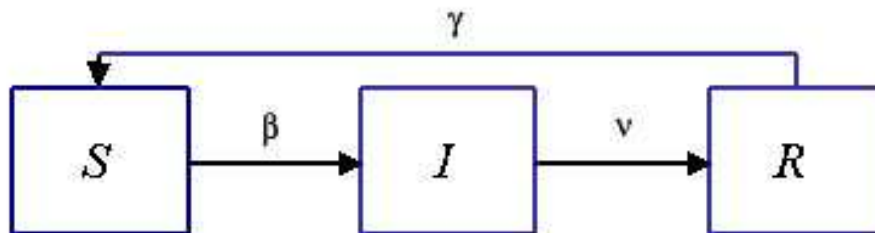


Epidemic Models

Modeling in the field of epidemiology has its roots in the early twentieth century. Sir Ronald Ross (1857-1932) studied the spread of malaria and developed the important idea that one didn't need to eradicate all mosquitoes to eliminate the disease. Modern epidemiology has its theoretical roots founded on modeling the spread of a disease and showing that if certain conditions are met, then a disease will go extinct.

This section examines infectious diseases caused by *microparasites*, such as bacteria and viruses, where the disease is transmitted from one host to another. This contrasts with the more complicated cycles of *macroparasitic* diseases, such as malaria, where intermediate vectors, like mosquitoes enter the dynamics of the model. We will examine simple mathematical models for infectious diseases such as measles, mumps, AIDS, chickenpox, and gonorrhea.

Some of the earliest mathematical models were developed by Kermack and McKendrick (1927). They examined a series of models based on healthy, infected, and immune individuals in a constant population (no births or deaths). These are called *SIR models without vital dynamics* (Hethcote, 1976).



Assume that we have a constant population, N , and that the population is divided into the three states, susceptibles S , infectives I , and removed or immune R . The first group are the individuals who are capable of becoming infected with a particular disease. The second group consists of individuals who are infected and can infect others. Sometimes these models include a class of exposed individuals, E , who are infected but cannot yet pass along the disease. Finally, the class R represents those who have recovered from the disease and are immune to infection. Using the diagram above, we can write the following system of differential equations:

$$\begin{aligned}\frac{dS}{dt} &= -\frac{\beta SI}{N} + \gamma R, \\ \frac{dI}{dt} &= \frac{\beta SI}{N} - \nu I, \\ \frac{dR}{dt} &= \nu I - \gamma R.\end{aligned}$$

The constant β represents the average number of contacts by an infected individual. The constants ν and γ represent the recovery rate and rate of loss of immunity, respectively.

SI Model

Consider the simplest of these models, where once someone is infected they remain infected.

This matches diseases such as genital herpes viruses. This model is written:

$$\begin{aligned}\frac{dS}{dt} &= -\frac{\beta SI}{N}, \\ \frac{dI}{dt} &= \frac{\beta SI}{N}.\end{aligned}$$

Since $N = S(t) + I(t)$, it follows that this model reduces to the simple model:

$$\frac{dI}{dt} = \beta I \left(1 - \frac{I}{N}\right),$$

which is simply the logistic growth model. It follows that the disease-free state ($S = N$ and $I = 0$) is unstable, and asymptotically, the entire population gets the disease ($I = N$ and $S = 0$). (This is like the carrying capacity for the logistic growth equation.)

SIS Model

There are several diseases, often caused by bacteria, that do not produce an immune response in the body, *e.g.*, gonorrhea or syphilis. These diseases, when given treatment leave the host unprotected, so the infected individuals return to the susceptible population. The simplest form of this model is given by:

$$\begin{aligned}\frac{dS}{dt} &= -\frac{\beta SI}{N} + \nu I, \\ \frac{dI}{dt} &= \frac{\beta SI}{N} - \nu I,\end{aligned}$$

where ν is the rate of treatment and $\frac{1}{\nu}$ is the average length of the disease in an individual. Again we let $S(t) = N - I(t)$, and the model above reduces to the first order differential equation:

$$\frac{dI}{dt} = (\beta - \nu)I \left(1 - \frac{\beta I}{(\beta - \nu)N}\right),$$

which is again a logistic growth equation. It follows that if $\beta > \nu$, then asymptotically the infected and susceptible populations go to:

$$\lim_{t \rightarrow \infty} I(t) = \frac{(\beta - \nu)N}{\beta} \quad \text{and} \quad \lim_{t \rightarrow \infty} S(t) = \frac{\nu N}{\beta}.$$

Thus, the disease remains endemic. However, if $\beta \leq \nu$, then the extinction equilibrium becomes stable and the disease dies out.

The ratio β/ν that appears in the SIS epidemic model is referred to as the *basic reproduction number* and is often denoted R_0 . This number relates the contact rate, β , to the cure rate, ν . Alternately, we see that R_0 represents the number of secondary infections caused by a single infected individual, β , during his/her infectious period, $1/\nu$. In the model above, we see that if $R_0 > 1$, then the disease is endemic, while if $R_0 \leq 1$, then the disease will go extinct.

SIR Model

Most viral diseases, such as measles or chickenpox, cause the body to mount an immune response. Once the body sees a particular disease, then a future infection is highly unlikely. For example, most people get chickenpox as children, but only rarely, do people get the disease

again in its more serious form, shingles. After a host becomes infected, then they develop a permanent immunity to the disease, R . The simplest form of this model is given by:

$$\begin{aligned}\frac{dS}{dt} &= -\frac{\beta SI}{N}, \\ \frac{dI}{dt} &= \frac{\beta SI}{N} - \nu I, \\ \frac{dR}{dt} &= \nu I.\end{aligned}$$

Notice that the differential equation in R is uncoupled from the other two equations. Also, we see that $\lim_{t \rightarrow \infty} I(t) = 0$, since the population has a fixed number, N , and R grows proportionally to I . It follows that the only equilibrium is $(S_e, 0, 0)$, where a certain number of susceptibles remain susceptible, depending on initial conditions.

If we consider the first two equations above, then we can compute the Jacobian matrix:

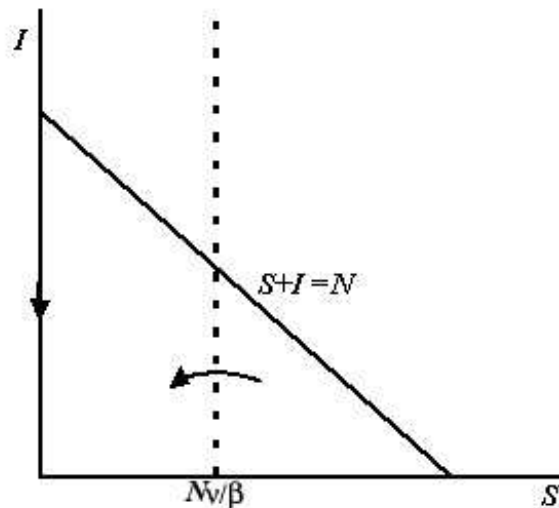
$$J(S, I) = \begin{pmatrix} -\frac{\beta I}{N} & -\frac{\beta S}{N} \\ \frac{\beta I}{N} & \frac{\beta S}{N} - \nu \end{pmatrix}.$$

It follows that the characteristic equation is

$$\lambda^2 - \left(\frac{\beta S_e}{N} - \nu\right) \lambda = 0,$$

so the eigenvalues are $\lambda_1 = 0$ and $\lambda_2 = \frac{\beta S_e}{N} - \nu$. Thus, the system is neutrally stable when $S_e = N\nu/\beta$. If $S_e > N\nu/\beta$ ($S_e < N\nu/\beta$), then $\lambda_2 > 0$ ($\lambda_2 < 0$) and the equilibrium, S_e , is unstable (stable).

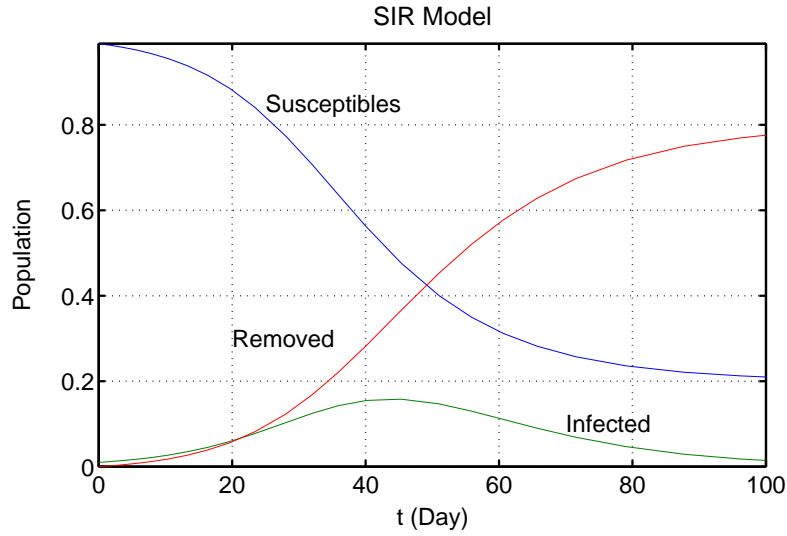
By using the assumption that $R(t) = N - S(t) - I(t)$, which implies $S(t) + I(t) \leq N$, we can draw the phase portrait for this system. The figure below shows the case when $R_0 = \frac{\beta}{\nu} > 1$.



In the figure above, we see that if most of the population is susceptible and a few infectives enter the population, then an epidemic will occur. The solution of the SIR model would begin in this case near the corner of the feasible region with $(S, I) = (N, 0)$, which has unstable equilibria. Thus, the solution initially increases until it crosses the line $S = N\nu/\beta$. Subsequently, the disease decreases, and the solution tends towards a stable equilibrium with $(S, I) = (S_e, 0)$ and $S_e < N\nu/\beta$.

If $R_0 = \frac{\beta}{\nu} < 1$, then the vertical line in the phase portrait is to the right of the feasible region ($S > N$), so all solutions tend to the stable equilibria along the S -axis. It follows that the disease can never become established.

Below is a figure showing a simulation of the model with $\beta = 0.2$ and $\nu = 0.1$. With $N = 1$ (normalized), the initial conditions given are $S(0) = 0.99$, $I(0) = 0.01$, and $R(0) = 0$, meaning that at the start of the simulation, 1% of the population is infected with the rest being susceptible. The simulation shows that the epidemic builds to where almost 15% of the population is infected shortly after 40 days, then the epidemic declines. In the end almost 80% of the population will have become infected and immune to any subsequent outbreak. About 20% of the population never gets the disease and remains susceptible to the infectious disease.



If we consider only the variables S and I , then we can combine the first two differential equations in the model to form the first order differential equation:

$$\frac{dI}{dS} = -1 + \frac{\nu N}{\beta S},$$

which is readily solved by integration to give

$$I(t) = N - R(0) - S(t) + \frac{\nu N}{\beta} \ln \left(\frac{S(t)}{S(0)} \right).$$

Since we know that the number of infected asymptotically approaches zero, it follows that the limiting value of $S(t)$ satisfies the implicit equation:

$$S(\infty) = N - R(0) + \frac{\nu N}{\beta} \ln \left(\frac{S(\infty)}{S(0)} \right).$$

The dynamics of the epidemic depends on the initial population of susceptibles, so an epidemic occurs only if

$$R = \frac{\beta S(0)}{\nu N} > 1.$$

This value of R is referred to as the *effective rate* (Anderson and May, 1991).

SIRS Model

Material for this type of model may be developed in the future.

Model for Gonorrhea

Gonorrhea ranks high among reportable communicable diseases in the United States. Public health officials estimate that more than 2,500,000 Americans contract the disease every year. This disease is spread by sexual contact and if untreated can result in blindness, sterility, arthritis, heart failure, and possibly death. Gonorrhea has a very short incubation time (3-7 days) and does not confer immunity to those individuals who have recovered from the disease. Thus, it is a classic example of an SIS model. It often causes itching and burning for males, particularly during urination, while it is often asymptomatic in females. Thus, males tend to seek treatment more often than females. Because the primary transmission is through sexual contact and mostly from heterosexual contacts, it follows that modeling is improved by dividing the population into males and females. Further divisions by sexual activity can also improve the model, but that is left as an exercise for the reader.

We will assume a sexually active heterosexual population with c_1 females and c_2 males. If the number of infective females is given by x and the number of infective males is given by y , then a mathematical model that describes this disease is given by the following system of differential equations:

$$\begin{aligned}\frac{dx}{dt} &= -a_1x + b_1(c_1 - x)y, \\ \frac{dy}{dt} &= -a_2y + b_2(c_2 - y)x.\end{aligned}$$

The cure rates for infective females and males are proportional to the infective populations with proportionality constants a_1 and a_2 , respectively. New infective females are added to the population at a rate proportional to the number of infective males and susceptible females, $b_1y(c_1 - x)$. (A similar term adds infectives to the male population.)

This model assumes a well-mixed steady population (no births or deaths) of equally sexually active people with exclusively heterosexual relationships. The group of people is closed with all people equally likely to encounter each other. The constant b_1/a_2 is the infective male contact rate. This is explained from the differential equation by noting that b_1 is the rate of contact by an infected male, while a_2 is the cure rate for a male, so the ratio provides the number of female contacts the average male has before becoming cured of the disease. Similarly, b_2/a_1 is the infective female contact rate.

[] Anderson, R. M. and May, R. M. (1991). *Infectious Diseases of Humans, Dynamics and Control*, Oxford University Press, Oxford, U.K.

[] Hethcote, H. W. (1976). Qualitative analyses of communicable disease models, *Math. Biosc.*, **28**, 335-356.

[] Kermack, W. O. and McKendrick, A. G. (1927). A contribution to the mathematical theory of epidemics, *Proc. Roy. Soc. Lond. A*, **115**, 700-721.