Vector-Host Model of Multi-Mediated Infectious

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ABSTRACT. There have been many outbreaks of infectious diseases in the history. Each outbreak had a great impact on public health and industrial production, which changes the progress of human history. Mathematical models can well describe the dynamic behavior of diseases. Through numerical modeling, studying the relationship between various parameters could help government to come up with more effective control measures that would mitigate the risk of diseases. By studying the impact of multi-media transmission mechanism of infectious diseases and constructing a vector host model under multiple vectors, this paper establishes a vector-borne infectious disease model and obtains the expression of basic regeneration number. In addition, this paper also discussed the existence and stability of the equilibrium point, and theoretically analyzes the dynamic model.

Keywords: Infectious Disease; Multimedia; Stability

1   Introduction

Infectious diseases have always been a major problem plaguing people in the world. The spread of infectious diseases brings great harm to public health and impacts industrial productions. Since the 21st century, various new infectious diseases emerged one after another and continue to ravage around the world. In particular, the outbreak of COVID-19 pandemic in 2019 has once again made people realize the dangers of infectious diseases. In order to control the spread of the epidemic, countries around the world have invested a lot of human, material and financial resources, and taken many measures, but at present, they are still unable to fully control the spread of the epidemic. Therefore, how to effectively control the spread of infectious diseases is of great significance.

Mathematical models play an important role in controlling the spread of infectious diseases, analysing the complex transmission patterns of infectious diseases, and evaluating the public health. The earliest mathematical model of infectious diseases was carried out by public health doctors. The first practice under the guidance of mathematical epidemiology was Daniel Bernouilli's vaccination against smallpox in 1760 [1]. A complete theoretical system was formed in 1911. Ross proposed a mathematical model of malaria to study the law of transmission between malaria and mosquitoes [2]. In 1927, Kermack and Mckendrick first proposed the infectious disease cabin model, built

the SIS infectious disease model in 1932, and put forward the concept of infectious disease threshold in 1933, laying the foundation for the study of the mathematical model of infectious diseases [3-5]. A large number of research on infectious disease models has been carried out [6-10]. Van den Driessche and Watmough gave a simple and feasible basic regenerative number calculation method in 2002 [11], which became one of the important cornerstones for the dynamic analysis of infectious diseases.In the past 20 years, international research on the dynamics of infectious diseases has made great progress, and mathematical models are increasingly used in infectious disease research. The establishment and research of these models provide a theoretical basis for the effective prevention and control of infectious diseases.

Most of the existing infectious disease models target specific infectious diseases and analyse a single vector involved in diseases. However, many infectious diseases may not have single vector, such as plague, that can be transmitted by flea bites or through contact with infected animals. Epidemic animals may be marmots, rabbits, Tibetan sheep, etc. For example, schistosomiasis, cattle and pigs are the main vectors in watery places, and in hilly areas, wild animals and mice are the main vectors. There are multiple vectors in these infectious diseases, so it is necessary to study the impact of multiple vectors on the spread of infectious diseases.

This paper builds a new mathematical model based on multi-media transmission mechanism, and focuses on the impact of multiple vector transmission factors on the spread of infectious diseases. For certain types of infectious diseases, the population is divided into two categories: susceptible S and infected person I. Media is divided into vector 1 and vector 2, vector 1 is divided into susceptible person A, infected person B, media 2 is divided into susceptible person M, infected person V. Assuming that the birth mortality rates of the population, media 1 and media 2 are the same, respectively b, d and c. By establishing a multimedia propagation model, the expression of the basic regenerative number of this model is obtained, the existence and stability of the equilibrium point of the model are discussed, and the established dynamic model is theoretically analyzed.

2   PRELIMINARIES

2.1   The SIR model

In 1927, Kermack and Mckendrick established a model of the spread of SIR infectious diseases [3]. Assuming that the total number of the population remains unchanged, denoted by the contant N, the total number of the population can be divided into three groups at the time t, the susceptible S(t), the infectious I(t) and the recovered R(t). Clearly, N=S(t)+I(t)+R(t). Secondly, if each infected person I infects a susceptible person S with a chance β, then the number of susceptible patients who eventually are infected is βSI. Finally, the cure rate of infected person is δ.

The corresponding system of differential equations is:

$$\frac{dS(t)}{dt} = -\beta S(t)I(t) \tag{1}$$

$$\frac{dI(t)}{dt} = \beta S(t)I(t) - \delta I(t) \tag{2}$$

$$\frac{dR(t)}{dt} = \delta I(t) \tag{3}$$

On this basis, in 1932, Kermack et al. built a SIS infectious disease model for diseases without immunity [4].

The corresponding ordinary differential equations are:

$$\frac{dS(t)}{dt} = -\beta S(t)I(t) + \delta I(t) \tag{4}$$

$$\frac{dI(t)}{dt} = \beta S(t)I(t) - \delta I(t) \tag{5}$$

And by analyzing the infectious disease model, we creatively put forward the "threshold theory" to judge whether the disease can be epidemic. This important threshold is the basic regeneration number R. If R is greater than 1, the disease will always exist. If R is less than 1, the disease will gradually disappear.

2.2   The SEIR model

For many important infections, there is a significant incubation period, during which the individual has been infected but has not yet been infected. During this period, the individual is in cabin E (for exposure).

Assuming that the incubation period is a random variable with an exponential distribution,the parameter is a(that is, the average incubation period is a^{-1}), we have a model:

$$\frac{dS}{dt} = \mu N - \mu S - \frac{\beta IS}{N} \tag{6}$$

$$\frac{dE}{dt} = \frac{\beta IS}{N} - (\mu + a)E \tag{7}$$

$$\frac{dI}{dt} = aE - (\gamma + \mu)I \tag{8}$$

$$\frac{dR}{dt} = \gamma I - \mu R \tag{9}$$

3   The model

Most of the existing models of infectious diseases such as SIR and SEIR are based on the research and analysis of the single vector involved in specific infectious diseases. However, there are many infectious diseases that have multiple vectors, so it is necessary to study the impact of multiple vectors on the spread of infectious diseases. This paper builds a mathematical model based on the situation of multiple media transmission, and focuses on the impact of multiple media transmission factors on the transmission of infectious diseases. For certain types of infectious diseases, the population is divided into two categories: susceptible S and infected I. The media is divided into

media 1 and media 2. Media 1 is divided into susceptible A, infected B, media 2 is divided into susceptible M, infected V. Assuming that the birth mortality rates of the population, media 1 and media 2 are the same, which are b, d and c respectively.$\beta_{SV}, \beta_{SI}, \beta_{SB}$ are the coefficients of infected vector 2, infected person I, and infected vector 1. $\beta_{AV}, \beta_{AI}, \beta_{AB}$ are the coefficients of infected vector 2, infected person I, and infected vector 1. $\beta_{MI}I, \beta_{MV}$ are the coefficients of infected with susceptible vector 2 infected people I and infected vector 2. γ is the rate of infected people I recovering to susceptible people S. The model established in this article is as follows:
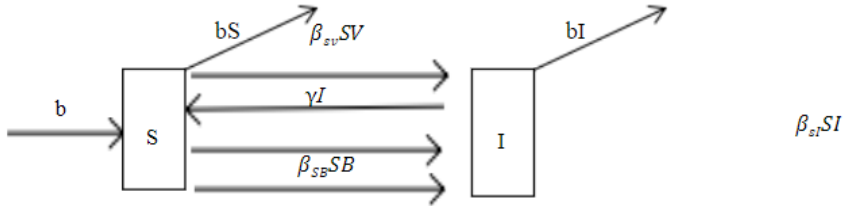

Fig. 1. Progression of susceptible person (S) into infected person(I)

As shown in Figure 1 the probability of susceptible person S is infected by infected V is β_{sv} and among susceptible person S The recovery rate of infected person I is γ, and among infected person I, γI of them are restored to susceptible person S.The probability of susceptible person S is infected by infected person I is β_{sI} and among susceptible person S, $\beta_{sI}SI$ of them transform into infected person I. The probability of susceptible person S is infected by infected B is - and $\beta_{SB}SB$. bS of susceptible person S die and bI of infected person die.
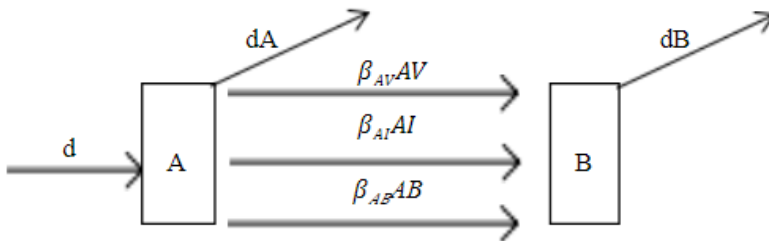

Fig. 2. Progression of susceptible A into infected B

As shown in Figure 2 the susceptible A have a chance of β_{AV} to be infected by infected V, so $\beta_{AV}AV$ of susceptible A transform into infected B. The susceptible A have a chance of β_{AI} to be infected by infected person I, so $\beta_{AI}AI$ of susceptible A transform into infected B. The susceptible A have a chance of β_{AB} to be infected by infected B, so $\beta_{AB}AB$ of susceptible A transform into infected B. dA of susceptible A die and dB of infected B die.
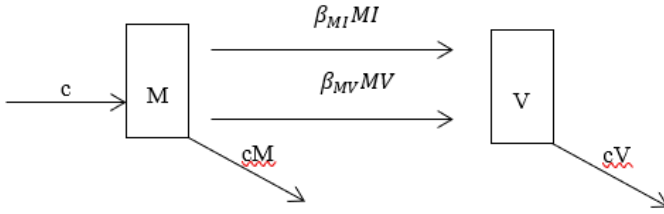
Fig. 3. Progression of susceptible M into infected V

As shown in Figure 3 the susceptible M has a chance of β_{MI} to be infected by infected person I, so β_{MI} of susceptible M transform into infected person I. the susceptible M has a chance of β_{MV} to be infected by infected V, so $\beta_{MV}MV$ of susceptible M transform into infected V. cM of susceptible M die and cV of infected V die.

As shown in figure 1,figure 2,figure 3, susceptible person S, susceptible A , and susceptible M entered the infected area at the rate of v_1, v_2, v_3, respectively, where $v_1 = \beta_{SV}SV + \beta_{SI}SI + \beta_{SB}SB$, $v_2 = \beta_{AV}AV + \beta_{AI}AI + \beta_{AB}AB$, $v_3 = \beta_{MI}MI + \beta_{MV}MV$.

Assuming that the incidence of disease is the standard incidence,the following differential equations are established according to the above model:

$$\frac{dI}{dt} = S(\beta_{SV}V + \beta_{SI}I + \beta_{SB}B) - I(\gamma + b) \tag{10}$$

$$\frac{dB}{dt} = A(\beta_{AV}V + \beta_{AI}I + \beta_{AB}B) - dB \tag{11}$$

$$\frac{dV}{dt} = M(\beta_{MI}I + \beta_{MV}V) - cV \tag{12}$$

$$\frac{dS}{dt} = b(1 - S) + \gamma I - S(\beta_{SV}V + \beta_{SI}I + \beta_{SB}B) \tag{13}$$

$$\frac{dA}{dt} = d(1 - A) - A(\beta_{AV}V + \beta_{AI}I + \beta_{AB}B) \tag{14}$$

$$\frac{dM}{dt} = c(1 - M) - M(\beta_{MI}I + \beta_{MV}V) \tag{15}$$

Among them: $\frac{dI}{dt}$ reflects the rate of change of average infection in human beings, $\frac{dB}{dt}$ reflects the rate of change of average infection in reaction vector 1, $\frac{dV}{dt}$ reflects the rate of change of change of average infection in reaction vector 2, $\frac{dS}{dt}$ reflects the rate of change rate of reaction susceptible people, $\frac{dA}{dt}$ reflects the rate of change rate of change of susceptibles in reaction vector 1, $\frac{dM}{dt}$ reflects the rate of change of susceptibles in reaction vector 2.

The disease-free equilibrium of this model is:
$S_0=1$
$A_0=1$

$M_0=1$
$I_0=0$
$B_0=0$
$V_0=0$

The model (4a-4f) always has a disease-free equilibrium point. We use van den Driessche and Watmough 's method to calculate the basic reproduction number of the model by the next generation matrix method. The basic reproduction number R_0 represents the number of people infected by a patient during the average disease period at the early stage of the disease when all people are susceptible. The infection items of the model are I, V, B.

$$\mathcal{F} = \begin{matrix} I \\ B \\ V \\ S \\ A \\ M \end{matrix} \begin{bmatrix} S(\beta_{SV}V + \beta_{SI}I + \beta_{SB}B) \\ A(\beta_{AV}V + \beta_{AI}I + \beta_{AB}B) \\ M(\beta_{MI}I + \beta_{MV}V) \\ -S(\beta_{SV}V + \beta_{SI}I + \beta_{SB}B) \\ -A(\beta_{AV}V + \beta_{AI}I + \beta_{AB}B) \\ -M(\beta_{MI}I + \beta_{MV}V) \end{bmatrix} \quad \text{and} \quad \mathcal{V} = \begin{matrix} I \\ B \\ V \\ S \\ A \\ M \end{matrix} \begin{bmatrix} I(\gamma + b) \\ dB \\ cV \\ b(S - 1) - \gamma I \\ d(A - 1) \\ c(M - 1) \end{bmatrix} \tag{16}$$

$$F = \begin{bmatrix} \beta_{SV}S & \beta_{SI}S & \beta_{SB}S \\ \beta_{AV}A & \beta_{AI}B & \beta_{AB}A \\ \beta_{MI}M & 0 & \beta_{MV}M \end{bmatrix}, \quad V = \begin{bmatrix} \gamma + b & 0 & 0 \\ 0 & d & 0 \\ 0 & 0 & c \end{bmatrix} \tag{17}$$

$$F = \begin{bmatrix} \beta_{SV} & \beta_{SI} & \beta_{SB} \\ \beta_{AV} & \beta_{AI} & \beta_{AB} \\ \beta_{MI} & 0 & \beta_{MV} \end{bmatrix}, \quad V = \begin{bmatrix} \gamma + b & 0 & 0 \\ 0 & d & 0 \\ 0 & 0 & c \end{bmatrix} \tag{18}$$

F is a nonnegative matrix, V is a nonsingular M-matrix. the basic reproduction number R_0 of the system is as follows:

$$R_0 = \rho(FV^{-1}) \tag{19}$$

where ρ represents the spectral radius of FV^{-1}.

The spectrum of a matrix, or the spectral radius of a matrix, plays an important role in the theory of eigenvalue estimation, generalized inverse matrix, numerical analysis and numerical algebra. In mathematics, the spectral radius of a matrix or bounded linear operator is the supremum of the set of absolute values of its eigenvalues. By simple calculation,

$$V^{-1} = \frac{1}{dc(\gamma+b)} \begin{bmatrix} dc & 0 & 0 \\ 0 & c(\gamma + b) & 0 \\ 0 & 0 & d(\gamma + b) \end{bmatrix} \tag{20}$$

$$FV^{-1} = \begin{bmatrix} \frac{\beta_{SV}S}{(\gamma+b)} & \frac{\beta_{SI}S}{d} & \frac{\beta_{SB}S}{c} \\ \frac{\beta_{AV}A}{(\gamma+b)} & \frac{\beta_{AI}A}{d} & \frac{\beta_{AB}A}{c} \\ \frac{\beta_{MI}M}{(\gamma+b)} & 0 & \frac{\beta_{MV}M}{c} \end{bmatrix} \qquad (21)$$

Order $\frac{\beta_{SV}S}{\gamma+b} = a$, $\frac{\beta_{SI}S}{d} = b$, $\frac{\beta_{SB}S}{c} = c$, $\frac{\beta_{AV}A}{\gamma+b} = d$, $\frac{\beta_{AI}A}{d} = e$, $\frac{\beta_{AB}A}{c} = f$, $\frac{\beta_{MI}M}{\gamma+b} = g$, $\frac{\beta_{MV}M}{c} = h$,the spectral radius of FV^{-1} that can be calculated using MATLAB.R_0=[a/3+e/3+h/3-((ae)/3-(bd)/3+(ah)/3-(cg)/3+(eh)/3-(a+e+h)²/9)/(((((a+e+h)(ae-bd+ah-cg+eh))/6-(a+e+h)³/27-(aeh)/2+(bdh)/2-(bfg)/2+(ceg)/2)²+((ae)/3-(bd)/3+(ah)/3-(cg)/3+(eh)/3-(a+e+h)²/9)³)^{1/2}-((a+e+h)(ae-bd+ah-cg+eh))/6+(a+e+h)³/27+(aeh)/2-(bdh)/2+(bfg)/2-(ceg)/2)^{1/3}+(((((a+e+h)(ae-bd+ah-cg+eh))/6-(a+e+h)³/27-(aeh)/2+(bdh)/2-(bfg)/2+(ceg)/2)²+((ae)/3-(bd)/3+(ah)/3-(cg)/3+(eh)/-(a+e+h)²/9)³)^{3/2}-((a+e+h)(ae-bd+ah-cg+eh))/6+(a+e+h)³/27+(aeh)/2-(bdh)/2+(bfg)/2-(ceg)/2)^{1/3}

When the basic regeneration number $R_0 < 1$, the disease-free equilibrium point P_0 of the system is asymptotically stable locally, and the disease will eventually disappear. When $R_0 > 1$, instability, disease will be prevalent.

4   Conclusion

This paper builds a new mathematical model based on multi-media transmission mechanism, and focuses on the impact of multiple vector transmission factors on the spread of infectious diseases. Assuming that the birth mortality rates of the population, media 1 and media 2 are the same, respectively b, d and c. By establishing a multimedia propagation model, the expression of the basic regenerative number of this model is obtained, the existence and stability of the equilibrium point of the model are discussed, and the established dynamic model is theoretically analyzed. This paper establishes a mathematical model of multimediated infectious diseases and derives the disease-free equilibrium point(DFE) and the threshold of disease transmission, which is the basic regeneration number R_0. The local stability of DFE is based on R_0. when R_0<1, DFE is locally asymptotic and stable; when R_0>1, DFE is unstable. This paper analyzes the stability of multi-mediated infectious diseases. The established model considers the impact of multi-mediated transmission and provides a theoretical basis for the control of infectious diseases in the case of multi-mediated transmission.

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