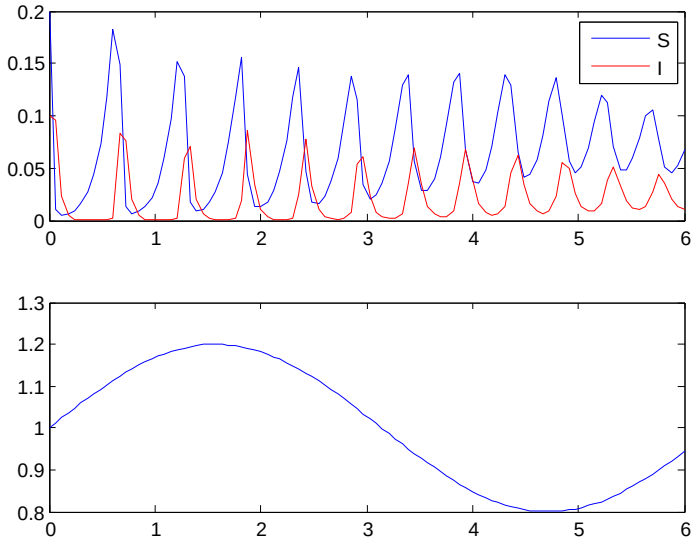


Figure 4.4: Variation in time with $\alpha(t) = 1 + \Gamma sin(t)$

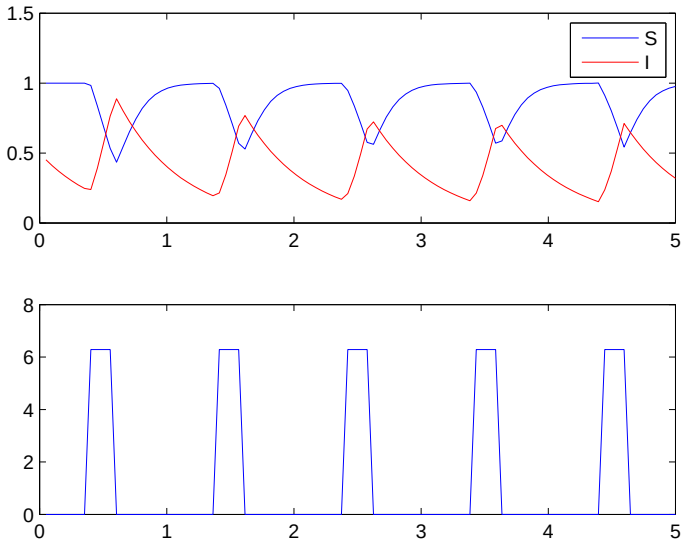


Figure 4.5: Variation in time with $\alpha(t)$ as in (4.13)

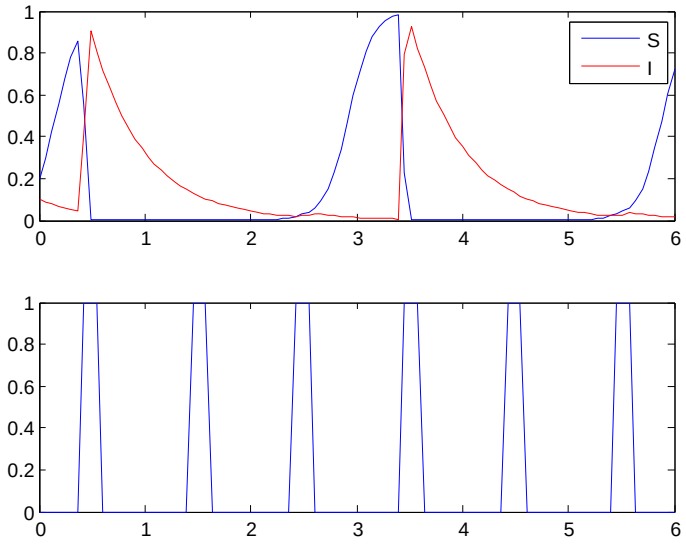


Figure 4.6: Variation in time with a big R_0

Chapter 5

Conclusions and future work

In our project we started off using the famous SIR model. We found that the key parameter for virulence is R_0. However, this model is only applicable when the timescale of the spread of the disease is much quicker than the birth of rabbits.

We introduced a growth term and found that logistic growth is more realistic than exponential one.

Modelling a rabbit population as perfectly mixed is unsatisfactory, as in reality rabbits live in communities. We introduced a spatial dependence and came up with a PDE model. Numerical simulations of the model show a very nice spreading wavefront.

We then decided to introduce more realistic transmission model. We used a third population of infected fleas to spread the disease, and incorporated a faster diffusion term for them.

Finally, we introduce time-dependent parameters, and gave numerical simulations that suggest that seasonal diseases give way to periodic variations in the population for certain R_0 values.

For future work, it would be interesting to move into two spatial dimensions. The spatial domain could include areas where it is more difficult for the disease to spread.

We would also like to include genetic immunities in our model, as this is a key factor that hampers the progress of diseases over long-time periods.