Mathematical modelling of infectious diseases: A story of influenza and TB

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What is modelling?

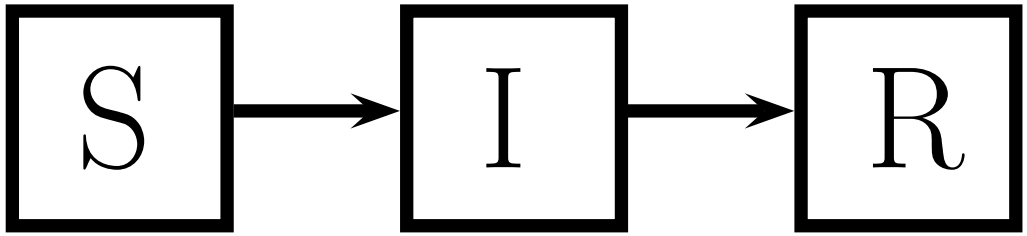
- Models are a simplified representation of a complex phenomenon
  - Mathematical models are caricatures
- Used for both prediction and understanding
- Modelling is an iterative process; always return to the original problem and assess the model
- Always remember that

> "Essentially, all models are wrong, but *some* are useful"
>
> G.E.P. Box and N.R. Draper, 1987, *Empirical Model-Building and Response Surfaces*. Wiley, pp424, 1987

Why model infectious diseases?

- The past: Understanding the underlying causes of disease dynamics
  - Did school closure impact influenza spread?
- The present: Real time data analysis and impact on policy decisions
  - Can we estimate how big an epidemic will be from early data?
- The future: Scenario planning
  - Different scenarios around infectivity, severity, timing, quarantine, etc.
  - Vaccination planning; best schedule, targeted populations, etc.

The SIR model



- S=Susceptible I=Infectious R=Removed
- Most often used in epidemiology studies
- No fine detail; low data demands
- Assumes a homogeneous, well mixed population

$$\frac{dS}{dt} = -\beta SI$$
$$\frac{dI}{dt} = \beta SI - \gamma I$$
$$\frac{dR}{dt} = \gamma I$$

  - β is the transmission rate
  - $1/\gamma$ is the average infectious period

Influenza – with Mick Roberts

- We explore the effect of heterogeneity in population susceptibility and infectivity on an epidemic in terms of:
  - size and timing of the peak
  - final size distribution
  - mortalities due to the pathogen
- An important question in epidemiology is "for whom should we prioritise vaccination?"
  - most susceptible?
  - most infectious?
  - highest mortality rate?

Compare how epidemic attributes are affected by vaccination prior to the epidemic for an influenza-like-illness.

Our model

$$\frac{\partial x(t,\theta)}{\mathrm{d}t} = -(\beta + \rho\theta)x\frac{1}{2}\int_{-1}^{1} c(\theta)y(t,\theta)\,d\theta$$

$$\frac{\partial y(t,\theta)}{\mathrm{d}t} = (\beta + \rho\theta)x\frac{1}{2}\int_{-1}^{1} c(\theta)y(t,\theta)\,d\theta - y$$

$$\frac{\partial z(t,\theta)}{\mathrm{d}t} = y$$

where

- x, y, z are the densities of susceptible, infectious, and removed (immune after infection or dead)
- $(\beta + \rho\theta)$ is the variation in the transmission rate
- $\theta \in [-1, 1]$, giving $\beta \pm \rho$
- $c(\theta)$ is the variation in infectivity

Results

Susceptibility profile	Final size	Deaths	$\mathcal{R}_{\text{eff}}$
Uniform	42.0%	1.01%	1.32
Youngest Vacc. (RH)	0.02%	0.00%	0.95
Oldest Vacc. (LH)	39.9%	0.81%	1.35
Both	20.8%	0.50%	1.15

Final size for classic SIR model with is 14.3%.

Tuberculosis and Diabetes

Tuberculosis (TB):

- TB is a contagious airborne disease
- 1/3 of the worlds population infected with TB
- Kiribati has the 2nd highest TB case notification rate in the Western Pacific Region
  - 339 TB cases /100,000 population (WHO 2012)

Diabetes Mellitus (DM):

- DM is a chronic, not infectious, disease
- In Kiribati adult prevalence has reached 28% for adults (25+) (WHO 2009)

The risk of TB is 1.5–8 times higher in patients with diabetes (Stevenson, Critchley et al. 2007)

The Pacific Islands

The Project – with Kerri Viney

- Our goal: to quantify the effect of an increasing burden of DM on TB prevalence – does this explain the data?
  - Develop a compartmental model – system of ODEs
- "What if" scenarios
  - E.g. If DM increases to x% of the population over the next 10 years, what is likely to happen with TB over this period?
- A sensitivity analysis;
  - what are the likely effects of assumptions made during the modelling process on the results?
  - on which parameters of TB and DM should future data collection focus?
  - Latin Hypercube Sampling with the Partial Rank Correlation Coefficient multivariate analysis

Any questions?

Image courtesy of `http://spikedmath.com/046.html`
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