CHAPTER – 2

LITERATURE SURVEY

2.1 Some Mathematical Models

The increasing study of realistic and practically useful mathematical models in population biology is a reflection of their use in helping to understand the dynamic processes involved and in making practical predictions. In Continuous Growth Models, Single-species models are of relevance to laboratory studies in particular but, in the real world, can reflect a telescoping of effects which influence the population dynamics. Let \( N(t) \) be the population of the species at time \( t \), then the rate of change is a conservation equation for the population.

\[
\frac{dN}{dt} = \text{births} - \text{deaths} + \text{migration} \quad \cdots(2.1)
\]

The form of the various terms on the right hand side of the above equation necessitates modelling the situation with which we are concerned. The simplest model has no migration and the birth and death terms are proportional to \( N \).

That is,

\[
\frac{dN}{dt} = bN - dN \quad \cdots(2.2)
\]

\[\Rightarrow N(t) = N_0e^{(b-d)t} \quad \cdots(2.3)\]

where \( b, d \) are positive constants and the initial population \( N(0) = N_0 \). Thus if \( b > d \) the population grows exponentially while if \( b < d \) it dies out. This approach, due to Malthus (1798), is fairly unrealistic. One of the interesting aspects of Thucydides’ account is that there is no mention of person-to-person contagion which we now accept so freely with diseases. It was only in the 19\(^{th}\) century that it was beginning to be discussed. Evil exhalations from the earth, aerial miasmata and so on were generally accepted. Many South-East Asians can be forgiven for believing that the smog and smoke belching from
the forest fires in Indonesia are responsible for the large upsurge of dengue. One of the major epidemics in the U.S.A. was the Yellow Fever epidemic in Philadelphia in 1793 in which about 5000 people died out of a population of around 50,000, although estimates suggest that about 20,000 fled the city, from the interesting Scientific American article by Foster et al. (1998) and the book by Powell (1993). The landmark book by McNeill (1989) is a fascinating story of the relation between disease and people. The modelling literature is now extensive and growing very quickly. Although now quite old, a good introduction to the variety of problems and models for the spread and control of infectious diseases is the book by Bailey (1975). The article by Hethcote (1984) reviews three basic epidemiological models. The book by Diekmann and Heesterbeek (2000) is a good introduction to the field. For example, they discuss how to use biological assumptions in constructing models and present applications; they cover both deterministic and stochastic modelling. A few useful sources for the latest information on specific diseases, either globally or for a specific country, include the WHO and the CDC; their search and information features are very efficient.

Models can also be extremely useful in giving reasoned estimates for the level of vaccination for the control of directly transmitted infectious diseases. Mathematical modeling of infectious disease began in 1911 with Ross’s model (Ross 1911), and major extensions are described in a book (MacDonald 1957). SIR (Susceptible(S), Infection (I), and Recovery (R)) is one of the most basic epidemiological models from 1927. This is the most widely used model for the spread of disease. First, models were two-dimensional with one variable representing human and the other representing mosquitoes. An important addition to the above said models was the inclusion of acquired immunity proposed by Dietz et.al (1990). Aron & May (1983), Nedelman (1985), Anderson & May (1991) and Koella (2003) have written some good reviews on the mathematical modeling of infectious diseases. Some works by Chen & Wilson (2006), and Yang HM & Ferreira, MU (2000) have also included environmental effects for the spread and resistance to drugs and the evolution of immunity.

Susceptible humans, \([SS]_h\), can be infected when they are bitten by infectious mosquitoes. They then progress through the exposed, \([EX]_h\), infectious, \([IF]_h\), and recovered, \([RC]_h\), classes, before reentering the susceptible class. Susceptible mosquitoes, \([SS]_m\), can become infected when they bite infectious or recovered humans. The infected
mosquitoes then move to the exposed state \([EX]_m\) and then move to infectious state \([IF]_m\).

Both species follow a logistic population model, with humans having additional immigration and disease-induced death. Birth, death, and migration into and out of the population are not shown in the figure.

Fig. 2.1. SEIRS Model


### 2.2 Ngwa model

\[
\frac{dS_h}{dt} = g_h \, N_h + \gamma \, R_h + rI_h - (\mu' + \mu_h \, N_h) \, S_h - \left( \frac{C_m a I_m}{N_h} \right) \, S_h \quad \ldots(2.4)
\]

\[
\frac{dE_h}{dt} = \left( \frac{C_m a I_m}{N_h} \right) \, S_h - (\nu_h + \mu' + \mu_i \, N_h) \, E_h \quad \ldots(2.5)
\]

\[
\frac{dI_h}{dt} = \nu_h E_h - \left( r + q + \mu_d + \mu_i + \mu_i \, N_h \right) \, I_h \quad \ldots(2.6)
\]

\[
\frac{dR_h}{dt} = qI_h - (\gamma + \mu_i + \mu_i \, N_h) \, R_h \quad \ldots(2.7)
\]

\[
\frac{dS_m}{dt} = g_m \, N_m - (\mu'_m + \mu_m \, N_m) \, S_m - \left( \frac{C_m a I_m}{N_h} \right) \, S_m - \left( \frac{\bar{C}_m a R_m}{N_h} \right) \, S_m \quad \ldots(2.8)
\]
\[
\frac{dE_m}{dt} = \left( \frac{C_{hm} \alpha I_h}{N_h} \right) S_m + \left( \frac{\hat{C}_{hm} \alpha R_h}{N_h} \right) S_m - \left( \nu_m + \mu'_z + \mu_z N_m \right) E_m \quad \text{...}(2.9)
\]
\[
\frac{dI_m}{dt} = \nu_m E_m - \left( \mu'_z + \mu_z N_m \right) I_m \quad \text{...}(2.10)
\]

Where, \( g_h \) - birth rate of human

\( g_m \) - Birth rate of mosquito,

\( \gamma \) - Rate of loss of immunity

\( \nu_h \) - infectious rate from exposed class for human

\( \nu_m \) - Infectious rate from exposed class for mosquito

\( \mu'_1 \) - density independent death rate of human

\( \mu_1 \) - density dependent death rate of human

\( \mu_d \) - disease induced death rate of human

\( \mu'_z \) - density independent death rate of mosquito

\( \mu_z \) - density dependent death rate of mosquito

\( q \) - acquire immunity rate

\( C_{mh} \) - infectivity of mosquito

\( C_{hm} \) - infectivity of infected human

\( \hat{C}_{hm} \) - infectivity of immune human

\( a \) - biting rate of mosquito on human

\( r \) - average recovery rate of human from infectious to susceptible class

\( N_h \) - total number of human

\( N_m \) - total number of mosquito

The extension of the Ngwa model (2004) includes human immigration, excludes direct human recovery from the infectious to the susceptible class, and generalizes the mosquito biting rate so that it applies to wider ranges of populations. In Ngwa and Shu model (2000), the total number of mosquito bites on humans depends only on the number of mosquitoes, while in the proposed model, the total number of bites depends on both the human and mosquito population sizes. Human migration is present throughout the world and plays a large role in the epidemiology of diseases. In many parts of the developing world, there is rapid urbanization as many people leave rural areas and migrate to cities in...
search of employment. This movement can be included as a constant immigration rate into the susceptible class. Immigration of infectious humans is not included based on the assumption that most people who are sick will not travel. The direct infectious-to-susceptible recovery stated by the model of Ngwa and Shu (2000) is excluded. This is a realistic simplifying assumption because most people show some period of immunity before becoming susceptible again. As the proposed model includes an exponential distribution of movement from the recovered to the susceptible class, it ensures the quick return to susceptibility of some individuals.

2.3 Simple Epidemic Models

In the classical models the total population is taken to be constant. If a small group of infected individuals is introduced into a large population, a basic problem is to describe the spread of the infection within the population as a function of time. Of course this depends on a variety of circumstances, including the actual disease involved, but as a first attempt at modelling directly transmitted diseases some not unreasonable general assumptions can be made. Consider a disease which, after recovery, confers immunity which, if lethal, includes deaths: dead individuals are still counted. Suppose the disease is such that the population can be divided into three distinct classes: the susceptibles, $S$, who can catch the disease; the infectives, $I$, who have the disease and can transmit it; and the removed class, $R$, namely, those who have either had the disease, or are recovered, immune or isolated until recovered. The progress of individuals is schematically represented by $S \rightarrow I \rightarrow R$. Such models are often called SIR models. The number of classes depends on the disease. SI models, for example, have only susceptible and infected classes while SEIR models have a susceptible class, $S$, a class in which the disease is latent, $E$, an infectious class, $I$, and a recovered or dead class, $R$. The assumptions made about the transmission of the infection and incubation period are crucial in any model; these are reflected in the terms in the equations and the parameters. With $S(t)$, $I(t)$, and $R(t)$ as the number of individuals in each class assume here that:

(i) The gain in the infective class is at a rate proportional to the number of infectives and susceptibles, that is, $rSI$, where $r > 0$ is a constant parameter. The susceptible are lost at the same rate.
(ii) The rate of removal of infectives to the removed class is proportional to the number of infectives, that is, \(aI\) where \(a > 0\) is a constant; \(1/a\) is a measure of the time spent in the infectious state.

(iii) The incubation period is short enough to be negligible; that is, a susceptible who contracts the disease is infective right away.

The various classes are considered as uniformly mixed; that is, every pair of individuals has equal probability of coming into contact with one another. This is a major assumption and in many situations does not hold good as in most sexually transmitted diseases (STD’s). The model mechanism based on the above assumptions is then

\[
\frac{dS}{dt} = -rSI \\
\frac{dI}{dt} = rSI - aI \\
\frac{dR}{dt} = aI
\]

where \(r > 0\) is the infection rate and \(a > 0\) the removal rate of infectives. This is the classic Kermack-McKendrick (1927) model. Here, of course, only interested in nonnegative solutions for \(S\), \(I\) and \(R\). This is a basic model but, even so, some highly relevant general comments about epidemics can be made and, in fact, adequately describe some specific epidemics with such a model. The constant population size is built into the system (2.11)–(2.13) since, on adding the equations,

\[
\frac{dS}{dt} + \frac{dI}{dt} + \frac{dR}{dt} = 0
\]

\(S(t) + I(t) + R(t) = N\)

where \(N\) is the total size of the population. Thus, \(S\), \(I\) and \(R\) are all bounded above by \(N\). The mathematical formulation of the epidemic problem is completed given initial conditions such as

\(S(0) = S_0 > 0\), \(I(0) = I_0 > 0\), \(R(0) = 0\)

A key question in any epidemic situation is, given \(r\), \(a\), \(S_0\) and the initial number of infectives \(I_0\), whether the infection will spread or not, and if it does how it develops with time, and crucially when it will start to decline. From (2.12),

\[
\left[ \frac{dI}{dt} \right]_{r=0} = I_0 (rS_0 - a)
\]
\[
\begin{align*}
\left[ \frac{dI}{dt} \right]_{t=0} &= I_0 (rS_0 - a) > 0 \text{ if } S_0 > \rho \quad \text{and} \quad \left[ \frac{dI}{dt} \right]_{t=0} = I_0 (rS_0 - a) < 0 \text{ if } S_0 < \rho , \text{ where} \\
\rho &= \frac{a}{r} 
\end{align*}
\]

From 2.11,
\[
\frac{dS}{dt} \leq 0, \quad S \leq S_0 , \quad \text{we have if} \quad S_0 < \frac{a}{r}, \quad \text{then} \quad \left[ \frac{dI}{dt} \right] = I (rS - a) \quad \text{for all} \quad t \geq 0 \quad \cdots (2.18)
\]

in which case \( I_0 > I (t) \rightarrow 0 \) as \( t \rightarrow \infty \) and so the infection dies out; that is, no epidemic can occur. On the other hand if \( S_0 > \frac{a}{r} \) then \( I (t) \) initially increases and hence there is an epidemic. The term ‘epidemic’ means that \( I (t) > I_0 \) for some \( t > 0 \). Hence there is a threshold phenomenon. If \( S_0 > S_c = \frac{a}{r} \) there is an epidemic while if \( S_0 < S_c \) there is not.

The critical parameter \( \rho = \frac{a}{r} \) is sometimes called the relative removal rate and its reciprocal \( \sigma = \frac{r}{a} \) the infection’s contact rate.

The above diagram shows the phase trajectories in the susceptibles (S)-inffectives (I) phase plane for the SIR model epidemic system (2.11) - (2.13). The curves are determined by the initial conditions \( I (0) = I_0 \) and \( S (0) = S_0 \). With \( R (0) = 0 \), all trajectories start on the line \( S + I = N \) and remain within the right triangle since \( 0 < S + I < N \) for all time. An epidemic situation formally exists if \( I (t) > I_0 \) for any time \( t > 0 \); this
always occurs if \( S_0 > \frac{a}{r} \) and \( I_0 > 0 \). Now, \( R_0 \) can be written as, \( R_0 = \frac{rS_0}{a} \) where \( R_0 \) is the basic reproduction rate of the infection, that is, the number of secondary infections produced by one primary infection in a wholly susceptible population. Here \( \frac{1}{a} \) is the average infectious period. If more than one secondary infection is produced from one primary infection, that is, \( R_0 > 1 \), clearly an epidemic ensues. The whole question of thresholds in epidemics is obviously important. The definition and derivation or computation of the basic reproduction rate is crucial and can be quite complicated. One such example is the population being heterogeneous (Diekman et al., 1990). The basic reproduction rate is a crucial parameter grouping for dealing with an epidemic or simply a disease which is currently under control with vaccination, for example. Although the following arguments are based on \( R_0 \) they are quite general. Clearly one way to reduce the reproduction rate is to reduce the number of susceptible \( S_0 \).

Stochastic processes are ways of quantifying the dynamic relationships of sequences of random events. Stochastic models play an important role in elucidating many areas of the natural and engineering sciences. They can be used to analyze the variability inherent in biological and medical processes, to deal with uncertainties affecting managerial decisions and with the complexities of psychological and social interactions, and to provide new perspectives, methodology, models, and intuition to aid in other mathematical and statistical studies. A quantitative description of a natural phenomenon is called a mathematical model of that phenomenon. Examples abound, from the simple equation \( S = Zgt^2 \) describing the distance \( S \) traveled in time \( t \) by a falling object starting at rest to a complex computer program that simulates a biological population or a large industrial system. In the final analysis, a model is judged using a single, quite pragmatic, factor, the model's usefulness. The word "stochastic" derives itself from the Greek meaning to aim, to guess and implies "random" or "chance." The antonym is "sure," "deterministic," or "certain." A deterministic model predicts a single outcome from a given set of circumstances. A stochastic model predicts all possible outcomes by their likelihoods, or probabilities (Howard M. Taylor & Samuel Karlin 1998).