Epidemiological Mathematics: Key Aspects of Modelling the Spread of Infectious Diseases

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1 Introduction

"We are not ready for the next epidemic."
— Bill Gates, 2015

Despite the advancements in the last century to improve our collective understanding of infectious diseases, they still pose a large and constant threat to significant portions of the human population. A crucial part of fighting any disease is studying how the disease moves through a population to prevent others from being infected.

Epidemiology has evolved a great deal in the past 150 years (appendix A.1) - from simple systems of two coupled ordinary differential equations to complex probabilistic models that attempt to replicate the dynamics of human behaviour. This rapid advancement of such a complex topic has mainly been motivated by a great need for understanding. It is clear, just looking at recent history (Spanish flu, measles, rubella, polio, Ebola, the list is almost never ending), that a global pandemic of a deadly disease is not unlikely, and the outcome could be as life changing and society altering as a nuclear war or climate change. One of the first lines of defence for tackling an outbreak of a disease are mathematicians studying the data and quantifying the dynamics, giving medical professionals information on which to base decisions.

1.1 What is Epidemiology?

Epidemiology is a branch of medicine that studies the occurrence, advancement and the (potential) control of disease spread through populations. To a mathematician, epidemiology comes down to using mathematical models to describe changes at the population level - splitting a population into groups and describing the dynamics and rates individuals transition between groups.

By analysing characteristics of a disease, such as the mechanisms through which it spreads, which groups are particularly susceptible, and the force of infection, mathematicians can build increasingly sophisticated models to more accurately forecast how a disease may spread. This has the potential to save many lives and hours of intense medical labour. Not only is epidemiology a vital branch of mathematics for saving human lives, it also plays a crucial role in many other areas of nature - population dynamics[3], economics[4], and biological systems, to name a few - and so a cursory understanding of the subject is almost a necessity for those who wish to study natural science.

1.1.1 Endemic, Epidemic, and Pandemic

Diseases mainly exhibit 3 different behaviours:

1. **Endemic**: the disease exists at a reasonably consistent (or cyclical) level in a particular area.
2. **Epidemic**: the disease spreads rapidly in a specific area or population over a short period of time.
3. **Pandemic**: a global version of epidemic - the disease spreads rapidly around the world.

These behaviours were motivated by Sir Ronald Ross (appendix A.2). It is important to note that these terms do not denote the severity of a disease. They simply describe the aggression of the spread, and area of impact.

1.2 Aim

This short paper will motivate the mathematics behind epidemiology and introduce key concepts, beginning with very simple models. Accompanying this will be a few examples to assist the reader's transition from theoretical understanding to practical application and intuition. Included in the appendix is code that the reader is free to use and edit to assist their learning, and a brief history on modern epidemiology.

2 Deterministic Models using Differential Equations

When mathematicians wish to model the dynamics of a system, the first usual point of contact is to describe the system as a set of differential equations. Epidemiology is no different, in this respect. Although aspects of epidemiology were used in the mid-to-late eighteenth century, it was first generalised by McKendrick and Kermack in 1926-1927[5], where they devised both a stochastic and deterministic approach to deducing the final size of an epidemic.

This section will assume that the reader has a good appreciation of *systems of ordinary differential equations*. If the reader finds themselves lost at any point, a recommended companion text is *Ordinary Differential Equations* by Morris Tenenbaun and Harry Pollard[6]. It is clear, reasonably concise, and contains many good exercises to cultivate understanding.

2.1 The Epidemic SIR Model

One of the simplest systems to describe the dynamics of an epidemic is the SIR model[7]. It takes its name from the three populations that individuals can move between:

1. **Susceptible**: individuals are who do not carry the disease, but are at risk of infection.
2. **Infected**: individuals who are carriers of the disease, potentially spreading it.
3. **Removed**: individuals who have had the disease and can no longer be infected (because they have recovered and become immune, for example).

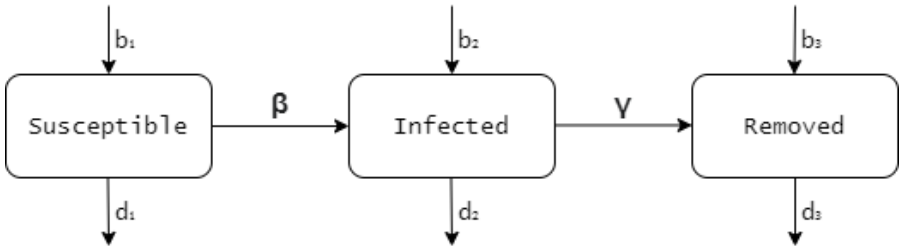

Figure 1: Transfer diagram of the SIR model with an open population (births and deaths)

Figure 1 is physical representation of this process, including birth rates b, death rates d, infection rate β, and removal rate γ. Individuals move between the three groups at some given rate over time t. In a closed population (ie, we assume that $b, d \lll \beta, \gamma$, so we can ignore natural births and deaths), the system of equations that describe the change in each group through time is:

$$\begin{aligned} \frac{dS}{dt} &= -\beta SI \\ \frac{dI}{dt} &= \beta SI - \gamma I \\ \frac{dR}{dt} &= \gamma I \end{aligned} \tag{1}$$

and so $\frac{dS}{dt} + \frac{dI}{dt} + \frac{dR}{dt} = 0$. We can interpret these equations to mean that:

- Individuals move from the susceptible group to the infected group proportional to the amount of people that can be infected S, the amount of people that can infect others I, and the "successful contact rate" β.
- Individuals move from the infected group to the removed group proportional to the current population of the infected group I and the removal rate γ.
- Once an individual enters the removed group R they remain there indefinitely.

Intuitively, the initial conditions of the system with a large population size N are such that, as a proportion of population, $S(0) = \frac{N-1}{N} \approx 1$, $I(0) = \frac{1}{N} \approx 0$, $R(0) = 0$, and, at any time t, $S(t) + I(t) + R(t) = 1$. In other words, at $t = 0$ most people are susceptible with very few (proportional to the size of the population) being infectious.

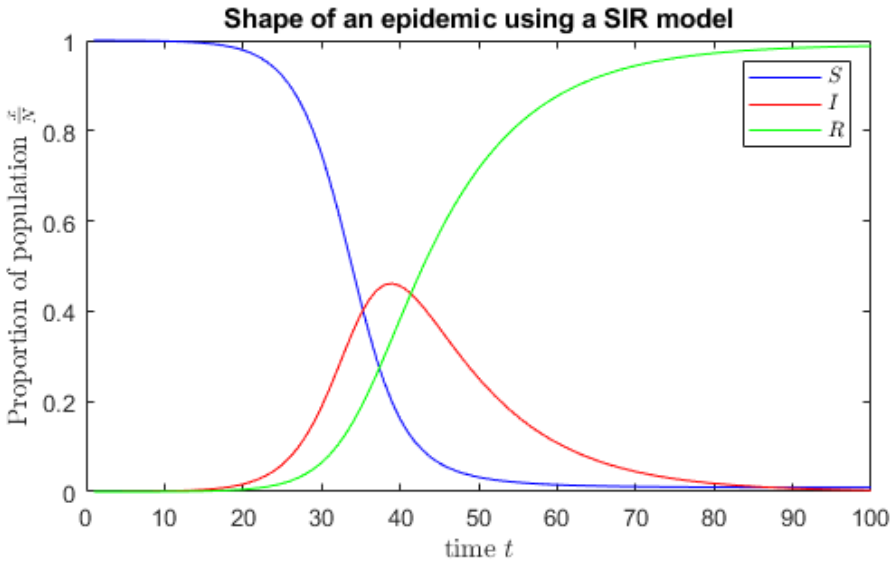

Figure 2: Epidemic using the SIR model for $S_0 = 9999$, $I_0 = 1$, $R_0 = 0$, $\beta = 0.4$, $\gamma = 0.09$. Code for the SIR model is included in appendix B.1

The SIR model, whilst perhaps being too simple to catch the day-to-day intricacies of an epidemic, does a good job at modelling the overall dynamics of a well-behaved system. It operates on a few assumptions

1. The population size N is constant (closed population).
2. The transition rates are constant.
3. There is no seperation in the demographic by things such as age, location or sex (which can alter transmission rates).
4. The population is well mixed, so all infected individuals have the same chance of passing on the disease.

Diseases (and people), unfortunately, are not always well behaved, and so the SIR model is not always the best type of model to use[8]. SIR models do, however, lay a good foundation to build more sophisticated models from.

2.2 The Three Threshold Quantities

When an infectious disease begins to spread, there a few key characteristics that dictate how self-sustaining the disease is at any given time, and therefore what trajectory the epidemic is currently on. These quantities are the basic reproduction number R_0, the contact number σ, and the replacement number R[10].

2.2.1 The Basic Reproduction Number R_0

The basic reproductive number R_0 is defined from the very beginning of an epidemic ($t = 0$), and is a fixed value. The value tells us this: given a perfectly susceptible population (ie, one infected individual, many susceptible individuals) how many secondary cases should be expected?

This number can be quantified by analysing the differential equation for the infected population in equation (1). For an epidemic to occur at small values of t, the disease needs to be spreading faster than the removal rate, so therefore it is required that $\frac{dI}{dt} > 0$, which means

$$\begin{aligned} \beta SI - \gamma I &> 0 \\ \frac{\beta SI}{\gamma} &> I \end{aligned} \tag{2}$$

However, at $t = 0$ it is known that $S \approx 1$. Substituting this in and dividing through by the infected population gives

$$R_0 = \frac{\beta}{\gamma} > 1 \tag{3}$$

which gives R_0. It is clear that, for an epidemic to occur, the first hurdle of the disease is to ensure that its R_0 exceeds one. This can be done through a variety of ways, such as[9]

- High transmissibility.
- Long recovery time.
- Existing in a population with a high average rate of contact (high mixing).

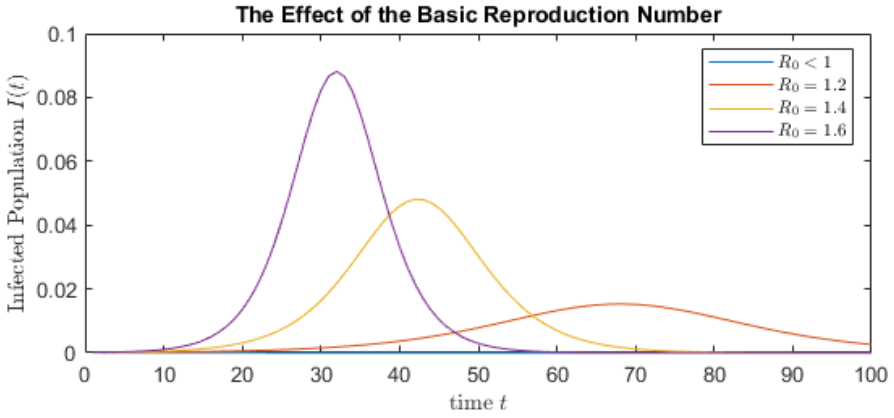

Figure 3: The different shapes the infected group can take, depending on the value of R_0

R_0 is a very important concept, as it has a huge impact on the size and length of an epidemic (figure 3). Large values of R_0 cause aggressive, short epidemics, where a large number of individuals could be infected at once. Values close to but exceeding 1 lead to long, slow epidemics, with a low peak.

2.2.2 The Contact Number σ

The contact number σ is simply the average number of adequate contacts between infected individuals and susceptible individuals. It is bounded by R_0, so

$$\sigma \leq R_0 \tag{4}$$

and at $t = 0$, $\sigma = R_0$. Unlike R_0, σ is variable through an epidemic, and therefore varies with t. This is because the number of adequate contacts can change for a variety of reasons as an epidemic unfolds. For most simple models, σ remains constant. In complex models, σ can vary. However, it does always follow the relationship established in (4).

2.2.3 The Replacement Number R

The replacement number R describes the current average number of secondary cases from a typical infection at a given time. It is very similar to R_0, but varies with time. Since $S \leq 1$ it therefore holds that, for very small t, $R_0 = \sigma = R$, and as the epidemic develops that for $t > 0$, $R_0 \geq \sigma \geq R$. If $R > 1$ then the epidemic is growing. If $R < 1$ then the epidemic is decaying.

2.3 The Endemic SIR Model

The endemic SIR model is very similar to the epidemic model. The major difference is that, in this model, we assume that the dynamics of the disease (β and γ) are on a scale similar to vital population dynamics (births and deaths), and so they are no longer negligible. For a simple endemic, take the follow assumptions

- $b = d = \mu$, so birth rates and death rates are equal, giving a closed population (constant N).
- Having or having had the disease does not change the rate of death.
- Individuals are only born susceptible. They cannot be born infected or with a natural immunity.

This means that equation (1) becomes

$$\begin{aligned} \frac{dS}{dt} &= -\beta SI + \mu - \mu S \\ \frac{dI}{dt} &= \beta SI - (\gamma + \mu)I \\ \frac{dR}{dt} &= \gamma I - \mu R \end{aligned} \tag{5}$$

2.3.1 Equilibrium

The new variable μ changes the dynamics, and introduces the possibility of an equilibrium - a point at which the level of the disease is constant[12].

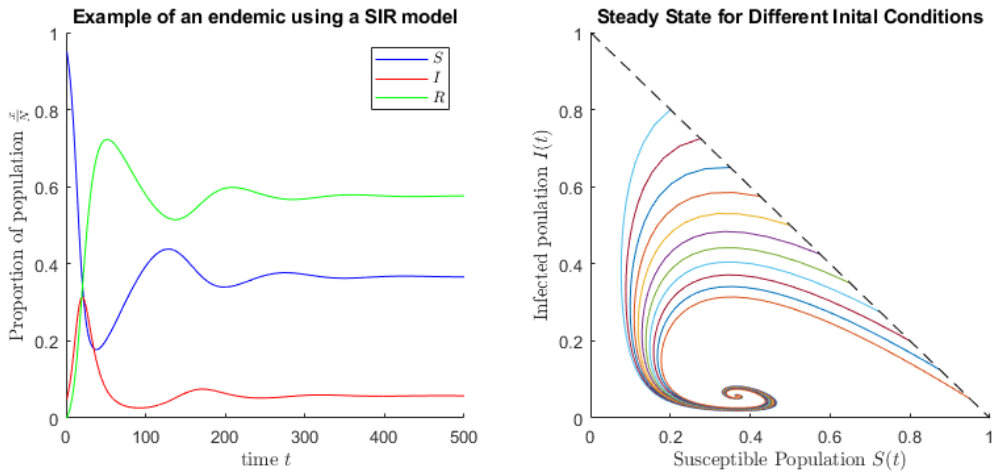

Figure 4: Left graph: an example endemic. Right graph: susceptible population against infected population for different initial conditions. Even if the initial conditions vary wildly, the steady state solution remains the same. For the all simulations, $\beta = 0.3$, $\gamma = 0.1$, $\mu = 0.01$. SIR endemic code is in appendix B.3

When at an equilibrium, there should be no change to the system, so $\frac{dS}{dt} = \frac{dI}{dt} = \frac{dR}{dt} = 0$. If the disease and population dynamics are known, the steady solution can be found using system

(5). If all equations equal 0, then

$$\begin{aligned} -\beta SI + \mu - \mu S &= 0 \\ \beta SI - (\gamma + \mu)I &= 0 \end{aligned} \tag{6}$$

and so $I\big(\beta S - (\gamma + \mu)\big) = 0$. For a non trivial equilibrium, $I > 0$, and so $\beta S - (\gamma + \mu) = 0$, which gives

$$S = \frac{\gamma + \mu}{\beta} \tag{7}$$

at the steady state. Putting in the parameters for the example in figure 4 gives $S = 0.3667$, which matches the values in the graphs. The infectious population equilibrium is then at

$$I = \frac{\mu(1 - S)}{\beta S} \tag{8}$$

Again, the parameters for figure 4 give $I = 0.0576$, which also matches the solution. Obviously, the removed population at the equilibrium is also easy to calculate as $R = 1 - S - I$. This means that, given the population and disease parameters, a value for S, I, and R can be calculated as $t \longrightarrow \infty$.

3 Discrete Time Stochastic Epidemic SIR Model

In the previous section, deterministic models were explored. These had fixed rates and quantities, which made modelling and predicting behaviour reasonably straightforward. Deterministic models work well when considering the dynamics of a large system, as the law of large numbers states that if enough probability driven events take place (such as a contact with another person successfully passing on a disease) the mean outcome should be close to the expectation[13].

Realistically, this process breaks down in the early stages of a breakout, and probability driven events can cause extreme shifts in the dynamics of a system (eg, another outbreak happening on the opposite side of the planet because, by chance, an infectious individual went on holiday). Because of this, stochastic models can be employed and simulated many times to find

1. The trajectory and behaviour in the average case.
2. The probability extinction vs epidemic vs pandemic.
3. The probability and behaviour of the average worst case scenario.

3.1 Adapted Reed-Frost Model[15]

The Reed-Frost model is one of the simplest practical cases of a stochastic epidemic model. Formulated originally in the 1920s, it wasn't solidified into a final theory and published until 1952[14]. The model considers the infectivity of a disease on an individual level. Much like in the previous simple SIR model, it uses a closed population that mixes of size N, where each individual has a fixed probability of having adequate contact with an infected individual each day (time step). Individuals are infectious for R time steps (also known as the recovery period) before they are removed.

3.1.1 The Model as a Markov Chain

Each individual i, from $i = 1, 2, 3, ..., N$ is labelled as

$$Y^{\{i\}} = \{Y(t), t = 0, 1, 2, ...\} \tag{9}$$

in such a way that $Y^{\{i\}}(t) = 0$ means that the individual is susceptible at time step t, and $Y^{\{i\}}(t) = 1$ means they are infectious. The infectious period R is fixed (although, one could

consider allowing R to be a random variable too). Therefore, if an individual is infected at time t they are removed at time $t + R$, and they cannot be reinfected. The total number of infectious individuals at time t is therefore

$$Y(t) = \sum_{i=1}^{N} Y^{\{i\}}(t) \quad \text{for} \quad t \geq 0 \tag{10}$$

For every time step $[t, t+1)$, each individual encounters a fixed number n_i of other individuals, and a random number of other individuals $M_i(t)$, making the total number of individuals encountered in $[t, t + 1)$

$$N_i(t) = n_i + M_i(t) \tag{11}$$

This is to ensure each susceptible person has more than 0 encounters in each time step, so $N_i(t) \geq n_i$. $M_i(t)$ is a mutally independent variable, and does not change, regardless of the current state of the population. If an individual has not been infected at time t and encounters an individual who is infectious at time $[t, t + 1)$ then the individual becomes infected with a probability $p \in [0, 1]$, with the infection starting at $t + 1$. They will remain infectious until $t + 1 + R$.

If $Y(t)$ is a known value then the probability of encountering an infectious individual is simply

$$f(t) = \frac{Y(t)}{N} \tag{12}$$

Since the model is calculating the probability of transitioning between states (susceptible, infectious, removed), and all $N_i(t)$ are independently identically distributed, this can be seen as an $(R + 1)$-dimensional *Markov chain*[16]. If $X(t)$ is the number of individuals yet be infected, $Z(t)$ is the number of removed individuals, and $Y_i(t)$ is the number of individuals who have been infected for i time units (so $i = 0, 1, ..., R - 1$) at time t, then

$$V(t) = \big(X(t), Y_0(t), Y_1(t), ..., Y_{R-1}(t)\big), \quad t = 0, 1, 2, ... \tag{13}$$

This means that the condition of $V(t)$ is dependent on the current state of the population - previous states have no impact. Clearly then

$$Z(t) = Z(t - 1) + Y_{R-1}(t - 1) \quad \text{and} \quad Y(t) = \sum_{k=0}^{R-1} Y_k(t) \tag{14}$$

Each individual can only be susceptible, infected, or removed at any one time, and so $X^{\{i\}} + Y^{\{i\}} + Z^{\{i\}} = 1$, as two variables must be 0.

3.1.2 Probability of Transmission

Since it has been shown that the process is a Markov chain, it is important to establish a general form to calculate the probability that an individual transitions to a different state each step. Firstly, it is assumed that the recovery period R is fixed, and so if an individual is first infectious at time t, they will recover at time $t + R$. Take $Y(t) = y$.

During each time step, a susceptible individual $X^{\{i\}}$ will meet N_i other individuals. The probability of transitioning from $X \to Y$ is proportional to the current number of infected contacts and the probability of a catching the disease, given that j infectious contacts have occurred. Given that j infectious contacts occur at time t for an individual i, and each infectious contact has a probability $p \in [0, 1]$ of causing an infection, the probability of transitioning from $X \to Y$ is

$$p_j = 1 - (1 - p)^j \tag{15}$$

Assuming that $N \gg N_i$, the probability of encountering j number of individuals can be approximated with the binomial distribution

$$P_j^{\{i\}}\big(y, N_i(t); N\big) \approx \binom{N_i(t)}{j} \left(\frac{y}{N-1}\right)^j \left(1 - \frac{y}{N-1}\right)^{N_i(t)-j} \tag{16}$$

This means that the probability of an individual transitioning from $X \to Y$ at time t is given by

$$P(Y_0^{\{i\}}(t+1) = 1 | N_i(t), X^{\{i\}}(t) = 1, Y(t) = y) = 1 - \left(1 - \frac{py}{N-1}\right)^{N_i(t)} \tag{17}$$

Given the probability of transmission, recovery rate, and initial conditions, one can calculate the probability of the disease going extinct against the probability of an epidemic occurring.

4 Conclusion

Both stochastic and deterministic models are useful for studying the behaviour and dynamics of an infectious disease. Deterministic models are quite modular, with layers of complexity (such as aging populations, multi disease systems, etc) being quite simple to add, provided parameters are known. They also allow different disease outbreaks to be directly compared. As stated in section 3, they do lose some of the small perturbations that clearly exist in real world diseases, and they cannot account for random events that can have a huge impact on the progression of an epidemic (eg a new variant). There is also the issue of fixed disease parameters. There is no room for the possibility of an extinction event of a disease at early t - it either will go extinct or it won't, dictated by the value of R_0.

Stochastic models can take these small, random behaviours into account, but sometimes at the cost of truly seeing the underlying dynamics of the system. There is also the fact that calculating the probability of an individual contact being a successful transmission is already quite a challenge. These probabilities could vary for a huge variety of reasons. However, Linda J. S. Allen [17] notes that "*Stochastic modeling of epidemics is important when the number of infectious individuals is small or when the variability in transmission, recovery, births, deaths, or the environment impacts the epidemic outcome.*" She goes on to say that it is especially important for *zoonotic* infectious diseases, where a pathogen jumps from animal to human transmission.

In reality, it is useful to use both models to forecast the behaviour of a disease[17]. Ideally, the stochastic model should match the general shape of the deterministic model, whilst displaying some interesting perturbations.

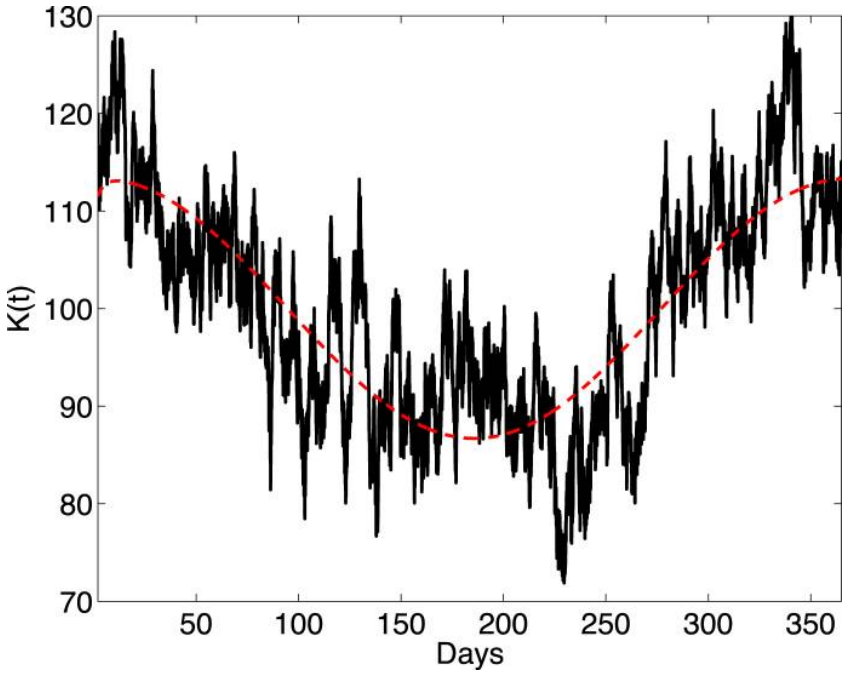

Figure 5: A graph on the predicted progression of malaria. The red dotted line is the ODE solution. The black line is a SDE solution that takes into account random environmental variability that effects birth, death, and transmission rates

A Celebrating Some of the Founders of Modern Epidemiology

A.1 John Snow (1813 - 1858)

John Snow was an English physician who played a crucial role in controlling a cholera outbreak in Soho, London in 1854. At this time many academics and physicians explained the spread of disease using *miasma*, meaning bad air. John Snow was one of a few skeptics of this concept, and actually displayed some understanding of germ theory in one of his early papers[1]. It was due to this skepticism that he began to suspect that cholera was spreading through the water, not the air. The local authorities, however, were not easy to convince.

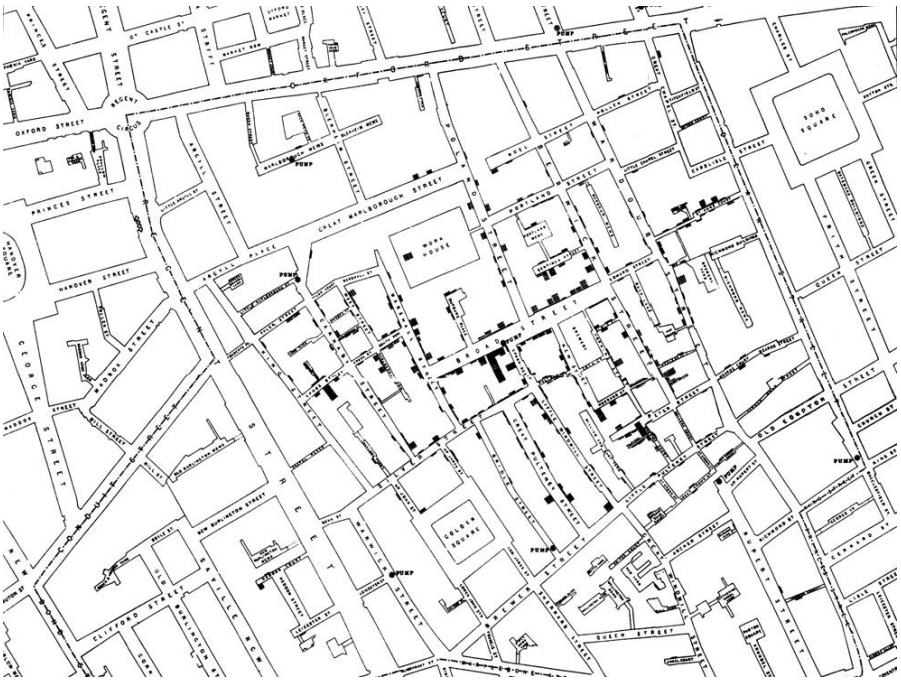

Figure 6: Map John Snow made to study the spread of cholera through the population of Soho

In an effort to convince them, John Snow embarked on one of the earliest recordings of modern epidemiology. He spent days going door to door in Soho to mark who and where the disease had spread to, creating the map above. It was during this mapping process that he noticed three things:

1. Most deaths and cases were recorded near the Broad Street water pump.
2. There were no cases in labourers who worked at the local brewery near the pump.
3. There were a few outliers - people who lived relatively far from Broad Street, but had still somehow caught the disease.

Upon further investigation, he discovered the two crucial pieces of evidence he needed.

1. The workers at the brewery consumed light beer during the day, not water.
2. The outlier's were brought water from the Broad Street pump because they preferred the taste.

Using this evidence, John Snow successfully lobbied to have the Broad Street pump handle removed, bringing the epidemic under control. Sadly, however, John Snow's (correct) theory that the disease was due to fecal matter in the water was ultimately rejected.

In honour of John Snow, a replica pump stands in Broadwick Street, where lectures on public health are held each year. The handle of the pump is ceremonially removed and reattached as a tribute to his efforts.

A.2 Sir Ronald Ross (1857 - 1932)

Sir Ronald Ross was a British doctor who was awarded the Nobel Prize for Physiology and Medicine in 1902 for his long work and study of malaria transmission. Whilst as a young man he studied and sat his medical exams in England, most of his experience was garnered in the Indian Medical Service, which he joined in 1881.

Inspired by the previous works of Sir Patrick Manson and Alphonse Laveran, he began studying Malaria in 1892. It wasn't until 1897 that he made the landmark discovery that Malaria was spread through a parasite found in the stomach of mosquitoes. He solidified his findings a year later by showing that mosquitoes who fed on infected birds developed the parasites, and could spread the disease to other birds on subsequent feedings.

This isn't his only great contribution, however. Sir Ronald Ross also laid some of the groundwork for the modern study of epidemics, motivating the patterns of disease spread known as *epidemic curves*[2], a graph that the modern world is now only too familiar with.

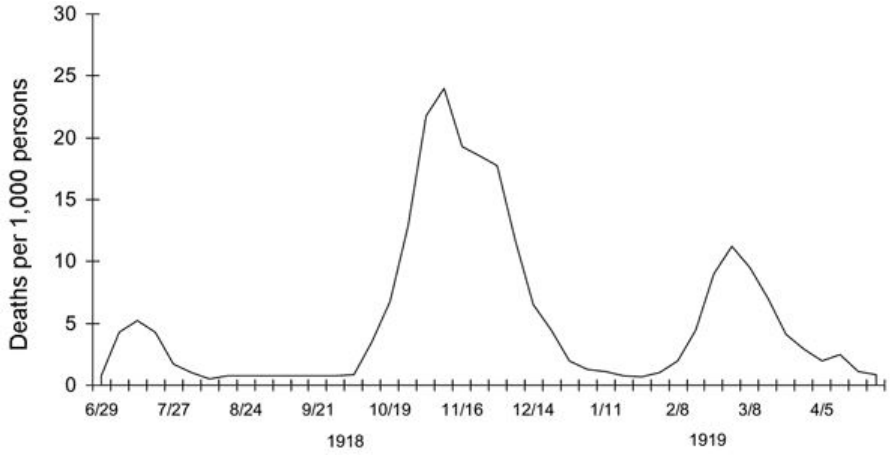

Figure 7: Epidemic curve of Spanish influenza, 1918-1919. The three waves are distinct

In the cited paper, Sir Ronald Ross highlighted that there were 3 key patterns in disease spreads

1. *Endemic*: Constant low level infections with small fluctuations.
2. *Outbreak*: Constant presence of disease with local flare ups in frequent intervals.
3. *Epidemic*: Incredibly intense outbreaks followed by (almost) complete disappearance.

In the same paper, he motivated two interesting questions:

- What causes these differences between disease spreads?
- In epidemics, what are the disease characteristics that shape the curve?

These two questions really do underpin the foundation of epidemiology, and, whilst a mathematician may not be too interested in the details of the biology, we are certainly interested in exploring how different variables may alter the length and ferocity of an epidemic.

B Code for Mathematical Models

B.1 Epidemic SIR Model

```
%% Preallocate vectors for population changes
n = 100; % Number of time steps
time = [1:1:n]; % Time steps
S = zeros(1, n); % Susceptible population changes
I = zeros(1, n); % Infected population changes
R = zeros(1, n); % Recovered population changes

%% Variables
beta = 0.4; % Contacts per person
gamma = 0.09; % Recovery rate
S(1) = 9999; %  Initial susceptible population
I(1)= 1; % Initial infected population
R(1) = 0; % Initial recovered population
N = S(1)+I(1)+R(1); % Population size
% Convert to proportion of population (closed system)
S(1) = S(1)/N;
I(1) = I(1)/N;
R(1) = R(1)/N;

%% Run similation
for t=2:100
    % Calculate changes in populations
    dS = -beta*I(t-1)*S(t-1)
    dR = gamma*I(t-1);
    dI = -dS - dR;

    % Apply changes
    S(t) = S(t-1) + dS;
    I(t) = I(t-1) + dI;
    R(t) = R(t-1) + dR;
end

%% plot data
plot(time, S, '-b')
hold on
plot(time, I, '-r')
plot(time, R, '-g')

%% Beautify plot
title('Shape of an epidemic using a SIR model')
ylabel('Proportion of population $\frac{x}{N}$', 'interpreter','latex')
xlabel('time $t$', 'interpreter','latex')
legend('$S$','$I$','$R$', 'interpreter','latex')
```

B.2 Basic Reproductive Number

```
%% Preallocate vectors for population changes
steps = 100; % Number of time steps
time = [1:1:steps]; % Time steps
pop_size = 10000;
n = length(time);

%% Variables
gamma = 0.5; % Recovery rate
betas = [0.45, 0.6, 0.7, 0.8]; % Contacts per person


%% Run similation
for i=1:length(betas)
    beta=betas(i);
    S = zeros(1, n); % Susceptible population changes
    I = zeros(1, n); % Infected population changes
    R = zeros(1, n); % Recovered population changes
```

```
    S(1) = 9999; %  Initial susceptible population
    I(1)= 1; % Initial infected population
    R(1) = 0; % Initial recovered population
    % Convert to proportion of population (closed system)
    S(1) = S(1)/pop_size;
    I(1) = I(1)/pop_size;
    R(1) = R(1)/pop_size;
    for t=2:n
        % Calculate changes in populations
        dS = -beta*I(t-1)*S(t-1);
        dR = gamma*I(t-1);
        dI = -dS - dR;

        % Apply changes
        S(t) = S(t-1) + dS;
        I(t) = I(t-1) + dI;
        R(t) = R(t-1) + dR;
    end
    %% plot data
    plot(time, I)
    hold on
end


%% Beautify plot
title('The Effect of the Basic Reproduction Number')
ylabel('Infected Population $I(t)$', 'interpreter','latex')
xlabel('time $t$', 'interpreter','latex')
legend('$R_0 < 1$', '$R_0 =  1.2$', '$R_0 = 1.4$', '$R_0 = 1.6$', 'interpreter'
    ,'latex')
```

B.3 Endemic SIR Model

```
%% Preallocate vectors for population changes
n = 500; % Number of time steps
time = [1:1:n]; % Time steps
S = zeros(1, n); % Susceptible population changes
I = zeros(1, n); % Infected population changes
R = zeros(1, n); % Recovered population changes
dS = zeros(1, n); % Susceptible population changes
dI = zeros(1, n); % Infected population changes
dR = zeros(1, n); % Recovered population changes


%% Variables
beta = 0.3; % Contacts per person
gamma = 0.1; % Recovery rate
mu = 0.01; % A natural birth and death rate (births = deaths)
N=10000; %population size
Sic = [2000:750:9500]; % initial conditions for S population

% simulate many different initial conditions to show equilibrium
for i =1:length(Sic)
    S(1) = Sic(i); %  Initial susceptible population
    I(1)= N-Sic(i); % Initial infected population
    R(1) = 0; % Initial recovered population
    % Convert to proportion of population (closed system)
    S(1) = S(1)/N;
    I(1) = I(1)/N;
    R(1) = R(1)/N;

    %% Run similation
    for t=2:n
    % Calculate changes in populations
       dS(t-1) = -beta*I(t-1)*S(t-1) + mu - mu*S(t-1);
      dI(t-1) = beta*I(t-1)*S(t-1) - (gamma + mu)*I(t-1);
       dR(t-1) = gamma*I(t-1) - mu*R(t-1);
```

```
35
36          % Apply changes
37           S(t) = S(t-1) + dS(t-1);
38          I(t) = I(t-1) + dI(t-1);
39          R(t) = R(t-1) + dR(t-1);
40      end
41
42      %% plot data
43      figure(1)
44      subplot(1, 2, 2)
45      hold on
46      plot(S,I)
47  end
48
49
50  %% Beautify plot
51  figure(1)
52  subplot(1, 2, 1)
53  hold on
54  plot(time, S, '-b')
55  hold on
56  plot(time, I, '-r')
57  plot(time, R, '-g')
58  title('Example of an endemic using a SIR model')
59  ylabel('Proportion of population $\frac{x}{N}$', 'interpreter','latex')
60  xlabel('time $t$', 'interpreter','latex')
61  legend('$S$','$I$','$R$', 'interpreter','latex')
62  xlim([0,500])
63
64  figure(1)
65  subplot(1, 2, 2)
66  plot([0:0.01:1], flip([0:0.01:1]), '--k')
67  xlim([0, 1])
68  ylim([0,1])
69  title('Steady State for Different Inital Conditions')
70  ylabel('Infected poulation $I(t)$', 'interpreter','latex')
71  xlabel('Susceptible Population $S(t)$', 'interpreter','latex')
```

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