

Mathematics for Infectious Diseases; Deterministic Models: A Key

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ABSTRACT

The occurrence of infectious diseases was the principle reason for the demise of the ancient India. The main infectious diseases were smallpox, measles, influenza and typhus. There were also other diseases such as whooping cough, the mumps and diphtheria. It would be very difficult to obtain current information regarding important diseases, methods of transmission, methods of control and the likes. Since the wrong theories or knowledge have hindered advances in understanding. Therefore, this paper seeks to give a simple and clear description of mathematical models for infectious diseases. It has become important tools in understanding the fundamental mechanisms that drive the spread of infectious diseases.

Key words: Deterministic models, infectious diseases, Mathematical modeling.

1. INTRODUCTION

Since the spread of infectious disease has always been of big concerns and poses a threat to public health, as well as the economic and social developments to human society. Therefore its prevention and control become extremely important,[5].

As, it is quite obvious that human or animal invasions of new ecosystems, increased international travel, and changes in social and economic patterns will continue to provide opportunities for new and existing infectious diseases. Scientific experiments are usually the way to obtain information and to test hypotheses. Experiments in epidemiology are often difficult or impossible to design. Even if we are able to arrange an experiment there are serious ethical questions involved in withholding treatment from a control group. Sometimes data may be collected from reports of epidemics or of endemic disease levels, but they are often incomplete or inaccurate. Hence, parameter estimation and model fitting are very difficult, [2].

Still or perhaps because of this, ecological and public health challenges that infectious diseases present have been addressed with mathematical models.

Mathematical models have become important tools in understanding the fundamental mechanisms that drive the spread of infectious diseases and suggesting strategies for their control. This understanding the transmission characteristics of infectious diseases can lead to better approaches to reducing the transmission of these diseases, [9]. More specifically, mathematical models:

- i. Help clarify assumptions, variables, and parameters e.g. pathways involved in parasite spreading or degree of heterogeneity needed;
- ii. Provide conceptual results such as thresholds for disease invasion or plausibility of parasite eradication;
- iii. Can contribute to the design and analysis of epidemiological surveys, especially by suggesting crucial data that should be collected;

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- iv. Can be used as experimental tools for testing control measures and determining sensitivities to changes in parameter values;
- v. Can be used to compare and optimize costs and efficiency of various detection, prevention and control programs;

When parasites are used as control agents, models can provide qualitative insights into the circumstances under which parasites are capable of regulating their host population, and of doing so in an adequate and stable manner, [1].

2. CLASSIFICATION OF MATHEMATICAL MODELS

The methods to model an epidemic have to take in consideration its aims and stage of development which finally divide the model into the following categories, [11]:

2.1 Compartmental and Distributional Models

For the infection dynamics, from the total population, you can take at least two big groups of populations (as *variables*), a susceptible population and the population with the infection, in these models each one of these subpopulations are called *compartments* of susceptibles and infectives respectively.

But also in the infected compartment we could explore the distribution of severity of the infection in *distributional models*, for example during the incubation period of infection; in consequence the total infected population is the sum of those in all the different levels of severity.

2.2 Discrete and Continuous Models

Changes in population involved in a infection transmission model can take place either in discrete steps or as a smooth continuous process. In discrete models, difference equations reflect the change over the whole time step, whereas in continuous models, differential equations are developed to explore

changes in one variable with a diminishingly small change in another variable. For example sexual interactions allowing the spread of HIV occur continuously, so most models of HIV epidemic are continuous.

2.3 Deterministic Models

In a *deterministic* model events are not subject to chance and two realizations of a model using the same parameters and exact starting conditions will give exactly the same results. However, results can diverge in the case of deterministic chaos because it is impossible to exactly specify both starting conditions and the value of variables.

2.4 Stochastic Models

In *stochastic* models chance is taken into account. There are a number of ways to allow the events in a model to be influenced by chance, but the most common and rigorous method is Monte Carlo simulation, where the set of possible next events is defined with a probability attached to each. A random number generator is then used to calculate when the next event will occur and which of the range of possible events it will take place.

3. BASIC ASSUMPTIONS TO INFECTION MODELS

Many epidemiological models of infections use the conventional assumption that the host population is held constant, independent of the presence or absence of the infection, by an unspecified mechanism. This assumption stems from a history of medical interest in human diseases, predominantly in developed countries, where population densities do usually remain roughly constant on time scales appropriate to the operation of most diseases, [8].

On the other hand, densities of human populations in developing countries and most animal populations need to be treated as a dynamic variable. As expected, models with a variable host population size are often

more difficult to analyze because this additional variable requires an extra equation, [4].

Two main groups can be singled out among the deterministic models for the spread of infectious diseases which are transmitted through person-to-person contact. For infections, modelers distinguish several classes of hosts according to their status with respect to the disease:

Susceptible, S The portion of the population that has not been affected by the disease but may be infected in case of contact with a sick person.

Exposed, E Latent period of the disease; individuals are infected but not yet infectious and hence not yet able to pass the disease to the others. During this period the parasite reproduces rapidly within the host but its abundance is still too low for active transmission to other susceptible hosts.

Infectious, I Individuals already infected and who are also responsible for the transmission of the disease to the susceptible group.

Do not commute for infected individuals which are those who are either exposed or infectious, $E + I$.

Recovered, R Individuals recovered from the disease who have temporary or permanent immunity or, eventually, those who have died from the illness and not from other causes.

Not all epidemiological models will include all of these classes, but some will include more. In addition, many host populations are structured to various extents, so the models must divide the heterogeneous population to classes within which the individuals have similar characteristics. This division into groups can be based not only on the mode of transmission,

contact pattern, latent period, infectious period, genetic susceptibility or resistance, type and amount of control, but also on social, cultural, economic, demographic (age or sex), or geographic (spatial location) factors, [3].

We also stress here that the exposed and infectious periods cannot be mistaken with the incubation (or asymptomatic) and symptomatic periods, respectively. This is because one can transmit parasites long before becoming symptomatic, and one can still be symptomatic while no longer infectious.

The choice of which classes to include in a model depends on the characteristics of the modeled disease and the purpose of the model, [10].

The Common assumptions that are used in the formation of mathematical models for infection include the following

- a. The disease is transmitted by contact between an infected individual with susceptible individuals.
- b. The disease is transmitted instantaneously upon contact, and the population under consideration.
- c. All susceptible individuals are equally susceptible and all infected individuals are equally infectives.

4. THE MATHEMATICAL MODELS

During the development of epidemiology in the population, deterministic (compartmental) models played a central role. Such models divide the population into homogeneous sub-populations. An ordinary differential equation classically corresponds to each class which describes the rate of change in the size of individuals in the respective class as a result of all processes affecting this rate.

For a generic state variable, we may formally write:

$$\frac{dX}{dt} = \text{rates of all processes affecting } X$$

The Prior Deterministic Mathematical models are described as:

4.1 SIR - Mathematical Model

SIR models are a traditional point of departure in exploration of infection dynamics in epidemiology. SIR model is composed with the assumption that hosts are born as susceptibles, as

$$\frac{dS}{dt} = \text{rates of.....births - natural deaths - new infections}$$

$$\frac{dI}{dt} = \text{rates ofnew infections - natural deaths - disease - induced deaths - recovery}$$

$$\frac{dR}{dt} = \text{rates ofrecovery - natural deaths}$$

The SIR model is used when the disease under study confers permanent immunity to infected individuals after recovery or, in extreme cases, it kills them. After the contagious period, the infected individual recovers and is included in the R group. These models are suitable to describe the behavior of epidemics produced by virus agent diseases (measles, chickenpox, mumps, HIV, poliomyelitis), [7].

$$\begin{aligned}\frac{dS}{dt} &= -kSI + b(S + R) + pb'I - rS \\ \frac{dI}{dt} &= kSI + qb'I - (c + r')I \\ \frac{dR}{dt} &= cI - rR\end{aligned}$$

4.2 SIS- Mathematical Model

The SIS model assumes that the disease does not confer immunity to infected individuals after recovery. Thus, after the infective period, the infected individual recovers and is again included in the S group. Therefore, the model presents only two epidemiological compartments, S and I. This model is suitable to describe the behavior of epidemics

produced by bacterial agent diseases (meningitis, plague, venereal diseases) and by protozoan agent diseases (malaria), [7].

$$\begin{aligned}\frac{dS}{dt} &= -kSI + bS + \gamma I - rS \\ \frac{dI}{dt} &= kSI + b'I - (\gamma + r')I\end{aligned}$$

Particularly

SI- Mathematical Model

$$\begin{aligned}\frac{dS}{dt} &= -kSI + bS - rS \\ \frac{dI}{dt} &= kSI + b'I - r'I\end{aligned}$$

And

SIRS- Mathematical Model

$$\begin{aligned}\frac{dS}{dt} &= -kSI + b(S + R) + pb'I - rS + gR \\ \frac{dI}{dt} &= kSI + qb'I - (c + r')I \\ \frac{dR}{dt} &= cI - rR - gR\end{aligned}$$

3. SEIR- Mathematical Model

The exposed class E is often omitted as not crucial for the host-parasite interaction. Acronyms are often used to name epidemiological models, and these are based on the classes they contain and the flow patterns between these classes. So, for example, in the SEIR model, first become susceptible, then exposed, then infectious, and finally recover with permanent immunity.

$$\begin{aligned}\frac{dS}{dt} &= -kSI + b(S + E + R) + pb'I - rS \\ \frac{dE}{dt} &= kSI + q_1b'I - (v + r)E \\ \frac{dI}{dt} &= vE + q_2b'I - (c + r')I \\ \frac{dR}{dt} &= cI - rR\end{aligned}$$

Particularly

SEIRS -Mathematical Model

An SEIRS model is similar, but there is no passively immune class, and the immunity is only temporary so that recovered individuals regain their susceptibility after the temporary immunity fades away.

$$\frac{dS}{dt} = -kSI + b(S + E + R) + pb'I - rS + gR$$

$$\frac{dE}{dt} = kSI + q_1b'I - (v + r)E$$

$$\frac{dI}{dt} = vE + q_2b'I - (c + r')I$$

$$\frac{dR}{dt} = cI - rR - gR$$

With initial data

$$S(0) = S_0 > 0; E(0) = E_0 > 0; I(0) = I_0 > 0 \text{ and } R(0) = 0.$$

Here, followings are initial populations and infection related parameters defined.

S_0 = Population of susceptibles at initial state.

I_0 = Population of infectives at initial state.

E_0 = Population of exposed individuals at initial state.

k = The transmission rate of infection from infectives to susceptibles.

b = The birth rate of susceptible, removed, exposed individuals.

r = The death rate of susceptible, exposed and removed individuals.

b' = The birth rate of infective individuals.

r' = The death rate of infected individuals.

v = The rate at which exposed individuals pass from latent to the Infective Class.

c = The cure rate of infectives.

g = The rate at which removed individuals loose their immunity.

γ = The rate at which infective hosts loose their temporary immunity.

p = The fraction of offsprings of infective parents, who are susceptible at birth.

q = The fraction of offsprings of infective parents, who are infective at birth.

q_1 = The fraction of offspring of infective parents who are in latent period.

q_2 = The fraction of offsprings of infective parents who are infective at birth.

$$1 = p + q \text{ and } 1 = p + q_1 + q_2$$

5. REMARK

From the above, it is observed that the modeling of infectious diseases has shown rich dynamic behavior and phenomena. The much understanding has been gained through the use of relative simple models capturing only the most critical biological mechanisms. The infection dynamics can employ well developed modern dynamics theory to better characterize the inherent patterns and also to investigate long term behavior of infection transmissions. Further the analysis of model parameters can help us to make more realistic simulations and reliable transmission prediction which may not be feasible by experiments or field studies.

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