

12 Epidemic modelling

Monday, March 23, 2020 10:06 PM

Let's consider a simple model of epidemics.

SIR model (3 component model)

Let $S(t)$ = # of susceptible individuals

$I(t)$ = # of infectious individuals

$R(t)$ = # of recovered individuals

$N = S(t) + I(t) + R(t)$ is the total population size.

β = transmission rate

γ = recovery rate

We will work in the well-mixed, where every person is equally likely to contact any other person in the population. You can think of this as a fully-connected social network.

Then the # of new infections per unit time is given by

$$\underbrace{\beta I}_{\text{chance of infection}} \cdot \underbrace{\frac{S}{N}}_{\text{chance of encounter with someone susceptible}}$$

and # of recoveries is γI .

$$\Rightarrow \frac{dS}{dt} = -\beta I \cdot \frac{S}{N}$$

$$\frac{dI}{dt} = \beta I \cdot \frac{S}{N} - \gamma I$$

$$\frac{dR}{dt} = \gamma I$$

The basic reproduction number $R_0 = \frac{\beta}{\gamma}$ ← transmission rate
← recovery rate

R_0 is the average number of new infections caused by an infected individual before they recover if everyone is susceptible.

$$\frac{dI}{dt} > 0 \Leftrightarrow 0 < \beta I \cdot \frac{S}{N} - \gamma I$$

$$\Leftrightarrow \beta I \cdot \frac{S}{N} > \gamma I$$

$$\Leftrightarrow \frac{\beta}{\gamma} \cdot \frac{S}{N} > 1$$

$$\Leftrightarrow \frac{\beta}{\gamma} \cdot \frac{S}{N} > 1$$

$$\Leftrightarrow \frac{R_0 \cdot S}{N} > 1$$

If the effective reproduction number $\frac{R_0 \cdot S}{N} > 1$, infection grows

$\frac{R_0 \cdot S}{N} < 1$, infection dies out.

Because $R(t) = N - S(t) - I(t)$, we can simplify to just the equations for $S(t)$, $I(t)$.

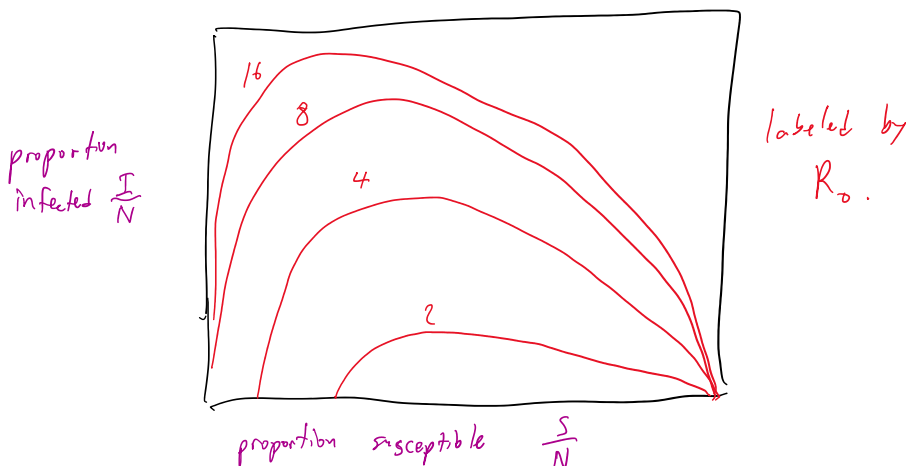
Solve the ODEs:
$$\frac{dI}{dS} = \frac{\beta I \cdot \frac{S}{N} - \gamma I}{-\beta I \cdot \frac{S}{N}} = -1 + \frac{N}{R_0 \cdot S}$$

$$I - I(0) = \left[-S + \frac{N}{R_0} \ln S \right]_0^t$$

$$I - I(0) = -S + \frac{N}{R_0} \ln S - \left(-S(0) + \frac{N}{R_0} \ln S_0 \right)$$

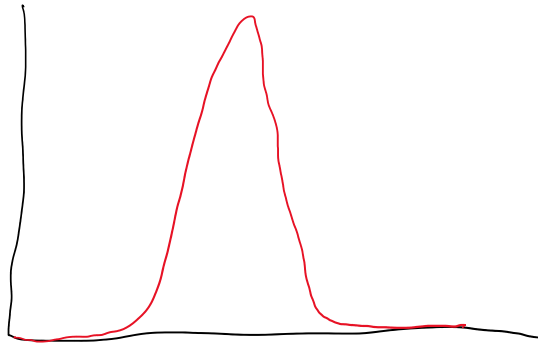
$$I = I(0) + S(0) - S + \frac{N}{R_0} \ln \frac{S}{S(0)}$$

This gives a phase portrait. We can graph $\frac{I}{N}$ vs. $\frac{S}{N}$



Unfortunately, we can't easily go from phase portrait to $I(t)$, so we don't actually know how long it takes.

Using numerical simulations, we can recover the expected epidemic curve.



Note: NOT Gaussian.

Note that the model we have described is similar to logistic growth, but not quite because individuals drop out of the infected population.

We have been considering only the deterministic model, but the real world is more stochastic

SIR stochastic differential equations

Let $S(t), I(t) \in [0, N]$ (continuous)

We will give a heuristic derivation, because this is not an SDE class.

Divide the time interval $[0, t]$ into subintervals of length Δt

Let $\Delta X(t) = \begin{bmatrix} \Delta S(t) \\ \Delta I(t) \end{bmatrix}$. Then $\Delta X(t) \in \left\{ \begin{bmatrix} 0 \\ 0 \end{bmatrix}, \begin{bmatrix} -1 \\ 1 \end{bmatrix}, \begin{bmatrix} 0 \\ -1 \end{bmatrix} \right\}$.

because

Event	Change $(\Delta S, \Delta I)$	Probability
Infection	$(-1, 1)$	$(\beta I S/N) \Delta t + o(\Delta t)$
Recovery	$(0, -1)$	$\gamma I \Delta t + o(\Delta t)$

Subdivide Δt further into smaller subintervals of length

$$\Delta t_i = t_i - t_{i-1}, \quad i=1, \dots, n, \quad \text{with } t_0 = t, \quad t_n = t + \Delta t$$

$$\text{and } \sum_{i=1}^n \Delta t_i = \Delta t.$$

→ ...

$\bar{i}=1$

Then $\Delta X(t) = \sum_{i=1}^n \Delta X(t_i)$, where $\Delta X(t_i) = X(t_i) - X(t_{i-1})$.

For Δt_i sufficiently small, it is reasonable to assume that the random variables $\{\Delta X(t_i)\}$ on the interval Δt are independent and identically distributed.

For n sufficiently large, we can apply the Central Limit Theorem.

$\Rightarrow \Delta X(t)$ is approximately normal with
 mean $E(\Delta X(t))$ and
 covariance matrix $CV(\Delta X(t))$.

$\Rightarrow \Delta X(t) - E(\Delta X(t)) \approx \text{Normal}(\vec{0}, CV(\Delta X(t)))$. ← continuous approximation to $\Delta X(t)$ because we use expectations

$$E(\Delta X) \approx \begin{pmatrix} -\beta SI/N \\ \beta SI/N - \gamma I \end{pmatrix} \Delta t \equiv f \Delta t = f(X(t)) \Delta t$$

taking the rate of change for $S(t)$ and $I(t)$.

and

$$CV(\Delta X) \approx E[(\Delta X)(\Delta X)^T] = E \begin{bmatrix} (\Delta S)^2 & \Delta S \Delta I \\ \Delta S \Delta I & (\Delta I)^2 \end{bmatrix}$$

$$= \begin{bmatrix} \beta SI/N & -\beta SI/N \\ -\beta SI/N & \beta SI/N + \gamma I \end{bmatrix} \Delta t \equiv C \Delta t = C(X(t)) \Delta t$$

because $(\Delta S)^2 = \Delta S$

$\Delta S \Delta I$ is nonzero iff $\Delta S = -1, \Delta I = 1$

$(\Delta I)^2 = \Delta I$

} can rewrite C as function of susceptible and infection states

Let $G = \begin{pmatrix} -\sqrt{\beta SI/N} & 0 \\ \sqrt{\beta SI/N} & -\sqrt{\gamma I} \end{pmatrix}$. Then $C = GG^T$

TI

. | continuous approximation

Then

continuous approximation

$$\Delta X(t) \approx \mathbb{E}(\Delta X(t)) + \text{Normal}(0, \text{CV}(\Delta X(t)))$$
$$= f(X(t)) \Delta t + G(X(t)) \Delta W(t),$$

where $\Delta W(t) = \begin{bmatrix} \Delta W_1(t) \\ \Delta W_2(t) \end{bmatrix}$ and $\Delta W_i(t) \sim \text{Normal}(0, \Delta t)$.

Letting $\Delta t \rightarrow 0$,

$$dX(t) = f(X(t)) dt + G(X(t)) dW(t),$$

where $W(t) = \begin{bmatrix} W_1(t) \\ W_2(t) \end{bmatrix}$ and $W_1(t), W_2(t)$ are independent Wiener processes.

i.e. $W_i(t) \sim \text{Normal}(0, t)$, or $dW_i(t) \sim \text{Normal}(0, dt)$.

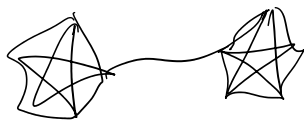
Note that $W(t)$ can be thought of as standard Brownian motion.

$$\Rightarrow \begin{aligned} dS(t) &= -[\beta S(t) I(t) / N] dt - \sqrt{\beta S(t) I(t) / N} \cdot dW_1(t) \\ dI(t) &= [\beta S(t) I(t) / N - \gamma I(t)] dt + \sqrt{\beta S(t) I(t) / N} \cdot dW_1(t) - \sqrt{\gamma I(t)} dW_2(t) \end{aligned}$$

If $I(t) = 0$, the epidemic stops.

Thus, sometimes even if $R_0 > 1$, an epidemic will randomly stop early.

Assume we do not have perfect mixing. i.e. that there are weakly connected communities



Then even if a disease has a high spread rate, you have a chance that the epidemic will not start in the 2nd community.

Linear to growth theory

Connections to graph theory

Can we model the SIR model using graph theory and/or percolation?

Suppose we have an Erdős-Renyi random graph.

How is this related to the SIR model?

Let's find the size of the connected component containing node 0.

1. Start at 0.
2. Include each node i w.p. p .
3. Go to each new node and create an edge to remaining nodes w.p. p .
4. Repeat.

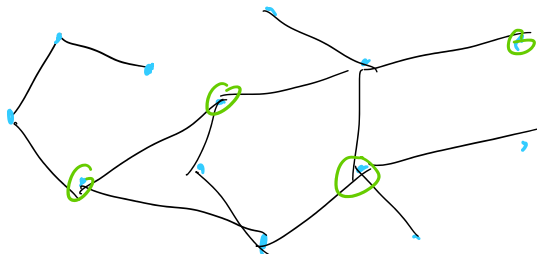
Once we have moved past a node, we never go back.

This process is equivalent to SIR infection model!

Connection to percolation theory

An Erdős Renyi random graph can be viewed as bond percolation on the fully connected graph, because you are turning on or off edges with probability p .

What happens if we additionally model site percolation on an Erdős Renyi random graph?



This is equivalent to immunizations, because we are removing nodes.

- Questions:
- (1) What is an appropriate model for social distancing?
 - (2) What about non-fully connected graphs?
 - (3) What are ways to model geographic borders?

- (2) What about non-rigid connections & maps.
- (3) What are ways to model geographic borders?
- (4) What type of mathematical phenomenon is "herd immunity" related to?