symmetry

MDPI

Article

Volterra–Lyapunov Stability Analysis of the Solutions of Babesiosis Disease Model

Fengsheng Chien [1,2] and Stanford Shateyi [3,*]

[1] School of Finance and Accounting, Fuzhou University of International Studies and Trade, Fuzhou 352020, China; jianfengsheng@fzfu.edu.cn
[2] Faculty of Business, City University of Macau, Macau 999078, China
[3] Department of Mathematics, University of Venda, Private Bag X5050, Thohoyandou 0950, South Africa
* Correspondence: stanford.shateyi@univen.ac.za

Abstract: This paper studies the global stability analysis of a mathematical model on Babesiosis transmission dynamics on bovines and ticks populations as proposed by Dang et al. First, the global stability analysis of disease-free equilibrium (DFE) is presented. Furthermore, using the properties of Volterra–Lyapunov matrices, we show that it is possible to prove the global stability of the endemic equilibrium. The property of symmetry in the structure of Volterra–Lyapunov matrices plays an important role in achieving this goal. Furthermore, numerical simulations are used to verify the result presented.

Keywords: global stability; Babesiosis disease; dynamical systems; Volterra–Lyapunov stability

check for updates

Citation: Chien, F.; Shateyi, S. Volterra–Lyapunov Stability Analysis of the Solutions of Babesiosis Disease Model. *Symmetry* **2021**, *13*, 1272. https://doi.org/10.3390/sym13071272

Academic Editor: Nicola Mastronardi

Received: 26 May 2021
Accepted: 8 July 2021
Published: 15 July 2021

Publisher's Note: MDPI stays neutral with regard to jurisdictional claims in published maps and institutional affiliations.

Copyright: © 2021 by the authors. Licensee MDPI, Basel, Switzerland. This article is an open access article distributed under the terms and conditions of the Creative Commons Attribution (CC BY) license (https://creativecommons.org/licenses/by/4.0/).

1. Introduction

The study of epidemiology plays an essential role in understanding the pattern of disease transmission and prevention. Therefore, the important role that mathematical models play in epidemiology should not be overlooked. A mathematical model can help researchers describe the dynamics of infectious diseases, predicting system behaviors and also adopting a strategy to control the disease [1–8].

Bovine babesiosis (BB) is a tick-borne disease of cattle caused by protozoan parasites of the genus Babesia, order Piroplasmida, phylum Apicomplexa. The Babesia bovis, Babesia bigemina and Babesia divergens are the principal species of Babesia that cause BB. Morbidity and mortality vary greatly and are influenced by several factors, such as prevailing treatments employed in an area, previous exposure to a species/strain of parasite, age, cattle breed, and vaccination status. All Babesia are transmitted by ticks with a limited host range. Blood infected with Babesia parasites and associated vectors of infected blood (especially ticks, but also by mechanical means) are the sources of infection. Bovine Babesia species are principally maintained by subclinically infected cattle that have recovered from disease and by tick vectors via transovarial transmission. The principal vectors of B. bovis and B. bigemina are Rhipicephalus spp. ticks and these are widespread in tropical and subtropical countries. The major arthropod vector of B. divergens is Ixodes Ricinus. Babesiosis has an important economic impact in the livestock sector of tropical regions [9–12]. The mathematical models of bovine Babesiosis have recently attracted an increasing amount of attention from researchers [13–17]. Aranda et al. [14] investigated the dynamical behavior of the tick-borne diseases. They determined the system's equilibria and performed stability analysis. In this model the total population of bovine $N_B(t)$ divides into three classes, namely bovines who may become infected ($\bar{S}_B(t)$-susceptible), bovine infected by the Babesia parasite ($\bar{I}_B(t)$-infected) and bovine who have been treated for Babesiosis ($\bar{C}_B(t)$-controlled) and the population of ticks $N_T(t)$ is divided into two categories, namely, ticks who may become infected ($\bar{S}_B(t)$-susceptible) and ticks infected by the Babesia parasite ($\bar{S}_B(t)$-infected).

One of the important issues in studying epidemiological models is to study the stability analysis of equilibrium points. Recently, the global stability of the endemic equilibrium has received considerable attention and several techniques were discussed [18–20]. Dang et al. [16] investigated the model of Babesiosis disease in more detail. They also examined the local stability of the disease-free equilibrium (DFE) by calculating eigenvalues. The authors in [16] stated "Since it is difficult to study the global stability property of this equilibrium point by analytical methods, we will do it by means of numerical simulations, as in Examples 1 and 2". Consequently, it is necessary to apply an analytical method to confirm that point E^* is asymptotically stable.

In order to extend the results of [16], we present the stability analysis of equilibria for this system with a simple approach. Liao and Wang [21] proposed a combination of the Lyapunov function method and Volterra–Lyapunov matrix properties and proved the global asymptotic stability of the endemic equilibria. This method does not meet the challenges of the Lyapunov function method including determining the appropriate Lyapunov function and coefficients. The method of Volterra–Lyapunov stable matrices [21], is modified using two Lemmas and reducing the dimensions of the matrices to overcome the problems exhibited by the standard technique (presented in [21]), while in some parts of the original method this technique is not used [22–24]. This approach transfers the analysis from differentiable functions to related matrices. A key point in the proposed method is its direct computational implementation. The importance of symmetry in computational science has been shown in recent research. In the process of proving the stability of equilibrium points in the present work, the symmetry of the matrices is one of the main conditions. This is mentioned in the definition of a positive (negative) definite matrix. Accordingly, when defining the Volterra–Lyapunov stable matrices, the symmetry of matrix $CA + A^T C^T$ is a fundamental precondition.

The structure of this article is as follows. Section 2, presents the model formulation, equilibrium points of the Babesiosis disease and the stability analysis of disease-free equilibrium (DFE). Section 3 discusses the global stability of the E^*. The simulation results for different values of the parameters are given as two examples in Section 4. Finally, Section 5 presents the final results.

2. Mathematical Model

We consider the mathematical model of Babesiosis disease for bovine and tick populations that was formulated by the following system of differential equations:

$$\begin{aligned}
\frac{d\bar{S}_1(t)}{dt} &= (\mu_1 + \alpha_1)\bar{C}(t) - \beta_1 \bar{S}_1(t)\frac{\bar{I}_2(t)}{N_2(t)},\\
\frac{d\bar{I}_1(t)}{dt} &= \beta_1 \bar{S}_1(t)\frac{\bar{I}_2(t)}{N_2(t)} - \lambda_1 \bar{I}_1(t),\\
\frac{d\bar{C}_1(t)}{dt} &= \lambda_1 \bar{I}_1(t) - (\mu_1 + \alpha_1)\bar{C}(t),\\
\frac{d\bar{S}_2(t)}{dt} &= \mu_2 p \bar{I}_2(t) - \bar{S}_2(t)\frac{\bar{I}_1(t)}{N_1(t)},\\
\frac{d\bar{I}_2(t)}{dt} &= \bar{S}_2(t)\frac{\bar{I}_1(t)}{N_1(t)} - \mu_2 p \bar{I}_2(t).
\end{aligned} \tag{1}$$

In order to describe the model, the assumptions are stated in Table 1.

Table 1. Description of the disease model.

Bovine Population	Description	Ticks Population	Description
$N_1(t)$	Total	$N_2(t)$	Total
$\bar{S}_1(t)$	Susceptible	$\bar{S}_2(t)$	Susceptible
$\bar{I}_1(t)$	Infected	$\bar{I}_2(t)$	Infected
$\bar{C}_1(t)$	Controlled	-	-

All the parameters are positive. A detailed discussion on the stability and optimal control of this system can be seen in [14,16].

Since $C_1(t) = 1 - S_1(t) - I_1(t)$, we focus our study on the following reduced system:

$$\begin{aligned} \frac{dS_1(t)}{dt} &= (\mu_1 + \alpha_1)(1 - S_1(t) - I_1(t)) - \beta_1 S_1(t) I_2(t), \\ \frac{dI_1(t)}{dt} &= \beta_1 S_1(t) I_2(t) - \lambda_1 I_1(t), \\ \frac{dI_2(t)}{dt} &= \beta_2(1 - I_2(t)) I_1(t) - \mu_2 p I_2(t). \end{aligned} \tag{2}$$

2.1. Equilibrium Points

The goal of this part is to find the equilibrium points of the system (2). Let us introduce the disease-free equilibrium E_0

$$E_0 = \left(S_1^0, I_1^0, I_2^0\right) = (1, 0, 0).$$

Furthermore, the endemic equilibrium (if it exists) is calculated

$$E_1 = (S_1^*, I_1^*, I_2^*),$$

where E_1 satisfies the following equilibrium equations

$$(\mu_1 + \alpha_1)(1 - S_1^* - I_1^*) - \beta_1 S_1^* I_2^* = 0, \tag{3}$$
$$\beta_1 S_1^* I_2^* - \lambda_1 I_1^* = 0, \tag{4}$$
$$\beta_2(1 - I_2^*) I_1^* - \mu_2 p I_2^* = 0. \tag{5}$$

Finally, we will have:

$$S_1^* = \frac{\beta_2\lambda_1(\mu_1 + \alpha_1) + p\lambda_1(\mu_1 + \alpha_1 + \lambda_1)\mu_2}{\beta_2[\alpha_1(\beta_1 + \lambda_1) + \lambda_1\mu_1 + \beta_1(\lambda_1 + \mu_1)]},$$

$$I_1^* = \frac{(\mu_1 + \alpha_1)(\beta_1\beta_2 - p\lambda_1\mu_2)}{\beta_2[\alpha_1(\beta_1 + \lambda_1) + \lambda_1\mu_1 + \beta_1(\lambda_1 + \mu_1)]},$$

$$I_2^* = \frac{(\mu_1 + \alpha_1)(\beta_1\beta_2 - p\lambda_1\mu_2)}{\beta_1\beta_2(\mu_1 + \alpha_1) + p\beta_1(\mu_1 + \alpha_1 + \lambda_1)\mu_2}.$$

Utilizing the next-generation matrix method [25], the basic reproductive rate can be calculated as

$$R_0 = \frac{\beta_1\beta_2}{\lambda_1\mu_2 p}.$$

2.2. Stability of the Disease-Free Equilibria

Here, we discuss the global asymptotic stability of E_0 by the following Theorem. Note that for the system (2) the positive invariant set is defined as

$$\Gamma = \Big\{(S_1, I_1, I_2) \in \mathbb{R}^3_+ : 0 \leq S_1 + I_1 \leq 1, 0 \leq I_2 \leq 1\Big\},$$

Theorem 1. *Consider the model (2) written in the form*

$$\begin{aligned} \frac{dS}{dt} &= F_1(S, I), \\ \frac{dI}{dt} &= F_2(S, I), \\ F_2\Big(S^{0,0}\Big) &= 0, \end{aligned} \tag{6}$$

where $S \in \mathbb{R}^m$ and $I \in \mathbb{R}^n$ indicate the uninfected and the infectious populations. Moreover, $E_0 = (S^0, 0)$ is the disease-free equilibrium of the model.

Furthermore, Hypotheses (H1) and (H2) are established:

Hypothesis 1 (H1). *For $\frac{dS}{dt} = F_1(S, 0)$, S^0 is globally asymptotically stable;*

Hypothesis 2 (H2). *$F_2(S, I) = AI - \hat{F}_1(S, I)$, with $\hat{F}_2(S, I) \geq 0$ for $(S, I) \in \Omega$, where the Jacobian matrix $A = \frac{\partial F_2}{\partial I}(S^0, 0)$ is an M-matrix, that is all off-diagonal elements of A are non-negative.*

Then provided that $R_0 < 1$ the disease-free equilibrium $E_0 = \left(S_1^0, I_1^0, I_2^0\right) = (1, 0, 0)$ is globally asymptotically stable

Proof. Let us consider $S = S_1$, $I = \begin{bmatrix} I_1 \\ I_2 \end{bmatrix}$. When $I_1 = I_2 = 0$, the uninfected subsystem (S_1) changes to

$$\frac{dS_1}{dt} = (\mu_1 + \alpha_1)(1 - S_1), \tag{7}$$

which has the solution

$$S_1(t) = 1 + e^{-(\mu_1 + \alpha_1)t}(S_1(0) - 1), \tag{8}$$

irrespective of the initial value $S_1(0)$, we obtain

$$\lim_{t \to \infty} S_1(t) = 1,$$

and this confirms that $S^0 = 1$ is globally asymptotically stable, that this condition (H_1) holds for this model.

Next, the infectious subsystem (I_1 and I_2) is equivalent to

$$\begin{aligned} \frac{dI}{dt} = F_2(S, I) &= \begin{bmatrix} \beta_1 S_1 I_2 - \lambda_1 \, I_1 \\ \\ \beta_2 I_1 - \beta_2 I_1 I_2 - \mu_2 p \, I_2 \end{bmatrix} \\ &= \begin{bmatrix} -\lambda_1 & \beta_1 \\ \\ \beta_2 & -\mu_2 \, p \end{bmatrix} \begin{bmatrix} I_1 \\ \\ I_2 \end{bmatrix} - \begin{bmatrix} 0 \\ \\ \beta_2 I_2 \, I_1 \end{bmatrix} \\ &= AI - \hat{F}_2(S, I), \end{aligned}$$

where

$$A = \begin{bmatrix} -\lambda_1 & \beta_1 \\ \beta_2 & -\mu_2\, p \end{bmatrix},$$

and

$$\hat{F_2}(S, I) = \begin{bmatrix} 0 \\ \beta_2 I_2\ I_1 \end{bmatrix}.$$

If $I(0) > 0$, then $I(t) \geq 0$. Since A is an M-matrix; therefore using the variation formula, we have

$$0 \leq I(t) = e^{At} I(0) - \int_0^t e^{A(t-r)} \hat{F_2}(S(r), I(r)) dr \leq e^{At} I(0). \tag{9}$$

Since A is M-matrix, when $R_0 < 1$ then A has a dominant eigenvalue $m(A) < 0$ and we have $\lim_{t\to\infty} ||e^{At}|| = 0$, thus

$$\lim_{t\to\infty} I(t) = 0. \tag{10}$$

Therefore, E_0 is globally stable and the proof is complete. □

3. Stability Analysis of the Endemic Equilibria

3.1. Notations

This section focuses on the stability analysis of E^*. The following definitions and notations are the requirements of this process.

Assumption 1. *Let M has the property of symmetry and is a positive (negative) definite, in this case M is written $M > 0 (< 0)$.*

Lemma 1 ([26])**.** *If there exists a positive diagonal matrix $C_{n\times n} > 0$ such that $CA + A^T C^T < 0$ then $A_{n\times n}$ is Volterra–Lyapunov stable.*

Lemma 2 ([26])**.** *If there exists the diagonal matrix $C_{n\times n} > 0$ such that $CA + A^T C^T > 0$ then $A_{n\times n}$ is diagonally stable.*

Proposition 1 ([26,27])**.** *The $A = \begin{bmatrix} a_{11} & a_{12} \\ a_{21} & a_{22} \end{bmatrix}$ is Volterra–Lyapunov stable if and only if:*

$$\begin{cases} a_{11} < 0, \\ a_{22} < 0, \\ \det(A) = a_{11}a_{22} - a_{12}a_{21} > 0. \end{cases}$$

Proposition 2 ([28,29])**.** *Consider the nonsingular $A_{n\times n} = [a_{ij}]$, $(n \geq 2)$, the positive diagonal matrix $B_{n\times n} = diag(b_1, \cdots, b_n)$ and $C = A^{-1}$, such that:*

$$\begin{cases} a_{nn} > 0, \\ \widetilde{B}\widetilde{A} + (\widetilde{B}\widetilde{A})^T > 0\,, \\ \widetilde{B}\widetilde{C} + (\widetilde{B}\widetilde{C})^T > 0, \end{cases}$$

then, there is $b_n > 0$ such that $BA + A^T B^T > 0$.

It is noteworthy that we delete the last row and column of the matrix A and signify it with matrix $\widetilde{A}_{(n-1)\times(n-1)}$.

3.2. Global Stability of the Endemic Equilibrium

The purpose of this section is to investigate the stability analysis of the E^* by the classical Lyapunov method with the aid of Volterra–Lyapunov stable matrices [24].

Let us introduce the biologically feasible domain of (2) as follows

$$\Omega = \left\{(S_1, I_1, I_2) \in \mathbb{R}^3_+ : 0 \leq S_1 + I_1 \leq 1, 0 \leq I_2 \leq 1\right\}.$$

which is clearly a positively invariant set in $\mathbb{R}^3$.

Consider the Lyapunov function

$$\mathscr{L} = z_1(S_1 - S_1^*)^2 + z_2(I_1 - I_1^*)^2 + z_3(I_2 - I_2^*)^2, \tag{11}$$

where z_1, z_2 and z_3 are positive constants. The derivative of $\mathscr{L}$ along solutions of (2) is

$$\begin{aligned}
\frac{d\mathscr{L}}{dt} &= 2z_1(S_1 - S_1^*)\frac{dS_1}{dt} + 2z_2(I_1 - I_1^*)\frac{dI_1}{dt} + 2z_3(I_2 - I_2^*)\frac{dI_2}{dt},\\
&= 2z_1(S_1 - S_1^*)[-(\mu_1 + \alpha_1)(S_1 - S_1^*) - (\mu_1 + \alpha_1)(I_1 - I_1^*) - \beta_1 S_1 I_2 + \beta_1 S_1^* I_2^*]\\
&\quad + 2z_2(I_1 - I_1^*)[\beta_1 S_1 I_2 - \beta_1 S_1^* I_2^* - \lambda_1((I_1 - I_1^*))]\\
&\quad + 2z_3(I_2 - I_2^*)[\beta_2(I_1 - I_1^*) - \beta_2 I_1 I_2 + \beta_2 I_1^* I_2^* - \mu_2 p(I_2 - I_2^*)].
\end{aligned}$$

By adding and subtracting expressions $\beta_1 S_1^* I_2$ and $\beta_2 I_1 I_2^*$ to the first and second brackets, one can obtain

$$\begin{aligned}
\frac{d\mathscr{L}}{dt} &= 2z_1(S_1 - S_1^*)[-(\mu_1 + \alpha_1)(S_1 - S_1^*) - (\mu_1 + \alpha_1)(I_1 - I_1^*) - \beta_1 S_1 I_2 + \beta_1 S_1^* I_2^* + \beta_1 S_1^* I_2\\
&\quad - \beta_1 S_1^* I_2] + 2z_2(I_1 - I_1^*)[\beta_1 S_1 I_2 - \beta_1 S_1^* I_2^* + \beta_1 S_1^* I_2 - \beta_1 S_1^* I_2 - \lambda_1(I_1 - I_1^*)]\\
&\quad + 2z_3(I_2 - I_2^*)[\beta_2(I_1 - I_1^*) - \beta_2 I_1 I_2 + \beta_2 I_1^* I_2^* + \beta_2 I_1 I_2^* - \beta_2 I_1 I_2^* - \mu_2 p(I_2 - I_2^*)].
\end{aligned}$$

therefore, we have

$$\begin{aligned}
\frac{d\mathscr{L}}{dt} &= -2z_1(\mu_1 + \alpha_1 + \beta_1 I_2)(S_1 - S_1^*)^2 - 2z_1(\mu_1 + \alpha_1)(S_1 - S_1^*)(I_1 - I_1^*)\\
&\quad - 2z_1\beta_1 S_1^*(S_1 - S_1^*)(I_2 - I_2^*) - 2\lambda_1 z_2(I_1 - I_1^*)^2 + 2z_2\beta_1 I_2(S_1 - S_1^*)(I_1 - I_1^*)\\
&\quad + 2z_2\beta_1 S_1^*(I_1 - I_1^*)(I_2 - I_2^*) - 2z_3(\mu_2 p + \beta_2 I_1^*)(I_2 - I_2^*)^2\\
&\quad + 2z_3\beta_2(1 - I_2)(I_1 - I_1^*)(I_2 - I_2^*)\\
&= X(ZQ + Q^T Z^T)X^T,
\end{aligned} \tag{12}$$

where $X = [S_1 - S_1^*, I_1 - I_1^*, I_2 - I_2^*]$, $Z = diag(z_1, z_2, z_3)$, and

$$Q = \begin{bmatrix} -(\mu_1 + \alpha_1 + \beta_1 I_2) & -(\mu_1 + \alpha_1) & -\beta_1 S_1^* \\ \beta_1 I_2 & -\lambda_1 & \beta_1 S_1^* \\ 0 & \beta_2(1 - I_2) & -(\mu_2 p + \beta_2 I_1^*) \end{bmatrix} \tag{13}$$

Remark 1. *The following theorem considers the stability analysis of E^*. To accomplishing it, we must meet the conditions of Proposition 2. Decrease the dimension of the matrix $-Q_{3\times3}$ to $-\widetilde{Q}_{2\times2}$ matrix, and then use Proposition 1. Through these steps, one can conclude that Q is Volterra–Lyapunov stable.*

Theorem 2. *Suppose that Equation (13) specifies the matrix $Q_{3\times3}$, then $Q_{3\times3}$ is Volterra–Lyapunov stable.*

Proof. By Proposition 2, one can derive the following conditions for proving the Volterra–Lyapunov stability of Q.

Condition 1. $-Q_{33} > 0$.

Condition 2. $-\tilde{Q}$ is diagonal stable.

Condition 3. $H = \widetilde{-Q^{-1}}$ is diagonal stable.

1. Clearly $-Q_{33} > 0$.

2. Let us delete the last row and last column of matrix $-Q$ and call it matrix $-\tilde{Q}$. It follows that

$$-\tilde{Q} = \begin{bmatrix} \mu_1 + \alpha_1 + \beta_1 I_2 & \mu_1 + \alpha_1 \\ -\beta_1 I_2 & \lambda_1 \end{bmatrix}. \tag{14}$$

   $-\tilde{Q}_{11} > 0$, and $-\tilde{Q}_{22} > 0$. And

$$\det(-\tilde{Q}) = \lambda_1(\mu_1 + \alpha_1 + \beta_1 I_2) + \beta_1 I_2(\mu_1 + \alpha_1) > 0$$

   Therefore, $-\tilde{Q}$ is diagonal stable.

3. Finally, using Proposition 1, the diagonal stability of $\widetilde{-Q^{-1}}$ is determined. Let us delete the last row and last column of matrix Q^{-1} and define the matrix $H = \widetilde{-Q^{-1}}$. One derives that

$$H = \widetilde{-Q^{-1}} = \frac{1}{\det(-Q)} \begin{bmatrix} \mathscr{H}_{11} & \mathscr{H}_{12} \\ \mathscr{H}_{21} & \mathscr{H}_{22} \end{bmatrix},$$

   where,

$$\mathscr{H}_{11} = (\mu_2 p + \beta_2 I_1^*)\lambda_1 - \beta_1 S_1^* \beta_2 (1 - I_2),$$

$$\mathscr{H}_{12} = -[(\mu_1 + \alpha_1 + \beta_1 I_2)(\mu_1 + \alpha_1) + \beta_1 S_1^* \beta_2 (1 - I_2)],$$

$$\mathscr{H}_{21} = \beta_1 I_2 (\mu_2 p + \beta_2 I_1),$$

$$\mathscr{H}_{22} = (\mu_1 + \alpha_1 + \beta_1 I_2)(\mu_2 p + \beta_2 I_1^*).$$

   Obviously, $\mathscr{H}_{22} > 0$. Now, we show $\mathscr{H}_{11} > 0$. Since, $0 \leq I_2 \leq 1$, therefore,

$$\beta_1 S_1^* \beta_2 I_2^* > \beta_1 S_1^* \beta_2 I_2^* (1 - I_2),$$

from the Equation (4), we obtain that

$$\lambda_1 I_1^* \beta_2 > \beta_1 S_1^* \beta_2 I_2^* (1 - I_2),$$

and (5) gives,

$$\lambda_1(\mu_2 p I_2^* + \beta_2 I_1^* I_2^*) > \beta_1 S_1^* \beta_2 I_2^* (1 - I_2),$$

consequently, we have

$$\lambda_1\mu_2 p + \lambda_1\beta_2 I_1^* - \beta_1 S_1^*\beta_2(1-I_2) > 0.$$

In addition, using $\mathscr{H}_{11} > 0$, we show that $\det(-Q) > 0$:

$$\begin{aligned}\det(-Q) = {} & (\mu_1+\alpha_1+\beta_1 I_2)[\lambda_1\mu_2 p + \lambda_1\beta_2 I_1^* - \beta_1 S_1^*\beta_2(1-I_2)] \\ & + (\mu_1+\alpha_1)[\beta_1 I_2(\mu_2 p + \beta_2 I_1^*)] + \beta_1 S_1^*(\beta_1\beta_2 I_2(1-I_2)) > 0. \end{aligned} \tag{15}$$

Hence, it is clear to see:

$$\det(\widetilde{-Q^{-1}}) = \frac{1}{\det(-Q)}(\mathscr{H}_{11}\mathscr{H}_{22} - \mathscr{H}_{12}\mathscr{H}_{21}) > 0,$$

$$\widetilde{-Q^{-1}}_{11} = \frac{1}{\det(-Q)}\mathscr{H}_{11} > 0,$$

$$\widetilde{-Q^{-1}}_{22} = \frac{1}{\det(-Q)}\mathscr{H}_{22} > 0.$$

Therefore, one can conclude that $\widetilde{-Q^{-1}}$ is diagonal stable.

In summary, the above steps confirm the conditions of Proposition 2 and one can conclude the Volterra–Lyapunov stability of matrix Q. This completes the proof. □

Theorem 3. *If $R_0 > 1$, then the endemic equilibrium $E^* = (S_1^*, I_1^*, I_2^*)$ of model (2) is globally stable in Ω.*

Proof. Theorem 2 concludes that there exists a positive diagonal matrix Z such that $ZQ + Q^T Z^T < 0$. Therefore, $\frac{d\mathscr{L}}{dt} < 0$ when $X \neq E^*$ and this ensures the global stability of the endemic equilibrium point. □

Remark 2. *The above results can be extended to other biological models. It is noted that, in [16], the authors did not discuss the globally stability of the disease free and endemic equilibrium and only discussed the global stability of E^* by means of numerical simulations.*

4. Numerical Results

In this section, we assess numerically Theorem 3 by means of two examples.

Example 1. *Suppose that system (2) has the parameters $\beta_1 = 0.003, \beta_2 = 0.00048, \mu_1 = 0.0002999, \mu_2 = 0.001609, \alpha_1 = 0.001, \lambda_1 = 0.0265, p = 0.5$.*

The basic reproduction number is such that $R_0 = 0.0675 < 1$ and the system (2) has only the disease free equilibrium of $E_0 = (1, 0, 0)$. The numerical simulation of system (2) with initial conditions is illustrated in Figures 1 and 2, all solutions converge to the E_0. In Figure 3, by the phase plane portrait of (S_1, I_1), we see that three solutions converge to the E_0 with three different initial conditions. Figures 4–6, show the phase diagram of (S_1, I_2), (I_1, I_2) and (S_1, I_1, I_2), with three initial conditions. Therefore, it can be seen from these figures that all solutions converge to the disease-free equilibrium point under the mentioned initial conditions.

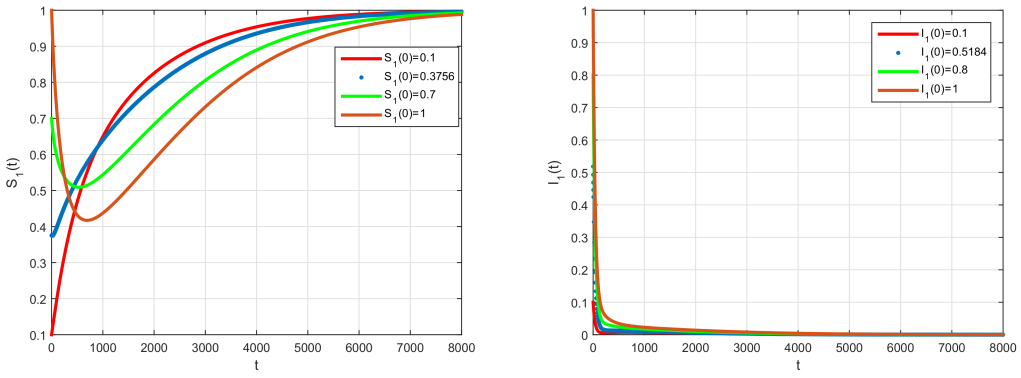


Figure 1. Time series evolution depicting the dynamical behaviour of the system, using $\beta_1 = 0.003$, $\beta_2 = 0.00048, \mu_1 = 0.0002999, \mu_2 = 0.001609, \alpha_1 = 0.001, \lambda_1 = 0.0265, p = 0.5, (R_0 = 0.0675 < 1)$.

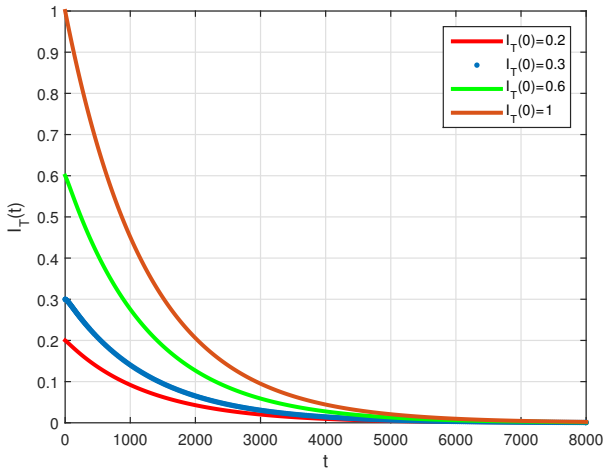


Figure 2. Time series evolution depicting the dynamical behaviour of the system (2), using $\beta_1 = 0.003$, $\beta_2 = 0.00048, \mu_1 = 0.0002999, \mu_2 = 0.001609, \alpha_1 = 0.001, \lambda_1 = 0.0265, p = 0.5, (R_0 = 0.0675 < 1)$.

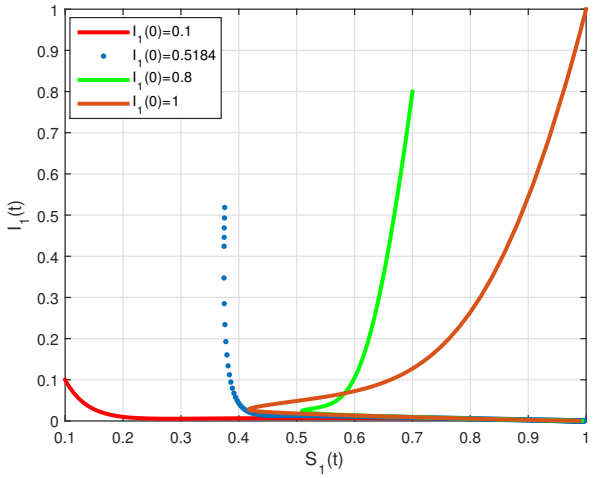


Figure 3. State diagrams of S_1 vs. I_1 for system (2), using $\beta_1 = 0.003, \beta_2 = 0.00048, \mu_1 = 0.0002999$, $\mu_2 = 0.001609, \alpha_1 = 0.001, \lambda_1 = 0.0265, p = 0.5, (R_0 = 0.0675 < 1)$.

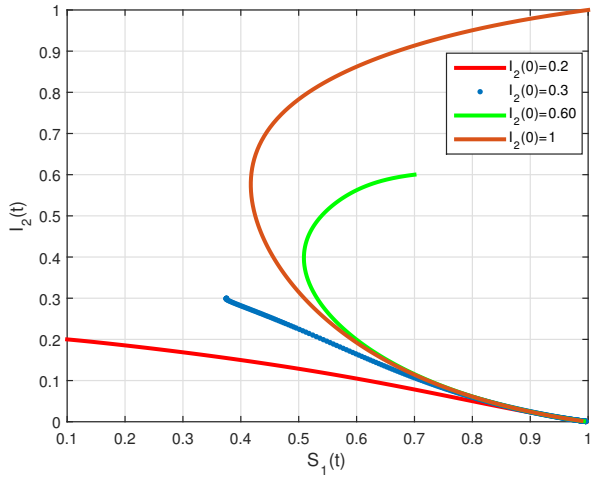


Figure 4. State diagrams of S_1 vs. I_2 for system (2), using $\beta_1 = 0.003, \beta_2 = 0.00048$, $\mu_1 = 0.0002999$, $\mu_2 = 0.001609, \alpha_1 = 0.001, \lambda_1 = 0.0265, p = 0.5, (R_0 = 0.0675 < 1)$.

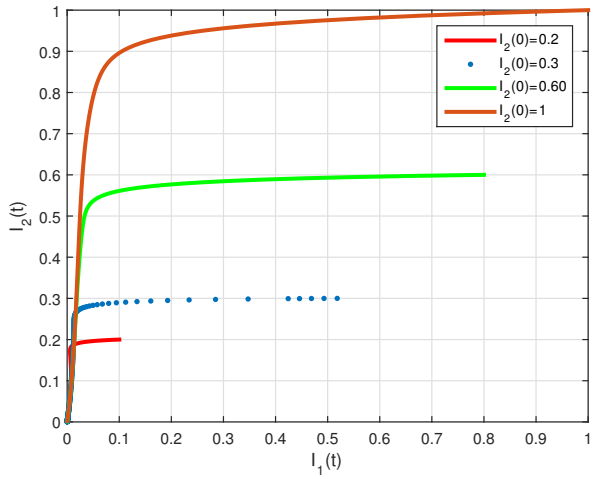


Figure 5. State diagrams of I_1 vs. I_2 for system (2), using $\beta_1 = 0.003, \beta_2 = 0.00048$, $\mu_1 = 0.0002999$, $\mu_2 = 0.001609, \alpha_1 = 0.001, \lambda_1 = 0.0265, p = 0.5, (R_0 = 0.0675 < 1)$.

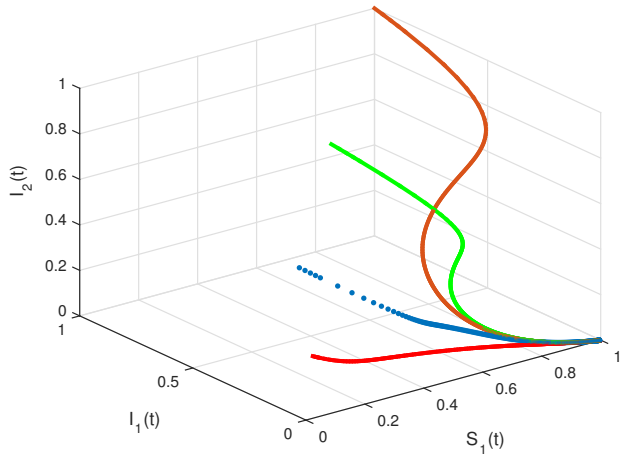


Figure 6. State diagrams of system (2), using $\beta_1 = 0.003, \beta_2 = 0.00048, \mu_1 = 0.0002999$, $\mu_2 = 0.001609$, $\alpha_1 = 0.001, \lambda_1 = 0.0265, p = 0.5, (R_0 = 0.0675 < 1)$.

Example 2. *Suppose that system (2) has the parameters* $\beta_1 = 0.006, \beta_2 = 0.00048, \mu_1 = 0.0002999, \mu_2 = 0.001609, \alpha_1 = 0.001, \lambda_1 = 0.000265, p = 0.1.$

The basic reproduction number is such that $R_0 = 67.5446 > 1$ and the system (2) has the endemic equilibrium of $E^* = (S_1^*, I_1^*, I_2^*)$. The numerical simulation of system (2) with initial conditions is illustrated in Figures 7 and 8, all these solutions converge to the $E^* \approx (0, 0498, 0.7893, 0.70)$. The phase diagram of (S_1, I_1) with different initial conditions is given in Figure 9. Figures 10–12, show the phase diagram of (S_1, I_2), (I_1, I_2) and (S_1, I_1, I_2), with three initial conditions. The figures confirm the endemic equilibrium E^* is globally asymptotically stable and all solutions converge to the endemic equilibrium point under the mentioned initial conditions.

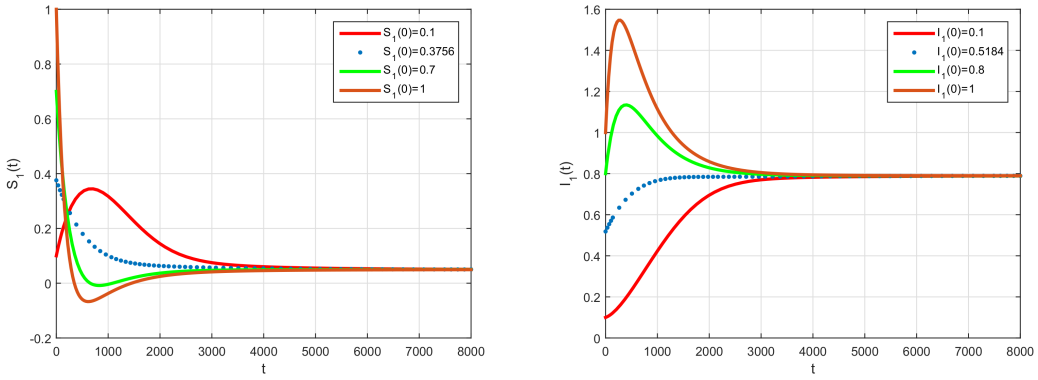


Figure 7. Time series evolution of S_1 and I_1 for system (2), using $\beta_1 = 0.006$, $\beta_2 = 0.00048$, $\mu_1 = 0.0002999, \mu_2 = 0.001609, \alpha_1 = 0.001, \lambda_1 = 0.000265, p = 0.1$, $(R_0 = 67.5446 > 1)$.

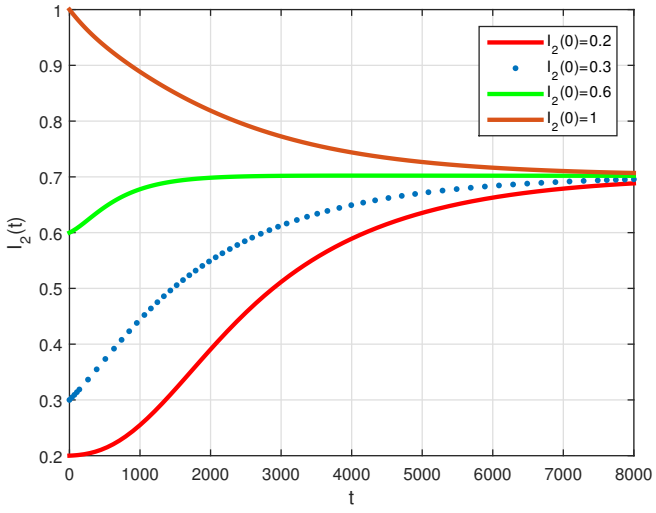


Figure 8. Time series evolution of I_2 for system (2), using $\beta_1 = 0.006$, $\beta_2 = 0.00048$, $\mu_1 = 0.0002999, \mu_2 = 0.001609, \alpha_1 = 0.001, \lambda_1 = 0.000265, p = 0.1$, $(R_0 = 67.5446 > 1)$.

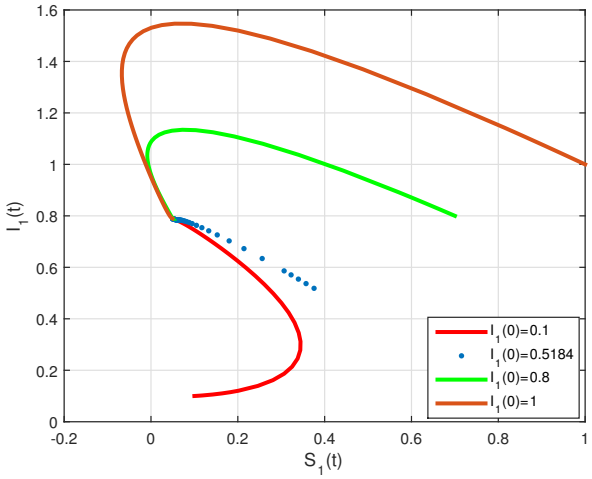


Figure 9. State diagrams of S_1 vs. I_1 for system (2), using $\beta_1 = 0.006, \beta_2 = 0.00048, \mu_1 = 0.0002999,$ $\mu_2 = 0.001609, \alpha_1 = 0.001, \lambda_1 = 0.000265, p = 0.1, (R_0 = 67.5446 > 1)$.

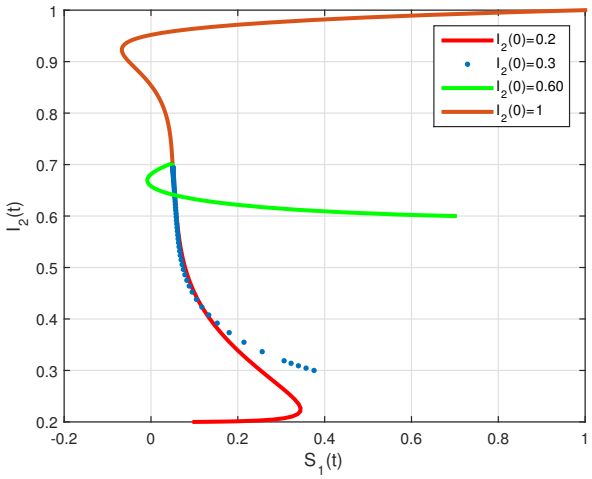


Figure 10. State diagrams of S_1 vs. I_2 for system (2), using $\beta_1 = 0.006, \beta_2 = 0.00048, \mu_1 = 0.0002999,$ $\mu_2 = 0.001609, \alpha_1 = 0.001, \lambda_1 = 0.000265, p = 0.1, (R_0 = 67.5446 > 1)$.

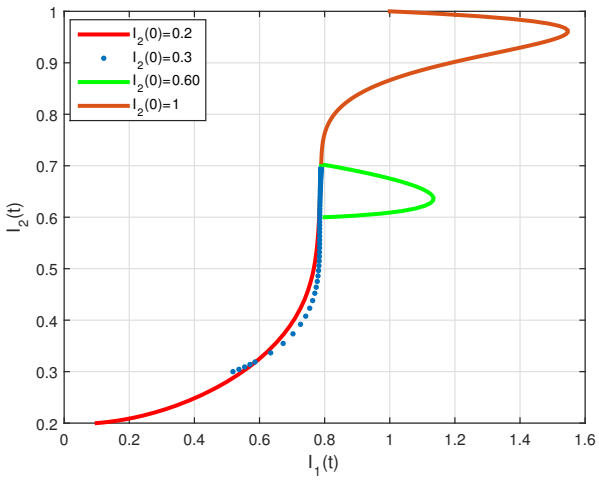


Figure 11. State diagrams of I_1 vs. I_2 for system (2), using $\beta_1 = 0.006, \beta_2 = 0.00048, \mu_1 = 0.0002999,$ $\mu_2 = 0.001609, \alpha_1 = 0.001, \lambda_1 = 0.000265, p = 0.1, (R_0 = 67.5446 > 1)$.

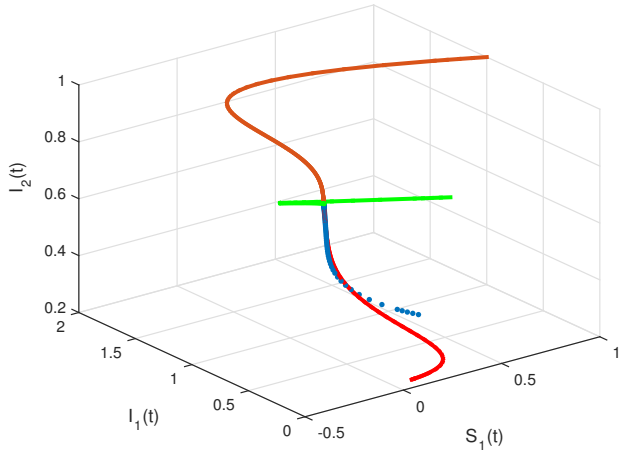


Figure 12. State diagrams of system (2), using $\beta_1 = 0.006, \beta_2 = 0.00048, \mu_1 = 0.0002999$, $\mu_2 = 0.001609, \alpha_1 = 0.001, \lambda_1 = 0.000265, p = 0.1, (R_0 = 67.5446 > 1)$.

5. Conclusions

In the presented work, conditions for the global stability analysis of the Babesiosis disease system are established by the properties of Volterra–Lyapunov stable matrices and the classical method of Lyapunov stability. We used the modified method of Volterra–Lyapunov matrices as mentioned in Remark 1. The main advantage of this modification is that the higher dimensional matrices can be easily implemented. In each step, we reduce the dimensions of the matrix and use the property of the Volterra–Lyapunov matrices. This strategy simplifies the calculations and the proofs. The simulations confirm the results of the theories presented in this method.

Author Contributions: The authors declare that the study was realized in collaboration with equal responsibility. All authors have read and agreed to the published version of the manuscript.

Funding: Not applicable.

Institutional Review Board Statement: Not applicable.

Informed Consent Statement: Not applicable.

Data Availability Statement: Not applicable.

Acknowledgments: The authors express their sincere thanks to the anonymous referees for their rigorous comments and valuable suggestions helping to improve this paper.

Conflicts of Interest: The authors declare no conflict of interest.

References

1. Sadiq, S.F.; Khan, M.A.; Islam, S.; Zaman, G.; Jung, I.H.; Khan, S.A. Optimal control of an epidemic model of leptospirosis with nonlinear saturated incidences. *Annu. Res. Rev. Biol.* **2014**, *4*, 560–576. [CrossRef]
2. Panigoro, H.S.; Suryanto, A.; Kusumawinahyu, W.M.; Darti, I. Dynamics of an eco-epidemic predator-prey model involving fractional derivatives with power-law and Mittag-Leffler kernel. *Symmetry* **2021**, *13*, 785. [CrossRef]
3. Ibrahim, O.M.; Ekundayo, D.D. COVID-19 pandemic in Nigeria: Misconception among individuals, impact on animals and the role of mathematical epidemiologists. *Preprint* **2020**. [CrossRef]
4. Hussain, T.; Ozair, M.; Okosun, K.O.; Ishfagh, M.; Awan, A.U.; Aslam, A. Dynamics of swine influenza model with optimal control. *Adv. Differ. Equ.* **2019**, *2019*, 508. [CrossRef]
5. Rohith, G.; Devika, K.B. Dynamics and control of COVID-19 pandemic with nonlinear incidence rates. *Nonlinear Dyn.* **2020**, *101*, 2013–2026. [CrossRef]
6. Momoh, A.A.; Abdullahi, H.M.; Abimbola, N.G.A.; Michael, A.I. Modeling, optimal control of intervention strategies and cost effectiveness analysis for buruli ulcer model. *Alex. Eng. J.* **2021**, *60*, 2245–2264. [CrossRef]
7. Rezapour, S.; Mohammadi, H.; Jajarmi, A. A new mathematical model for zika virus transmission. *Adv. Differ. Equ.* **2020**, *2020*, 589. [CrossRef]

8. Benz, K.; Rech, C.; Mercorelli, P.; Sergiyenko, O. Two cascaded and extended kalman filters combined with sliding mode control for sustainable management of marine fish stocks. *J. Autom. Mob. Robot. Intell. Syst*. **2020**, *14*, 28–35.
9. World Organisation for Animal Health. *Manual of Diagnostic Tests and Vaccines for Terrestrial Animals*; OIE: Paris, France, 2020.
10. Johnson, N.; Phipps, L.P.; McFadzean, H.; Brlow, A.M. An outbreak of bovine babesiosis in February, 2019, triggered by above average winter temperatures in southern England and co-infection with Babesia divergens and Anaplasma phagocytophilum. *Parasites Vectors* **2020**, *13*, 305. [CrossRef]
11. Solorio-Rivera, J.L.; Rodrıguez-Vivas, R.I. Epidemiology of the babesiosis bovis. II. Epidemiologic indicators and elements for the design of strategies of control. *BioMed* **1997**, *8*, 95–105.
12. Benavides, E. *Considerations with Respect to the Epizootilogia of Anaplasmosis and Babesiosis in the Bovines*; ACOVEZ: Bogotá, Colombia, 1985; Volume 9, pp. 4–11.
13. Hartemink, N.A.; Randolph, S.E.; Davis, S.A.; Heesterbeek, J.A. The basic reproduction number for complex disease systems: Defining R_0 for tick-borne pathogens. *Am. Nat.* **2008**, *171*, 743–754. [CrossRef] [PubMed]
14. Aranda, D.F.; Trejos, D.Y.; Valverde, J.C.; Villanueva, R.J. A mathematical model for Babesiosis disease in bovine and tick populations. *Math. Methods Appl. Sci.* **2012**, *35*, 249–256. [CrossRef]
15. Zafar, Z.U.A.; Rehan, K.; Mushtaq, M. Fractional-order scheme for bovine babesiosis disease and tick populations. *Adv. Differ. Equ*. **2017**, 86. [CrossRef]
16. Dang, Q.A.; Hoang, M.T.; Trejos, D.Y.; Valverde, J.C. Feedback control variables to restrain the Babesiosis disease. *Math. Methods Appl. Sci.* **2019**, *42*, 7517–7527. [CrossRef]
17. Pourbashash, H. Global analysis of the babesiosis disease in bovine and tick populations model and numerical simulation with multistage modified sinc method. *Iran. J. Sci. Technol. Trans. Sci.* **2018**, *42*, 39–46. [CrossRef]
18. Bentaleb, D.; Amine, S. Lyapunov function and global stability for a two-strain SEIR model with bilinear and nonmonotone incidence. *Int. J. Biomath.* **2019**, *12*, 1950021. [CrossRef]
19. Upadhyay, R.K.; Pal, A.K.; Kumari, S.; Roy, P. Dynamics of an SEIR epidemic model with nonlinear incidence and treatment rates. *Nonlinear Dyn.* **2019,** *96*, 2351–2368. [CrossRef]
20. Chen, X.; Cao, J.; Park, J.H.; Qiu, J. Stability analysis and estimation of domain of attraction for the endemic equilibrium of an SEIQ epidemic model. *Nonlinear Dyn.* **2017**, *87*, 975–985. [CrossRef]
21. Liao, S.; Wang, J. Global stability analysis of epidemiological models based on Volterra-Lyapunov stable matrices. *Chaos Solitons Fractals* **2012**, *45*, 966–977. [CrossRef]
22. Masoumnezhad, M.; Rajabi, M.; Chapnevis, A.; Dorofeev, A.; Shateyi, S.; Karga, N.S.; Nik, H.S. An approach for the global stability of mathematical model of an infectious disease. *Symmetry* **2020**, *12*, 1778. [CrossRef]
23. Parsaei, M.R.; Javidan, R.; Kargar, N.S.; Nik, H.S. On the global stability of an epidemic model of computer viruses. *Theory Biosci.* **2017**, *136*, 169–178. [CrossRef] [PubMed]
24. Zahedi, M.S.; Kargar, N.S. The Volterra-Lyapunov matrix theory for global stability analysis of a model of the HIV/AIDS. *Int. J. Biomath*. **2016**, *10*, 1750002. [CrossRef]
25. Driessche, V.D.; Watmough, J. Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission. *Math. Biosci.* **2002**, *180*, 29–48. [CrossRef]
26. Cross, G.W. Three types of matrix stability. *Linear Algebra Appl.* **1978**, *20*, 253–263. [CrossRef]
27. Rinaldi, F. Global stability results for epidemic models with latent period. *IMA J. Math. Appl. Med. Biol.* **1990**, *7*, 69–75. [CrossRef]
28. Redheffer, R. Volterra multipliers I. *SIAM J. Algebr. Discret. Methods* **1985**, *6*, 592–611. [CrossRef]
29. Redheffer, R. Volterra multipliers II. *SIAM J. Algebr. Discret. Methods* **1985**, *6*, 612–623. [CrossRef]