

# The University of Calgary Biostatistics Centre (UCBC)

# **Bayesian Modelling of Epidemics**

Part I: Intro to Deterministic Epidemic Models
Part II: Stochastic Population-level Models
Part III \ IV: Individual-level Models

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ISBA, Montréal, June 2022





# Bayesian Modelling of Epidemics Part I: Intro to Deterministic Epidemic Models

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### Outline

1. Introduction to Transmission modelling

2. Classic Model: SIR Compartmental Model

3. Reproduction Number (R)

4. ODEs: Extensions & Alternatives



#### Outline

1. Introduction to Transmission modelling

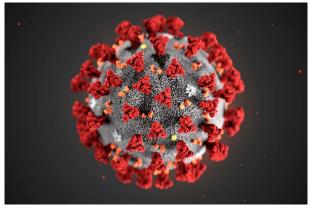
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# Example: COVID-19



CDC/SCIENCE PHOTO LIBRARY



# Infectious (Communicable) Diseases

#### Examples:

measles, AIDS, SARS, ebola, monkeypox (human) influenza (avian, swine, equine, human, etc.) foot-and-mouth disease (cloven-hooved animals) PRRS, PED, PHF (swine) citrus canker, tomato rust (crops) COVID-19

#### We may also be interested in modelling:

invasive animal species
(e.g. Asian giant hornet, emerald ash borer, mountain pine beetle)
invasive plant species
(e.g. giant hogweed, Japanese knotweed)
fire spread
internet / financial market contagion



# Example: Asian giant hornet



https://www.cnn.com/2020/10/23/us/asian-giant-murder-hornet-nest-scn-trnd/index.html



# Topic of Today's Workshop

#### Mechanistic models of disease transmission

These are models built to mimic underlying biological/ecological mechanisms associated with how transmission occurs

They are not designed with the goal of data analysis in mind (although we will consider how to inform them via data)

Parameters are usually biologically / ecologically interpretable.

Parameters can be 'estimated' in a piecemeal fashion

Typically used to simulate and forecast epidemics and the effect of control strategies upon those epidemics



# Topic of Today's Wokshop

Many other data-driven techniques exist for analyzing infectious disease data

We might collectively termed these disease surveillance methods/models

#### For example:

CUSUM / process control type methods spatial / spatio-temporal disease mapping (longitudinal) regression models

We will not be discussing disease surveillance methods today



# Types of Transmission Model

#### Examples of model types:

#### 1) Homogenous models

any infectious individual has equal chances of infecting any susceptible individual

#### 2) Population Structured models

homogeneous mixing within strata (e.g. age-category) homogeneous mixing between strata but rates within and between different strata can vary



# Types of Transmission Model

#### Examples of model types:

#### 3) Individual-level models

chances of any particular infectious individual infecting any particular susceptible individual depends upon covariates about the individuals concerned

#### 4) Agent-based models

"high dimensional, high complexity ILMs"



# Important characteristic: Non-independence of infection events

#### Non-independence of infection events

As more individuals are infectious, the risk of infection for any remaining needs to go up (and vice versa)

This invalidates assumptions of most off-the-shelf statistical models, so we require something a bit more specialized



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W. Kermack & A. McKendrick (1927). A contribution to the mathematical theory of epidemics. Proc. R. Soc. London A 115, 700-721.

M. Keeling & P. Rohani (2007). Modelling Infectious Diseases In Humans and Animals. Princeton University Press

Here, we assume a **closed population** of *n* individuals

Time is considered **continuous** (not discrete)

Units of time whatever is most suitable to our disease (e.g., hours, days, weeks, months, years, etc.)

Individuals are assumed to be homogeneous with respect to the disease

Individuals are assumed to mix homogeneously

Individuals are assumed to become infectious as soon as they are infected with the disease (i.e., no **latent period**)



We assume that at a given time point t, each individual can be in one, and only one, of three states (or compartments):

S	Susceptible	doesn't have disease;
		can contract it
I	Infectious	has contracted the disease;
		can pass it on
R	Removed	been removed from the susceptible population
		e.g. died from the diease;
		e.g. isolated from the susceptible population;
		e.g. recovered and developed immunity

#### Notation:

 $S_t$  is the proportion of susceptible individuals in the population at time t  $I_t$  is the proportion of infectious individuals in the population at time t  $R_t$  is the proportion of removed individuals in the population at time t

Note:  $S_t + I_t + R_t = 1$  for all t



These assumptions lead us to a set of three ordinary differential equations for  $S_t$ ,  $I_t$ , and  $R_t$ :

$$\frac{dS_t}{dt} = -\beta S_t I_t \tag{1}$$

$$\frac{dl_t}{dt} = \beta S_t l_t - \gamma l_t \tag{2}$$

$$\frac{dH_t}{dt} = \gamma I_t.$$
(3)

 $eta \geq 0$  is the **transmission rate** (rate at which individuals come together in a way disease can be transmitted)

 $\gamma \geq$  0 is the **removal rate** (e.g., rate of recovery with immunity, death, etc.)

Note:  $\frac{1}{\gamma}$  defines the **infectious period** (the length of time individuals spend in the infectious state)



A flow diagram for the SIR model (at the individual level):

$$oxed{\mathsf{S}} \xrightarrow{\beta I_t} oxed{\mathsf{I}} \xrightarrow{\gamma} oxed{\mathsf{R}}$$



```
## Install and access deSolve package
install.packages ("deSolve")
library (deSolve)
## Create an SIR function
SIR = function(time, state, parameters) {
  with (as.list(c(state, parameters)), {
    dS = (-beta * S * I)
    dI = (beta * S * I) - (gamma * I)
    dR = qamma * I
    return(list(c(dS, dI, dR)))
```

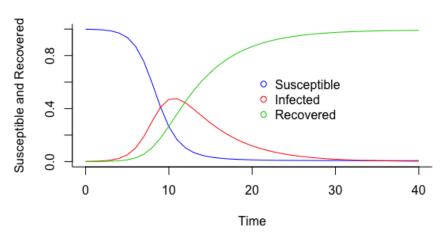


```
init = c(S = 0.999, I = 0.001, R = 0.0)
parameters = c(beta = 1, gamma = 0.2)
times = seq(0, 40, bv = 1)
## Solve using ode (essentially produce output)
out = ode(y = init, times = times, func = SIR, parms = parameters)
## plot data
out = as.data.frame(out)
out$time = NULL #remove as I don't want this in plot
matplot(x = times, y = out, type = "l",
         xlab = "Time", vlab = "Susceptible and Recovered",
         main = "SIR Model", lwd = 1, lty = 1, bty = "l", col = c(4,2,3))
legend(20, 0.7, c("Susceptible", "Infected", "Recovered"),
         pch = 1, col = c(4,2,3), btv = "n")
```

### Initialization









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The **basic reproduction number** or  $R_0$  is:

the (expected) number of infections that result directly from a single infection in an otherwise wholly susceptible population.

Note: deterministic in a deterministic model (e.g. our SIR-ODE); an average in a stochastic model

In our SIR-ODE model it is given by

$$R_0 = rac{eta}{\gamma}$$



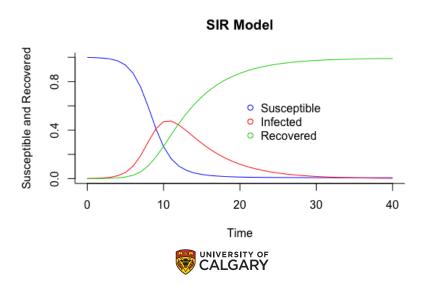
 $R_0$  is mathematically and epidemiologically interesting due to the behaviour of epidemics in relation to the threshold  $R_0=1$ 

Specifically, under a deterministic ODE model:

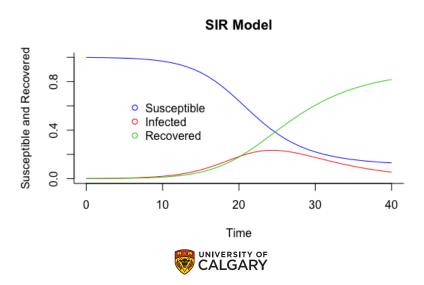
if  $R_0 > 1$  an epidemic will take off if  $R_0 < 1$  an epidemic will die out



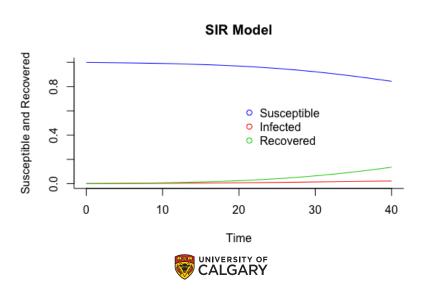
In our model  $\beta = 1$  and  $\gamma = 0.2$ , so  $R_0 = 5$ :



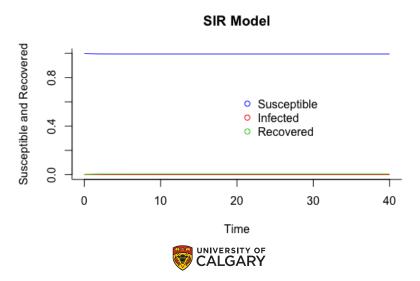
If we change our parameters to  $\beta = 0.5$  and  $\gamma = 0.2$ , so  $R_0 = 2.5$ :



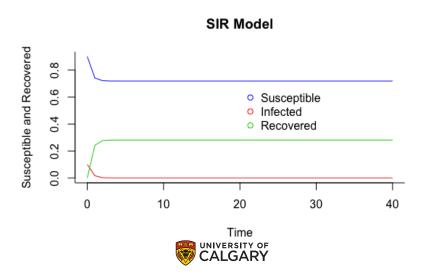
If we change our parameters to  $\beta = 0.5$  and  $\gamma = 0.4$ , so  $R_0 = 1.25$ :



If we change our parameters to  $\beta=2$  and  $\gamma=2.5$ , so  $R_0=0.8$ :



Now, keep the parameters as  $\beta=5$  and  $\gamma=4$ , so  $R_0=0.8$  but also infect 10% of the population at time 0, rather than 0.1%:



# Effective Reproduction Number $(R_t)$

Conceptually  $R_0$  is concerned with what happens at the intialization of the epidemic in a population that is behaving in its "normal" state

Of course, as the number of susceptibles goes down the number of individuals which can be **directly infected** via each new infection goes down.

This is captured by the **effective reproduction number** or  $R_t$ :

the expected number of infections to result directly from a new single infection in the population at time t.

It is given by:

$$R_t = \frac{\beta}{\gamma} S_t$$
$$= R_0 S_t$$

And **changes in population behaviour** can effect the number of direct infections occurring from each new infection via changes in both  $\beta$  and  $\gamma$ . (e.g., social distancing, vaccination, quarantine, mask wearing, etc)



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# 1. Frequency vs. Density Dependence

Our model here is known as a **frequency dependent** or **mass action** model. This results from the fact that  $S_t$ ,  $I_t$  and  $R_t$  are **proportions** of the population So transmission dynamics don't depend on population size

An alternative approach is to use a **density dependent** model In such a model,  $S_t$ ,  $I_t$  and  $R_t$  are the **numbers** of the population within each respective state Transmission dynamics do depend on population size,

or assuming a fixed area in which the population resides, population density



#### 2. Other Possible Disease Timelines

#### A number of **compartmental frameworks** are possible:

SI

SIR

SEIR

SIS

SIRS

etc.

Which we choose will depend on the disease we are studying (and maybe the quality of our data...)



# Example: SEIR Compartmental Model

These assumptions lead us to a set of four ordinary differential equations for  $S_t$ ,  $E_t$ ,  $I_t$ , and  $R_t$ :

$$\frac{dS_t}{dt} = -\beta S_t I_t 
\frac{dE_t}{dt} = \beta S_t I_t - \sigma E_t 
\frac{dI_t}{dt} = \sigma E_t - \gamma I_t 
\frac{dR_t}{dt} = \gamma I_t.$$

$$eta \geq$$
 0 is the transmission rate  $\sigma \geq$  0 is the "infectious rate"  $\gamma \geq$  0 is the removal rate

$$\frac{1}{\sigma}$$
 defines the **latent period**  $\frac{1}{\gamma}$  defines the **infectious period**



#### 3. Discrete versus continuous time

We have a choice between modelling our transmission process in either discrete or continuous time.

In **discrete time**, model generates counts / list of individuals infected each discrete time unit (event times are **INTEGERS**)

e.g., days, weeks, etc.

In **continuous time** model generates EXACT times of infection for individuals (event times are **REAL NUMBERS**)

e.g., a particular point in in time on a particular day, week, etc.

