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Mathematical Modeling and Role of Dynamics in Epidemiology

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Abstract

This study aims at providing the Considerable role of correlation of mathematical modeling and dynamical aspects of some epidemic diseases. This study emphasizes an understanding of deterministic modelling applied to the population dynamics of infection diseases. Here we are mainly emphasizing the historical background of mathematical modelling and role of dynamics in different infection diseases such as measles, AIDS, Cholera, Plague, Malaria, T.B., and Dengue etc. Our investigation is focusing on historical aspects of bioepidemiological mathematical survey.

Keyword: Mathematical modelling, Epidemic disease, Biomathematical aspects, Dynamics.

1. Introduction

The field of infectious diseases is ever long been concerned with epidemiological aspects and considerable with correlation of biomathematical historical background. Infection diseases have been a great concern of human kind since the very beginning of our history. At present, we still have a deal with some infectious diseases i.e. measles, AIDS, Plague, Malaria, T.B., Dengue etc. Millions of people die annually from above mentioned diseases and billions of other is infected. There was a belief since last from previous time that infection disease would be soon eliminated with the improvement in antibiotics, vaccinations, medical science and medical care. Together with the threat of biological weapon, whose research is lately concerned about microorganisms and lethal infectious disease and their transmission characteristics.

The spread of infection diseases has always been of concerns and a threat to public health [22, 23, 24].

2. Historical Background of Mathematical Modeling in Epidemiology

The historical aspects of epidemiological mathematical modeling were initiated from records of historians and scholars are the Plague of Athens (430-428 BC). The most precise description is provided by the scientific historian – Thucydides – (460-400 BC) including the symptoms, disease progression and number of death. Hippocrates's (459-337 BC) work, "On the epidemics", tells us about the factors which were affecting the disease spreading and ways of the spreading at that time.

Epidemics which were killing in millions occurred in 14^{th} century when 25 million people died in Europe due to Bubonic Plague. The Antonina Plague (165-180 AD) was and ancient pandemic, either of smallpox or measles, brought back to the Roman Empire by troops returning from campaigns in the Near-East. The epidemic invaded the Roman Empire, claimed the lives of two Roman emperors and caused drastic population reduction and economic hardships which led to disintegration of the empire because of disorganization that facilitated invasions of barbarians [25]. In the early 1500s smallpox was introduced into the Caribbean by the Spanish armies led by Cortez, from were it spread to Mexico, Peru and Brazil. The population of Mexico was reduced from up to 30 million to less than 2 million during a period of 50 years after the Spanish invasion, smallpox being the principal cause of death [4].

While mathematical modeling of infectious diseases can be traced back to 1760 when Bernoulli used mathematical models for smallpox [3], the research on infectious diseases, using deterministic mathematical models, actually began in the 20th century. Hamer formulated a discrete-time model for the spread of measles in 1906. A physician, Dr. Ross, used a differential equation model to describe the transmissions of malaria between human beings and mosquitoes in 1911, and determined that there exists a threshold of the size of mosquitoes below which the spread of malaria can be controlled. It was because of his outstanding contributions in the research of the transmission dynamics of malaria Dr. Ross was awarded his second Nobel Prize in medicine. Kermack and McKendrick formulated a well-known and well-recognized SIR (susceptible-infective–recovered) compartmental model, in 1926, to study the outbreak of Black Death in London during the period of 1665–1666, and the outbreak of plague in Mumbai in 1906.

In middle 1800's Louis Pasteur confirmed experimentally the germ theory of disease and he created the first vaccine for rabies. At the same time, Robert became famous for the discovery of the anthrax bacillus (1877), the tuberculosis bacillus (1882) and the cholera vibrio (1883) and for his development of Koch's postulates. Diseases were no more punishment of gods or some kind of witchcraft. The science could explain "why" and mathematics could explain "how". Pragmatic approaches were limited and there was appropriate theory to explain the mechanism by which epidemics spread. The idea of passing on a bacterial disease through contact between an infected and healthy individual became familiar.

More developments and progresses have been particularly made during the past 20 years. Massive mathematical models have been formulated and developed to study various infectious diseases, ranging from more theoretic, general ones Waltman [20]; Burnett and White [6]; Hoppensteadt [17]; Frauenthal [14]; Anderson and May[1]; Evans[13]; Busenberg and Cooke[8]; Capasso[9]; Isham and Medley[19]; Daley and Gani[11]; Diekman and Heesterbeek [12] to more specific ones especially for measles, malaria, tuberculosis, sexually transmitted diseases (STD), or AID/HIV [4,5,15,16,18,]. The modeling of infectious diseases has shown rich dynamic behavior and phenomena.

In india the drastic effects of epidemic disease were remarkable in the field of epidemiology. Human viruses in ancient Indian literature such as the Rigveda (c. 8000 BC), Charaka Sahara (c. 700 BC) and several other Ayurvedic texts until 1600 AD, Puranas (c. 200 BC to 750 AD), travel accounts of visitors to India, and some British records.

3. Spread of Disease in India

In India, the drastic effects of epidemic disease were remarkable in the field of epidemiology. Overflood of population is a major cause of spread of any disease. In the 1891census the population of Bombay was counted to be 820,000. Most of the immigrant workers lived in chawls. The cities of services were not geared toward the well being of this part of the population and various diseases were endemic to the slums.

In September 1896 the first case of Bubonic plague was detected in Mandvi by Dr. Acacio Gabriel Viegas , the death toll was estimated at 1,900 people per week through the rest of the year. Many people fled from Bombay at this time, and in the census of 1901, the population had actually fallen to 780,000. On 9 December 1898 the Bombay City Improvement Trust was created by an act of the British Parliament. It was entrusted with the job of creating a healthier city. In the first year of the plague, a research laboratory was set up at the JJ Hospital. It moved in 1899 to the Government House in Parel under the directorship of Haffkine. This was the beginning of the Haffkine Institute. In 1900, the mortality rate from plague was about 22 per thousand. In the same year, the corresponding rates from tuberculosis were 12 per thousand, from cholera about 14 per thousand, and about 22 per thousand from what were classified as "fevers". The plague was fearsome only because it was contagious. More mundane diseases took a larger toll.

In 1974 smallpox epidemic of India was one of the worst smallpox epidemics of 20th century. At least 15,000 people died of smallpox between January to May 1974, mainly in the Indian states of Bihar, Orissa and West Bengal. There were thousands who survived but were disfigured or blinded. India reported 61,482 cases of smallpox to WHO in these five months. India had over 86% of the world's smallpox cases in 1974.Donald Henderson, who was a U.S. Public Health Services Officer stationed in New Delhi, said that by June of 1975, we hope we will be finished with smallpox in Asia according to WHO smallpox eradicated program.

In 1994, total 693 suspected bubonic or pneumonic plague cases were reported to WHO by Government of India. These cases were from Maharashtra, Gujarat, Karnataka, MadhyaPradesh, Uttar Pradesh and New Delhi. Positive laboratory test results for Yersinia pestis were reported by India. The outbreak of suspected plague disease lasted from August 26, 1994 to October 18, 1994.

In 1994, there was a pneumonic plague epidemic in Surat, India that resulted in 52 deaths and in a large internal migration of about 300,000 residents, who fled fearing quarantine.

The early 2009 Gujarat hepatitis B outbreak is a hepatitis-B epidemic that spread in Modasa, northern Gujarat, India. Over 125 people were infected and up to 49 people were killed in the epidemic.

4. Role of Dynamics in Epidemiology

Epidemic dynamics is an important method of studying the spread of infection disease. It is based on the specific property of population growth, spread rule of infection disease, and the related social factors etc. To construct mathematical models reflecting the dynamic properties of infection disease, to analyze the dynamical behavior and to do some simulations. The research result is helpful to predict the growth of infection disease, to determine the key factors of the spread of infection disease and to seek the optimum strategies of preventing and controlling the spread of infection diseases.

4.1 Compartmental Models in Epidemiology.

The dynamic models for infectious disease are mostly based on their compartment structure. The compartment structures for dynamic models are firstly given by Kermack and Mckendrick in 1927 and are developed by many other biomathematicians in 1932.

For viral disease like influenza, measles, swine flu, and chikengunia, the recovered individuals gain immunity to the same virus. For these diseases the SIR model is applicable. Moreover, for Bacterial diseases like gonorrhea, Bubonic Plague, Tuberculosis, Syphilis, etc, the recovered individuals gain no immunity and can reinfected. To study the dynamics if these diseases the SIS model is applicable.

4.1.1 Fundamental forms of Compartmental Models.

(1) Models without latent periods.

In these models the infected individuals becomes infectious immediately. These models are as follows:

(2) SI Model.

In this model, the infectives cannot be recovered from infection. It is represented by following diagram:

The model equations are as follows:

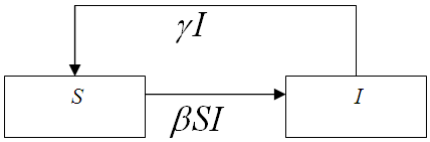
$$\frac{dS}{dt} = -\beta SI$$

And

$$\frac{dI}{dt} = \beta SI$$

(3) SIS Model

In this model, the infectives are recovered but gain no immunity from infection. It is represented by following diagram:



The model equations are as follows:

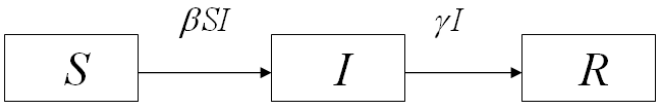
$$\frac{dS}{dt} = -\beta SI + \gamma I$$

And

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

(4) SIR Model.

In this model, the infectives obtain permanent immunity to the disease after recovered from infection. It is represented by following diagram:



The model equations are as follows:

$$\frac{dS}{dt} = -\beta SI$$

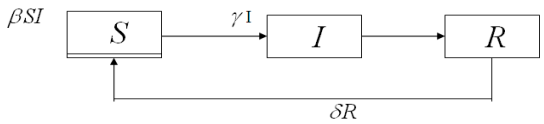
$$\frac{dI}{dt} = \beta SI - \gamma I = \beta I(S - \rho)$$

where $\rho = \frac{\gamma}{\beta}$

And $\frac{dR}{dt} = \gamma I$

(5) SIRS Model.

In this model, the recovered individuals have only temporary immunity after they recovered from infection. It is represented by following diagram:



The model equations are as follows:

$$\frac{dS}{dt} = -\beta SI + \delta R$$

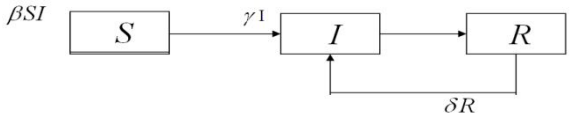
$$\frac{dI}{dt} = \beta SI - \gamma I = \beta I(S - \rho)$$

where $\rho = \frac{\gamma}{\beta}$

And $\frac{dR}{dt} = \gamma I - \delta R$

(6) SIRI Model.

In this model, the infectives cannot obtain permanent immunity to the disease after recovered from infection. It is represented by following diagram:



The model equations are as follows:

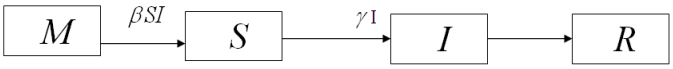
$$\frac{dS}{dt} = -\beta SI$$

$$\frac{dI}{dt} = \beta SI - \gamma I + \delta R = \beta I(S - \rho) + \delta R \text{ ,where } \rho = \frac{\gamma}{\beta}$$

And $\frac{dR}{dt} = \gamma I - \delta R$

(7) MSIR Model

For many infections, including measles, babies are not born into the susceptible compartment but are immune to the disease for the first few months of life due to protection from maternal antibodies (passed across the placenta or through colostrum). This added detail can be shown by including an M class (for maternally derived immunity) at the beginning of the model. It is represented by following diagram:

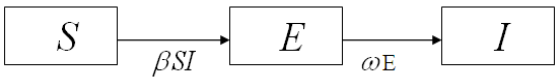


4.2 Models with latent periods

For many important infections there is a significant period of time during which the individual has been infected but is not yet infectious themselves. During this latent period the individual is in compartment (E) exposed compartment. These models are as follows:

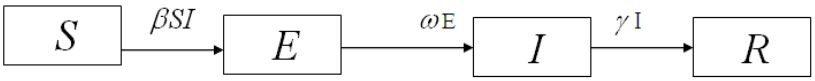
1) SEI Model.

This model is represented by following diagram:



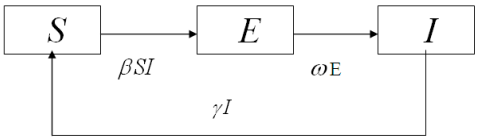
2) SEIR Model.

In this model the population is broken into four compartments: susceptible, exposed, infectious and recovered. This model is represented by following diagram:



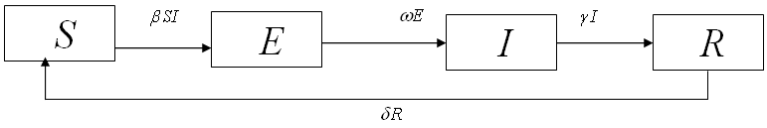
3) SEIS Model.

In this model the population is broken into four compartments: susceptible, exposed, and infectious again susceptible. This model is represented by following diagram:



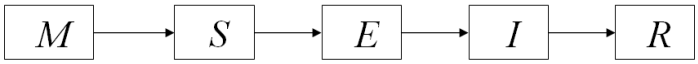
4) SEIRS Model.

In this model the population is broken into five compartments: susceptible, exposed, infectious, recovered and again susceptible. This model is represented by following diagram:



5) MSEIR Model

For the case of a disease, with the factors of passive immunity, and latency period there is the MSEIR model are used for epidemiological classes. This model is represented by following diagram:



Where the symbol stands for
M = Births and passive immunity
S = susceptible class
E = Exposed class
I = Infective class
R = Recovered class
γ = Recovery rate
β = Transmission rate
ω = Progression rate
δ = Immunity rate

4.1.2 Basic Concepts of Epidemiologic dynamics.

We often come across the terms like contact rate, adequate contact rate, infection rate, simple mass action incidence, standard incidence, saturation incidence, basic reproduction number, threshold numbers etc whose definitions is as follows:

An infectious disease transmitted through direct contacts. The number of individuals contacted by an infectives per unit of time is called a **contact rate** of infection and is denoted by P(N). It is depends on the total population N. If the individuals contacted by an infectives are susceptible, they may be infected. Suppose that the probability of infection by each contact is β_0. Then the function β_0 N is called an **adequate contact rate**, which describes the infection strength of the infectives and is usually depends on the toxicity of the virus or bacteria and the situation of the environment. Since disease are only transmitted to susceptible by contacting with infectives and the fraction of the susceptible with the population is $\frac{S}{N}$, then the **mean adequate contact rate** is $\beta_0 P(N)\frac{S}{N}$. This rate is called an **infection rate**. Then the total new infectives in the infected compartment is $\beta_0 P(N)\frac{SI}{N}$, which is called an **incidence** of the disease. There are three types of incidence are used in disease modelling:

1. If the contact rate is proportional to the total population size i.e. P(N)=kN then the incidence βSI, where $\beta = \beta_0 k$ is called the transmission coefficient. This type of incidence is called **bilinear incidence or simple mass action incidence.**

2. If the contact rate is constant i.e. P(N)=k then the incidence $\beta SI/N$, where $\beta = \beta_0 k'$, then it is called the **standard incidence.**

If the number of susceptible is large compared to H, then the incidence $\beta \frac{SI}{H+S}$, where H is constant, is called the **saturation incidence.**

A **basic reproduction number** is the number of secondary cases produced in a totally susceptible population by a single infective individual during the time span of infection. **Thresholds** are also numbers which are capable of forecasting either the disease persists or not.

Conclusion

This study emphasizes an understanding of deterministic modelling applied to the population dynamics of infection diseases and the role of dynamics in different infection diseases such as measles, AIDS, Cholera, Plague, Malaria, T.B., and Dengue etc. Our investigation is focusing on historical aspects of bioepidemiological mathematical survey. This study also provides the Considerable role of Correlation of mathematical modelling and dynamical aspects of some epidemic diseases.

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