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Role of Mathematical Modeling To Study the Dynamics of Malarial Epidemiology

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Abstract:
Infection and vector born diseases have been a great concern of human kind since the very beginning of our history. Mosquito, a creature that causes human deaths more than other creatures in the world, spread lethal diseases called Mosquito-Borne Diseases (MBDs). Mosquito-borne diseases have been the cause of concern for the whole world for centuries. In India, malaria and dengue are the center of focus as confirmed cases of these diseases are more than the other MBDs. According to the National Center for Vector-Borne Diseases (NCVBD), India has witnessed over 18 lakh confirmed cases and over 2500 deaths since 2019 caused by mosquitoes, approximately11% of confirmed cases and 9% of deaths were from Uttar Pradesh. This study aims at providing the Considerable role of correlation of mathematical modeling and dynamical aspects of epidemic disease. This study emphasizes an understanding of deterministic modelling applied to the population dynamics of infection disease. Here we are mainly emphasizing the historical background of mathematical modelling and role of dynamics in Malaria.

Keywords: Mathematical modeling, Mosquito-Borne Diseases (MBDs), Malaria, Epidemic disease, Bio-mathematical aspects, Dynamics.

Introduction:

The field of infectious diseases is ever long been concerned with epidemiological aspects and considerable with correlation of bio-mathematical historical background [1,2,3]. The spread of infection diseases has always been of concerns and a threat to public health.

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. 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. 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. Massive mathematical models have been formulated and developed to study various infectious diseases. 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.

In India, malaria affects more than a million people annually, a figure that amounts to about 4% of the global malaria burden (World Health Organization, 2018). With its extensive geographic and climatic diversity, the epidemiology of malaria ranges from endemic areas with perennial transmission to outbreak-prone, unstable areas. The situation is further complicated due to the presence of a wide distribution of anopheline vectors transmitting three major *Plasmodium* species: *Plasmodium falciparum*, *Plasmodium vivax*, and *Plasmodium malariae* (14). Though the share of *P. falciparum* (66%) is more than *P. vivax* (34%) in the country, about 48% of the estimated global *vivax* malaria cases in 2017 occurred in India (24).

Malaria is a deadly mosquito-borne disease caused by Plasmodium parasites transmitted by Anopheles female mosquitoes between humans. Once an infected mosquito bites a human, the parasites multiply in the host's liver, destroying red blood cells during infection [6]. Parasites that infect humans are Plasmodium falciparum (P. falciparum), Plasmodium vivax (P. vivax), Plasmodium ovale (P. ovale), Plasmodium knowlesi (P. knowlesi) and Plasmodium malariae (P. malariae) [11, 4]. P. falciparum and P. vivax are unicellular protozoan parasites of humans, and are the most important Plasmodium species causing malaria in humans [11, 5,7.8]]. There are 58 species of Indian anophelines out of which six—Anopheles culicifacies, An. fluviatilis, An. stephensi, The WHO has set an ambitious goal of malaria elimination in 35 countries and at least a 90% reduction in malaria cases by 2030. Within the WHO Southeast Asia region, India remains the leading contributor to the malaria burden, with 79% of cases and 83% of total malaria deaths. Under the auspices of the Global Technical Strategy, adopted by the World Health Assembly in May 2015[24,25], India launched its malaria elimination program in 2016 under the National Framework for Malaria Elimination in India 2016–2030[15].
In this study, we have derived and analyzed a mathematical model in order to better understand the transmission and spread of the malaria disease, and tried to suggest possible ways for its prevention and control.

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. [17, 18,19]. To construct mathematical models reflecting the dynamic properties of infection disease, to analyze the dynamical behavior and to do some simulations [19,20,21,22,23]. 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.

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

$$S \xrightarrow{\beta SI} I$$

The model equations are as follows:

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

and

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

(3) SIS Model
In this model, the infective are recovered but gain no immunity from infection. It is represented by following diagram:

$$S \xrightarrow[\beta SI]{\gamma I} I$$

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:

$$S \xrightarrow{\beta SI} I \xrightarrow{\gamma I} R$$

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:

$$\beta SI \quad S \xrightarrow{\gamma I} I \longrightarrow R$$

The model equations are as follows:

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

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

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:

$$\beta SI \quad S \xrightarrow{\gamma I} I \longrightarrow R$$

The model equations are as follows:

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

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

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:

$$M \xrightarrow{\beta SI} S \xrightarrow{\gamma I} I \longrightarrow R$$

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:

$$S \xrightarrow[\beta SI]{} E \xrightarrow[\omega E]{} I$$

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:

$$S \xrightarrow{\beta SI} E \xrightarrow{\omega E} I \xrightarrow{\gamma I} R$$

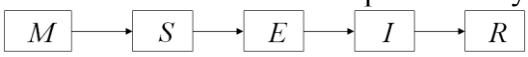
3) SEIS Model.
In this model the population is broken into four compartments: susceptible, exposed, and infectious again susceptible. This model is represented by following diagram:

$$S \xrightarrow[\beta SI]{} E \xrightarrow[\omega E]{} I$$

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:

$$S \xrightarrow{\beta SI} E \xrightarrow{\omega E} I \xrightarrow{\gamma I} R$$

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 symbols stands for -

M = Births and passive immunity	R = Recovered class
S = Susceptible class	γ = Recovery rate
E = Exposed Class	β = Transmission rate
I = Infective class	ω = Progression rate
	δ = Immunity rate

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

$$\beta_0 P(N) \frac{SI}{N}$$, 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.

$$P(N) = kN \text{ then the incidence } \beta SI,$$

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 $\frac{\beta SI}{N}$, where $\beta = \beta_0 k'$, then it is called the **standard incidence.**

   If the constant i.e. $P(N) = k$ 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 Malaria. 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. Mathematically we recommend the use of higher and more compartments in the modelling of diseases by future researchers in order to be able to capture the complex interactions amongst the human and vector compartments more extensively.

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