DYNAMICAL ANALYSIS OF A CLASS OF MONKEYPOX EPIDEMIC MODEL

by

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In this paper, we proposed and investigated a class of Monkeypox infectious mathematical model between human and animal populations, with a particular focus on interventions targeting early-exposed population. The model involves a more realistic incidence term and the possible stochastic perturbations. We conducted a detailed mathematical analysis of the corresponding deterministic model, including the existence of solutions to the equations, the existence of equilibria, the basic reproduction number, $\mathcal{R}_0$, and the local stability of equilibria. Then we turned to the stochastic model, and obtained the sufficient conditions of the disease eradication and sustained persistence of the stochastic system. Finally, we conducted numerical simulations to validate the proposed models and validated that the stochastic interaction is a crucial factor for studying the infectious disease. The results indicated that the detection and intervention of early-stage infected individuals have significant impact on the control of the disease transmission.

Key words: *deterministic model, stochastic perturbation, Monkeypox, stability analysis*

Introduction

Monkeypox (Mpox) is a zoonotic disease caused by the Mpox virus which belongs to the Orthopoxvirus genus. This disease is found in rodents such as squirrels, rats and mice rather than monkeys and is transmitted from them to humans [1]. Monkeypox virus (MPXV) can be transmitted from animals to human beings through direct contact with infected animal's fluid, meat, and scratches or bites from animals. The Mpox typically has three phases: incubation, prodrome, and the eruptive stage. The MPXV has an average incubation period of 13 days (range from 5-21 days). The fatality rate ranges from 0-15% [2]. Early signs of Mpox may resemble those of chickenpox but lymphadenopathy is a distinctive feature of Mpox. During the eruptive phase, skin lesions appear in a centrifugal distribution and progress through several stages: macules, papules, vesicles, and finally, pustules [3].

Mathematical models have been proved to enhance our understanding of the spread and control of infectious diseases, especially those capable of capturing the multi-stage aspects of the disease [4]. In recent research work on modelling Mpox transmission [5], the incidence rate of animal to human infection is $\beta_a I_a/N_a$. The author also considered the boundary equilibrium and analyze using numerical methods in [6]. However, the incidence rate from infected-animals to human beings is typically lower than the human-to-human transmission. Therefore,

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using $\beta_a I_a/N_a$ to depict the infection term from animals-to-humans is more appropriate which is rarely used [7]. In practical scenarios, numerous confounding factors affect disease transmission. Many researchers employ stochastic mathematical models to better understand the dynamics of infectious diseases [8]. A minority of references have considered the impact of stochastic factors on propagation. Fractional-order stochastic modelling [9], probabilistic formulation with Levy jumps model are also be used to modelling the transmission of Mpox [10].

In our study, it is considered that the Mpox infection can be more easily traced to early-stage infected individuals due to its typical symptoms. Hence, it's crucial to involve the exposed population and removed population in modelling due to early identity, artificial treatment and isolation measures.

Deterministic model

In this section, we have proposed a deterministic model of Mpox infection while considering early-exposed populations. The human host population is categorized into four compartments: susceptible, $S_h(t)$, exposed in the early stages, $E_h(t)$, infected, $I_h(t)$, and removed, $R_h(t)$. We divided the animal host populations into three variables in model: susceptible populations, $S_a(t)$, infected populations, $I_a(t)$, and removed populations, $R_a(t)$. As humans can be infected by both infected animals and infected humans, sub-humans population, S_h, is decreased by infection via two modes: transmission from infected animals, denoted by $\beta_{a_2} I_a S_a/N_a$, and transmission from infected humans, denoted by $\beta_h I_h S_h/N_h$. We consider the removed rate, γ_1, due to human intervention or isolation. Since exposed animals may not be isolated or treated, the system does not explicitly consider exposed compartments and uniformly classifies them as infected animal populations. Table 1 provides a summary of the parameters and their respective meanings. The transmission diagram is listed in fig. 1. Then, the model can be represented by ODE:

$$\begin{aligned}
S_h' &= \Pi_h - \mu_h S_h - \left(\frac{\beta_{a_2} I_a}{N_a} + \frac{\beta_h I_h}{N_h}\right) S_h \\
E_h' &= \left(\frac{\beta_{a_2} I_a}{N_a} + \frac{\beta_h I_h}{N_h}\right) S_h - \left(\delta_h + \gamma_1 + \mu_h\right) E_h \\
I_h' &= \delta_h E_h - \left(d_h + \gamma_2 + \mu_h\right) I_h \\
R_h' &= \gamma_1 E_h + \gamma_2 I_h - \mu_h R_h \\
S_a' &= \Pi_a - \frac{\beta_{a_1} S_a I_a}{N_a} - \mu_a S_a \\
I_a' &= \frac{\beta_{a_1} S_a I_a}{N_a} - \left(d_a + \gamma_a + \mu_a\right) I_a \\
R_a' &= \gamma_a I_a - \mu_a R_a
\end{aligned} \tag{1}$$

where N_a, N_h are the total population of human and animal, respectively:

$$\begin{aligned}
N_h(t) &= S_h(t) + E_h(t) + I_h(t) + R_h(t) \\
N_a(t) &= S_a(t) + I_a(t) + R_a(t)
\end{aligned} \tag{2}$$

Table 1. Description of population division and the parameter in the system (1)

Variable/ Parameter	Description
N_h	Total population of humans
S_h	Population of susceptible humans
E_h	Population of humans exposed in the early stages
I_h	Population of infected humans
R_h	Population of removed humans
N_a	Total population of animals
S_a	Population of susceptible animals
I_a	Population of infected animals
R_a	Population of removed animals
Π_h/Π_a	Recruitment rate of humans/animals
μ_h/μ_a	The nature death rate of humans/aimals
β_{a_1}	Contact rate from infectious animals to susceptible animals
β_{a_2}	Contact rate from infectious animals to susceptible humans
β_h	Contact rate from infectious humans to susceptible humans
δ_h	Progression rate of exposed humans to the infectious humans
d_h/d_a	Disease-induced death rate of humans/animals
γ_1	Removed rate due to human intervention or isolation
γ_2/γ_a	Recovered rate of infectious humans/animals

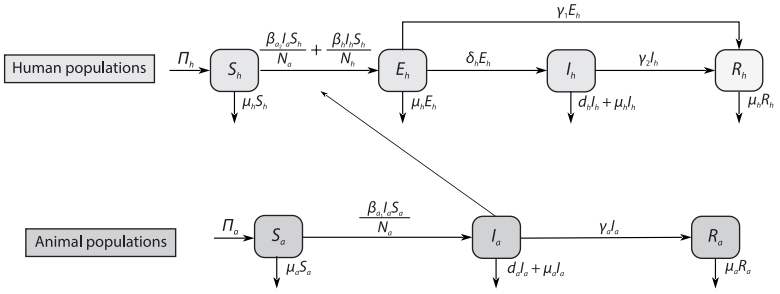

Figure 1. A schematic diagram of the system (1)

It can be verified by examining directions of the vector fields on the boundary of $\mathbb{R}^7_+$ that solutions to system (1) with non-negative initial conditions remain non-negative for $t \geq 0$ and that the system is well defined. It's easy to verified that the limit sets of system (1) are contained in the bounded and positively invariant region:

$$\Gamma = \left\{ \left(S_h, E_h, I_h, R_h, S_a, I_a, R_a\right) \in \mathbb{R}^7_+ \middle| 0 \leq N_h \leq \frac{\Pi_h}{\mu_h},\ 0 \leq N_a \leq \frac{\Pi_a}{\mu_a} \right\} \tag{3}$$

Basic reproduction number

System (1) has three possible equilibria in Γ:

– Disease-free equilibrium $P_0 = (\overline{S}_h, 0, 0, 0, \overline{S}_a, 0, 0) = (\Pi_h/\mu_h, 0, 0, 0, \Pi_a/\mu_a, 0, 0)$.

– Endemic equilibrium $P^* = (S_h^*, E_h^*, I_h^*, R_h^*, S_a^*, I_a^*, R_a^*) > 0$.
– Boundary equilibrium $P_1 = (\tilde{S}_h, \tilde{E}_h, \tilde{I}_h, \tilde{R}_h, \tilde{S}_a, 0, 0)$.

Using the next generation matrix method [11], we define the basic reproduction number of the system (1) as $\mathcal{R}_0 = \max\{\mathcal{R}_h, \mathcal{R}_a\}$, where:

$$\mathcal{R}_h = \frac{\delta_h \beta_h}{(\delta_h + \gamma_1 + \mu_h)(d_h + \gamma_2 + \mu_h)}, \quad \mathcal{R}_a = \frac{\beta_{a_1}}{d_a + \gamma_a + \mu_a}$$

are the basic reproduction numbers of the single human and animal population, respectively. We have observed that the removed rate γ_1 of early-exposed populations impact the basic reproduction number $\mathcal{R}_h$. Therefore, isolating or treating a larger proportion of early-exposed individuals is advantageous for disease control.

The existence and uniqueness of equilibrium

The disease-free equilibrium P_0 is always exist. If $\mathcal{R}_a > 1$, $\mu_h^2 > \beta_{a_2} d_h$, we can let the constant:

$$c = \frac{\beta_{a_2} I_a^*}{N_a^*}$$

then we can obtain that

$$S_a^* = \frac{\left(1 + \dfrac{\gamma_a}{\mu_a}\right)\Pi_a}{\beta_{a_1} - d_a}, \quad I_a^* = \frac{\Pi_a - \mu_a S_a^*}{d_a + \gamma_a + \mu_a}, \quad R_a^* = \frac{\gamma_a}{\mu_a} I_a^*, \quad S_h^* = \frac{\Pi_h}{\mu_h} - \frac{\delta_h + \gamma_1 + \mu_h}{\mu_h} E_h^*$$

$$E_h^* = \frac{d_h + \gamma_2 + \mu_h}{\delta_h} I_h^*, \quad N_h^* = \frac{1}{\mu_h}\left(\Pi_h - d_h I_h^*\right)$$

and I^*_h is the positive root of the eq. (4):

$$-\frac{n}{\mu_h}\left(\beta_h - d_h - m d_h\right) I_h^{*2} + \frac{\Pi_h}{\mu_h}\left(\beta_h - mn - m d_h - n\right) I_h^* + m\frac{\Pi_h^2}{\mu_h} = 0 \tag{4}$$

where

$$n = \frac{(\delta_h + \gamma_1 + \mu_h)(d_h + \gamma_2 + \mu_h)}{\delta_h}, \quad m = \frac{c}{\mu_h}$$

Then we can conclude that the endemic equilibrium P^* is the only equilibrium of (1) in the interior of Γ.

Similar to the previous proof, we will prove the uniqueness of the boundary equilibrium P_1. If $\mathcal{R}_h > 1$, we have that $\beta_h - n > 0$, then:

$$\tilde{I}_h = \frac{\Pi_h(\mathcal{R}_h - 1)}{\beta_h - d}$$

The boundary equilibrium P_1 is the only equilibrium of (1) in the boundary of Γ.

Theorem 1. (1) If $\mathcal{R}_0 < 1$, then the disease-free equilibrium P_0 is locally asymptotically stable in Γ; if $\mathcal{R}_0 > 1$, then the disease-free equilibrium P_0 is unstable. (2) If $\mathcal{R}_h > 1$, $\mathcal{R}_a < 1$, the boundary equilibrium P_1 is locally asymptotically stable.

The proof is provided in the *Appendix A*.

Theorem 2. If $\mathcal{R}_0 \leq 1$, then the disease-free equilibrium P_0 is globally asymptotically stable in Γ. If $\mathcal{R}_0 > 1$ then the disease-free equilibrium P_0 is unstable.

Proof. We choose the Lyapunov function:

$$L = L_1 + L_2 = I_a + \left(E_h + \frac{\delta_h + \gamma_1 + \mu_h}{\delta_h} I_h \right) \tag{5}$$

We can get that $\dot{L} < 0$ if $\mathcal{R}_0 \leq 1$. Furthermore, $\dot{L} = 0 \Leftrightarrow E_h = I_h = R_h = I_a = R_a = 0$ and $S_h = \overline{S}_h$, $S_a = \overline{S}_a$. Therefore, the largest invariant set in the closure $\overline{\Gamma}$ of Γ where $\dot{L} = 0$ is the singleton $\{P_0\}$. By LaSalle's Invariance Principle, P_0 is globally asymptotically stable in Γ, completing the proof.

Stochastic model

In this section, to account for the impact of stochastic factors in the transmission of Mpox, we extended the deterministic system and developed a stochastic perturbation system:

$$\begin{aligned}
\mathrm{d}S_h(t) &= \left[\Pi_h - \mu_h S_h - \left(\frac{\beta_{a_2} I_a}{N_a} + \frac{\beta_h I_h}{N_h} \right) S_h \right] \mathrm{d}t + \sigma_1 S_h \mathrm{d}B_1(t) \\
\mathrm{d}E_h(t) &= \left[\left(\frac{\beta_{a_2} I_a}{N_a} + \frac{\beta_h I_h}{N_h} \right) S_h - (\delta_h + \gamma_1 + \mu_h) E_h \right] \mathrm{d}t + \sigma_2 E_h \mathrm{d}B_2(t) \\
\mathrm{d}I_h(t) &= [\delta_h E_h - (d_h + \gamma_2 + \mu_h) I_h] \mathrm{d}t + \sigma_3 I_h \mathrm{d}B_3(t) \\
\mathrm{d}R_h(t) &= (\gamma_1 E_h + \gamma_2 I_h - \mu_h R_h) \mathrm{d}t + \sigma_4 R_h \mathrm{d}B_4(t) \\
\mathrm{d}S_a(t) &= \left(\Pi_a - \frac{\beta_{a_1} S_a I_a}{N_a} - \mu_a S_a \right) \mathrm{d}t + \sigma_5 S_a \mathrm{d}B_5(t) \\
\mathrm{d}I_a(t) &= \left[\frac{\beta_{a_1} S_a I_a}{N_a} - (d_a + \gamma_a + \mu_a) I_a \right] \mathrm{d}t + \sigma_6 I_a \mathrm{d}B_6(t) \\
\mathrm{d}R_a(t) &= (\gamma_a I_a - \mu_a R_a) \mathrm{d}t + \sigma_7 R_a \mathrm{d}B_7(t)
\end{aligned} \tag{6}$$

where $B_i(t)$ are mutually independent standard Brownian motions and $\sigma_i^2 > 0$, ($i = 1,..., 7$) denote the intensities of the white noise. We also assume that the Brownian motion may fulfil the basic postulates of $B_i(0) = 0$. Some basic theory in the following text can be found in [12], and are omitted here for brevity.

Existence and uniqueness of the positive solution

It can be verified that for any initial value

$$X(0) = (S_h(0), E_h(0), I_h(0), R_h(0), S_a(0), I_a(0), R_a(0)) \in \mathbb{R}_+^7$$

there is a unique positive solution

$$X(t) = [S_h(t), E_h(t), I_h(t), R_h(t), S_a(t), I_a(t), R_a(t)]$$

of stochastic system (6) on $t \geq 0$ and the solution will maintain in $\mathbb{R}_+^7$ with probability one. The method can used by [13].

Extinction of disease

We find the sufficient conditions for the extinction in this section. For simplicity and comfort in reading the next results, we define:

(1) $\mathcal{R}_0 = \max\{2\mathcal{R}_h, \mathcal{R}_a\}, \ f = \max\{0, 1-\mathcal{R}_0\}$

(2) $H_1 = \min\left\{\dfrac{(\delta_h+\gamma_1+\mu_h)(d_h+\gamma_2+\mu_h)}{\delta_h}, d_a+\gamma_a+\mu_a\right\}$

(3) $H_2 = \dfrac{(\delta_h+\gamma_1+\mu_h)(d_h+\gamma_2+\mu_h)}{\delta_h}(2\mathcal{R}_h-1)+(d_a+\gamma_a+\mu_a)(\mathcal{R}_a-1)$

(4) $H_3 = \dfrac{1}{2\left(\dfrac{1}{\sigma_2^2}+\dfrac{1}{\sigma_3^2}+\dfrac{1}{\sigma_6^2}\right)}$

Theorem 3. [11, 12] Let

$$(S_h(t), E_h(t), I_h(t), R_h(t), S_a(t), I_a(t), R_a(t))$$

be the solution of system (6) with any initial value

$$\left[S_h(0), E_h(0), I_h(0), R_h(0), S_a(0), I_a(0), R_a(0)\right] \in \mathbb{R}_+^7$$

we have

$$\lim_{t\to+\infty}\sup\frac{1}{t}\ln\left(E_h(t)+\frac{\delta_h+\gamma_1+\mu_h}{\delta_h}I_h(t)+I_a(t)\right) \le v \quad a.s.$$

where

$$v = H_1 f + H_2 - H_3 \tag{7}$$

Especially, if $v < 0$, then the diseases in E_h, I_h, and I_a go to extinction with probability one, *i.e.*

The proof is provided in the *Appendix B*.

Stationary distribution and ergodicity

In this section, based on the theory of Has'minskii [14], we verify that there is an ergodic stationary distribution, which reveals that the disease will persist.

Defining a parameter:

$$\mathcal{R}_0^s = \mathcal{R}_h^s \cdot \mathcal{R}_a^s \tag{8}$$

where

$$\mathcal{R}_h^s = \frac{2\delta_h\beta_h\mu_h\Pi_h}{(\Pi_h+\Pi_a)\left(\mu_h+\dfrac{\sigma_1^2}{2}\right)\left(\delta_h+\gamma_1+\mu_h+\dfrac{\sigma_2^2}{2}\right)\left(d_h+\gamma_2+\mu_h+\dfrac{\sigma_3^2}{2}\right)}$$

$$\mathcal{R}_a^s = \frac{\beta_{a_1}\mu_a\Pi_a}{(\Pi_h+\Pi_a)\left(\mu_a+\dfrac{\sigma_5^2}{2}\right)\left(d_a+\gamma_a+\mu_a+\dfrac{\sigma_6^2}{2}\right)}$$

Theorem 4. If $\mathcal{R}_0^s > 1$, then for any initial value

$$\left[S_h(0), E_h(0), I_h(0), R_h(0), S_a(0), I_a(0), R_a(0)\right] \in \mathbb{R}_+^7$$

system (6) admits a unique stationary distribution $\pi(.)$ and it has the ergodic property.

The proof is provided in the *Appendix C*.

Numerical simulations

To validate diverse analytical outcomes in our research, we employ numerical methods to investigate both deterministic and stochastic systems. Our simulations consider the initial conditions: $S_h(0) = 120$, $E_h(0) = 0$, $I_h(0) = 0$, $R_h(0) = 0$, $S_a(0) = 150$, $I_a(0) = 30$, and $R_a(0) = 150$. Parameter values are chosen based on those in [5, 6, 10] and summarized in tab. 2.

Table 2. Parameter values of the system (1)

Parameter	Interpretation	values
Π_h	Recruitment rate of humans	10
Π_a	Recruitment rate of animals	10
μ_h	The nature death rate of humans	0.015/0.02/0.015
μ_a	The nature death rate of animals	0.03/0.03/0.03
β_{a_1}	Contact rate from infectious animals to susceptible animals	0.027/0.27/0.27
β_{a_2}	Contact rate from infectious animals to susceptible humans	0.09
β_h	Contact rate from infectious humans to susceptible humans	0.06/0.06/0.2
δ_h	Progression rate of exposed humans to the infectious humans	0.2
d_h	Disease-induced death rate of humans	0.002
d_a	Disease-induced death rate of animals	0.002
γ_1	Removed rate due to human intervention or isolation	0.6/0.06/0.06
γ_2	Recovered rate of infectious humans	0.01/0.01/0.01
γ_a	Recovered rate of infectious animals	0.02/0.2/0.02

In fig. 2 dynamics behaviour comparisons of $[S_h(t), E_h(t), I_h(t), R_h(t), S_a(t), I_a(t), R_a(t)]$ in the deterministic and stochastic system: $\beta_h = 0.06$, $\gamma_1 = 0.6$, $\mu_h = 0.015$, $\gamma_2 = 0.01$, $\beta_{a_1} = 0.027$, $d_a = 0.002$, $\mu_a = 0.03$, $\gamma_a = 0.02$, $\sigma_i = 0.2$ ($i = 1,...,7$), $\mathcal{R}_0 = 0.5453 < 1$, and $v = -0.0217 < 0$ (furthermore $\mathcal{R}_0^s = 0.0147 < 1$). In fig. 2, we can find that the result confirm our hypothesis that effective early intervention in this population can control disease transmission, resulting in the attainment of a disease-free equilibrium. The trend of the disease is similar to that of the deterministic system. In fig. 3, $\beta_h = 0.06$, $\gamma_1 = 0.06$, $\mu_h = 0.02$, $\gamma_2 = 0.01$, $\beta_{a_1} = 0.27$, $d_a = 0.002$, $\mu_a = 0.03$, $\gamma_a = 0.2$, $\sigma_1 = 0.2$, $\sigma_2 = 0.85$, $\sigma_3 = 0.85$, $\sigma_4 = 0.2$, $\sigma_5 = 0.2$, $\sigma_6 = 0.85$, $\sigma_7 = 0.2$, $\mathcal{R}_0 = 1.3393 > 1$ and $v = -0.0072 < 0$ (furthermore $\mathcal{R}_0^s = 0.0032 < 1$). The fig. 3(e) resulting in disease stabilization after reaching a certain threshold. However, due to the significant perturbations introduced by the stochastic system, it allows for disease eradication. Thus, studying stochastic systems is essential for the same infectious disease. From fig. 4, $\beta_h = 0.2$, $\gamma_1 = 0.06$, $\mu_h = 0.015$, $\gamma_2 = 0.01$, $\beta_{a_1} = 0.27$, $d_a = 0.002$, $\mu_a = 0.03$, $\gamma_a = 0.02$, $\sigma_1 = 0.2$, ($i = 1,..., 7$). The $\mathcal{R}_0 = 5.3872 > 1$ and $\mathcal{R}_0^s = 1.3910 > 1$ (furthermore $v = 0.5742 > 0$,) $\mathcal{R}_0^s = 0.0032 < 1$), fig. 4(f), we known that in the stochastic system, when the conditions for disease persistence are met, indicating that the disease will continue to exist.

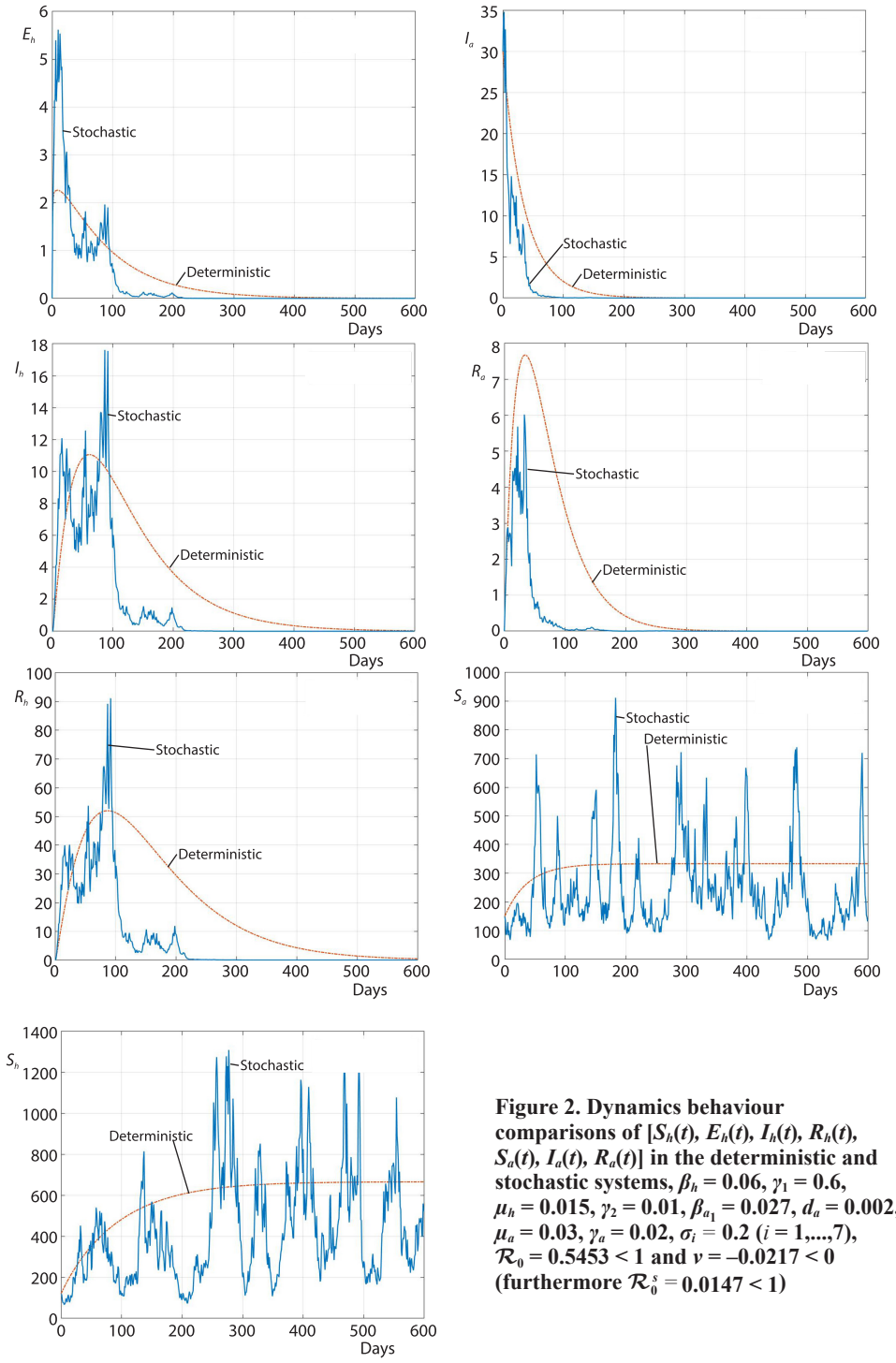

Figure 2. Dynamics behaviour comparisons of [$S_h(t)$, $E_h(t)$, $I_h(t)$, $R_h(t)$, $S_a(t)$, $I_a(t)$, $R_a(t)$] in the deterministic and stochastic systems, $\beta_h = 0.06$, $\gamma_1 = 0.6$, $\mu_h = 0.015$, $\gamma_2 = 0.01$, $\beta_{a_1} = 0.027$, $d_a = 0.002$, $\mu_a = 0.03$, $\gamma_a = 0.02$, $\sigma_i = 0.2$ ($i = 1,...,7$), $\mathcal{R}_0 = 0.5453 < 1$ and $v = -0.0217 < 0$ (furthermore $\mathcal{R}_0^s = 0.0147 < 1$)

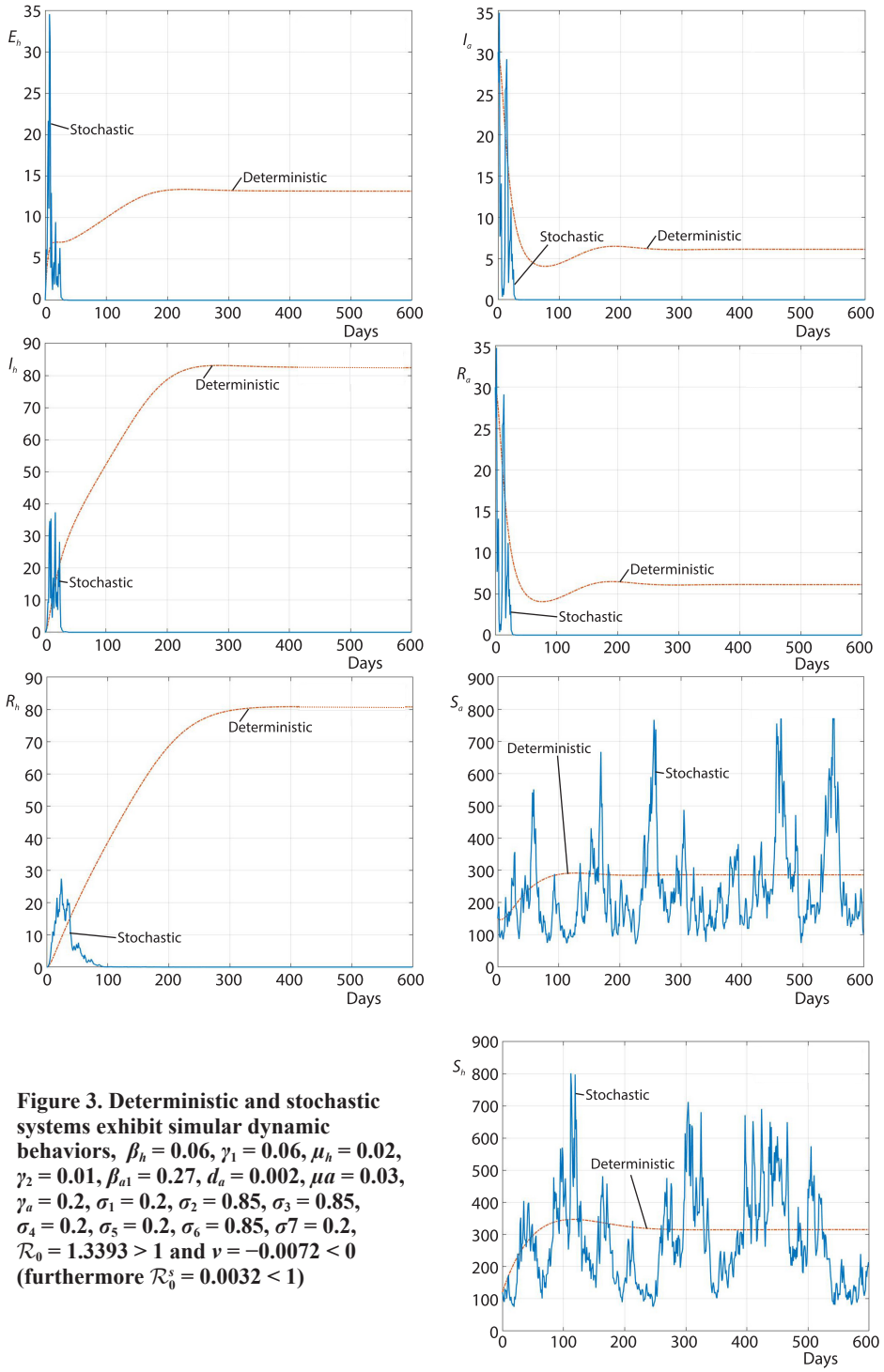

Figure 3. Deterministic and stochastic systems exhibit simular dynamic behaviors, $\beta_h = 0.06$, $\gamma_1 = 0.06$, $\mu_h = 0.02$, $\gamma_2 = 0.01$, $\beta_{a1} = 0.27$, $d_a = 0.002$, $\mu a = 0.03$, $\gamma_a = 0.2$, $\sigma_1 = 0.2$, $\sigma_2 = 0.85$, $\sigma_3 = 0.85$, $\sigma_4 = 0.2$, $\sigma_5 = 0.2$, $\sigma_6 = 0.85$, $\sigma 7 = 0.2$, $\mathcal{R}_0 = 1.3393 > 1$ and $v = -0.0072 < 0$ (furthermore $\mathcal{R}_0^s = 0.0032 < 1$)

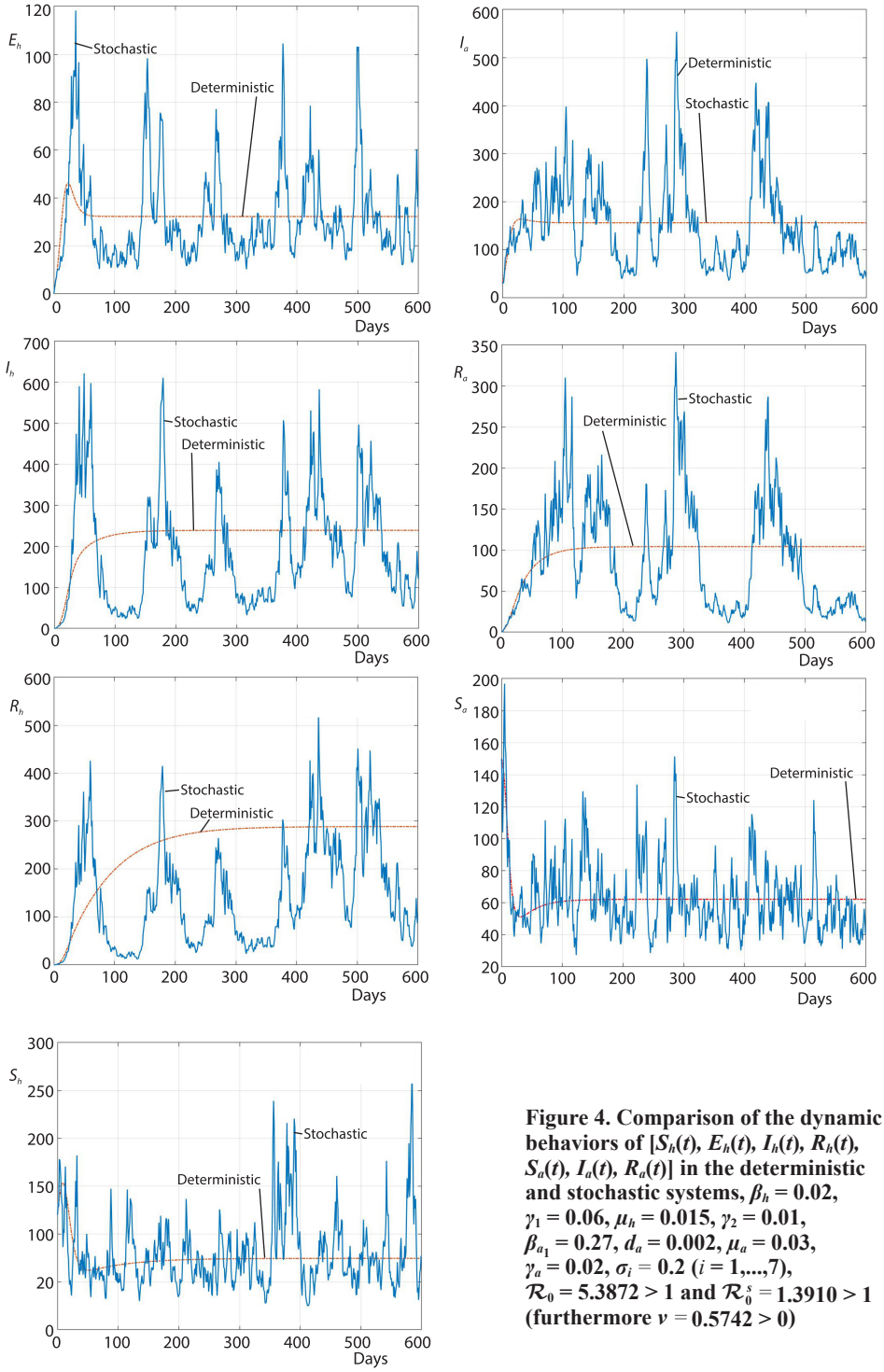

Figure 4. Comparison of the dynamic behaviors of [$S_h(t)$, $E_h(t)$, $I_h(t)$, $R_h(t)$, $S_a(t)$, $I_a(t)$, $R_a(t)$] in the deterministic and stochastic systems, $\beta_h = 0.02$, $\gamma_1 = 0.06$, $\mu_h = 0.015$, $\gamma_2 = 0.01$, $\beta_{a_1} = 0.27$, $d_a = 0.002$, $\mu_a = 0.03$, $\gamma_a = 0.02$, $\sigma_i = 0.2$ ($i = 1,...,7$), $\mathcal{R}_0 = 5.3872 > 1$ and $\mathcal{R}_0^s = 1.3910 > 1$ (furthermore $v = 0.5742 > 0$)

Appendix A
The proof of *Theorem 2*

Proof. The proof of Part (1). The Jacobian matrix at disease-free equilibrium P_0 is:

$$J(P_0)=\begin{pmatrix} -\mu_h & 0 & -\beta_h & 0 & 0 & -\beta_{a_2}\dfrac{\overline{S}_h}{\overline{S}_a} & 0 \\ 0 & -(\delta_h+\gamma_1+\mu_h) & \beta_h & 0 & 0 & \beta_{a_2}\dfrac{\overline{S}_h}{\overline{S}_a} & 0 \\ 0 & \delta_h & -(d_h+\gamma_2+\mu_h) & 0 & 0 & 0 & 0 \\ 0 & \gamma_1 & \gamma_2 & -\mu_h & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -\mu_a & -\beta_{a_1} & 0 \\ 0 & 0 & 0 & 0 & 0 & \beta_{a_1}-(d_a+\gamma_a+\mu_a) & 0 \\ 0 & 0 & 0 & 0 & 0 & \gamma_a & -\mu_a \end{pmatrix} \tag{9}$$

It not difficult to find that the five eigenvalues which are $-\mu_h$, $-\mu_h$, $-\mu_a$, $-\mu_a$, and $\beta_{a_1}-(d_a+\gamma_a+\mu_a)-\mu_a=(\mathcal{R}_a-1)(d_a+\gamma_a+\mu_a)$. Then we can simplify the Jacobian matrix to the following form $J(P_0)_{2\times 2}$ and the characteristic equation is:

$$\begin{aligned}&\lambda^2+(\delta_h+\gamma_1+\mu_h)+(d_h+\gamma_2+\mu_h)\lambda+\\&+(\delta_h+\gamma_1+\mu_h)(d_h+\gamma_2+\mu_h)\left(1-\mathcal{R}_h\right)=0\end{aligned} \tag{10}$$

According to the Vieta's theorem, we can infer that all the eigenvalues of the Jacobian matri $J(P_0)$ are negative if $\mathcal{R}_0<1$. Thus we have derived that the disease-free equilibrium P_0 is locally asymptotically stable by the Routh-Hurwitz condition. If $\mathcal{R}_0>1$, then there exists un-negative eigenvalue, thus P_0 is unstable.

The proof of Part (2). It is known that when $\mathcal{R}_a<1$, the disease will die out in the animal population. Therefore, we only consider the spread of the disease in the human population. Let $\mu dt=d\tau$, we obtain the following system:

$$\begin{aligned}\frac{dE_h}{d\tau}&=\left(c+\frac{\tilde{\beta}_h I_h}{N_h}\right)(N_h-E_h-I_h-R_h)-\omega_1 E_h\\ \frac{dI_h}{d\tau}&=\tilde{\delta}_h E_h-\omega_2 I_h\\ \frac{dR_h}{d\tau}&=\tilde{\gamma}_1 E_h+\tilde{\gamma}_2 I_h-R_h\\ \frac{dN_h}{d\tau}&=\frac{\Pi_h}{\mu_h}-N_h-\tilde{d}_h I_h\end{aligned} \tag{11}$$

where

$$c=\frac{\beta_a I_a}{\mu_h N_a},\ \tilde{\beta}_h=\frac{\beta_h}{\mu_h},\ \tilde{\delta}_h=\frac{\delta_h}{\mu_h},\ \tilde{\gamma}_1=\frac{\gamma_1}{\mu_h},\ \tilde{\gamma}_2=\frac{\gamma_2}{\mu_h}$$

$$\tilde{d}_h=\frac{d_h}{\mu_h},\ \omega_1=(\tilde{\delta}_h+\tilde{\gamma}_1+1),\ \omega_2=(\tilde{d}_h+\tilde{\gamma}_2+1)$$

Similar to the proof of the Part (1), the characteristic equation of $J(P_1)$ is:

$$(\lambda+1)(\lambda^3+A_1\lambda^2+A_2\lambda+A_3)=0 \tag{12}$$

where

$$A_1=\frac{\tilde{\beta}_h\tilde{I}_h}{\tilde{N}_h}+1+\omega_1+\omega_2>0,\quad A_2=(\omega_1+\omega_2)\left(1+\frac{\tilde{\beta}_h\tilde{I}_h}{\tilde{N}_h}\right)>0$$

$$A_3=\frac{\tilde{\beta}_h\tilde{I}_h}{\tilde{N}_h}\left[\tilde{\delta}_h(1+\tilde{\gamma}_2)+\omega_2(1+\tilde{\gamma}_1)+\tilde{\delta}_h\tilde{d}_h\frac{\tilde{E}_h+\tilde{I}_h+\tilde{R}_h}{\tilde{N}_h}\right]>0$$

and we can conclude that $A_1A_2 - A_3 > 0$. The Routh-Hurwitz conditions are satisfied. Thus we have derived that the boundary equilibrium P_1 , which exists if $\mathcal{R}_h > 1$, $\mathcal{R}_a < 1$, is always locally asymptotically stable.

Appendix B

The proof of *Theorem 3*

Proof. Define C^2-function V: $\mathbb{R}^7_+ \to \mathbb{R}$: $V(E_h, I_h, I_a) = E_h + kI_h + I_a$

where

$$k=\frac{\delta_h+\gamma_1+\mu_h}{\delta_h}$$

Since the contact rate between animals and humans is much smaller than the contact rate between people ($\beta_{a_2} < \beta_h$) , in other words, there is a constant $0 < \epsilon \leq 1$ that makes

$$\frac{\beta_{a_2}I_aS_h}{N_a}=\epsilon\frac{\beta_hI_hS_h}{N_h}$$

As we have stated in the previous process, applying Itô's formula to $\ln V$ then we have:

$$\mathrm{d}(\ln V)=L(\ln V)\mathrm{d}t+\frac{1}{V}\left[\sigma_2\mathrm{d}B_2(t)+\sigma_3\mathrm{d}B_3(t)+\sigma_4\mathrm{d}B_4(t)\right] \tag{13}$$

where

$$\begin{aligned}L(\ln V)\leq\frac{1}{V}\left[k(d_h+\gamma_2+\mu_h)I_h\left(2\mathcal{R}_h-1\right)+(d_a+\gamma_a+\mu_a)I_a\left(\mathcal{R}_a-1\right)\right]-\\-\frac{\sigma_2^2E_h^2+\sigma_3^2k^2I_h^2+\sigma_6^2I_a^2}{2V^2}\end{aligned} \tag{14}$$

Obviously, we can find that

$$\frac{I_h}{V}\leq 1,\quad \frac{I_a}{V}\leq 1,\quad \text{and}\quad V^2\leq\left(\frac{1}{\sigma_2^2}+\frac{1}{\sigma_3^2}+\frac{1}{\sigma_6^2}\right)\left(\sigma_2^2E_h^2+\sigma_3^2k^2I_h^2+\sigma_6^2I_a^2\right)$$

Then we can infer that:

$$\mathrm{d}(\ln V)\leq\left(H_1f+H_2-H_3\right)\mathrm{d}t+\frac{1}{V}\left[\sigma_2\mathrm{d}B_2(t)+\sigma_3\mathrm{d}B_3(t)+\sigma_4\mathrm{d}B_4(t)\right] \tag{15}$$

Integrating (13) from 0 to t and then dividing by t on both sides, we have:

$$\frac{\ln V(t)}{t}-\frac{\ln V(0)}{t}\leq H_1f+H_2-H_3+\frac{M_1(t)}{t}+\frac{M_2(t)}{t}+\frac{M_3(t)}{t} \tag{16}$$

where

$$M_1(t)=\int_0^t \frac{\sigma_2 E_h(s)}{V(s)}\mathrm{d}B_2(s), M_2(t)=\int_0^t \frac{\sigma_3 k I_h(s)}{V(s)}\mathrm{d}B_3(s), M_3(t)=\int_0^t \frac{\sigma_6 I_a(s)}{V(s)}\mathrm{d}B_6(s)$$

Taking the superior limit on both sides of eq. (16), we have:

$$\lim_{t\to+\infty}\sup\frac{\ln V(t)}{t}\le v \quad a.s.$$

Again we know that the simple meaning of the above equation implies that:

$$\lim_{t\to+\infty} E_h(t)=0, \quad \lim_{t\to+\infty} I_h(t)=0, \quad \lim_{t\to+\infty} I_a(t)=0 \quad a.s.$$

We can say that when v is negative, the diseases die out with probability one.

Appendix C

The proof of *Theorem 4*

Based on the theory of Has'minskii [14], we assume $X(t)$ be a regular time-homogenous Markov process in E_d as described:

$$\mathrm{d}X(t)=b(X)\mathrm{d}t+\sum_r^k \sigma_r \mathrm{d}B_r(t)$$

The diffusion matrix is defined

$$A(X)=[a_{ij}(x)],\ a_{ij}(x)=\sum_{r=1}^k \sigma_r^i(x)\sigma_r^j(x)$$

Proof. Similar to the lemma used in [8], we can give the diffusion matrix of the system (6).

$$B=\begin{pmatrix}
\sigma_1^2 S_h^2 & 0 & 0 & 0 & 0 & 0 & 0\\
0 & \sigma_2^2 E_h^2 & 0 & 0 & 0 & 0 & 0\\
0 & 0 & \sigma_3^2 I_h^2 & 0 & 0 & 0 & 0\\
0 & 0 & 0 & \sigma_4^2 R_h^2 & 0 & 0 & 0\\
0 & 0 & 0 & 0 & \sigma_5^2 S_a^2 & 0 & 0\\
0 & 0 & 0 & 0 & 0 & \sigma_6^2 I_a^2 & 0\\
0 & 0 & 0 & 0 & 0 & 0 & \sigma_7^2 R_a^2
\end{pmatrix}$$

Choosing

$$M=\min_{(S_h,E_h,I_h,R_h,S_a,I_a,R_a)\in\overline{D}_\sigma\subset\mathbb{R}_+^7}\left\{\sigma_1^2 S_h^2,\sigma_2^2 E_h^2,\sigma_3^2 I_h^2,\sigma_4^2 R_h^2,\sigma_5^2 S_a^2,\sigma_6^2 I_a^2,\sigma_7^2 R_a^2\right\}$$

we can get that

$$\sum_{i,j=1}^7 a_{ij}(S_h,E_h,I_h,R_h,S_a,I_a,R_a)\xi_i\xi_j\ge M\left\|\xi\right\|^2$$

for any

$$(S_h, E_h, I_h, R_h, S_a, I_a, R_a) \in \bar{D}_\sigma, \quad \xi = (\xi_1, \cdots, \xi_7) \in \mathbb{R}_+^7$$

where

$$\bar{D}_\sigma = \left(\frac{1}{k}, k\right) \times \left(\frac{1}{k}, k\right) \times \left(\frac{1}{k}, k\right) \times \left(\frac{1}{k}, k\right) \times \left(\frac{1}{k}, k\right) \times \left(\frac{1}{k}, k\right) \times \left(\frac{1}{k}, k\right)$$

and $k > 1$ is a sufficiently large integer.

Next, we construct C^2-function V_1, V_2, and V_3: $\mathbb{R}_+^7 \to \mathbb{R}$ such as:

$$\begin{aligned} V_1 &= S_h + E_h + I_h + R_h + S_a + I_a + R_a - c_1 \ln S_h - c_2 \ln E_h - c_3 \ln I_h - c_4 \ln S_a - c_5 \ln I_a \\ V_2 &= c_6 V_1 - \ln S_h - \ln E_h - \ln R_h - \ln S_a - \ln I_a - \ln R_a + S_h + E_h + I_h + R_h + S_a + I_a + R_a \\ V_3 &= V_2(S_h, E_h, I_h, R_h, S_a, I_a, R_a) - V_2[S_h(0), E_h(0), I_h(0), R_h(0), S_a(0), I_a(0), R_a(0)] \end{aligned} \tag{17}$$

where c_1-c_6 are some positive constants. Making use of Ito's formula, we have:

$$\begin{aligned} L(V_1) \le -7\left(\frac{c_1\Pi_h}{S_h}\frac{c_2\beta_h I_h S_h}{N_h E_h}\frac{c_3\delta_h E_h}{I_h}\mu_h N_h \frac{c_4\Pi_a}{S_a}\frac{c_5\beta_{a_1} S_a}{N_a}\mu_a N_a\right)^{1/7} + c_1\left(\frac{\beta_{a_2} I_a}{N_a} + \frac{\beta_h I_h}{N_h}\right) + \\ + \Pi_h + \Pi_a + c_1\mu_h + c_2(\delta_h + \gamma_1 + \mu_h) + c_3(d_h + \gamma_2 + \mu_h) + c_4\left(\mu_a + \frac{\sigma_5^2}{2}\right) + \\ + c_4\frac{\beta_{a_1} I_a}{N_a} + \frac{c_1\sigma_1^2}{2} + \frac{c_2\sigma_2^2}{2} + \frac{c_3\sigma_3^2}{2} + c_5\left(d_a + \gamma_a + \mu_a + \frac{\sigma_6^2}{2}\right) \end{aligned} \tag{18}$$

Moreover, we assume that:

$$c_1 = \frac{2(\Pi_h + \Pi_a)}{\mu_h + \frac{\sigma_1^2}{2}}, \quad c_2 = \frac{\Pi_h + \Pi_a}{\delta_h + \gamma_1 + \mu_h + \frac{\sigma_2^2}{2}}, \quad c_3 = \frac{\Pi_h + \Pi_a}{d_h + \gamma_2 + \mu_h + \frac{\sigma_3^2}{2}}$$

$$c_4 = \frac{\Pi_h + \Pi_a}{\mu_a + \frac{\sigma_5^2}{2}}, \quad c_5 = \frac{\Pi_h + \Pi_a}{d_a + \gamma_a + \mu_a + \frac{\sigma_6^2}{2}}$$

Consequently, using the parameter $\mathcal{R}_0^s$ in eq. (18) which take the form:

$$L(V_1) \le -7(\Pi_h + \Pi_a)\left[(\mathcal{R}_0^s)^{1/7} - 1\right] + c_1\left(\frac{\beta_{a_2} I_a}{N_a} + \frac{\beta_h I_h}{N_h}\right) + c_4\frac{\beta_{a_1} I_a}{N_a} \tag{19}$$

The application of the Ito's formula and the use of the system (6) we get:

$$\begin{aligned} LV_3 \le -c_6\kappa + \Pi_h + \Pi_a + 3\mu_h + \gamma_1 + \delta_h + 3\mu_a + d_a + \gamma_a - d_h I_h - \mu_a N_a - d_a I_a + \\ + (c_1 c_6 + 1)\left(\frac{\beta_{a_2} I_a}{N_a} + \frac{\beta_h I_h}{N_h}\right) + (c_4 c_6 + 1)\frac{\beta_{a_1} I_a}{N_a} - \frac{\beta_{a_2} I_a S_h}{N_a E_h} - 2\left(\frac{\mu_h\beta_h S_h I_h}{E_h}\right)^{1/2} - \\ - \frac{\gamma_1 E_h}{R_h} - \frac{\gamma_2 I_h}{R_h} - \frac{\Pi_h}{S_h} - \frac{\Pi_a}{S_a} - \frac{\beta_{a_1} S_a}{N_a} - \frac{\gamma_a I_a}{R_a} + \frac{1}{2}\left(\sigma_1^2 + \sigma_2^2 + \sigma_4^2 + \sigma_5^2 + \sigma_6^2 + \sigma_7^2\right) \end{aligned} \tag{20}$$

where

$$\kappa = 7(\Pi_h + \Pi_a)\left[(\mathcal{R}_0^s)^{1/7} - 1\right] > 0$$

Then we define a set is given:

$$D=\left\{\epsilon_1\le S_h\le\frac{1}{\epsilon_1},\epsilon_2\le E_h\le\frac{1}{\epsilon_2},\epsilon_3\le I_h\le\frac{1}{\epsilon_3},\epsilon_4\le R_h\le\frac{1}{\epsilon_4},\epsilon_5\le S_a\le\frac{1}{\epsilon_5},\epsilon_6\le I_a\le\frac{1}{\epsilon_6},\epsilon_7\le R_a\le\frac{1}{\epsilon_7}\right\}$$

where $\epsilon_i > 0$ (i = 1,...,7) are sufficiently small constants. Let

$$\hat{\omega}=(S_h,E_h,I_h,R_h,S_a,I_a,R_a)$$

then we divide the domain $\mathbb{R}^7_+/D$ into the 13 categories is given:

$$D_1=\left\{\hat{\omega}\in\mathbb{R}^7_+,0<S_h<\epsilon_1\right\},\quad D_2=\left\{\hat{\omega}\in\mathbb{R}^7_+,0<E_h<\epsilon_2,S_h\ge\epsilon_1\right\}$$

$$D_3=\left\{\hat{\omega}\in\mathbb{R}^7_+,0<I_h<\epsilon_3,0<I_a<\epsilon_6,E_h\ge\epsilon_2\right\},\quad D_4=\left\{\hat{\omega}\in\mathbb{R}^7_+,0<R_h<\epsilon_4,I_h\ge\epsilon_3\right\}$$

$$D_5=\left\{\hat{\omega}\in\mathbb{R}^7_+,0<S_a<\epsilon_5\right\},\quad D_6=\left\{\hat{\omega}\in\mathbb{R}^7_+,0<R_a<\epsilon_7,I_a\ge\epsilon_6\right\},\quad D_7=\left\{\hat{\omega}\in\mathbb{R}^7_+,S_h>\frac{1}{\epsilon_1}\right\}$$

$$D_8=\left\{\hat{\omega}\in\mathbb{R}^7_+,E_h>\frac{1}{\epsilon_2}\right\},\quad D_9=\left\{\hat{\omega}\in\mathbb{R}^7_+,I_h>\frac{1}{\epsilon_3}\right\},\quad D_{10}=\left\{\hat{\omega}\in\mathbb{R}^7_+,R_h>\frac{1}{\epsilon_4}\right\}$$

$$D_{11}=\left\{\hat{\omega}\in\mathbb{R}^7_+,S_a>\frac{1}{\epsilon_5}\right\},\quad D_{12}=\left\{\hat{\omega}\in\mathbb{R}^7_+,I_a>\frac{1}{\epsilon_6}\right\},\quad D_{13}=\left\{\hat{\omega}\in\mathbb{R}^7_+,R_a>\frac{1}{\epsilon_7}\right\}$$

Next, we will show that:

$$LV_3(S_h,E_h,I_h,R_h,S_a,I_a,R_a)<0$$

on $\mathbb{R}^7_+$, which is equivalent to proving it on the above 13 domains.

Case 1. If (S_h, E_h, I_h, R_h, S_a, I_a, R_a) $\in D_1$ by eq. (20),

$$\begin{aligned}LV_3&\le\Pi_h+\Pi_a+(c_1c_6+1)(\beta_{a_2}+\beta_h)+(c_4c_6+1)\beta_{a_1}+3(\mu_h+\mu_a)+\gamma_1+\\&+\delta_h+d_a+\gamma_a+\frac{1}{2}(\sigma_1^2+\sigma_2^2+\sigma_4^2+\sigma_5^2+\sigma_6^2+\sigma_7^2)-\frac{\Pi_h}{S_h}\le\\&\le\Pi_h+\Pi_a+(c_1c_6+1)(\beta_{a_2}+\beta_h)+(c_4c_6+1)\beta_{a_1}+3(\mu_h+\mu_a)+\gamma_1+\\&+\delta_h+d_a+\gamma_a+\frac{1}{2}\left(\sigma_1^2+\sigma_2^2+\sigma_4^2+\sigma_5^2+\sigma_6^2+\sigma_7^2\right)-\frac{\Pi_h}{\epsilon_1}\end{aligned}\tag{21}$$

let $\epsilon_1 > 0$ and sufficiently small, so $LV \le 0$ for any (S_h, E_h, I_h, R_h, S_a, I_a, R_a) $\in D_1$.

Cases 2-13 share a similar methodology with *Case 1* . All of them can lead to similar conclusions, and are omitted here for brevity. In conclusion, from all cases it could be noted that for a sufficiently small, ϵ_i, there are:

$$LV_3(S_h,E_h,I_h,R_h,S_a,I_a,R_a)<0\ \text{ for all }\ (S_h,E_h,I_h,R_h,S_a,I_a,R_a)\in\frac{\mathbb{R}^7_+}{D}$$

According to [14], we can obtain that system (6) is ergodic and has a unique stationary distribution. This completes the proof.

Conclusions

This research aimed to investigate monkeypox (Mpox) transmission dynamics between human and animal populations through mathematical modeling. Firstly, we refined a de-

terministic Mpox model (1) by incorporating early-exposed populations, analyzed the existence of solutions, identified three equilibrium points P_0, P^*, P_1, and derived the basic reproduction number $\mathcal{R}_0 = \max\{\mathcal{R}_h, \mathcal{R}_a\}$. Obviously, our study high lights the key role of $\mathcal{R}_0$, which suggests that the disease-free equilibrium is globally asymptotically stable when $\mathcal{R}_0 \leq 1$. Furthermore, we developed a stochastic model (6), which considers the impact of random factors, and provided conditions for disease eradication and persistence. We conducted numerical simulations to compare both the deterministic and stochastic models, and the results indicated that, under significant perturbations, the stochastic model (6) demonstrated the potential for disease extinction, whereas the deter ministic model (1) did not exhibit such a trend. This finding underscores the importance of early detection and intervention strategies, as they may be crucial in mitigating the impact of random factors and preventing disease outbreaks.

Nonetheless, our study is limited in that it primarily focuses on the stability of the disease-free equi librium, neglecting the analysis of endemic stability. Additionally, we relied on parameter settings from previous studies for the numerical simulations. It is crucial to acknowledge these limitations when interpret ing and applying the findings of our research

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Nomenclature

d_a – disease-induced death rate of animals
d_h – disease-induced death rate of humans
E_h – population of exposed in the early stages humans
I_a – population of infected animals
I_h – population of infected humans
N_a – total population of animals
N_h – total population of humans
R_a – population of removed animals
R_h – population of removed humans
S_a – population of susceptible animals
S_h – population of susceptible humans

Greek symbols

β_{a_1} – contact rate from infectious animals to susceptible animals
β_{a_2} – contact rate from infectious animals to susceptible humans
β_h – contact rate from infectious humans to susceptible humans
γ_a – recovered rate of infectious animals
γ_1 – removed rate due to human intervention or isolation
γ_2 – recovered rate of infectious humans
δ_h – progression rate of exposed humans to the infectious humans
μ_a – nature death rate of aimals
μ_h – nature death rate of humans
Π_a – recruitment rate of animals
Π_h – recruitment rate of humans

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