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Research Article

A Mathematical Model for Transmission of Hantavirus among Rodents and Its Effect on the Number of Infected Humans

Asep K. Supriatna,[1] **Herlina Napitupulu**,[1] **Meksianis Z. Ndii**,[2] **Bapan Ghosh**,[3] **and Ryusuke Kon**[4]

[1]*Department of Mathematics, Faculty of Mathematics and Natural Sciences, Universitas Padjadjaran, Sumedang 45363, Indonesia*
[2]*Department of Mathematics, Faculty of Mathematics and Natural Sciences, Universitas Nusa Cendana, Kupang 85001, Indonesia*
[3]*Department of Mathematics, Indian Institute of Technology Indore, Khandwa Road, Simrol, Indore 453552, Madhya Pradesh, India*
[4]*Faculty of Engineering, University of Miyazaki, Miyazaki 889-2192, Japan*

Correspondence should be addressed to Asep K. Supriatna; a.k.supriatna@unpad.ac.id

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In this paper, we present a mathematical model for the transmission of hantavirus among rodents and its effect on the number of hantavirus-infected human population. We investigate the model and present a standard analysis in mathematical epidemiology, such as determining the equilibria of the system and their stability analysis, together with the relationship to the basic reproduction number. It is found that the endemic equilibrium exists and is locally asymptotically stable when the basic reproduction number is greater than one; otherwise, the disease-free equilibrium is stable. Later on, we also show that by constructing a suitable Lyapunov function, the endemic equilibrium is globally asymptotically stable whenever it exists. Based on the basic reproduction number, we present a critical level of intervention to control the spread of the disease to humans. We found a significant finding from the present model that if the basic reproduction number is greater than one, then it is impossible to completely eliminate hantavirus disease in the system by solely focusing on any intervention for humans, like vaccination and curative action, without paying any attention to interventions for rodent populations. However, we can still decrease the density of infected humans with those interventions. Hence, we suggest that a combination of several interventions is needed to obtain effective control in eliminating the hantavirus. This information is useful for further study in finding an optimal control strategy to reduce or eliminate the transmission of hantavirus to humans.

1. Introduction

Some examples of important zoonoses include zoonotic influenza, salmonellosis, West Nile virus, plague, rabies, brucellosis, dengue, and hantavirus. Rats and mice are among the animals that spread most zoonoses to more than 35 diseases. The diseases are transmitted to humans via direct contact with rodents or extensive contact with rodent excreta-contaminated material [1]. Among the zoonoses spread by rodents is the hantavirus disease which is caused by the Hantaan virus. There are more than one species of rodents that can transmit hantavirus, including rats and mice.

Hantavirus pulmonary syndrome (HPS), or shortly hantavirus disease, is a fatal disease for humans. The virus is spread worldwide and is regarded as an important zoonotic pathogen that may cause severe and adverse effects in humans. It is transmitted to humans via direct contact with rodents or indirectly by rodent excreta (feces, urine, and aerosols). Humans may become infected once they inhale aerosolized droplets of urine or have extensive contact with rodent excreta-contaminated materials [2]. The disease is mainly circulated among rodents of different species and is also able to transmit from rodents to humans. However, there is no evidence of human-to-human transmission [3] or human-to-rodent transmission.

Hantaviruses are a group of viruses consisting of several strains that have been identified as infectious agents that can cause serious illness. Examples of Hantaan viruses are Dobrava, Puumala (PUU), and Seoul (SEOV) subtypes which may cause HFRS and Sin Nombre (SNV), Bayou virus (BAY), Black Canal Creek (BCC), and New York virus (NY) subtypes which may cause HPS [4]. To date, hantavirus infection is still regarded as a global zoonotic challenge, with an estimated more than 20,000 cases of hantavirus disease occurring annually worldwide, especially in Asia [5]. Sin Nombre virus, for example, is a type of hantavirus identified as the infectious agent that caused the deadly outbreak of hantavirus pulmonary syndrome in southwestern North America in 1993. Each hantavirus is harbored by an infected rodent species. Rodents do not lose infection and infect humans who come into contact with them or with their feces [6]. Each hantavirus generally associates with a primary rodent host where substantial coevolutionary adaptation is possible [7, 8].

There are about 30 different hantaviruses worldwide, some of which cause infections in humans [9]. Infection in humans is incidental, usually due to indirect transmission through contact with infectious rodent feces, but can cause hantavirus pulmonary syndrome with a mortality rate of up to 37% [10]. There are two characteristics of hantavirus infection observed in the field. For the first one, it is reported that infections can disappear entirely from rodent populations if environmental conditions are unsuitable, only to reappear when environmental conditions change and become favorable. This is a temporary feature. There are also spatial characteristics in the second one, in which there is evidence of focal infection. This "refugia" of rodent populations can be expanded or reduced [11].

The geographical distribution of hantavirus is mainly in Asia [5], such as in China, the Republic of Korea, and the Far East Region of the Russia Federation. As the most endemic country, more than 1,400,000 clinical cases of HFRS caused by HTNV and Seoul Virus (SEOV), with about 45,000 deaths, were reported in China during the period 1950 to 2001 [12]. This is about 70% to 90% of the total reported worldwide HFRS cases [13]. The remaining cases are reported from 18 countries (Asia), 32 countries (Europe) [13], and 7 countries (America) [14].

Other examples of countries that have already been invaded by hantavirus are Japan, Indonesia, and India (doi:10.1038/nindia.2008.104). Among several known strains of Hantaan viruses, one of them is Puumala (PUU) which may cause hemorrhagic fever with renal syndrome (HFRS) and hantavirus pulmonary syndrome (HPS) in humans [15]. Currently, there are no reported recent HFRS cases in Japan, but there may be some undiagnostic cases since infected brown rats are distributed throughout Japan and grey red-backed voles are massively infected with the PUU virus in Hokkaido [15]. Indonesia is also home to approximately 171 species of rats, and 22 species among them live on Java Island [16]. Wibowo [4] reported that at least 5 species of rats are among the reservoirs of the hantavirus. Recently, Mulyono et al. [17] added 4 more new species of rats which act as the reservoir of the hantavirus. Sendow et al. [18] and the reference therein pointed out the occurrence of HPS in Indonesia. The first reported cases of HPS in humans were in 2002, and initially, all the patients were suspected of contracting DHF [19]. The prevalence of hantavirus in rodents varies among cities in Indonesia, with the highest being 28.9% in Maumere, the eastern part of Indonesia [4]. This high prevalence of hantavirus in rodents also happens in other parts of the world, indicating the importance of rodents as a source of hantavirus transmission to humans and establishing the disease as a rodent-born disease.

Considering the danger of the disease to humans, some interventions have been proposed to control the spread of the disease to humans. This includes prevention and treatment. Prevention is done mainly by avoiding exposure to materials carrying hantavirus such as rodent's feces, urine, bodily secretions, and tissues. People who have a high risk of this exposure, such as those who are occupationally exposed to rodents, should take extra precautions to avoid this exposure. Some apparatus like gloves, goggles, rubber boots or disposable shoe covers, and coveralls or gowns may be used during their activities [20, 21], and ventilation of the room should be sufficiently good [22]. In general, rodent control to prevent high exposure to hantavirus in any building or house is recommended [23], which includes the use of rodent traps and poisons and the removal of possible nesting sites around the home [22]. Other examples of prevention have been described by Kerins et al. [24] related to pet rats, including euthanasia of the entire colony or testing and culling of infected animals. Intervention in humans to prevent hantavirus infection usually takes the form of vaccination, in which an inactivated hantavirus is injected [25]. While some therapeutics, concerned with the treatment of disease and the action of remedial agents, are given to cure infected humans [26], safe and effective vaccines and immunotherapy as preventives and treatments for hantavirus disease are still being developed [27, 28].

Hantavirus disease now begins to receive much attention from scientists, including mathematicians, in the attempt to understand, to control, and to eliminate it—if possible. In regard to the application of mathematics in the study of disease transmission, there are some authors that have constructed some mathematical models, such as Abramson and Kenkre [11, 29], Sauvage et al. [30], Abramson et al. [29], Allen et al. [31–34], Alvarez et al. [35], Escudero et al. [36], Chu et al. [37], Wesley [38, 39], Abramson [40], Rida et al. [41], Goh et al. [42], Karim et al. (2009), Kaplan et al. [43], Bürger et al. [44], and Yusof et al. [45, 46]. The most influential work in this area is written by Allen et al. whose subsequential works on mathematical modeling of hantavirus transmission have high citations. In this paper, we propose a new model for the transmission of hantavirus among rodents and its effect on the number of hantavirus-infected humans.

2. Formulation of New Model

In this section, we formulate a mathematical model for the transmission of hantavirus by considering the following

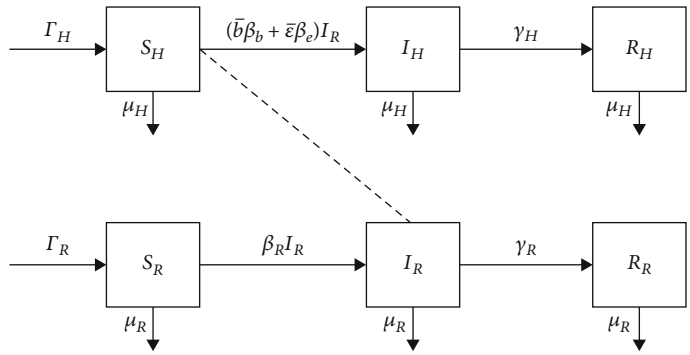

Figure 1: A schematic diagram of SIR-SIR transmission of hantavirus between humans and rodents.

assumptions: (i) there is only one species of rodents; (ii) the transmission happens only among rodents and from rodents to humans, and there is no transmission among humans and from humans to rodents since there is no evidence of human-to-human transmission [3]; (iii) transmission from rodents to humans occurs in two different modes, direct and indirect. Direct transmission occurs when there is direct contact between humans and infected rodents that may cause human infection by rodent bites, while indirect transmission can be done through the contact of humans and rodents excreta [2]. (iv) The recruitment to both human and rodent susceptible populations is constant, and (v) there is no vertical transmission [21].

Let us consider a human population, which, due to the circulation of hantavirus, is divided into three compartments, namely, the susceptible (S_H), the infected (I_H), and the recovered (R_H), who are assumed to be immune with $S_H(\mathrm{t}) + I_H(t) + R_H(t) = N_H(t)$. For all variables in the model (i.e., $X = S, I, R, N$), the notation $X(t)$ means the number of individuals in X class at time t. The rodent population is also assumed to have similar compartments with S_R denotes the susceptible rodents, I_R denotes the infective rodents, and R_R denotes the recovered rodents with $S_R(t) + I_R(t) + R_R(t) = N_R(t)$. A schematic diagram of disease transmission is shown in Figure 1.

The notations and parameters used in the schematic diagram above are presented in Table 1:

As there are two different routes of infection from rodent to human, i.e., by rodent biting and by contacting the rodent excreta, hence, we have the following equations as the governing hantavirus transmission among rodents and humans:

$$\frac{dS_H(t)}{dt} = \Gamma_H - \bar{b}\beta_b S_H(t) I_R(t) - \bar{\varepsilon}(I_R(t)) S_H(t)\beta_\varepsilon - \mu_H S_H(t), \tag{1}$$

$$\frac{dI_H(t)}{dt} = \bar{b}\beta_b S_H(t) I_R(t) + \bar{\varepsilon}(I_R(t)) S_H(t)\beta_\varepsilon - (\mu_H + \gamma_H) I_H(t), \tag{2}$$

$$\frac{dR_H(t)}{dt} = \gamma_H I_H(t) - \mu_H R_H(t), \tag{3}$$

Table 1: Parameters and notations used in the model formulation.

Γ_H; Γ_R	Recruitment rates (human; rodent)
$\bar{b}$	Number of bites/direct contact with rodent
$\bar{\varepsilon}$	Rodent excreta density
β_b; β_ε	Probability of successful contact (bites/direct contact; excreta)
β_H; β_R	Probability of successful contact (human; rodent)
μ_H; μ_R	Death rates (human; rodent)
γ_H; γ_R	Recovery rates (human; rodent)

$$\frac{dS_R(t)}{dt} = \Gamma_R - \beta_R S_R(t) I_R(t) - \mu_R S_R(t), \tag{4}$$

$$\frac{dI_R(t)}{dt} = \beta_R S_R(t) I_R(t) - (\mu_R + \gamma_R) I_R(t), \tag{5}$$

$$\frac{dR_R(t)}{dt} = \gamma_R I_R(t) - \mu_R R_R(t). \tag{6}$$

Let us consider the first case in which the number of rodent excreta is a linear function of the number of infective rodents, i.e., $\bar{\varepsilon}(I_R(t)) = \varepsilon I_R(t)$. Hence, the per capita successful contact rate between a susceptible and the rodent excreta rate, with the successful probability of transmission β_ε, is given by $\varepsilon I_R(t)\beta_\varepsilon$. Furthermore, if we also assume the successful probability of transmissions is the same regardless of its mode of transmission (via biting by the rodent or contact with rodent excreta and aerosol), i.e., $\beta_\varepsilon = \beta_b = \beta_H$, then, we have the total transmission rate from the two different modes given by

$$\begin{aligned} &\bar{b}\beta_b S_H(t) I_R(t) + \bar{\varepsilon}(I_R(t)) S_H(t)\beta_\varepsilon \\ &\quad = \bar{b}\beta_H S_H(t) I_R(t) + \varepsilon I_R(t) S_H(t)\beta_H \\ &\quad = \left(\bar{b} + \varepsilon\right)\beta_H S_H(t) I_R(t) = b\beta_H S_H(t) I_R(t). \end{aligned} \tag{7}$$

Hence, the complete equations for the SIR-SIR hantavirus transmission in this special case are given by Equations (3)–(6) plus the following equations:

$$\frac{dS_H(t)}{dt} = \Gamma_H - b\beta_H S_H(t) I_R(t) - \mu_H S_R(t), \tag{8}$$

$$\frac{dI_H(t)}{dt} = b\beta_H S_H(t) I_R(t) - (\mu_H + \gamma_H) I_H(t). \tag{9}$$

In fact, the last two equations can also be derived in more general ways without assuming $\beta_\varepsilon = \beta_b = \beta_H$. In this case, we let $b\beta'_H = (\bar{b}\beta_b + \varepsilon\beta_\varepsilon)$. In the subsequent section, we analyze the model by showing its steady-state solutions, their stability, and their relation to the basic reproduction number, which is central in mathematical epidemiology studies.

3. Results

In this section, we analyze the system of Equations (3)–(6), (8), and (9) by showing the equilibria and their stability. The relation of the existence of the equilibria and its stability to the basic reproduction number is also presented. Furthermore, we present the sensitivity analysis of the equilibria and the basic reproduction number to the change of parameters to find the most critical parameters affecting the dynamics of the system.

3.1. The Equilibria. An endemic-free or nonendemic equilibrium always exists for any parameters of the model. However, we show that there is a threshold that determines the existence of an endemic equilibrium, say $\mathcal{T}^\varepsilon$, so that the endemic equilibrium exists only if $\mathcal{T}^\varepsilon$ is above a certain value; otherwise, an endemic equilibrium does not exist. We sum up this property in the following theorem.

Theorem 1. *In the SIR-SIR hantavirus model (Equations (3)–(6) and Equations (8) and (9)), the following properties hold:*

(a) The trivial nonendemic equilibrium of the system always exists, given by $(S_{0H}{}^*, I_{0H}{}^*, R_{0H}{}^*, S_{0R}{}^*, I_{0R}{}^*, R_{0R}{}^*,) = (\Gamma_H/\mu_H, 0, 0, \Gamma_R/\mu_R, 0, 0)$

(b) An endemic equilibrium is given by $(S_{eH}{}^*, I_{eH}{}^*, R_{eH}{}^*, S_{eR}{}^*, I_{eR}{}^*, R_{eR}{}^*)$ *with* $S^*_{eR} = \Gamma_R/\mu_R\mathcal{T}^0$, $I^*_{eR} = \beta_R/\mu_R(\mathcal{T}^0 - 1)$, $R^*_{eR} = \gamma_R\beta_R/\mu_R^2(\mathcal{T}^0 - 1)$, $S^*_{eH} = \beta_R\Gamma_H/b\mu_R\beta_H(\mathcal{T}^0 - 1) + \mu_H\beta_R$, $I^*_{eH} = (\mu_R b\beta_H(\mathcal{T}^0 - 1)/\beta_R(\mu_H + \gamma_H))S^*_H$, *and* $R^*_H = \gamma_H/\mu_H I^*_H$, *and* $\mathcal{T}^0 = \beta_R\Gamma_R/\mu_R(\mu_R + \gamma_R)$ *is a threshold such that the endemic equilibrium exists only if* $\mathcal{T}^0 > 1$; *otherwise, the endemic equilibrium does not exist.*

Proof of Theorem 1. By solving Equations (3)–(6) and Equations (8) and (9) simultaneously under steady-state conditions (i.e., when all LHSs of the equations are equal to zero), the system has two equilibria, i.e., $(S_{0H}{}^*, I_{0H}{}^*, R_{0H}{}^*, S_{0R}{}^*, I_{0R}{}^*, R_{0R}{}^*,) = (\Gamma_H/\mu_H, 0, 0, \Gamma_R/\mu_R, 0, 0)$ and $(S_{eH}{}^*, I_{eH}{}^*, R_{eH}{}^*, S_{eR}{}^*, I_{eR}{}^*, R_{eR}{}^*)$ given by

(a) $(S_{0H}{}^*, I_{0H}{}^*, R_{0H}{}^*, S_{0R}{}^*, I_{0R}{}^*, R_{0R}{}^*,) = (\Gamma_H/\mu_H, 0, 0, \Gamma_R/\mu_R, 0, 0)$ which is a nonendemic equilibrium, since all of the infected classes ($I_{0H}{}^*$ and $I_{0R}{}^*$) are zero. Clearly, this trivial one always occurs

(b) $(S_{eH}{}^*, I_{eH}{}^*, R_{eH}{}^*, S_{eR}{}^*, I_{eR}{}^*, R_{eR}{}^*)$ is an endemic equilibrium, with $S^*_{eH} = \beta_R(\mu_R + \gamma_R)\Gamma_H/b\beta_H(-\mu_R(\mu_R + \gamma_R) + \beta_R\Gamma_R) + \beta_R(\mu_R + \gamma_R)\mu_H$, $I^*_{eH} = b\beta_H\Gamma_H(-\mu_R(\mu_R + \gamma_R) + \beta_R\Gamma_R)/(b\beta_H(-\mu_R(\mu_R + \gamma_R) + \beta_R\Gamma_R) + \beta_R(\mu_R + \gamma_R)\mu_H)(\mu_H + \gamma_H)$, $R^*_{eH} = (b\beta_H\Gamma_H(-\mu_R(\mu_R + \gamma_R) + \beta_R\Gamma_R)/(b\beta_H(-\mu_R(\mu_R + \gamma_R) + \beta_R\Gamma_R) + \beta_R(\mu_R + \gamma_R)\mu_H)(\mu_H + \gamma_H))\gamma_H/\mu_H$, $S^*_{eR} = \mu_R + \gamma_R/\beta_R$, $I^*_{eR} = -\mu_R(\mu_R + \gamma_R) - \beta_R\Gamma_R/\beta_R(\mu_R + \gamma_R)$, and $R^*_{eR} = -(\mu_R(\mu_R + \gamma_R) - \beta_R\Gamma_R)\gamma_R/\beta_R(\mu_R + \gamma_R)\mu_R$.

□

To find the condition for the existence of the endemic equilibrium, let us look for a threshold number, so that $S_{eH}{}^* \geq 0$, $I^*_{eH} > 0$, $R^*_{eH} > 0$, $S_{eR}{}^* \geq 0$, $I^*_{eR} > 0$, and $R^*_{eR} > 0$. Note that by using some algebraic manipulation, it is easy to show that the components of the equilibrium can be rewritten in the following forms.

(i) First, we focus on $S^*_{eR} = \mu_R + \gamma_R/\beta_R$. This can be written as $S^*_{eR} = \mu_R + \gamma_R/\beta_R = \Gamma_R/\mu_R(\beta_R\Gamma_R/\mu_R(\mu_R + \gamma_R))$. If we define $\mathcal{T}^0 = \beta_R\Gamma_R/\mu_R(\mu_R + \gamma_R)$, then we have $S^*_{eR} = \Gamma_R/\mu_R\mathcal{T}^0$ as required

(ii) Keeping in mind $\mathcal{T}^0 = \beta_R\Gamma_R/\mu_R(\mu_R + \gamma_R)$, then, we have the following-
$I^*_{eR} = -\mu_R(\mu_R + \gamma_R) - \beta_R\Gamma_R/\beta_R(\mu_R + \gamma_R) = \beta_R/\mu_R(\mathcal{T}^0 - 1)$

(iii) Similarly, $R^*_{eR} = -(\mu_R(\mu_R + \gamma_R) - \beta_R\Gamma_R)\gamma_R/\beta_R(\mu_R + \gamma_R)\mu_R = \gamma_R\beta_R/\mu_R^2(R_0 - 1)$

(iv) Next, we have $S^*_{eH} = \beta_R(\mu_R + \gamma_R)\Gamma_H/b\beta_H(-\mu_R(\mu_R + \gamma_R) + \beta_R\Gamma_R) + \beta_R(\mu_R + \gamma_R)\mu_H$. Let us look at the inverse which can be manipulated as

$$\begin{aligned}
\frac{1}{S^*_{eH}} &= \frac{b\beta_H(-\mu_R(\mu_R + \gamma_R) + \beta_R\Gamma_R) + \beta_R(\mu_R + \gamma_R)\mu_H}{\beta_R(\mu_R + \gamma_R)\Gamma_H} \\
&= \frac{b\beta_H(-\mu_R(\mu_R + \gamma_R) + \beta_R\Gamma_R)}{\beta_R(\mu_R + \gamma_R)\Gamma_H} + \frac{\beta_R(\mu_R + \gamma_R)\mu_H}{\beta_R(\mu_R + \gamma_R)\Gamma_H} \\
&= \frac{b\mu_R\beta_H}{\beta_R\Gamma_H}\frac{(-\mu_R(\mu_R + \gamma_R) + \beta_R\Gamma_R)}{\mu_R(\mu_R + \gamma_R)} + \frac{\mu_H}{\Gamma_H} \\
&= \frac{b\mu_R\beta_H}{\beta_R\Gamma_H}(R_0 - 1) + \frac{\mu_H}{\Gamma_H} \\
&= \frac{b\mu_R\beta_H(R_0 - 1) + \mu_H\beta_R}{\beta_R\Gamma_H}.
\end{aligned} \tag{10}$$

Hence, $S^*_{eH} = \beta_R\Gamma_H/b\mu_R\beta_H(\mathcal{T}^0 - 1) + \mu_H\beta_R$ as requested.

(v) Next $I^*_{eH} = b\beta_H\Gamma_H(-\mu_R(\mu_R + \gamma_R) + \beta_R\Gamma_R)/(b\beta_H(-\mu_R(\mu_R + \gamma_R) + \beta_R\Gamma_R) + \beta_R(\mu_R + \gamma_R)\mu_H)(\mu_H + \gamma_H)$

can be rewritten as $I^*_{eH} = b\beta_H\Gamma_H(-\mu_R(\mu_R+\gamma_R)+\beta_R\Gamma_R/\mu_R(\mu_R+\gamma_R))/(\mu_H+\gamma_H)(b\beta_H(-\mu_R(\mu_R+\gamma_R)+\beta_R\Gamma_R)+\beta_R(\mu_R+\gamma_R)\mu_H)/\mu_R(\mu_R+\gamma_R) = b\beta_H\Gamma_H(\mathcal{T}^0-1)/(\mu_H+\gamma_H)(b\beta_H(\mathcal{T}^0-1)+\beta_R\mu_H/\mu_R)$ as required

(vi) Finally, we have the following algebraic expression

$$R^*_{eH} = \left(\frac{b\beta_H\Gamma_H(-\mu_R(\mu_R+\gamma_R)+\beta_R\Gamma_R)}{(b\beta_H(-\mu_R(\mu_R+\gamma_R)+\beta_R\Gamma_R)+\beta_R(\mu_R+\gamma_R)\mu_H)(\mu_H+\gamma_H)}\right)\frac{\gamma_H}{\mu_H} = \frac{\gamma_H}{\mu_H}I^*_H \tag{11}$$

which completes the proof.

3.2. The Basic Reproduction Number and Stability of the Equilibria. Let us have a look at the form of the threshold $\mathcal{T}^0 = \beta_R\Gamma_R/\mu_R(\mu_R+\gamma_R)$ which can be written as $\mathcal{T}^0 = \beta_R(1/(\gamma_R+\mu_R))\Gamma_R(1/\mu_R)$ and can be read verbally as the multiplication of four rodent epidemiological factors. The multiplication of the four factors mentioned above is (the force of infection from an infectious rodent to a healthy rodent) × (the average length of stay of an infective rodent within the infectious period) × (the life expectancy of a healthy rodent) × (the constant rate of susceptible rodent recruitment). Interestingly, here, human epidemiological factors do not appear in the threshold parameter $\mathcal{T}^0$.

To provide a deeper interpretation of this threshold, let us consider a clinical intervention. In the health context, any intentional action designed to obtain an outcome is called a clinical intervention. If, in the absence of clinical intervention, we have $\mathcal{T}^0 > 1$ (hence, an endemic equilibrium exists), then we could apply a clinical intervention (such as vaccination), so that it is possible to reduce the threshold to be less than 1 by changing $\mathcal{T}^0$ to $\mathcal{T}^\varepsilon$ for a certain choice of $\varepsilon > 0$, resulting in $\mathcal{T}^\varepsilon < 1$ (removing the endemic equilibrium from the system). In the case of hantavirus, intervention other than clinical intervention is also possible such as reducing the rodent recruitment rate, reducing the life expectancy of the rodent, trapping, and culling infective rodents. This is the basic idea behind controlling/eliminating contagious diseases from a mathematical point of view. Finding this kind of threshold is vital in the study of mathematical epidemiology. In modern literature, this threshold is usually called the basic reproduction number (sometimes the basic reproduction/reproductive ratio). It is not easy to find this number for more complex transmissions of a disease. There are some good and rigorous literature studies regarding this concept, such as Diekmann and Heesterbeek [47], Diekmann et al. [48, 49], Van den Driessche and Watmough [50], and Zhao [51], that provide a more systematic way of constructing the basic reproduction number. We prove, by standard theory, that $\mathcal{T}^0$ mentioned above are indeed the basic reproduction number. We begin by defining the basic reproduction number.

The basic reproduction number of an infection is the expected number of cases produced by one case in a population where all the individuals are susceptible to infection. The authors of [19] (p. 4) defined the basic reproduction number, with the symbol $\mathcal{R}_0$, as the expected number of secondary cases per primary case in a "virgin" population. In the same book, they showed that $\mathcal{R}_0 \coloneqq \lim_{n\longrightarrow\infty}\|K^n\|^{1/n}$ ([47], p. 75), where K is the next-generation matrix defined therein. According to the authors, this is a natural definition of the basic reproduction number from which its value can be computed. However, there is another way to compute the basic reproduction number other than from this definition. In fact, there are some methods that are easier to use to obtain the basic reproduction number. As an example, the following method is suggested in Van den Driessche and Watmough [50]. The authors looked at an epidemic multicompartment model $dx_i/dt = f_i(x) = \mathcal{F}_i(x) - \mathcal{V}_i(x)$, $i = 1, \cdots, n$ (as in Equations (3)–(6) and Equations (8) and (9) above). They showed that the function $f_i(x)$ can be decomposed into the rate of appearance of new infections in the ith compartment, $\mathcal{F}_i(x)$, and the rate of transfer of individuals from/into the ith compartment, $\mathcal{V}_i(x)$. Furthermore, they defined F and V to be the Jacobian matrix evaluated at the nonendemic equilibrium and showed that the basic reproduction number can be calculated as the spectral radius $\mathcal{R}_0 = \rho(FV^{-1})$. The following theorem provides the basic reproduction number of the SIR-SIR hantavirus model in Equations (3)–(6) and Equations (8) and (9), which in this case is exactly the same as the threshold $\mathcal{T}^0$ in Theorem 1.

Theorem 2. *The SIR-SIR hantavirus model (Equations (3)–(6) and Equations (8) and (9)) has the basic reproduction number* $\mathcal{R}_0 = \beta_R\Gamma_R/\mu_R(\mu_R+\gamma_R)$.

Proof of Theorem 2. Following the method in [50], with reference to Equations (3)–(6) and Equations (8) and (9), we have the rate of appearance of new infection vectors $\mathcal{F}(x)$ and the rate of transfer of individual vectors $\mathcal{V}(x)$:

$$\mathcal{F} = \begin{pmatrix} 0 \\ b\beta_H S_H I_R \\ 0 \\ 0 \\ \beta_R S_R I_R \\ 0 \end{pmatrix}, \tag{12}$$

$$\mathcal{V} = \begin{pmatrix} -\Gamma_H + b\beta_H S_H I_R + \mu_H S_R \\ (\mu_H+\gamma_H)I_H \\ -\gamma_H I_H + \mu_H R_H \\ -\Gamma_R + \beta_R S_R I_R + \mu_R S_R \\ (\mu_R+\gamma_R)I_R \\ -\gamma_R I_R + \mu_R R_R \end{pmatrix}. \tag{13}$$

Next, from the two vectors, we obtain two matrices

$$F = \begin{pmatrix} 0 & b\beta_H S_H \\ 0 & \beta_R S_R \end{pmatrix}, \tag{14}$$

$$V = \begin{pmatrix} \mu_H + \gamma_H & 0 \\ 0 & \mu_R + \gamma_R \end{pmatrix}. \tag{15}$$

Consequently,

$$V^{-1} = \begin{pmatrix} \dfrac{1}{\mu_H + \gamma_H} & 0 \\ 0 & \dfrac{1}{\mu_R + \gamma_R} \end{pmatrix}, \tag{16}$$

$$FV^{-1} = \begin{pmatrix} 0 & \dfrac{b\beta_H S_H}{\mu_R + \gamma_R} \\ 0 & \dfrac{\beta_R S_R}{\mu_R + \gamma_R} \end{pmatrix}, \tag{17}$$

which gives rise to the effective reproduction number $\mathcal{R}_0 = \rho(FV^{-1}) = \beta_R S_R/\mu_R + \gamma_R$ where $S_R = \Gamma_R/\mu_R$. Hence, $\mathcal{R}_0 = \rho(FV^{-1}) = \beta_R \Gamma_R/\mu_R(\mu_R + \gamma_R)$ which completes the proof. □

Theorem 3. *The SIR-SIR model in Equations (3)–(6) and Equations (8) and (9) always has a trivial equilibrium, while the nontrivial equilibrium exists only if the basic reproduction number is greater than 1, i.e., $\mathcal{R}_0 = \beta_R \Gamma_R/\mu_R(\mu_R + \gamma_R) > 1$.*

Proof of Theorem 3. It is obvious as a consequence of Theorems 1 and 2. □

Theorem 4. *The nonendemic equilibrium $(S_{0H}{}^*, I_{0H}{}^*, R_{0H}{}^*, S_{0R}{}^*, I_{0R}{}^*, R_{0R}{}^*)$ of Equations (3)–(6) and Equations (8) and (9) is locally asymptotically stable whenever $\mathcal{R}_0 = \beta_R \Gamma_R/\mu_R(\mu_R + \gamma_R) < 1$ and unstable otherwise.*

Proof of Theorem 4. It is easy to show that the eigenvalues of the Jacobian matrix at the disease-free are $\lambda_1 = -\mu_R$, $\lambda_2 = -\mu_H$, $\lambda_3 = -(\mu_H + \gamma_H)$, and $\beta_R \Gamma_R - \mu_R(\mu_R + \gamma_R)/\mu_R$. The last eigenvalues is certainly negative if $\mathcal{R}_0 = \beta_R \Gamma_R/\mu_R(\mu_R + \gamma_R) < 1$. □

Theorem 5. *If the endemic equilibrium $(S_{eH}{}^*, I_{eH}{}^*, R_{eH}{}^*, S_{eR}{}^*, I_{eR}{}^*, R_{eR}{}^*)$ of Equations (3)–(6) and Equations (8) and (9) exists (i.e., whenever $\mathcal{R}_0 = \beta_R \Gamma_R/\mu_R(\mu_R + \gamma_R) > 1$), then it is locally asymptotically stable.*

Proof of Theorem 5. As before, it can be shown the eigenvalues of the Jacobian matrix at the disease-free are $\lambda_1 = -\mu_R$, $\lambda_2 = -\mu_H$, $\lambda_3 = -(\mu_H + \gamma_H)$, and $\lambda_4 = -b\beta b_H(\beta_R \Gamma_R - \gamma_R \mu_R - \mu_R^2) + \beta_R \mu_H(\mu_R + \gamma_R)/\beta_R(\mu_R + \gamma_R) = -b\beta b_H(\beta_R \Gamma_R/\mu_R(\mu_R + \gamma_R) - 1) + \beta_R \mu_H(\mu_R + \gamma_R)/\beta_R(\mu_R + \gamma_R)$ which is clearly negative if $\mathcal{R}_0 = \beta_R \Gamma_R/\mu_R(\mu_R + \gamma_R) > 1$. □

Further, we can also show that the endemic equilibrium, if exists, is globally asymptotically stable as follows.

Theorem 6. *If the endemic equilibrium $(S_{eH}{}^*, I_{eH}{}^*, R_{eH}{}^*, S_{eR}{}^*, I_{eR}{}^*, R_{eR}{}^*)$ of Equations (3)–(6) and Equations (8) and (9) exists (i.e., whenever $\mathcal{R}_0 = \beta_R \Gamma_R/\mu_R(\mu_R + \gamma_R) > 1$), then it is globally asymptotically stable.*

Proof of Theorem 6. The system of the last three equations is the famous SIR model. So, by using a standard Lyapunov function for the SIR model, we can show that the endemic equilibrium point of Equations (3)–(6) and Equations (8) and (9) is globally attractive in Ω defined by

$$\Omega = \left\{ (S_H, I_H, R_H, S_R, I_R, R_R) \in \mathbb{R}_+^6 : S_R > 0, I_R > 0 \right\}. \tag{18}$$

Note that the following equations are satisfied at the endemic equilibrium:

$\Gamma_R = \beta_R S_{eR}^* I_{eR}^* + \mu_R S_{eR}^*$ and $\mu_R + \gamma_R = \beta_R S_{eR}^*$. Define the function $V : \Omega \longrightarrow \mathbb{R}$ by $V(S_H, I_H, R_H, S_R, I_R, R_R) = S_R - \log S_R + I_R - \log I_R$. The derivative of V along the trajectories of (3)–(6), (8), and (9) is given by

$$\begin{aligned} \frac{dV}{dt} &= \left(1 - \frac{S_{eR}^*}{S_R}\right)\frac{dS_R}{dt} + \left(1 - \frac{I_{eR}^*}{I_R}\right)\frac{dI_R}{dt} \\ &= \left(1 - \frac{S_{eR}^*}{S_R}\right)(\beta_R S_{eR}^* I_{eR}^* + \mu_R S_{eR}^* - \beta_R S_R I_R - \mu_R S_R) \\ &\quad + \left(1 - \frac{I_{eR}^*}{I_R}\right)(\beta_R S_R I_R - \beta_R S_{eR}^* I_R) \\ &= -\mu_R \frac{(S_R - S_{eR}^*)^2}{S_R} + \beta_R S_{eR}^* I_{eR}^* \left(1 - \frac{S_{eR}^*}{S_R}\right)\left(1 - \frac{S_R I_R}{S_{eR}^* I_{eR}^*}\right) \\ &\quad + \beta_R S_{eR}^* I_{eR}^* \left(1 - \frac{I_{eR}^*}{I_R}\right)\left(\frac{S_R I_R}{S_{eR}^* I_{eR}^*} - \frac{I_R}{I_{eR}^*}\right) \\ &= -\mu_R \frac{(S_R - S_{eR}^*)^2}{S_R} + \beta_R S_{eR}^* I_{eR}^* \left(2 - \frac{S_{eR}^*}{S_R} - \frac{S_R}{S_{eR}^*}\right). \end{aligned} \tag{19}$$

To proceed with the last expression, $2 - S_{eR}^*/S_R - S_R/S_{eR}^*$, let us consider the following arithmetic-geometric mean (AGM) relation $x + y/2 \geq \sqrt{xy}$, where the equality holds if and only if $x = y$. Using this AGM relation $x = S_{eR}^*/S_R$ and $y = S_R/S_{eR}^*$ we obtain the expression $S_{eR}^*/S_R + S_R/S_{eR}^*/2 \geq 1$ or equivalently $2 - S_{eR}^*/S_R + S_R/S_{eR}^* \leq 0$, where the equality holds if and only if $S_{eR}^* = S_R$. Thus, we can conclude that $dV/dt = 0$ if and only if $S_{eR}^* = S_R$ otherwise $dV/dt < 0$. By LaSalle's invariant principle, the ω-limit set of any trajectory starting in Ω is contained in the maximal invariant set of Ω. It is straightforward to show that the maximal invariant set of Ω is the singleton consists of the endemic equilibrium point. Since every forward orbit in Ω is bounded, we can conclude that the endemic equilibrium is globally attractive in Ω. □

3.3. The Critical Level of Intervention. When an intervention is carried out to control the spread of the disease, the basic

Table 2: Critical intervention level found by setting the effective reproduction number to be less than one.

Intervention to rodent population	Objective	Critical intervention level
Culling/poisoning/trapping	Increase rodent death rate from μ_R to a higher mortality $\mu'_R = c\mu_R$	$c^* = (\gamma_R/2\mu_R)\left(\sqrt{(4\beta_R\Gamma_R/\gamma_R^2)+1}-1\right)$ $= (\gamma_R/2\mu_R)\left(\sqrt{(\mu_R(\mu_R+\gamma_R)/\gamma_R^2)4\mathscr{R}_0+1}-1\right)$
Curing	Increases rodent recovery rate from γ_R to a higher recovery $\gamma'_R = c\gamma_R$	$c^* = \beta_R\Gamma_R - \mu_R^2/\gamma_R\mu_R = \mathscr{R}_0(\gamma_R + \mu_R/\gamma_R) - (\mu_R^2/\gamma_R\mu_R)$
Isolation/transmission inhibitor	Reduces successful contact rate among rodents from or infection probability of rodent contact from β_R to a lower contact rate $\beta'_R = (1-c)\beta_R$, with $0 < c < 1$.	$c^* = 1 - 1/\mathscr{R}_0$

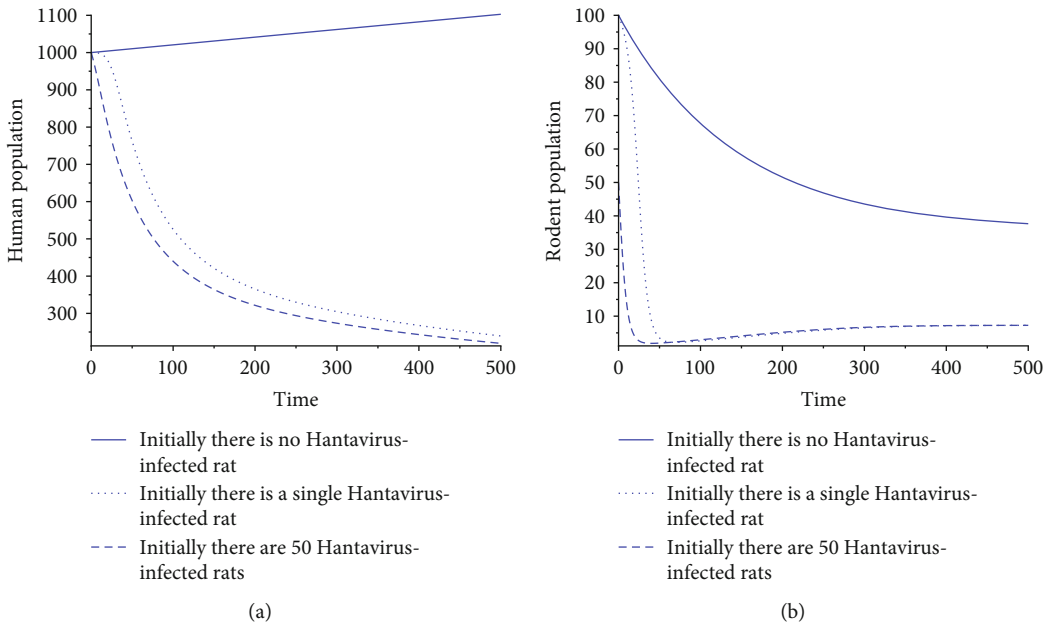

Figure 2: The growth of susceptible humans and rodents in the absence of hantavirus (solid lines) and in the presence of hantavirus (dashes and dots).

reproduction number $\mathscr{R}_0 = \beta_R\Gamma_R/\mu_R(\mu_R+\gamma_R)$ in Theorem 2 will change to the effective reproduction number $\mathscr{R}_0^e$ with the exact formula depending on the intervention being used. For example, if we are able to control so that only a portion of rodents could interact, say by a constant c, or decrease the probability successful contact from β_R to $\beta'_R = (1-c)\beta_R$, then the basic reproduction number will reduce to $\mathscr{R}_0^e = (1-c)\beta_R\Gamma_R/\mu_R(\mu_R+\gamma_R)$. To stop the spread of the disease, we need $\mathscr{R}_0^e < 1$ which is equivalent to $c > 1 - 1/\mathscr{R}_0$. We call $c^* = 1 - 1/\mathscr{R}_0$ as the critical intervention level that will be able to change the stability of the endemic equilibrium to an unstable equilibrium whenever $\mathscr{R}_0 > 1$.

In the case above, the critical intervention level has a simple form as a function of the basic reproduction number. Other forms are also possible, for example, when we take rodent culling as the intervention then basically it increases the natural mortality μ_R to a higher mortality μ'_R so that the effective reproduction number becomes $\mathscr{R}_0^e = \beta_R\Gamma_R/\mu'_R(\mu'_R+\gamma_R) = \beta_R\Gamma_R/c\mu_R(c\mu_R+\gamma_R) < 1$. In this case, the critical intervention level c^* is obtained by solving $c\mu_R(c\mu_R+\gamma_R)/\beta_R\Gamma_R > 1$, and given by the following, $c^* = (-1/2\gamma_R + 1/2\sqrt{4\beta_R\Gamma_R+\gamma_R^2})/\mu_R = \gamma_R/2\mu_R(\sqrt{4\beta_R\Gamma_R/\gamma_R^2+1}-1)$ which is positive.

We summarize the formulas for the critical intervention level in the following Table 2.

The critical level of intervention in Table 2 is derived using the effective reproduction number by equalizing it to one and solving for c as described in Section 3.3. Hence, it can only be used to undertake an intervention in the rodent population since the reproduction number does not contain parameters for the human population. The critical level of

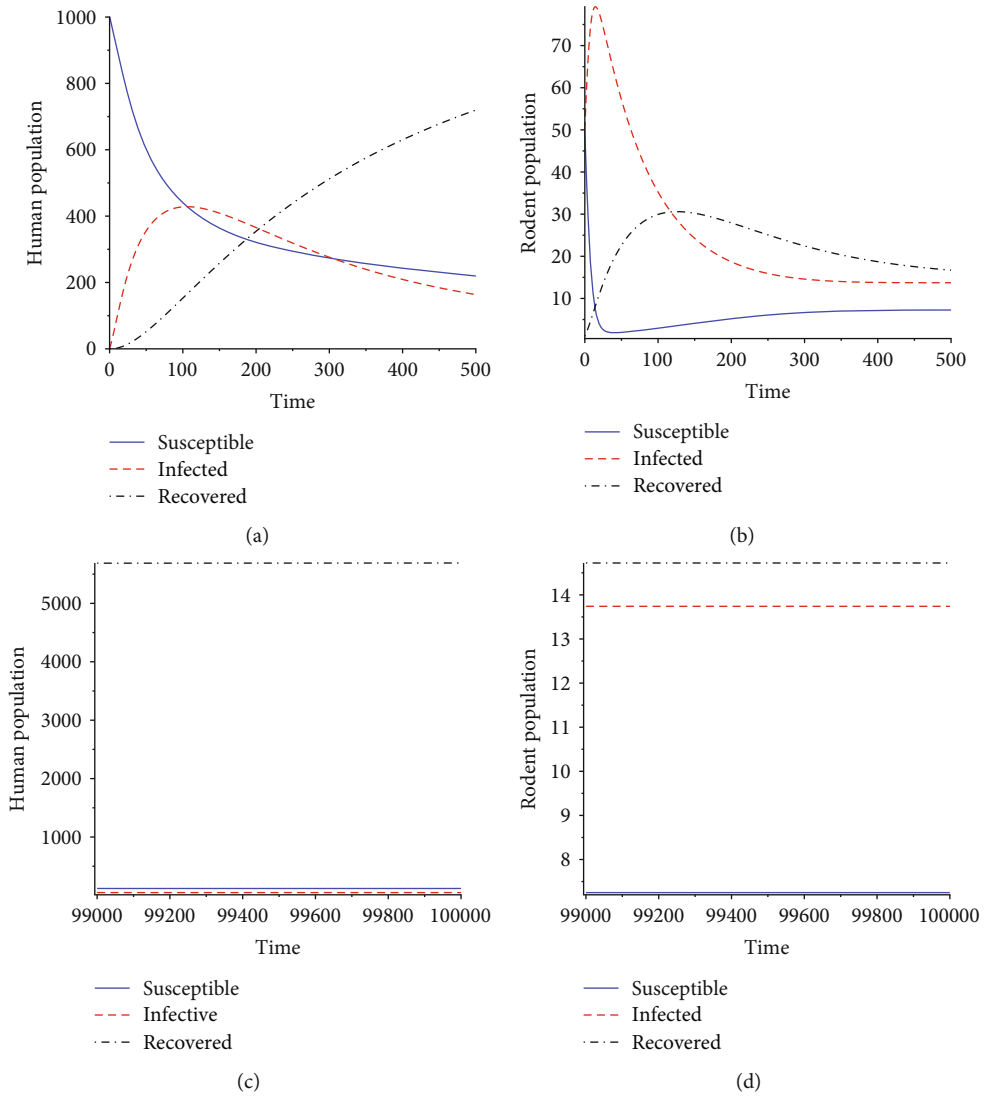

FIGURE 3: Transient solution of the system with $\mathscr{R}_0 = 4.926108374$ for (a) human subpopulations and (b) rodent subpopulations. The lower figures show the near equilibrium for (c) human subpopulations and (d) rodent subpopulations.

intervention above is aimed at eliminating the hantavirus so that the endemic state I^*_{eR} is zero and hence I^*_{eH} also vanishes. In fact, by observing the endemic state $I^*_{eH} = b\beta_H \Gamma_H(-\mu_R(\mu_R + \gamma_R) + \beta_R\Gamma_R)/(b\beta_H(-\mu_R(\mu_R + \gamma_R) + \beta_R\Gamma_R) + \beta_R(\mu_R + \gamma_R)\mu_H)(\mu_H + \gamma_H)$, we can only decrease this endemic state but will not make it vanish without the intervention of the rodent population. This can be seen as, whenever $\mathscr{R}_0 > 1$, $I^*_{eH} \leq 0$ only if $(-\mu_R(\mu_R + \gamma_R) + \beta_R\Gamma_R) \leq 1$ which is equivalent to either we make $\mathscr{R}_0 \leq 1$ by doing an intervention to the rodent population or making one of b, β_H, Γ_H parameters zero. The following section gives some numerical examples to illustrate the results presented above.

4. Numerical Examples

In this section, we present numerical examples to show the behavior of the SIR-SIR hantavirus model with and without the presence of clinical/nonclinical intervention (trapping/culling/poisoning the rodents, educating people to increase awareness regarding the danger of hantavirus so they avoid contact with rodents and their excreta, etc.). We use the following parameter values in the simulations:

$$\begin{aligned} b &= 0.1, \\ \beta_H &= 0.0015, \\ \Gamma_H &= 0.25, \\ \gamma_H &= \frac{1}{200}, \\ \mu_H &= \frac{1}{65^*365}, \\ \beta_R &= 0.002, \\ \Gamma_R &= 0.25, \\ \gamma_R &= 0.0075, \\ \mu_R &= 0.007. \end{aligned} \tag{20}$$

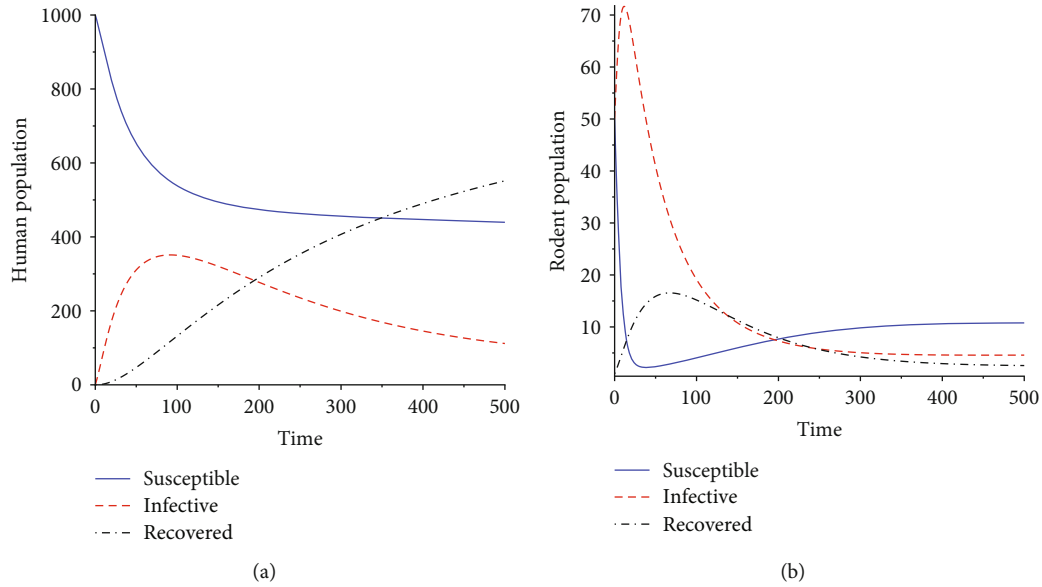

Figure 4: Transient solution of the system with the presence of culling to the rodent so that the death rate of the rodents increases up to twice the existing death rate, resulting in an effective reproduction number as low as $\mathcal{R}_0^c = 1.66$. However, this level of culling rate is not sufficient to eliminate the hantavirus.

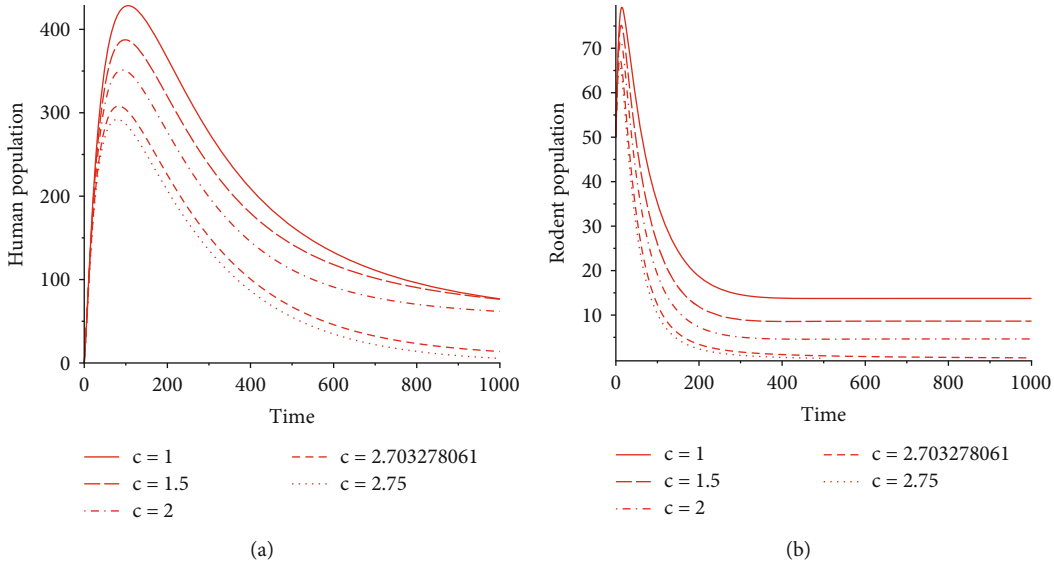

Figure 5: The effect of different culling levels of c to the number of infected human and infected rodent subpopulations. The critical culling level is $c = 2.703278061$, meaning that culling with a level lower than that level will not be effective in eliminating the hantavirus.

For comparison, we initially consider that there are 1,000 human individuals and 100 rodents in an environment. If it is assumed that there are no hantavirus-infected rats, both populations grow independently towards their respective equilibrium, as illustrated in Figure 2. In the absence of hantavirus, the growth of healthy human and rodent populations is depicted in the solid lines in Figures 2(a) and 2(b), respectively. Now, in the presence of hantavirus, if it is assumed that there is 1 infected rodent entering the system, the growth of the healthy or susceptible populations is shown in dash-dot lines in Figures 2(a) and 2(b), respectively. Compared to the case of the absence of hantavirus infection, both subpopulations are lower due to the infection of hantavirus and change their status to infected population. The dashed lines in the figures show the growth for different bigger infected rodent initial values (50 individuals). In the long term, in the presence of hantavirus, all subpopulations will converge to the equilibrium state as predicted by the stability theorem of the endemic state.

For the purpose of comparison, the following examples will assume a high basic reproduction number (chosen by the appropriate parameters above), and the hantavirus is heavily circulated among rodents, as indicated by the high initial value of infected rodents $S_R(0) = 50$, $I_R(0) = 50$, and

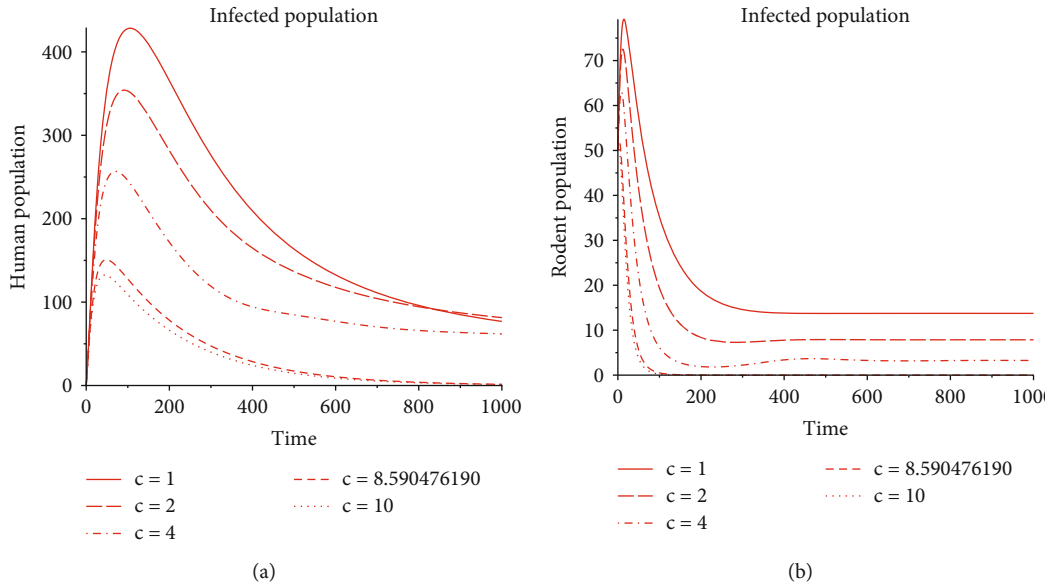

FIGURE 6: The effect of different curing levels of c to the number of infected human and infected rodent subpopulations. The critical curing level is $c = 8.590476190$, meaning that curing with a level lower than that level will not be effective in eliminating the hantavirus.

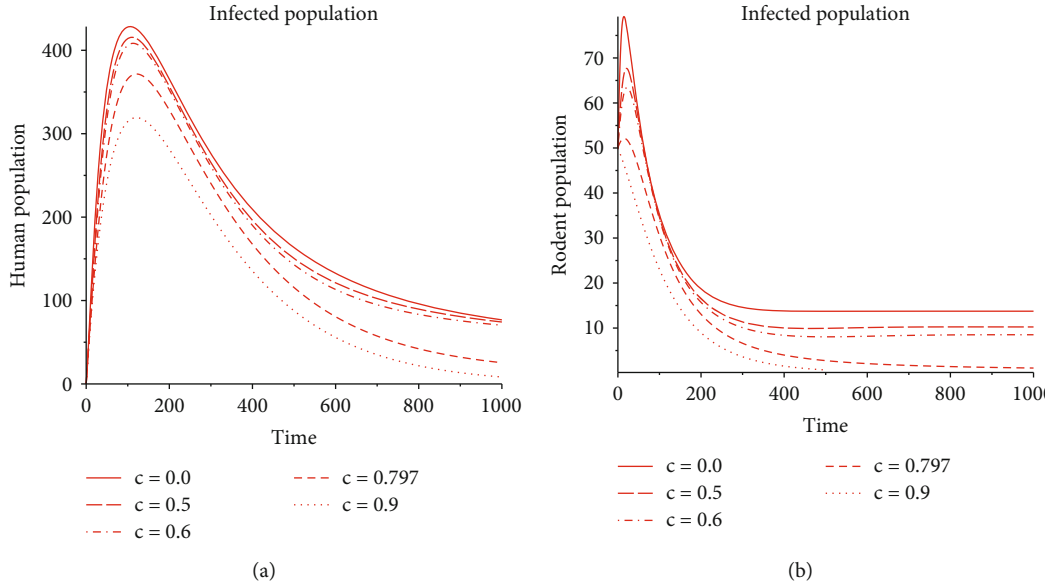

FIGURE 7: The effect of different isolation levels of c to the number of infected human and infected rodent subpopulations. This intervention can also be interpreted as vaccination. The critical isolation level is $c = 0.797$.

$R_R(0) = 1$. These initial values are chosen arbitrarily, just for illustration. The growth of all subpopulations is shown in Figure 3.

Figure 3 shows the transient solution of the system for human subpopulations (Figure 3(a)) and rodent subpopulations (Figure 3(b)). The lower figures show a near-equilibrium solution for human subpopulations (Figure 3(c)) and rodent subpopulations (Figure 3(d)). The resulting basic reproduction number for the chosen parameters indicates that the disease will become endemic eventually. To control the transmission of the hantavirus, we assume that culling is done to increase the death rate of the rodent up to twice the current death rate. The resulting solution of the system is shown in Figure 4 with the effective reproduction number $\mathcal{R}_0^e = 1.66$. This culling is not effective in eliminating the hantavirus, both in rodent and human population. This level of culling is not sufficient to drive the hantavirus to extinction. In fact, by referring to Table 2 regarding the critical intervention level, to increase the rodent death rate from μ_R to a higher mortality $\mu'_R = c\mu_R$, we need to set $c > 2.703278061$ which makes the effective reproduction number less than one. Figure 5 shows the resulting solution of the system when culling as the intervention on the rodent population is undertaken at various levels of c. Figure 6 shows the resulting solution of the system

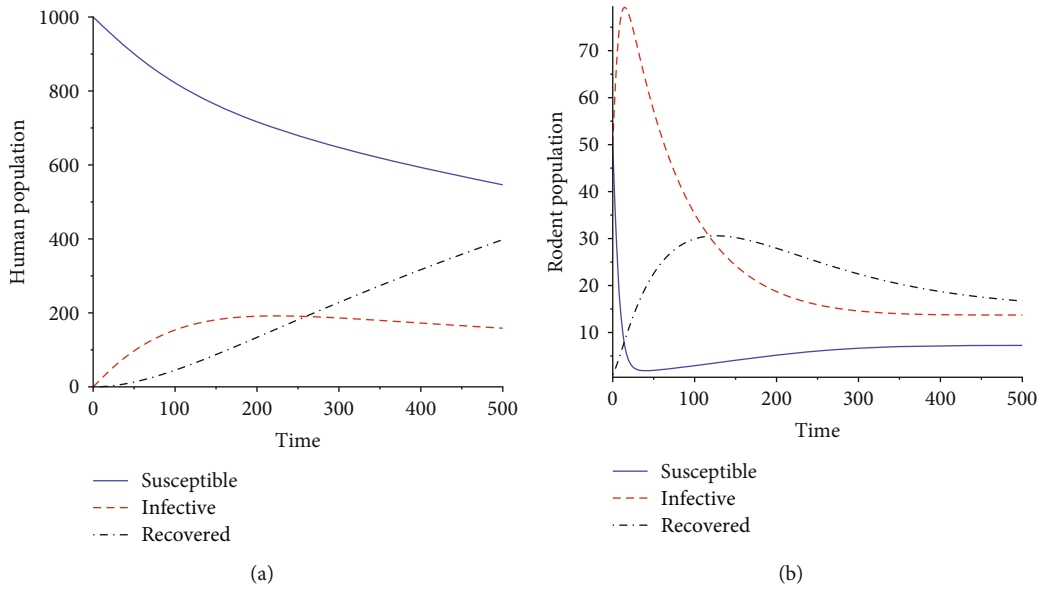

Figure 8: An example of the effect of saturated incidence on the number of infected human and infected rodent subpopulations. In this example, we use the functional form $\beta_H S_H(t)(I_R(t)/1 + \alpha I_R(t))$ with $\alpha = 0.05$ instead of $\beta_H S_H(t) I_R(t)$.

for the same parameters as in Figures 4 and 5, when curing as the intervention to the rodent population is undertaken for various levels of c, with the critical curing level is $c = 8.590476190$. This kind of intervention is not common, but it is feasible in terms of application technique since it is analogous to poisoning but with a different objective, i.e., to increase the recovery rate of the infected rodents. Figure 7 shows the resulting solution of the system when isolation to make contact among rodents is applied for various levels of c, with a critical isolation level of $c = 0.767$. This intervention can be viewed mathematically as similar to vaccination; hence, the critical isolation level is analogous to herd immunity to some extent. In reality, this intervention is also uncommon and difficult to implement since we have to vaccinate at least 76.7% of the rodent population unless vaccination can be implemented orally in the form of food bait for the rodents.

The results above are derived by assuming a mass action incidence rate and ignoring the presence of time delays. The results may be different if we do a fine-tuning to the model with the inclusion of more detailed and relevant factors. As an example, we show that if a saturated incidence rate as in Zhang et al. [52] is used in the present model, the solutions in Figure 3 change to those in Figure 8. Other information that also needs to be uncovered is the effect of the uncertainty of the parameters. In the following section, we present one way to analyze the effect of parameter uncertainty on the number of infected human population. We would like to derive the sensitivity indices to see which parameters are most influential on the results of the model (in this case, the number of infected human population).

4.1. Sensitivity Analysis. As most of the parameters have strong uncertainty, we perform a global sensitivity analysis to identify the most influential parameters of the model. It is measured against the increasing number of infected individuals. There are a lot of sensitivity analysis methods, such as Chitnis et al. [53], Marino et al. [54], and the references therein. Here, we use Latin hypercube sampling (LHS) in combination with the partial rank correlation coefficient (PRCC) [54] since it is among the most popular, reliable, and efficient sensitivity analyses to provide global sensitivity indexes. By following the method of Marino et al. [54], we simulate 2,000 samples, and the result is given in Figure 9. The range of the parameters used is given in Table 3.

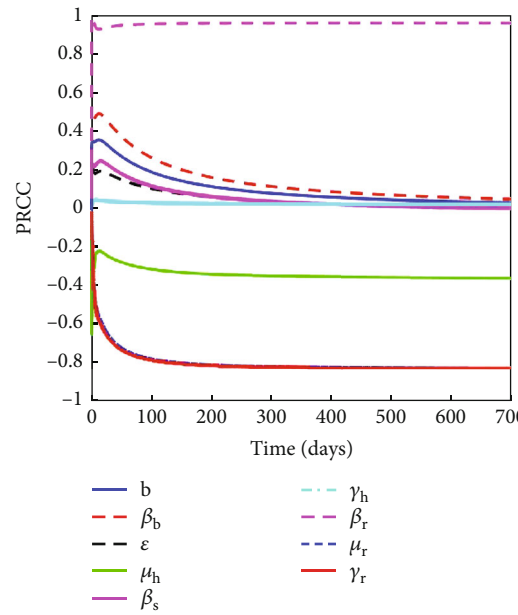

Figure 9: The PRCC plot shows the probability of successful contact between rodents β_r is the most influential parameter and has a positive relationship. Meanwhile, the parameters μ_r and γ_r have a negative relationship, which indicates that an increase in these parameter values results in a decrease in the number of hantavirus infections.

Table 3: Parameters and the range of parameter values used in sensitivity analysis.

Parameters	Minimum	Expected	Maximum
b	0	1	2
β_b	0	0.1	1
ϵ	0	0.1	1
μ_h	$1/(80\times365)$	$1/(65\times365)$	$1/(50\times365)$
β_ε	0	0.02	1
γ_h	0.05	0.082	0.1
β_r	0	0.02	1
μ_r	0	0.02	0.5
γ_r	0	0.02	0.5

Note that there is a slight difference to notation in the schematic diagram (Figure 1). This difference is clarified by recalling $\bar{\varepsilon}(I_R(t)) = \varepsilon I_R(t)$ and $b\,\beta'_H = (\bar{b}\beta_b + \varepsilon\beta_\varepsilon)$ as indicated in the text.

Figure 9 shows that the probability of successful contact between rodents β_r is the most influential parameter and has a positive relationship. This is realistic as the rodent is the source of infections, and hence, when humans and rodents interact and they successfully transmit viruses, the number of hantavirus cases increases. On the other hand, the parameters μ_r and γ_r have a negative relationship, which indicates that an increase in these parameter values results in a decrease in the number of hantavirus infections. An increase in the death rate of rodents aids in minimizing the number of hantavirus cases.

The figure shows that the greatest effect of intervention to control the spread of hantavirus is by reducing the contact rate between rodents β_r and by increasing the death rate and recovery rate parameters μ_r and γ_r. In reality, interventions to control the contact rate between rodents are difficult to implement, but increasing the death rate can be done much easier by nonclinical interventions, such as trapping, culling, and poisoning. Theoretically, increasing rodent recovery can also be implemented by using "drug food," although uncommon.

5. Conclusion

We have constructed a simple mathematical model for the transmission of hantavirus among rodents. Apart from the simpleness of the model, we arrive at the following useful insight. The analysis of the model shows that if the basic reproduction number is greater than one, then it is impossible to completely eliminate hantavirus disease in the system by solely focusing on any intervention to humans, like vaccination and curative action, without paying any attention to interventions to the rodent population unless there is no contact at all between human and rodent or between human and rodents' excreta or the successful probability contact rate is zero. However, we can still decrease the density of infected humans with those interventions. Hence, we suggest that a combination of several interventions is needed to obtain effective control in eliminating the hantavirus. Further, to determine the most significant parameters that can be used as control variables to reduce or eliminate hantavirus transmission, we use the Latin hypercube sampling in combination with partial rank correlation coefficient sensitivity analysis and found that the contact rate between rodents, the death rate, and the recovery rate of rodent's parameters are among the most significant parameters in determining both the numbers of infected rodents and infected humans. This justifies our first finding that solely focusing on intervention to humans may not succeed in completely eliminating hantavirus infection in the system. Moreover, this information is useful for further study in finding an optimal control strategy to reduce or eliminate the transmission of hantavirus to humans.

Data Availability

The data supporting the results of our study can be found in the article itself.

Conflicts of Interest

The authors declare that there are no conflicts of interest regarding the publication of this paper.

Acknowledgments

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