The effect of toxicant exposure on infected Flying Foxes

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Contents

Introduction

Heavy metal pollution due to urbanization has a serious impact on humans and animals. In addition, the exposure to those toxicants can influence the effects of virus infections on individual hosts and alter animal movement (Kessler et al. 2018). To understand this, a hypothetical flying fox species is infected with a virus and a mathematical model explores the infection dynamics in toxicant-contaminated landscapes.

A lot of data from flying foxes species are available, because flying foxes are often used as bioindicators for heavy metal pollution. To use flying foxes as an bioindicator the demography of flying foxes has been closely studied. They have a broad range of different habitats (Pulscher et al. 2020) and feed on fruiting plants in human environments, where they face exposure to heavy metals. Flying fox species are also used in this model is because they are reservoir hosts of a virus named henipavirus. This virus can be transmitted to humans and other animals, which also known as zoonotic spillover (Croser and Marsh 2013).

The exposure to toxicants can increase infection susceptibility, this has an effect on the transmission of viruses from one host to another. Toxicants can also influence animal movement. Toxicants can directly influence physical activity, or indirectly affect memory or navigation (Breed et al. 2006). In human-altered habitats food availability is more stable (and toxicant exposure is higher), this means that it is unlikely that groups of flying foxes move back to pristine environments.

The model gives an insight in how the flying fox population size, infection prevalence and zoonotic spillover depend on the contamination rate of the landscape and the effect of toxicant exposure on infection, movement and survival. The model was obtained from (Sánchez, Altizer, and Hall 2020). This research is divided into two parts. First the research by Sánchez et al. is being reproduced in which the effect of different transmission rates of the virus in the toxicant-contaminated habitat is being modulated. Then the effect of the cost of toxicants to dispersal of the animal is being modulated.

Methods

The model describes the infection dynamics in toxicant-contaminated landscapes and distinguishes the population size of flying foxes in different habitats. A landscape is divided into a contaminated (T) and a pristine (P) area, and the flying foxes are classified by their infection status; infected (I) or susceptible (S). In all of these cases the population size is influenced by the demography, infection and movement of the animal. This is illustrated in figure 1. The model is written in R (version 4.1.3) and the DeSolve package (version 1.32) is imported in order to model the differential equations.

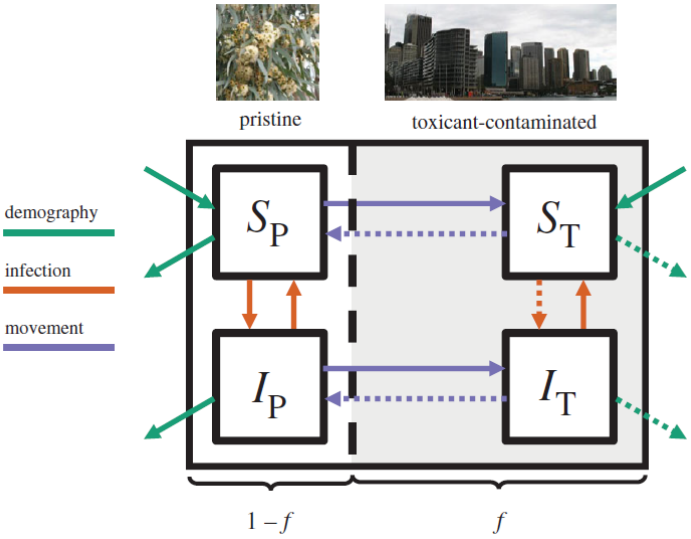

Figure 1: *biological model schematic. Each box represents a different population, where S stands for susceptible to infection, I for infected, P for pristine habitat and T for toxic-contaminated habitat. The arrows represent factors that influence the population size. Green represents demography factors, orange infection factors and purple describe movement factors.* f *is the fraction of toxic-contaminated habitat and therefore* f *- 1 is the fraction of pristine habitat*

$$\frac{dS_P}{dt} = (b_0 - \frac{b_1(S_P+I_P)}{1-f})(S_P+I_P) - mS_P - \beta_P S_P I_P + \gamma I_P - \sigma f S_P + \sigma(1-c_\sigma)(1-f)S_T$$

$$\frac{dI_P}{dt} = \beta_P S_P I_P - \gamma I_P - (m+\mu)I_P - \sigma f I_P + \sigma(1-c_\sigma)(1-f)I_T$$

$$\frac{dS_T}{dt} = (b_0 - \frac{b_1(S_T+I_T)}{f})(S_T+I_T) - \frac{m}{1-c_m}S_T - \beta_T S_T I_T + \gamma I_T + \sigma f S_P - \sigma(1-c_\sigma)(1-f)S_T$$

$$\frac{dI_T}{dt} = \beta_T S_T I_T - \gamma I_T - \frac{m+\mu}{1-\alpha c_m} I_T + \sigma f I_P - \sigma(1-c_\sigma)(1-f)I_T$$

Figure 2: *differential equations used by the model. Each differential equation is highlighted by different color; green represents demography paramters, orange represents infection parameters and purple represents movement paramters. Each equation calculates the population size of a different group. The groups are: susceptible animals in a pristine habitat (*S_P*), infected animals in a pristine habitat (*I_P*,) susceptible animals in a toxic contaminated habit (*S_T*) and infected aminals in a toxid contamindated area (*I_T*). Image obtained from Sánchez et al. (2020)*

In figure 2 four differential equations are shown. Firstly, $\frac{dS_P}{dt}$ calculates the population size of the susceptible flying foxes which are located in a pristine area. Secondly, $\frac{dI_P}{dt}$ calculates the amount of infected flying foxes in a pristine area. Next, $\frac{dS_T}{dt}$ calculates the amount of flying foxes which are susceptible and in a toxic area. Finally, $\frac{dI_T}{dt}$ calculates the population size of the flying foxes which are infected and located in a toxic area.

Additionally, parts of the differential equations in figure 1 are colored. Each color represents a different process which may affect the population size with that infection status and habitat. Green is used to indicate demography, orange to indicate infection and purple to indicate movement.

Each equation is broken down into its parameters and are listed in figure 3.

process	parameter	definition	units	value
demography	m	natural mortality rate	year^{-1}	0.1
	b_0	maximum *per capita* birth rate	host^{-1} year^{-1}	0.4
	b_1	density-dependent per capita birth rate	year^{-1}	$(b_0 - m)/50000 = 6e-6$
	c_m	cost of toxicants to survival		0.2
infection	β_P	transmission rate in pristine habitat	host^{-1} year^{-1}	0.006
	β_T	transmission rate in toxicant-contaminated habitat	host^{-1} year^{-1}	0.0015, 0.006, 0.0105
	γ	recovery rate	year^{-1}	36.5
	μ	disease-induced mortality rate	year^{-1}	0.25
	α	synergistic effect of infection and toxicants on survival		2
movement	f	fraction of the landscape that is toxicant-contaminated		0.01–0.99
	σ	*per capita* dispersal rate	year^{-1}	–log 0.1
	c_σ	cost of toxicants to dispersal		0.2, 0.8

Figure 3: *all of the paramters, definitoins, units and values used in the differential equations. The paramters are divided by demography, infection and movement. Image is obtained from Sánchez et al. (2020)*

As shown in figure 3; β_T, f and c_σ have different parameter values. These are used to modulate six different scenarios in which f is being changed, ranging from 0.01 to 0.99. This is the fraction of a landscape that is contaminated by toxicants. For each f, the model is initiated with a population of 50 000 , of which 100 where infected, and has run for a timespan of 50 years, the last values are then stored. This timespan is chosen because all differential equations are then at equilibrium. All of the parameter values are obtained from Sánchez et al. (2020), which is based on flying fox data (Sánchez, Altizer, and Hall 2020).

The six scenarios can be divided into two parts. These parts are based upon the two different values of c_σ (see figure 3). c_σ is the cost of toxicants to dispersal of the animal. That is, the ability of an animal of switching from a toxic contaminated habitat to a pristine habitat. When c_σ is low the animal can easily move from the toxic to the pristine habitat. On the contrary, if c_σ is high it is hard for the animal to move from the toxic to the pristine habitat.
In both parts the effect of different transmission rates in a toxic contaminated habitat with respect to the transmission rate in a pristine habitat are simulated. This is done by keeping a constant β_P value and the changing the β_T values, resulting in three different scenarios per part:

- $\beta_T < \beta_P$: transmission in toxicant-contaminated habitat is lower than in pristine habitat.
- $\beta_T = \beta_P$: transmission is equal in both habitats.
- $\beta_T > \beta_P$: transmission in toxicant-contaminated habitat is enhanced.

Transmission in this case is the transferring of a pathogen or virus from host to host.

For all six scenarios the population size, infection prevalence and spillover risk are being calculated using the following equations:

$$\text{Population size} = S_P + I_P + S_T + I_T$$

$$\text{Infection prevelance} = \frac{I_T}{\text{population size}}$$

$$\text{Spillover risk} = \frac{I_T}{f}$$

Results

As mentioned earlier, six scenarios are being modulated. These scenarios are divided into two parts, in the first part a c_σ value of 0.2 is used and in the second part a value of 0.8. In both parts the different transmission rates are simulated. For each simulation the the population size, infection prevalence and spillover risk are shown in a plot.
The first part of the results section compares all the different transmission rates to one and another. The second part compares the c_σ values.

Transmission rates

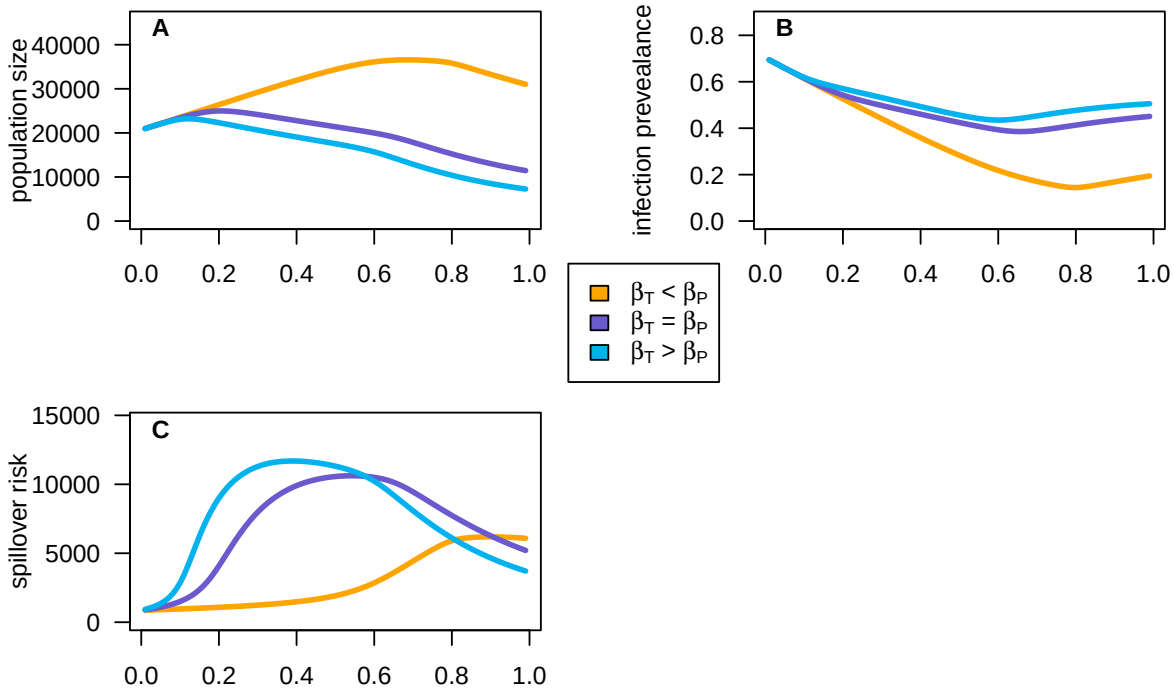

Figure 4: *modulated population size (A), infection prevalence (B) and spillover risk (C) with respect to the fraction of toxic-contaminated habitat. In all plots the line colors orange, purple and blue indicate* $beta_T < beta_P$, $beta_T = beta_P$ *and* $beta_T < beta_P$, *respectively. A* c_{sigma} *value of 0.2 was used.*

In figure 4 the population size, infection prevalence and spillover risk are shown with varying β_t values.

In figure 4A $\beta_T = \beta_P$ and $\beta_T > \beta_P$ show a similar trend. When the fraction of contaminant habitat (f) increases, the population size first increases a little and then declines slowly. However, the population size of $\beta_T = \beta_P$ has a longer period in which it increases, resulting in a higher population size at different f's. Interestingly, when looking at $\beta_T < \beta_P$ a significant longer increase can be seen, which results in a higher maximum of population size.

In figure 4B $\beta_T = \beta_P$and $\beta_T > \beta_P$ are again very similar. A slow decline of infection prevalence can be seen until an f of approximately 0.65 is reached, after which it increases. Furthermore, $\beta_T < \beta_P$ shows a steeper and longer decrease as f increases. It then increases in the same way as $\beta_T = \beta_P$ and $\beta_T > \beta_P$.

In figure 4C $\beta_T = \beta_P$ and $\beta_T > \beta_P$ repeatedly show similar trends. They both have a sharp S-shaped increase with respect to an increasing value of f. Hereafter it declines. The same S-shaped increase can be seen when look-

ing at $\beta_T < \beta_P$. However, this increases is less steep and the optimum that is reached is lower and at a higher f. Furthermore, after the optimum is reached it does not decline at all.

Altogether it seems like a similar trend in all graphs of figure 4 can be seen. $\beta_T = \beta_P$ and $\beta_T > \beta_P$ show very similar curves, where $\beta_T = \beta_P$ is delayed or more spread out. When looking at $\beta_T < \beta_P$ the same phenomenon can be seen, but more extreme. The increases and decreases are less steep and take longer with respect to an increasing f.

Comparing c_σ values

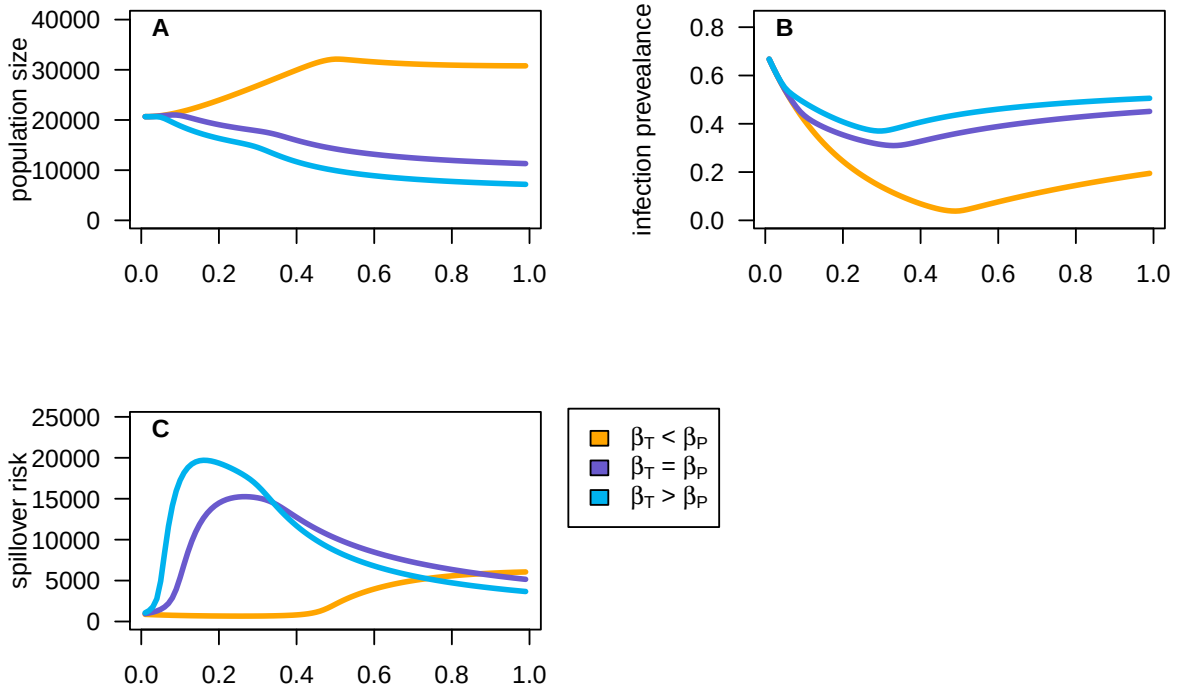

Figure 5: *modulated population size (A), infection prevalence (B) and spillover risk (C) with respect to the fraction of toxic-contaminated habitat. In all plots the line colors orange, purple and blue indicate* $beta_T < beta_P$, $beta_T = beta_P$ *and* $beta_T < beta_P$, *respectively. A* c_{sigma} *value of 0.8 was used.*

In figure 5 the population size, infection prevalence and spillover risk are shown with varying β_T values. These graphs will be mostly compared to the graphs in figure 4. In figure 5 a c_σ value of 0.8 was used, where as figure 4 uses a c_σ of 0.2.

Figure 5A and figure 4A the lines of $\beta_T = \beta_P$ and $\beta_T > \beta_P$ are in both graphs very close together and show a similar trend. These lines show in figure 4A a small initial increase, which can not be seen in figure 5A. $\beta_T < \beta_P$ does have an initial increase in figure 5A and shows more of a kink at the inflection point, compared to a curve in figure 4A. This inflection point is also at a lower f, that is, it is shifted to the right. $\beta_T < \beta_P$ also has a lower maximum at this point of inflection.

The graph illustrated in figure 5B and figure 4B show a similar pattern in a way that $\beta_T = \beta_P$, $\beta_T > \beta_P$ and $\beta_T < \beta_P$ and first decline and then increase. In addition, the values of where the lines start and end are the same in figure 5B and 4B. However, the point of inflection happens at a lower f and the minimum of the line is lower. This results in a more stiff decrease and increase.

When looking at figure 5C compared to figure 4C, the following can be noticed. The lines in figure 5C are similar but more compressed and shifted to the left. In addition, the spillover risk values of the peaks are higher. This results in a more steep increase. Interestingly, when looking at $\beta_T < \beta_P$ in figure 5C a flat-line can be seen until an f of 0.4 is reached.

Collectively, all graphs in figure 5 are unlike in way that the lines are shifted to the right when compared to figure 4. On the contrary, similar values of the start and stop values can be seen in all graphs (A, B and C) and lines.

Discussion

With the model, two different figures were created. The first one had a lower cost of toxicants to dispersal, in which three different transmission rates were shown. In the second figure these different transmission rates with a higher cost of toxicants to dispersal are shown. First a possible explanation for the differences in transmission rates is given. Hereafter the effect of the cost of dispersal rate is reviewed.

When the virus is less transmissible in toxicant-contaminated habitat than in pristine habitat ($\beta_T < \beta_P$), very low fractions of the toxic-contaminated habitat have a positive effect the population size. This may be due to the fact that the population density in this small toxic-contaminated area is little, resulting in less virus transmission between the animals. This means that less flying foxes are infected with the virus and therefore less flying foxes die from infection (higher population size). The infection prevalence decreases with f because there are less infected flying foxes in a toxic-contaminated habitat.

But when a virus is equally ($\beta_T = \beta_P$) or more ($\beta_T > \beta_P$) transmissible in toxicant-contaminated habitat, there is also a small increase in population size and a decrease in infection prevalence. However, in both scenarios the population size decreases sooner. This is because toxicants might amplify the transmission of the virus, causing the virus to go from host to host sooner and therefore infect more flying foxes with the virus. This amplification may be due to a weakened immune system caused by these toxicants, which might make the animal more susceptible to infection. When looking at the infection prevalence plot this might seem contradictory, because it decreases. However, the infection prevalence is the ratio between infected animals in toxic-contaminated habitat and the population size, More infected animals results in a lower population size, causing less infected animals in toxic-contaminated habitat and a lower population size, therefore a decline in infection prevalence.

It appears that in all three scenarios when there is a very high fraction of contaminated habitat, the population size (further) decreases. When looking at the equations there is a direct relation between f and demography and movement. In the equations about the toxicant-contaminated habitat, a higher f leads to less movement from the contaminated habitat to pristine habitat (- σ * (1 - c_σ) * (1 - f)I_T). This means that toxicant-contaminated habitats contain more flying foxes than pristine habitats. Assumed that a higher toxicant-contaminated habitat is also more urbanized, leading to more food sources for flying foxes, the exposure of toxicants to the flying foxes are higher and can cause more fatal cases. This leads to a decline in population size. Then the ratio of population size and amount of infected flying foxes in the toxicant habitat increase.
It seems that, when a virus is equally or more transmissible in toxicant-contaminated habitat that the spill-over risk occur at lower proportions of toxicant-contaminated habitat. Meaning that less flying foxes are needed to spill over diseases to humans. However when the transmission rate is less, the virus is only being spilled over at a high f. This could be because a higher fraction of contaminated habitat could mean more urbanization and more human-wildlife contact, and therefore an increase in spill-over risk.

When comparing these results to the results of Sánchez et al. (2020) the same results are found. The graphs of the population size, infection prevalence and spillover risk are identical ($c_\sigma = 0.2$). However, these three graphs are squeezed together in the paper of Sánchez et al (2020). This causes that the increases and decreases looks more steep and extreme, when this is not the case. For example, in figure 4B of this research the increase in infection prevalence at the end is moderate whilst this increase in figure 2b of their research is steeper. This can be a bit misleading.

In addition to the research of Sánchez et al. (2020), the cost of toxicants to dispersal was also changed (c_σ). When the cost of toxicants to dispersal is increased, it is harder for an animal to move from a toxic-contaminated habitat to a pristine habitat. This means that the chance of animal staying in the toxic-contaminated habitat is greater. This results in that the survival chance of an animal exposed to toxicants decreases, because it is difficult for the animal to move out of this harmful habitat. This effect can be seen in the results (figure 5AB), where the maximum population size is lower and this maximum is reached at a lower fraction of toxic-contaminated habitat. The rest of the explanations for the plots as described for c_σ = 0.2 are similar to the the movement of the lines in figure 5. A higher cost of toxicants to dispersal simply means that the overall population size and prevalence is reduced, and that infections could be transferred to humans at even lower fractions of toxicant-contaminated habitat.

In this research only data from the flying fox is used. However, this model can also be used for different animals and viruses. To do this, data must be gathered of a different animal and/or virus. Keep in mind when choosing a virus that

is not able to go from animal to human that the spillover graph is abundant. Nonetheless, the population size and infection prevalence can be analysed.

Furthermore, some parameters values can be changed to simulate other scenarios. For example, the recovery rate can be lowered and the disease-induced mortality rate increased to simulate a more severe virus.

Lastly, the model can be expanded by adding more factors. Sànches at el. recommended that factor such as infection-dependent movement decisions, age-related effects or gradual increase or decrease to toxicant exposure when moving in or out a toxicant-contaminated habitats might improve the model (Sánchez, Altizer, and Hall 2020).

Conlusion

High levels of urbanization causes a decrease in population size. The effects of the urbanization negatively influences the chance of survival of the animal when it is exposed to a virus. The magnitude of this decrease is highly affected by the transmission rate in a toxic-contaminated habitat when compared to the transmission rate in a pristine habitat. Lower levels of urbanization sometimes positively influence the chance of survival.

When the cost of toxicants to the dispersal is higher, the population size decreases because the animal is more likely to stay in a polluted area for a longer period of time. This also results in that there is a high chance of zoonotic spillover even at low levels of urbanization and therefore it becomes more dangerous for humans as well. Collectively, the cost of toxicants to the dispersal is an important parameter.

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Apendix

ODE Function

```
## ODE FUNCTION

# Load deSolve package
library(deSolve)

# Function with the models differential equations
wildlife_urbanization_model <- function(t, state, parameters) {
  with(as.list(c(state, parameters)),{

    dSp <- (b0 - (b1 * (Sp + Ip)) /(1-f) ) * (Sp + Ip) - m * Sp -  # demography
      beta_p * Sp * Ip + gamma * Ip -  # infection
      sigma * f * Sp + sigma * (1 - c_sigma) * (1 - f) * St  # movement


    dIp <-  beta_p * Sp * Ip - gamma * Ip -  # infection
      (m + mu) * Ip -  # demography
      sigma * f * Ip + sigma * (1 - c_sigma) * (1 - f) * It  # movement


    dSt <- (b0 - (b1 * (St + It) / f)) * (St + It) - (m / (1 - c_m)) * St -  # demography
      beta_t * St * It + gamma * It +  # infection
      sigma * f * Sp - sigma * (1 - c_sigma) * (1 - f) * St  # movement


    dIt <- beta_t * St * It - gamma * It -  # infection
      ((m + mu) / (1 - alpha * c_m)) * It +  # demography
      sigma * f * Ip - sigma * (1 - c_sigma) * (1 - f) * It  # movement


    list(c(dSp, dIp, dSt, dIt))
  })
}
```

Running the ODE function

```
## MODEL FUNCTION

run_model <- function(population = 50000, infected = 100, m = 0.1, b0 = 0.4,
                      c_m = 0.2, beta_p = 0.006, beta_t = 0.006, gamma = 36.5,
                      mu = 0.25, alpha = 2, sigma = -log(0.1), c_sigma = 0.2){

  # Create a data frame to store needed data
  df <- data.frame(matrix(nrow = 0, ncol = 8))
  colnames(df) <- c('f', 'Sp', 'Ip', 'St', 'It',
                    'pop_size', 'inf_prevalence', 'spillover')

  for (f in seq(0.01, 0.99, 0.01)){
    # Calculate b1 based on the function parameters
    b1 <- (b0 - m) / population

    # Calculate infection status per habitat
    Sp <- (population - infected) * (1 - f)
    Ip <- infected * (1 - f)
    St <- (population - infected) * f
    It <- infected * f

    # Creating a vector with the parameter values
    parameters <- c(m = m, b0 = b0, b1 = b1, c_m = c_m, beta_p = beta_p,
                    beta_t = beta_t, gamma = gamma, mu = mu, alpha = alpha,
                    f = f, sigma = sigma, c_sigma = c_sigma)

    # Creating a vector with the initial values
    state<- c(Sp = Sp, Ip = Ip, St = St, It = It)

    # Time frame of 50 years
    times <- seq(0, 50, 0.05)

    out <- tail(ode(y = state, times = times, func = wildlife_urbanization_model,
                    parms = parameters), 1)

    df[nrow(df) + 1,] <- c(f, out[2:5], sum(out[2:5]),
                           (out[3] + out[5])/sum(out[2:5]), out[5]/f)
    }

  return(df)
}
```

Obtaining data function

```
## OBTAIN DATA FUNCTION
obtain_data <- function(c_sigma){
  for (beta_t in c(0.0015, 0.006, 0.0105)){
    df <- run_model(beta_t = beta_t, c_sigma = c_sigma)
    if (beta_t == 0.0015){
      population_all <- data.frame('0.0015' = df$pop_size)
      infection_all <- data.frame('0.0015' = df$inf_prevalence)
      spillover_all <- data.frame('0.0015' = df$spillover)
    }
    if (beta_t == 0.006){
      population_all <- data.frame(population_all, '0.006' = df$pop_size)
      infection_all <- data.frame(infection_all, '0.006' = df$inf_prevalence)
      spillover_all <- data.frame(spillover_all, '0.006' = df$spillover)
    }
    if (beta_t == 0.0105){
      population_all <- data.frame(population_all, '0.0105' = df$pop_size)
      infection_all <- data.frame(infection_all, '0.0105' = df$inf_prevalence)
      spillover_all <- data.frame(spillover_all, '0.0105' = df$spillover)
    }
  }
  return(list(population_all, infection_all, spillover_all))
}
```

Plotting scenarios function

```
## PLOT FUNCTION
plot_scenario <- function(datasets, y_labels, line_cols, legends){
  plot_labels = c("A", "B", "C")
  f_seq = seq(0.01, 0.99, 0.01)
  for (item in 1:length(datasets)){
    data <- datasets[[item]]
    for (scenario in 1:3){
      if (scenario == 1){
        plot(data[,scenario] ~ f_seq, col = line_cols[scenario],
             type = 'l', lwd = 3, ylim = c(0, max(data) * 1.25),
             xlab = 'f', las = 1, ylab = "")
        title(ylab = y_labels[item], line = 3.3)
        text(x=0.05, y=max(data) * 1.2, labels = plot_labels[item], font = 2)
      }
      else{
        lines(data[,scenario] ~ f_seq, col = line_cols[scenario], lwd = 3)
      }
    }
  }
}
```

Running β_t scenarios

```
beta_t_data <- obtain_data(c_sigma = 0.2)
beta_t_labels <- c('population size', 'infection prevealance', 'spillover risk')
beta_t_cols <- c('orange', 'slateblue', 'deepskyblue2')
beta_t_legends <- c(expression(paste(beta[T], " < ", beta[P])),
                    expression(paste(beta[T], " = ", beta[P])),
                    expression(paste(beta[T], " > ", beta[P])))

par(mfrow=c(2,2), mar=c(3.1,5.1,3.1,2.1))
plot_scenario(datasets = beta_t_data, y_labels = beta_t_labels,
              line_cols = beta_t_cols, legends = beta_t_legends)
legend(1.1, 26000, legend = beta_t_legends, fill = beta_t_cols, xpd=NA)
```

Running c_σ scenarios

```
beta_t_data <- obtain_data(c_sigma = 0.8)
beta_t_labels <- c('population size', 'infection prevealance', 'spillover risk')
beta_t_cols <- c('orange', 'slateblue', 'deepskyblue2')
beta_t_legends <- c(expression(paste(beta[T], " < ", beta[P])),
                    expression(paste(beta[T], " = ", beta[P])),
                    expression(paste(beta[T], " > ", beta[P])))


par(mfrow=c(2,2), mar=c(3.1,5.1,3.1,2.1))
plot_scenario(datasets = beta_t_data, y_labels = beta_t_labels,
              line_cols = beta_t_cols, legends = beta_t_legends)
legend(1.1, 26000, legend = beta_t_legends, fill = beta_t_cols, xpd=NA)
```