1 Epidemiological Models

Max-Coloring. In this lecture, mathematics model is used to study epidemiological problem such as how diseases are spreading out, how the population are infected etc. Historically, we all believe in gum theory of disease, e.g., Pathogem: a theory of disease. Here is a brief view of History: in 1546, miasma fumes is a hot topic. According the theory, people thought disease might come from silkworms. In 1854, John Snow at London cholera, found out people contact and interaction may account for this disease. In 2002, in paper The Mathematics of Infectious Diseases, Herbert Hethcote tried to apply mathematical model into this area. Here is the model.

Background: Since people contact each other, when one or several members have been infected, the disease spreads out among the population. This process has been described in the model as below:

SI model Based on the assumption that a people can either be healthy or be infected.

Overview: S —— I

Definition: fixed population size N. S=Susceptible I=Infected S+I=N usually, let —I—=1 initially,

The story is, when a person in S contact another person in I, an infection might happen subjected to a rate β, which can be intuitively regarded as susceptible rate. There are two assumptions here: 1, An infected person connect an susceptible person in an sufficiently way. 2, Random mixing. Everyone is equally likely to reach every one else.

Model: A person in I meets another one in S by rate S/N. Therefore, the average rate of new infection per unit is βI(S/N). The notion here is: β is a very complex variable which hides a lot information including disease-specific parameters, rate at which you meet people, etc.

In terms of how S and I change with time, we have: dI/dt = βSI/N (growing) dS/dt = -βSI/N(shrinking) s=S/N fractional susceptible i=I/N fractional infect s + i =1 So dS/dt=-β(1-i)i logistic growth equation

$$i_t = \frac{i_0 e^{\beta t}}{1 - i_0 + i_0 e^{\beta t}}$$

2 SIR model

Based on assumption that a people will recover after disease and can not be infected again.

Overview: S—— I ——R

Definition: β governs contact/transmission γ governs recovery/death

Note: if we know r, we can compute t = length of illness therefore, the number of people who will recover in time t=γ*t the number of people who will not recover is (1-γ)*t

logistic growth curve
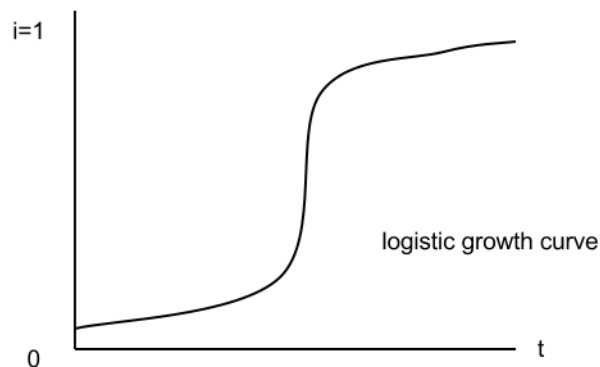
Probability that someone is still infected after t units of time is

$$(1 - \Delta \delta t)^{t/\delta t} = e^{-\Delta t}$$

Probability that someone stays infected for V but revoer by t+t P(t)dt=γdt

Still not a good disease model. ds/dt = -βsi stands for the loss from I di/dt = βsi - γi gain from I minus loss to R dr/dt = γi stands for gain from s s+i+r=1

Now deduct by two steps: i)eliminate i in the first equation using the last equiation. Then integrate these together by selecting a constant of integration so that the number recovered @t=0 is 0

$$S = S_0 e^{-\beta r/\Delta}$$

solve for r by eliminating i in second equation. The answer is: r=1-S

From the simulation we can see:

Here is an interesting question to ask: what happens if β and γ changes over time? It operates in 2 different phases. The cutoff is β/γ: β/γ =1 epidemic transition. β/γ ¡1 people are getting better faster than others all getting sick. epidemic will die out. β/γ ¿1 epidemic curve. In SI model: γ =0 so β can not be ¡ γ. β/γ =R correspond to the number of new cases created on average by each new case.

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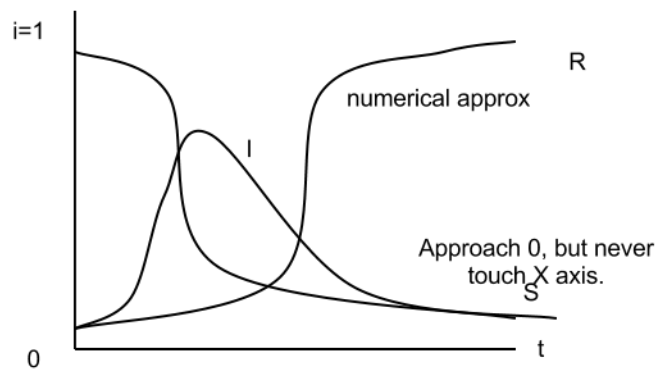
better faster than others all getting sick. epidemic will die out. $\beta/\gamma > 1$ epidemic curve. In SI model: $\gamma = 0$ so β can not be $\leq \gamma$. $\beta/\gamma = R$ correspond to the number of new cases created on average by each new case.

3 SIS model

Based on assumption that a people will recover after disease and may be infected again. Overview: S—— I ——S

Definition: β governs contact/transmission γ governs recovery/death Model: $ds/dt = \gamma i - \beta si$ $di/dt = (\beta\text{-}\gamma\text{-}\beta i)i$ $di/dt = \beta si - \gamma i$ s=1- i solve: final $i = (\beta\text{-}\gamma)/\beta$, which is also endemic disease state
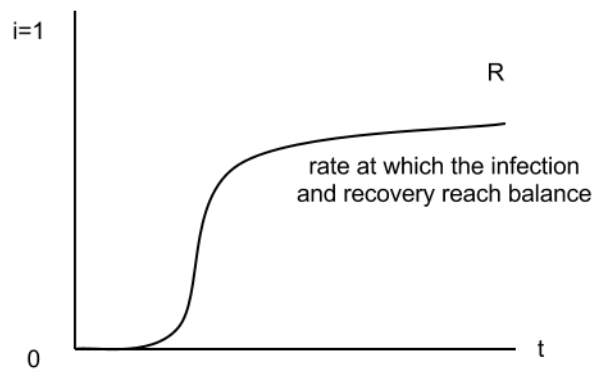
$$i_t = i_0 \frac{(\beta - \Delta)e^{(\beta-\Delta)t}}{\beta - \Delta + \beta i_0 e^{(\beta-\Delta)t}}$$

if $\beta\gamma$, then basically equivalent to R=β/γ=1

4 SIRS model

This model will take more and more factors into account. Overview: S—— I ——R —— S

The model yields different behavior depending on ,β,γ 1)if ¡1 . disease can die out present in endemic state oscillation: waves of infection.

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The future topic will be how to fix inadequacies of the model? The way is to make it more complex. nature of β -could be a function of N -people tend to saturate -could be climate depend -could be time depend

N needt be fixed births, immigration/emigration, natural deaths, deaths due to disease will effect N. For instance, for SEIR, there might me vertical incidence like born/die to S, die to E etc. Therefore, we may have some further models like SI, SIS, SIR, SEI, SEIS, SEIR, SEIRS.

Also, environment may constrain N P-¿ newborn A-¿ asymptomatic infected C-¿ carriers V-¿ Vaccinated ... also zoonotic disease.

E, I, R may have temporal components. We may use other, non exponential distribution.

Demographics. E.g., kids mix at different rates newborn might be S or E etc.

References

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[2] H. Herbert, The Mathematics of Infectious Diseases. SIAM Review Vol 42:4, 599-653.