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Mathematical Insights into Zoonotic Disease Spread: Application of the Milstein Method

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Abstract. This study develops a mathematical model to analyze the transmission dynamics of zoonotic diseases between baboons and humans in the Al-Baha region, incorporating key intervention strategies such as sterilization, restricted food access, and reduced human-baboon interactions. The model is formulated using both deterministic and stochastic frameworks to capture the effects of environmental and social variability. Theoretical analysis establishes the existence, uniqueness, and stability of the solutions, while the basic reproduction number R_0 determines conditions for disease eradication or persistence. A stochastic extension, implemented via the Milstein method, accounts for random fluctuations in disease dynamics. Sensitivity analysis identifies critical transmission parameters influencing disease spread, and numerical simulations evaluate the effectiveness of different intervention strategies. The findings provide actionable insights for public health and wildlife management, offering a robust framework for controlling zoonotic disease risks in human-wildlife interactions.

2020 Mathematics Subject Classifications: 92D30, 60H10

Key Words and Phrases: Zoonotic disease transmission, Milstein method, stochastic modeling, basic reproduction number, intervention strategies

1. Introduction

Zoonotic diseases, which are transmitted between animals and humans, pose a significant threat to global public health. Human populations' increasing encroachment into wildlife habitats has escalated zoonotic transmission risk. This is driven by factors such as habitat loss, competition for resources, and direct human-animal interactions [1, 2]. Among wildlife reservoirs, baboons (*Papio spp.*) are particularly noteworthy for their frequent interactions with humans in regions like Al-Baha, Saudi Arabia, where shared use

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of resources exacerbates the potential for disease spillover [3, 4]. Human-baboon interactions, often facilitated by food provisioning from residents and tourists, create conditions for zoonotic disease transmission. Diseases such as tuberculosis and viral infections have been identified as significant risks, spreading through direct contact or contaminated resources [5, 6]. The complexity of these transmission pathways underscores the urgent need for effective intervention strategies to mitigate zoonotic disease risks at human-wildlife interfaces.

Mathematical modeling is a powerful tool for understanding infectious disease dynamics. Deterministic models, which assume a fixed transmission pattern, have traditionally been used to analyze disease propagation [7, 8]. Mathematical modeling has gained significant attention for its applications in engineering [9], plant epidemiology [10], mathematical biology [11], medicine [12], as well as psychological and life sciences [13], viscoelasticity [14], electromagnetic wave propagation [15], quantum dynamics [16], Langevin systems [17], physics [18–26], Diabetes [27–37], Computer Virus [38], tobacco smoking models [39], medicine [40, 41], influenza [42], infectious diseases [43], epidemics [44], cancer [45], coronavirus [46], monkeypox [47], zoonotic viral infections [48], and alcohol-related models [49]. In many disciplines, including medicine, engineering, chemistry, physics, economics, and many more, epidemiology plays an important role, seee [50, 51].

Stochastic models provide a more realistic representation by incorporating randomness into disease transmission processes in biological systems [52–59]. The Michaelis-Menten function, frequently employed in enzyme kinetics, is particularly suitable for modeling the nonlinear degradation rate of insulin, providing a more nuanced understanding of diabetes pathogenesis [60]. By using numerical techniques such as the Milstein method to solve SDEs, these models achieve a high level of accuracy in simulating zoonotic disease dynamics [61, 62].

In this study, we develop a mathematical model to describe the transmission dynamics of zoonotic diseases between baboons and humans, incorporating key control measures such as sterilization, restricted food access, and reduced human-baboon interactions. Extending beyond a deterministic framework, we introduce a stochastic model using the Milstein method to capture the effects of environmental and social variability on disease dynamics. Through theoretical analysis, we establish the fundamental properties of the model, including the existence, uniqueness, and boundedness of solutions. Stability analysis determines the conditions for disease eradication or persistence based on the basic reproduction number, R_0. Sensitivity analysis identifies the most influential transmission parameters, guiding the development of effective intervention strategies. Numerical simulations assess the impact of different control measures, evaluating the effectiveness of sterilization, food access restrictions, and interaction reductions in mitigating disease spread. By integrating deterministic and stochastic approaches, this study provides a comprehensive understanding of zoonotic disease transmission. The incorporation of stochasticity allows for a more realistic representation of disease spread by accounting for environmental and social uncertainties. This research offers actionable insights for public health officials and wildlife managers to design robust, evidence-based strategies for minimizing disease risks in human-baboon interactions.

The primary objective of this study is to develop a compartmental mathematical model that captures the dynamics of zoonotic disease transmission between baboons and humans. The model's fundamental properties, including the existence, uniqueness, and boundedness of solutions, are analyzed to ensure its well-posedness. The basic reproduction number (R_0) is determined, and the local and global stability of both the disease-free and endemic equilibria are examined. To account for environmental and social variability, the model is extended into a stochastic framework using the Milstein method. A sensitivity analysis is conducted to identify key parameters influencing disease spread and control effectiveness. Finally, different intervention strategies—such as sterilization, restricted food access, and reduced human-baboon interactions—are simulated to assess their impact on disease mitigation.

The findings of this study provide insights into optimal public health and wildlife management strategies to mitigate zoonotic disease risks. By integrating deterministic and stochastic approaches, this study offers a robust framework for understanding and controlling zoonotic disease risks at the human-wildlife interface. The findings provide actionable insights for public health officials and wildlife managers, aiding in the design of effective intervention strategies to mitigate zoonotic disease transmission in the Al-Baha region and similar ecological settings.

2. Deterministic model

It aims to simulate baboon and human populations dynamics in the context of zoonotic disease transmission. It incorporates control strategies such as sterilization, restricted food access, and reduced interactions between humans and baboons. We model baboon population dynamics using these equations, which account for natural growth, disease transmission, and control actions. To model baboon and human populations with zoonotic disease transmission, the following equations are used:

$$\begin{aligned}
\frac{dx_1}{dt} &= rx_1\left(1-\frac{x_1+x_2+x_3}{K}\right)-\eta_b x_1x_2-H_s(t)x_1-H_f(t)x_1,\\
\frac{dx_2}{dt} &= \eta_b x_1x_2-\kappa_b x_2-H_s(t)x_2-H_f(t)x_2,\\
\frac{dx_3}{dt} &= \kappa_b x_2-H_f(t)x_3,\\
\frac{dx_4}{dt} &= -\eta_h x_4x_2-H_i(t)x_4,\\
\frac{dx_5}{dt} &= \eta_h x_4x_2-\kappa_h x_5-H_i(t)x_5,\\
\frac{dx_6}{dt} &= \kappa_h x_5.
\end{aligned} \tag{1}$$

The parameters of the model, existed in Table 1, were determined through empirical data, assumptions, and numerical calibration. Epidemiological data from the Al-Baha region informed estimates for key parameters such as the transmission rate from baboons

Parameter	Description	Units
x_1	Susceptible baboons	Individuals
x_2	Infected baboons	Individuals
x_3	Recovered baboons	Individuals
x_4	Susceptible humans	Individuals
x_5	Infected humans	Individuals
x_6	Recovered humans	Individuals
r	Intrinsic growth rate of baboons	1/time
K	Carrying capacity of baboons	Individuals
η_b	Transmission rate among baboons	1/time
η_h	Transmission rate from baboons to humans	1/time
κ_b	Recovery rate of infected baboons	1/time
κ_h	Recovery rate of infected humans	1/time
H_s	Sterilization control measure	1/time
H_f	Food restriction control measure	1/time
H_i	Interaction reduction measure	1/time

Table 1: Definitions of variables and parameters used in the model.

to humans (β_h) and the human recovery rate (γ_h), while control parameters (H_s, H_f, H_i) were based on plausible intervention strategies and validated through sensitivity analysis. Numerical simulations ensured model outputs aligned with observed disease dynamics. Human and baboon populations share similar susceptibility and interaction rates, simplifying analysis while retaining essential disease dynamics. Food access control parameters like (H_f) represent direct contact and indirect pathways, such as contamination of shared food resources. Sterilization (H_s), food restrictions (H_f), and reduced human-baboon interactions (H_i) are considered constant over time. Furthermore, the model assumes a closed system, excluding human and wildlife migration to focus on internal disease dynamics without external influences.

3. Stochastic model

The parameters of the system (1) are all deterministic and do not take into account randomness or environmental fluctuations. It is more appropriate and reasonable to consider how environmental noise affects Zoonotic Disease in a biological environment. Environmental fluctuations affect the propagation of epidemics on the population (see, [63–69]). Therefore, there are various approaches to consider random fluctuations in deterministic systems. One of them assumes that the epidemic is subject to some small and standard random fluctuations. This paper examines the effect of environmental noise on the Zoonotic Disease model (1) by assuming that the stochastic perturbations are white noise in nature and proportional to the variable. Next, we introduce a stochastic Zoonotic

Disease model defined by the following system of differential equations

$$\begin{aligned}
dx_1 &= \Big(rx_1\left(1-\frac{x_1+x_2+x_3}{K}\right)-\eta_b x_1x_2-H_s(t)x_1-H_f(t)x_1\Big)dt+\sigma_1x_1(t)dB_1(t),\\
dx_2 &= \Big(\eta_b x_1x_2-\kappa_b x_2-H_s(t)x_2-H_f(t)x_2\Big)dt+\sigma_2x_2(t)dB_2(t),\\
dx_3 &= \Big(\kappa_b x_2-H_f(t)x_3\Big)dt+\sigma_1S(t)dB_1(t)dt+\sigma_3x_3(t)dB_3(t),\\
dx_4 &= \Big(-\eta_h x_4x_2-H_i(t)x_4\Big)dt+\sigma_4x_4(t)dB_4(t),\\
dx_5 &= \Big(\eta_h x_4x_2-\kappa_h x_5-H_i(t)x_5\Big)dt+\sigma_5x_5(t)dB_5(t),\\
dx_6 &= \Big(\kappa_h x_5\Big)dt+\sigma_6x_6(t)dB_6(t),
\end{aligned} \tag{2}$$

where $B_i(t),(i=1,2,3,4,5,6)$ are a standard Brownian motion (see Definition 1) defined on a complete probability space $\left(\Omega,\mathcal{F},\{\mathcal{F}_t\}_{t\geq 0},\mathbb{P}\right)$ be a complete probability space with a filtration $\{\mathcal{F}_t\}_{t\geq 0}$ satisfying the usual conditions (i.e. it is increasing and right continuous while $\mathcal{F}_0$ contains all $\mathbb{P}$-null sets), σ_i, $(i=1,2,3,4,5,6)$ represent the intensity of noise.

Definition 1 ([70])**.** The positive real-time numbers process $(B(t))_{t\geq 0}$ is called the Wiener process if

(i) $B(0)=0,\mathbb{P}-$ a.s.

(ii) $\forall 0\leq s<t, B(t)-B(s)$ are independent of $\mathcal{F}_s$.

(iii) $\forall 0\leq s<t, B(t)-B(s)\backsim\mathcal{N}(0,t-s)$.

The n-dimensional stochastic differential equation (briefly, SDE) is defined by:

$$dy(t)=F(t,y(t))dt+G(t,y(t))dB(t),\quad with\quad y_0=y(0),$$

where $B(t)$ is a n-dimensional standard Wiener process, F is the drift coefficient, and G is the diffusion coefficient.

4. Existence, uniqueness, non-negativity of the solution

This section provides evidence that model (1) is epidemiologically relevant by demonstrating its non-negativity and boundedness. According to Derrick and Groosman theorem [71], if Lipchitz's condition as in Definition 1 is satisfied, then the solution of model (1) exists and is unique.

Definition 2 ([72])**.** *In system (1), $\vec{f}$ satisfies Lipchitz's condition if there exists a positive constant k, such as*

$$\left\|\vec{f}\left(\vec{X}_1\right)-\vec{f}\left(\vec{X}_2\right)\right\|<k\left\|\vec{X}_1-\vec{X}_2\right\|,\forall\vec{X}_1,\vec{X}_2\in\Omega.$$

The following theorem guarantee the existence and uniqueness of solution of model (1).

Theorem 1. *Under non-negative initial conditions, the model (1) is unique in* Ω *for all* $t \geq 0$.

Proof. The right side of model (1) can be written as follows.

$$\begin{aligned}
f_1 &= rx_1\left(1 - \frac{x_1 + x_2 + x_3}{K}\right) - \eta_b x_1 x_2 - H_s(t)x_1 - H_f(t)x_1,\\
f_2 &= \eta_b x_1 x_2 - \kappa_b x_2 - H_s(t)x_2 - H_f(t)x_2,\\
f_3 &= \kappa_b x_2 - H_f(t)x_3,\\
f_4 &= -\eta_h x_4 x_2 - H_i(t)x_4,\\
f_5 &= \eta_h x_4 x_2 - \kappa_h x_5 - H_i(t)x_5,\\
f_6 &= \kappa_h x_5.
\end{aligned}$$

Then, it can be shown that $\frac{\partial f_i}{\partial x_j}$ is continuous and $\left|\frac{\partial f_i}{\partial x_j}\right| < \infty$ for all $i, j = 1, 2, .., 6$. As a result of Derrick and Groosman theorem in [71], the system (1) is Lipchitz's unique solution.

Theorem 2. *The solutions of model (1) are all non-negative and ultimately bound under the assumption that the initial values are non-negative.*

Proof. Let us consider the model equations. Since $f_1, f_2, f_3, f_4, f_5, f_6$ are constructed from non-negative parameters and functions, we conclude that if the initial conditions are non-negative, then the solution remains non-negative for all $t \geq 0$. In other words, we analyze the equations for the fractional-order baboon-human population model at the boundary conditions where the population variables are zero:

$$\begin{aligned}
\dot{x}_1(\mathtt{t})\big|_{x_1=0} &= 0,\\
\dot{x}_2(\mathtt{t})\big|_{x_2=0} &= 0,\\
\dot{x}_3(\mathtt{t})\big|_{x_3=0} &= \gamma_{\mathtt{b}} x_2 \geq 0,\\
\dot{x}_4(\mathtt{t})\big|_{x_4=0} &= -\beta_{\mathtt{h}} x_4 x_2 \geq 0,\\
\dot{x}_5(\mathtt{t})\big|_{x_5=0} &= \beta_{\mathtt{h}} x_4 x_2 \geq 0,\\
\dot{x}_6(\mathtt{t})\big|_{x_6=0} &= \gamma_{\mathtt{h}} x_5 \geq 0.
\end{aligned}$$

From these equations, we can deduce that for any non-negative initial conditions, the deterministic model ensures that the solution remains non-negative for all $t \geq 0$. By employing similar techniques as demonstrated in Lemmas 5 and 6 in [73], it follows that the baboon-human population model solutions are non-negative.

Theorem 3. *The model (1) possesses uniformly bounded solutions within the region defined as:*

$$\mathcal{B} = \left\{ (x_1, x_2, x_3, x_4, x_5, x_6) \in \mathbb{R}^6_+ \cup \overrightarrow{0} : N = x_1 + x_2 + x_3 + x_4 + x_5 + x_6 \leq \frac{\mathtt{rK}}{\lambda} \right\}.$$

Proof. Let the total population at time t be defined as:

$$\mathtt{N(t)} = x_1(\mathtt{t}) + x_2(\mathtt{t}) + x_3(\mathtt{t}) + x_4(\mathtt{t}) + x_5(\mathtt{t}) + x_6(\mathtt{t}).$$

By summing the equations of the baboon-human population model, we arrive at:

$$\frac{dN(t)}{dt} = \mathtt{r}x_1 \left(1 - \frac{x_1 + x_2 + x_3}{\mathtt{K}}\right) - \mathrm{H}_s(\mathtt{t})x_1 - \mathrm{H}_f(\mathtt{t})x_1 - \mathrm{H}_i(\mathtt{t})x_4 - \gamma_{\mathtt{b}}x_2 - \gamma_{\mathtt{h}}x_5.$$

This leads to:

$$\frac{dN(t)}{dt} \leq \mathtt{rK} - \mu\mathtt{N(t)}, \tag{3}$$

where μ represents the minimum of the death rates and control actions.

The solution of the equation (3) satisfies

$$N(t) \leq \frac{\mathtt{rK}}{\mu} + \left(N(0) - \frac{\mathtt{rK}}{\mu}\right) \exp(-\mu t),$$

where $N(0)$ is the initial value. It is clear that

$$\lim_{t\to\infty} N(t) \leq 0,$$

and thus $N(t)$ is bounded with $N(t) \leq \frac{\mathtt{rK}}{\mu}$. Hence, we can see that the feasible region of model (1) is $\mathcal{B}$ which is positively invariant region.

Remark 1. *The region $\mathcal{B}$ is positively invariant concerning the baboon-human population model.*

Theorem 4. *Consider the stochastic differential equation (SDE) system:*

$$dX_t = f(X_t)dt + g(X_t)dW_t,$$

where $X_t \in \mathbb{R}^n$ is the state vector, W_t is an m-dimensional Wiener process, and the functions $f : \mathbb{R}^n \to \mathbb{R}^n$ and $g : \mathbb{R}^n \to \mathbb{R}^{n\times m}$ satisfy the following conditions:

(i) (Lipschitz Continuity) There exists a constant $L > 0$ such that for all $x, y \in \mathbb{R}^*n$,

$$\|f(x) - f(y)\| + \|g(x) - g(y)\| \leq L\|x - y\|.$$

(ii) (Linear Growth Condition) There exists a constant $C > 0$ such that for all $x \in \mathbb{R}^n$,

$$\|f(x)\|^2 + \|g(x)\|^2 \leq C(1 + \|x\|^2).$$

Under these conditions, there exists a unique global solution X_t for all $t \geq 0$, and if the initial condition $X_0 \geq 0$, then $X_t \geq 0$ almost surely for all $t \geq 0$.

Proof. The existence and uniqueness of a global solution follow from the standard results of stochastic differential equations based on the Lipschitz continuity and linear growth conditions (see, e.g., [74]).

To prove the nonnegativity of the solution, we apply Itô's formula to the function $\phi(X_t) = \max(0, X_t)$ component-wise. Since the drift term $f(X_t)$ and the diffusion term $g(X_t)$ are assumed to preserve nonnegativity (i.e., they do not push the solution into negative values if started from a nonnegative initial condition), we conclude that X_t remains nonnegative almost surely. A more rigorous approach involves considering a stopping time $\tau_\epsilon = \inf\{t \geq 0 : X_t \ngeq 0\}$ and using a contradiction argument, showing that $\mathbb{P}(\tau_\epsilon < \infty) = 0$.

Theorem 5. *For any initial value $(S_b(0), I_b(0), R_b(0), S_h(0), I_h(0), R_h(0)) \in \mathbb{R}^6_+$, there is a unique solution $(S_b(t), I_b(t), R_b(t), S_h(t), I_h(t), R_h(t))$ to the model (2). The solution will remain in $\mathbb{R}^6_+$ with probability one, namely $(S_b(t), I_b(t), R_b(t), S_h(t), I_h(t), R_h(t)) \in \mathbb{R}^6_+$ for all $t \geq 0$ almost surely (briefly a.s.).*

By using the same steps of proof of Theorem 2.1 in [75] we can prove the above theorem.

5. Equilibrium points and their stability analysis

At equilibrium, we solve for x_1 and x_4, leading to the infection-free equilibrium of model (1):

$$E_0 = (x_1^0, 0, 0, x_4^0, 0, 0),$$

where:

$$x_1^0 = \frac{Kr}{H_s + H_f}, \quad x_4^0 = \frac{K}{H_i}.$$

The basic reproduction number $\mathscr{R}_0$ is given by:

$$\mathscr{R}_0 = \frac{\eta_b x_1^0}{\kappa_b + H_s} + \frac{\eta_h x_4^0}{\kappa_h + H_i}.$$

Now, we prove the local and global stability of the infection-free equilibrium point.

Lemma 1. *$E_0 = (x_1^0, 0, 0, x_4^0, 0, 0)$ is locally asymptotically stable in Ω if $\mathscr{R}_0 < 1$, and unstable if $\mathscr{R}_0 > 1$.*

Proof. For the baboon-human population model at $E_0 = (x_1^0, 0, 0, x_4^0, 0, 0)$, the Jacobian matrix $J(E_0)$ is given by:

$$J(E_0) = \begin{bmatrix} -r & 0 & -\eta_b x_1^0 & 0 & 0 & 0 \\ 0 & -H_i & 0 & -\eta_h x_4^0 & 0 & 0 \\ 0 & 0 & \eta_b x_1^0 - (\kappa_b + H_s) & 0 & 0 & 0 \\ 0 & 0 & 0 & \eta_h x_4^0 - (\kappa_h + H_i) & 0 & 0 \\ 0 & 0 & 0 & 0 & -H_f & 0 \\ 0 & 0 & 0 & 0 & 0 & -H_f \end{bmatrix}.$$

The characteristic equation is obtained by solving $\det(J(E_0) - \kappa I) = 0$, where κ is the eigenvalue:

$$\det\left(J(E_0) - \kappa I\right) = 0.$$

The characteristic equation becomes:

$$(\kappa + r)(\kappa + H_i)(\kappa - \eta_b x_1^0 + \kappa_b + H_s)(\kappa - \eta_h x_4^0 + \kappa_h + H_i) = 0.$$

Thus, the eigenvalues are:

$$\kappa_1 = -r, \quad \kappa_2 = -H_i, \quad \kappa_3 = \eta_b x_1^0 - (\kappa_b + H_s), \quad \kappa_4 = \eta_h x_4^0 - (\kappa_h + H_i).$$

For stability, all eigenvalues must have negative real parts. κ_1 and κ_2 are always negative. The eigenvalues κ_3 and κ_4 depend on the basic reproduction number $\mathscr{R}_0$:

$$\mathscr{R}_0 = \frac{\eta_b x_1^0}{\kappa_b + H_s} + \frac{\eta_h x_4^0}{\kappa_h + H_i}.$$

If $\mathscr{R}_0 < 1$, then $\kappa_3 < 0$ and $\kappa_4 < 0$, and E_0 is locally asymptotically stable. If $\mathscr{R}_0 > 1$, then $\kappa_3 > 0$ or $\kappa_4 > 0$, and E_0 is unstable.

Lemma 2. *E_0 in Ω is globally asymptotically stable if $\mathscr{R}_0 < 1$, and unstable if $\mathscr{R}_0 > 1$.*

Proof. To establish the global asymptotic stability, we construct an appropriate Lyapunov function:

$$L = x_2 + x_5,$$

where x_2 and x_5 represent the infected populations of baboons and humans, respectively.

The time derivative of L can be expressed as:

$$\frac{dL}{dt} = \frac{dx_2}{dt} + \frac{dx_5}{dt}.$$

Using the model equations for the dynamics of x_2 and x_5:

$$\frac{dx_2}{dt} = \eta_b x_1 x_2 - (\kappa_b + H_s)x_2,$$
$$\frac{dx_5}{dt} = \eta_h x_4 x_2 - (\kappa_h + H_i)x_5,$$

we obtain:

$$\frac{dL}{dt} = \left(\eta_b x_1 - (\kappa_b + H_s)\right) x_2 + \left(\eta_h x_4 - (\kappa_h + H_i)\right) x_5.$$

At the infection-free equilibrium, we have $x_1 = x_1^0$ and $x_4 = x_4^0$. Thus, the derivative simplifies to:

$$\frac{dL}{dt} = \left(\eta_b x_1^0 - (\kappa_b + H_s)\right) x_2 + \left(\eta_h x_4^0 - (\kappa_h + H_i)\right) x_5.$$

If $\mathscr{R}_0 < 1$, then it follows that:

$$\frac{dL}{dt} \leq 0.$$

The largest invariant set where $\frac{dL}{dt} = 0$ corresponds to the infection-free equilibrium E_0. Therefore, by LaSalle's Invariance Principle, E_0 is globally asymptotically stable if $\mathscr{R}_0 < 1$.

Now, we prove the local and global stability of the endemic equilibrium point. For $I^{>}0$, $E^* = (x_1^*, x_2^*, x_3^*, x_4^*, x_5^*, x_6^*)$ is the endemic equilibrium point and is defined as the steady-state solution for the model (1).

$$\frac{dx_i}{dt} = 0, \quad i = 1, 2, 3, 4, 5, 6.$$

From the system equations, we can use the second and third equations to express x_2^* and x_3^*:

$$x_3^* = \frac{\kappa_b}{H_f} x_2^*.$$

Substituting this expression into the fourth equation yields:

$$x_5^* = \frac{\eta_h x_4^*}{\kappa_h + H_i} x_2^*.$$

Next, substituting into the first equation allows us to solve for x_1^* as:

$$x_1^* = \frac{Kr}{H_s} + \frac{\eta_b \, \mathrm{a}_2}{H_s} x_2^*.$$

Thus,

$$E^* = (x_1^*, x_2^*, x_3^*, x_4^*, x_5^*, x_6^*).$$

Lemma 3. *If $\mathscr{R}_0 > 1$, E^* is locally asymptotically stable in Ω.*

Proof. For the first-order integer model (1), the Jacobian matrix $J(E^*)$ at the endemic equilibrium point E^* is given by:

$$J(E^*) = \begin{bmatrix} -r & 0 & -\eta_b x_1^* & 0 & 0 & 0 \\ 0 & -H_i & 0 & -\eta_h x_4^* & 0 & 0 \\ 0 & 0 & \kappa_b - H_f & 0 & 0 & 0 \\ 0 & 0 & 0 & \kappa_h - H_i & 0 & 0 \\ 0 & 0 & 0 & 0 & -H_f & 0 \\ 0 & 0 & 0 & 0 & 0 & -H_f \end{bmatrix}.$$

Evaluating the Jacobian matrix at E^* gives the characteristic equation:

$$P(\xi) = \det\left(\xi I - J(E^*)\right) = 0,$$

where I is the identity matrix. The characteristic polynomial is:

$$P(\xi) = \xi^6 + \mathrm{a}_1 \, \xi^5 + \mathrm{a}_2 \, \xi^4 + \mathrm{a}_3 \, \xi^3 + \mathrm{a}_4 \, \xi^2 + \mathrm{a}_5 \, \xi + \mathrm{a}_6,$$

where the coefficients $a_1, a_2, \ldots, a_6$ are defined in terms of the parameters of the model (1).

If the Routh-Hurwitz criterion is satisfied, the endemic equilibrium is locally stable:

$$a_1 > 0, \quad a_2 > 0, \quad a_3 > 0, \quad a_4 > 0, \quad a_5 > 0, \quad a_6 > 0.$$

Thus, $E^*\ast$ is locally asymptotically stable when $\mathscr{R}_0 > 1$, as all conditions of the Routh-Hurwitz criterion are satisfied.

Lemma 4. *E^* is globally asymptotically stable if $\mathscr{R}_0 > 1$.*

Proof. We construct a suitable Lyapunov function L to prove the global asymptotic stability of $E^*\ast$:

$$L = (x_1 - x_1^*) + (x_2 - x_2^*) + (x_3 - x_3^*) + (x_4 - x_4^*) + (x_5 - x_5^*) + (x_6 - x_6^*).$$

As a result

$$\frac{dL}{dt} = \frac{dx_1}{dt} + \frac{dx_2}{dt} + \frac{dx_3}{dt} + \frac{dx_4}{dt} + \frac{dx_5}{dt} + \frac{dx_6}{dt}.$$

Thus

$$\frac{dx_1}{dt} = rx_1\left(1 - \frac{x_1 + x_2 + x_3}{K}\right) - \eta_b x_1 x_2,$$

and similarly for the other variables. At equilibrium, the terms involving x_2, x_5, and x_3 vanish, leading to:

$$\frac{dL}{dt} \leq 0.$$

Thus, $\frac{dL}{dt} = 0$ if and only if $x_1 = x_1^*$, $x_2 = x_2^*$, $x_3 = x_3^*$, $x_4 = x_4^*$, $x_5 = x_5^*$, and $x_6 = x_6^*$. E^* is asymptotically stable under the LaSalle's Invariance Principle when $\mathscr{R}_0 > 1$.

6. An optimal control of the proposed model

In this section, we extend the baboon-human population model (1) into an optimal control framework to determine the most effective intervention strategies for disease eradication over a finite time period. The extended model (1) introduces three control variables, each representing a different intervention method:

- $u_1(t)$: Preventive measures to reduce contact between susceptible and infected individuals.
- $u_2(t)$: Treatment efforts aimed at reducing the number of infectious individuals.
- $u_3(t)$: Screening efforts to identify and isolate infected individuals early.

The inclusion of these controls modifies the original baboon-human disease transmission model (1) into the following system of differential equations:

$$\begin{aligned}
\frac{dx_1}{dt} &= rx_1\left(1-\frac{x_1+x_2+x_3}{K}\right)-\frac{(1-\mathrm{u}_1)\eta_b x_1 x_2}{N}-H_s x_1,\\
\frac{dx_2}{dt} &= \frac{(1-\mathrm{u}_1)\eta_b x_1 x_2}{N}-(\mathrm{u}_2+\kappa_b)x_2,\\
\frac{dx_3}{dt} &= \mathrm{u}_2\, x_2 - H_f x_3,\\
\frac{dx_4}{dt} &= -\frac{(1-\mathrm{u}_1)\eta_h x_4 x_2}{N}-H_i x_4,\\
\frac{dx_5}{dt} &= \frac{(1-\mathrm{u}_1)\eta_h x_4 x_2}{N}-(\mathrm{u}_2+\kappa_h)x_5,\\
\frac{dx_6}{dt} &= \mathrm{u}_2\, x_5 - H_f x_6,
\end{aligned} \tag{4}$$

where $N = x_1 + x_2 + x_3 + x_4 + x_5 + x_6$ represents the total population of baboons and humans. The control variables $\mathrm{u}_1, \mathrm{u}_2$, and u_3 are bounded between 0 and 1, representing the proportion of effort applied in each intervention strategy.

To determine the optimal control strategy, we aim to minimize the number of infected individuals while balancing the costs associated with implementing the control measures. The objective functional $J(\mathrm{u}_1, \mathrm{u}_2, \mathrm{u}_3)$ is defined as:

$$J = \min_{\mathrm{u}_1,\mathrm{u}_2,\mathrm{u}_3} \in \mathrm{t}_0^*\, \mathrm{t}_f \left(b_1 x_2 + b_2 x_5 + \frac{1}{2}\sum_{i=1}^{*} 3 w_i u_i^2\right) dt,$$

where:

- b_1 and b_2 are weights that reflect the relative importance of reducing the infected baboon and human populations, respectively.
- w_1, w_2, w_3 represent the costs associated with each control measure (prevention, treatment, and screening).
- The quadratic terms $\frac{1}{2} w_i u_i^2$ capture the increasing cost of higher control efforts.

Our objective is to determine which control $\mathrm{u}_1^*, \mathrm{u}_2^*, \mathrm{u}_3^*$ that minimize J.

Given these conditions, an optimal control $(\mathrm{u}_1^{,}\, \mathrm{u}_2^{,}\, \mathrm{u}_3^{)}$ exists, ensuring that the objective functional J is minimized.

We examine the following control scenarios to evaluate the impact of different interventions on the disease dynamics:

- **Control with Prevention Only:** Preventing with u_1 without treatment ($\mathrm{u}_2 = 0$) and screening ($\mathrm{u}_3 = 0$). The results show a significant reduction in the infected population, as prevention reduces contact between susceptible and infected individuals. Implementing only prevention measures reduces the contact rate between susceptible and infected individuals, leading to a significant reduction in infections.

- **Control with Treatment Only:** Using only the treatment effort u_2 without prevention ($u_1 = 0$) and screening ($u_3 = 0$). The treatment reduces the number of infectious individuals initially, but without prevention, new infections still occur. Treatment efforts (u_2) decrease the number of infectious individuals, but without prevention, the infection still spreads.

- **Control with Screening Only:** Using only the screening effort u_3, without prevention or treatment. Screening helps identify infected individuals and reduce the spread of the disease, but without prevention, new infections occur. Screening efforts (u_3) help to isolate infected individuals but are less effective without simultaneous prevention measures.

- **Control with Prevention and Treatment:** Using both prevention (u_1) and treatment (u_2) without screening. This strategy effectively reduces both infectious individuals and intervention costs. The combination of prevention and treatment significantly reduces infection and is more cost-effective than either measure alone.

- **Control with Prevention, Treatment, and Screening:** Using all three control measures (u_1, u_2, u_3) together. The results show that this strategy provides the most effective disease control, with the number of infected individuals rapidly declining. The combined use of prevention, treatment, and screening provides the best outcome, rapidly decreasing the number of infections.

The extension of the baboon-human population model (4) into an optimal control framework allows us to identify the most effective strategies for disease prevention and treatment. By simulating different combinations of control measures, we can minimize both the number of infected individuals and the associated costs. Implementing all three control measures simultaneously provides the best outcomes for disease eradication.

The optimization output provides the best values for control parameters u_1, u_2, u_3, which are used to define the time-dependent control terms $H_s(t), H_f(t)$, and $H_i(t)$ in the differential equations. These controls, applied to susceptible, infected, and recovered populations, directly influence the disease dynamics between baboons and humans. As the optimization progresses, the objective function J decreases, indicating effective control measures that reduce infection rates while adhering to model (1) constraints. The convergence in the optimality column shows that the algorithm finds near-optimal control values, ensuring minimal disease transmission between the populations, thereby achieving the goal of the model (4).

Figure 1: Population Dynamics with Different Control Scenarios. Subfigures (a-f): These subfigures depict the dynamics of compartments for baboons (x_1, x_2, x_3) and humans (x_4, x_5, x_6) under varying intensities of control measures H_s, H_f, and H_i.

- **(a)-(d):** Moderate control intensities ($H_s = 0.1, H_i = 0.01$, varying H_f). These scenarios show gradual reductions in infected populations, demonstrating the effectiveness of sterilization and food restriction.

- **(e)-(f):** Higher control intensities ($H_s = 0.5, H_f = 0.5$) lead to significant declines in infections, highlighting the impact of increased intervention efforts.

Increased control measures reduce infection rates in both populations. Sterilization (H_s) and food restriction (H_f) significantly affect infected populations, while interaction reduction (H_i) supports prevention. Figure 1: Baseline Dynamics of Populations with Different

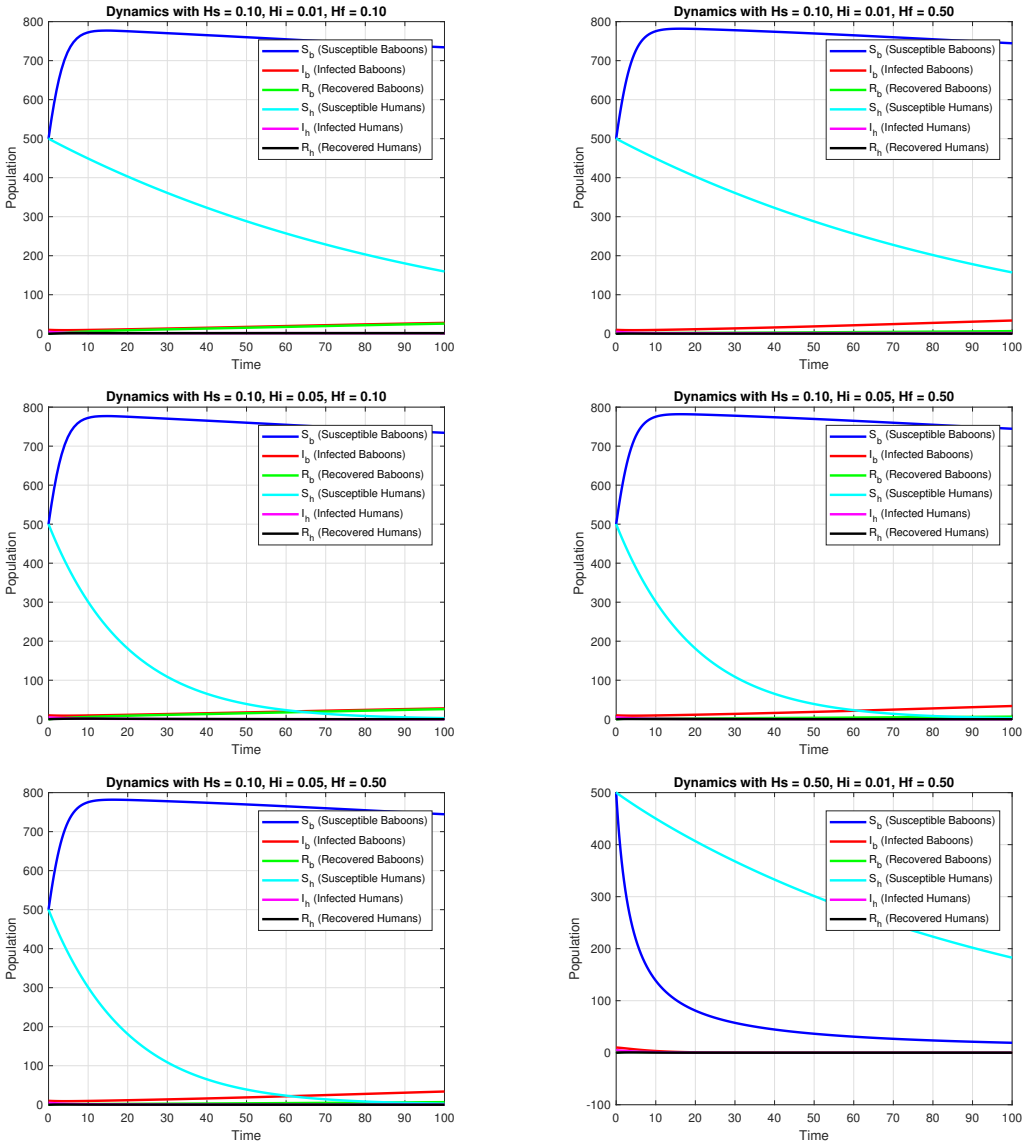

Figure 1: Comparision between the deterministic and stochastic model (2) for $x_1(t)$ and $x_4(t)$.

Control Scenarios. Figures 1(a) to 1(f) depict the population dynamics for compartments of baboons and humans over time with various values of H_s, H_f, and H_i: Subfigures (a)-(d) display scenarios with moderate control intensities (e.g., $H_s = 0.1$, $H_i = 0.01$,

and varying values of H_f). Subfigures (e)-(f) represent higher-intensity controls, such as $H_s = 0.5$ and increased H_f, showing a pronounced reduction in infected populations over time. Increased control values generally reduce infection rates in both populations. Particularly, high values of H_s and H_f significantly lower the infected baboon and human populations, demonstrating the effectiveness of sterilization and food restriction controls.

7. Sensitivity Analysis

The values of parameters and initial populations used in this study are obtained from various sources, including WHO situation reports and previous literature. The initial population values are set as follows: $x_1(0) = 5000$, $x_2(0) = 150$, $x_3(0) = 50$, $x_4(0) = 8000$, $x_5(0) = 100$, and $x_6(0) = 100$. The following Table 1, provides a summary of the parameter values and their respective sources: A sensitivity statistical analysis is essential

Parameter	η_b	η_h	κ_b	κ_h	H_s	H_i	H_f
Value	0.3	0.1	0.07	0.15	0.1	0.05	0.05
Source	Estimated	Estimated	Estimated	Estimated	Assumed	Assumed	Assumed

Table 2: Parameter values for the baboon-human population model (1).

to evaluating whether various factors affect a model (1) stability if data is unknown. Analyzing these parameters helps identify critical parameters. With the baboon-human model (1), we compute the sensitivity indices of the basic reproduction number, $\mathscr{R}_0$. Local sensitivity analysis uses the normalized forward sensitivity index $\mathscr{R}_0$. According to our model (1), $\mathscr{R}_0$ has the following sensitivity index:

$$\Gamma_{\upsilon}^{\mathscr{R}_0} = \frac{\partial \mathscr{R}_0}{\partial \upsilon} \times \frac{\upsilon}{\mathscr{R}_0},$$

where υ represents a parameter from Table 3, and $\mathscr{R}_0$ is derived from the model (1) dynamics. The basic reproduction number $\mathscr{R}_0$ is calculated as follows:

$$\mathscr{R}_0 = \frac{\eta_b x_1 x_2}{\kappa_b + H_s} + \frac{\eta_h x_4 x_5}{\kappa_h + H_i}.$$

Substituting the parameter values from Table 1 gives:

$$\mathscr{R}_0 = \mathscr{R}_{0b} + \mathscr{R}_{0h} = \frac{0.3 \cdot 5000 \cdot 150}{0.07 + 0.1} + \frac{0.1 \cdot 8000 \cdot 100}{0.15 + 0.05} = 1723529.41.$$

Also

$$\frac{\partial \mathscr{R}_0}{\partial \eta_b} = \frac{x_1^0}{\kappa_b + H_s} > 0, \quad \frac{\partial \mathscr{R}_0}{\partial \eta_h} = \frac{x_4^0}{\kappa_h + H_i} > 0, \quad \frac{\partial \mathscr{R}_0}{\partial \kappa_b} = -\frac{\eta_b x_1^0}{(\kappa_b + H_s)^2} < 0,$$
$$\frac{\partial \mathscr{R}_0}{\partial \kappa_h} = -\frac{\eta_h x_4^0}{(\kappa_h + H_i)^2} < 0, \quad \frac{\partial \mathscr{R}_0}{\partial H_s} = -\frac{\eta_b x_1^0}{(\kappa_b + H_s)^2} < 0, \quad \frac{\partial \mathscr{R}_0}{\partial H_i} = -\frac{\eta_h x_4^0}{(\kappa_h + H_i)^2} < 0,$$
$$\frac{\partial \mathscr{R}_0}{\partial \mu} = -\frac{\eta_b x_1(0) + \eta_h x_4(0)}{\mu^2} < 0.$$

These equations illustrate that increases in the transmission parameters η_b and η_h result in higher values of $\mathscr{R}_0$, indicating a greater potential for disease spread. In contrast, increases in recovery rates κ_b and κ_h, or in the handling rates H_s and H_i, lead to a decrease in $\mathscr{R}_0$, implying a reduction in disease transmission potential. The computed sensitivity indices of $\mathscr{R}_0$ for the parameters in the baboon-human model (1) are summarized in Table 1. The

Parameter	$x_1(0)$	$x_4(0)$	η_b	η_h	κ_b
Sensitivity Index	Positive	Positive	Positive	Positive	Negative

Parameter	κ_h	H_s	H_i	μ
Sensitivity Index	Negative	Negative	Negative	Negative

Table 3: Sensitivity indices of $\mathscr{R}_0$ for the baboon-human model (1).

sensitivity indices indicate that $\mathscr{R}_0$ increases with the values of $x_1(0)$, $x_4(0)$, η_b, and η_h, suggesting an increased risk of disease transmission as these parameters rise. Conversely, increases in κ_b, κ_h, H_s, H_i, and μ lead to a decrease in $\mathscr{R}_0$, reflecting reduced transmission potential and suggesting effective control measures. By focusing on these key parameters, we can significantly influence baboon-human disease dynamics. The results indicate that the values of $\mathscr{R}_0$ increase when the parameters x_1, x_2, x_4, x_5, η_b, and η_h increase, while the values of κ_b and κ_h have a decreasing effect. As a result of the sensitivity analysis, potential strategies for disease control and prevention are identified as critical parameters that affect the dynamics of the baboon-human model (1).

8. Numerical investigation

8.1. Application of the Runge-Kutta method on the deterministic model

The Runge-Kutta method of order 4 can be expressed as follows:

$$\mathrm{y}_{n+1} = \mathrm{y}_n + \frac{h}{6}\left(\mathrm{k}_1 + 2\,\mathrm{k}_2 + 2\,\mathrm{k}_3 + \mathrm{k}_4\right),$$

where the intermediate stages $\mathrm{k}_1, \mathrm{k}_2, \mathrm{k}_3$, and k_4 are defined as follows:

$$\mathrm{k}_1 = f(\mathrm{t}_n, \mathrm{y}_n),$$

$$\mathrm{k}_2 = f\left(\mathrm{t}_n + \frac{h}{2}, \mathrm{y}_n + \frac{h}{2}\,\mathrm{k}_1\right),$$

$$\mathrm{k}_3 = f\left(\mathrm{t}_n + \frac{h}{2}, \mathrm{y}_n + \frac{h}{2}\,\mathrm{k}_2\right),$$

$$\mathrm{k}_4 = f(\mathrm{t}_n + h, \mathrm{y}_n + h\,\mathrm{k}_3),$$

where: h is the time step, y_n is the approximation of the solution at time step t_n, $f(t, y)$ is the function representing the system of ODEs.

Now, we apply the Runge-Kutta method on the determinstic model: By using the values of the parameters existed in Table 1 with Initial Conditions: $x_1(0) = 5000$, $x_2(0) = 150$,

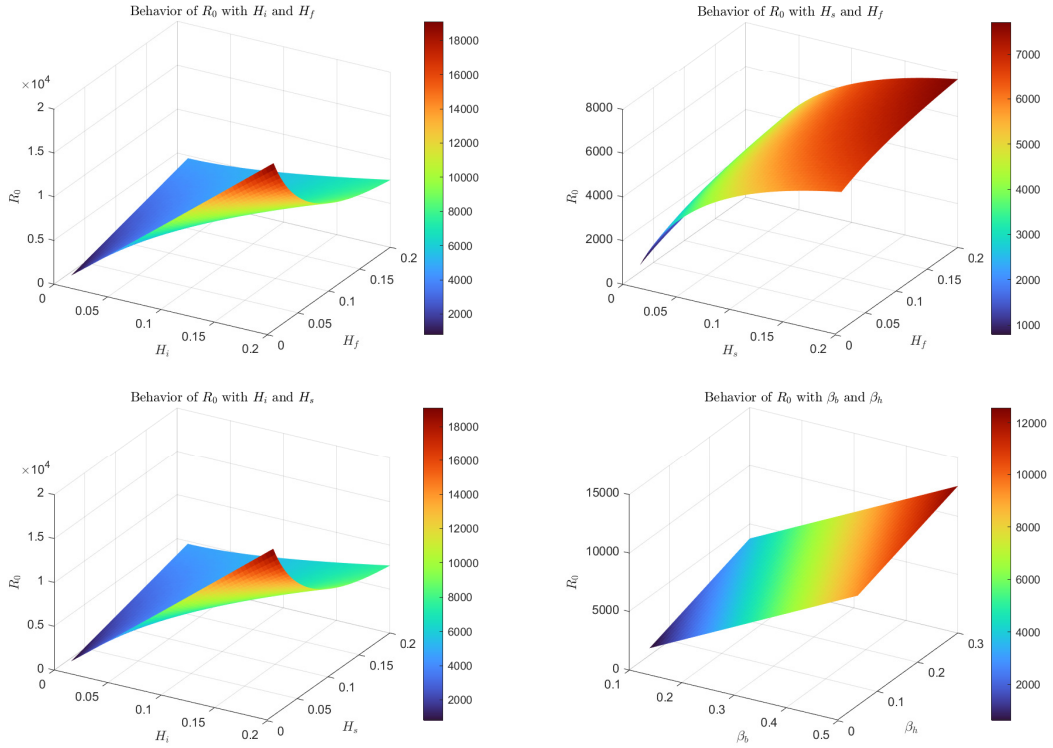

Figure 2: Sensitivity indices and behaviour of $\mathscr{R}_0$

$x_3(0) = 50$, $x_4(0) = 8000$, $x_5(0) = 100$, $x_6(0) = 100$. The time step: $\Delta t = 0.01$ and the noise intensities: $\sigma_1, \sigma_2, \sigma_3, \sigma_4, \sigma_5, \sigma_6$ set to desired levels (e.g., 0.1, 0.3, 0.5, 0.8). Simulate the model (1) for 100 time units. Compare deterministic and stochastic trajectories. Plot $x_1, x_2, x_3, x_4, x_5, x_6$ over time for different noise levels. The numerical simulations in this study explore the dynamics of zoonotic disease transmission between baboons and humans under various control scenarios. The simulations focus on how changes in control measures, represented by parameters H_s, H_f, and H_i, impact susceptible, infected, and recovered populations for both baboons and humans. These parameters correspond to sterilization, food access restriction, and interaction reduction controls, respectively. Below is an interpretation of each figure in the manuscript, followed by explanations of the trends and outcomes observed in each simulation. Finally, the simulations reveal that control measures targeting baboon populations—such as sterilization and limiting food access—significantly reduce infection rates in both populations. In addition, strategies that reduce human-baboon interactions are highly effective in minimizing zoonotic transmission risks. In light of this, holistic control strategies that consider the environmental and behavioral factors influencing disease dynamics between humans and wildlife are essential. Figure 3 examines how different levels of the control parameters H_s, H_f, and H_i affect the infected baboon population: Subfigure (a) shows how increasing H_s gradually reduces x_2, indicating that sterilization effectively limits disease spread. Subfigure (b) shows similar effects for varying H_f values, suggesting that limiting food access substan-

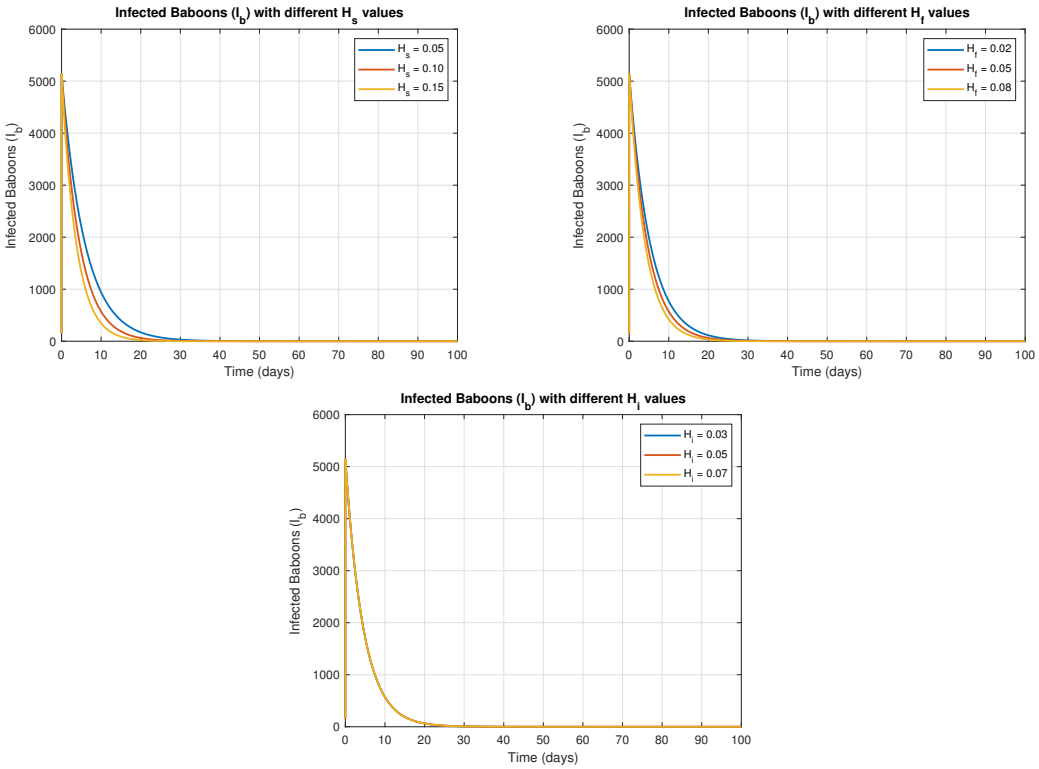

Figure 3: The effect of varying $H_s(t), H_f(t), H_i(t)$ on x_2.

tially decreases infection rates. Subfigure (c) illustrates that increased human interaction control (H_i) leads to lower infection levels in baboons, as reduced contact with humans helps prevent disease spillover. Figure 3 (a-c): Effect of Varying $H_s(t), H_f(t), H_i(t)$ on x_2 (Infected Baboons).

- **(a):** Increasing H_s reduces x_2, showing that sterilization effectively limits disease spread.
- **(b):** Increasing H_f (food restriction) significantly lowers x_2, emphasizing the role of reducing food-based human-baboon interactions.
- **(c):** Increasing H_i (interaction reduction) decreases x_2, as fewer interactions reduce direct transmission risks.

All control measures reduce infected baboons, with H_f having the greatest impact. Figure 4 shows the impact of different control intensities H_s, H_f, and H_i on the infected human population (x_5): Subfigure (a) illustrates that increasing H_s has a limited direct effect on infected humans. Subfigure (b) indicates that food restriction (H_f) significantly reduces x_5, as this measure limits baboon-human encounters around food sources. Subfigure (c) highlights that higher H_i values effectively lower x_5, demonstrating that reducing baboon-human interactions is crucial for controlling zoonotic transmission. Figure 4 (a-c) illustrate the effect of varying $H_s(t), H_f(t), H_i(t)$ on x_5 (Infected Humans).

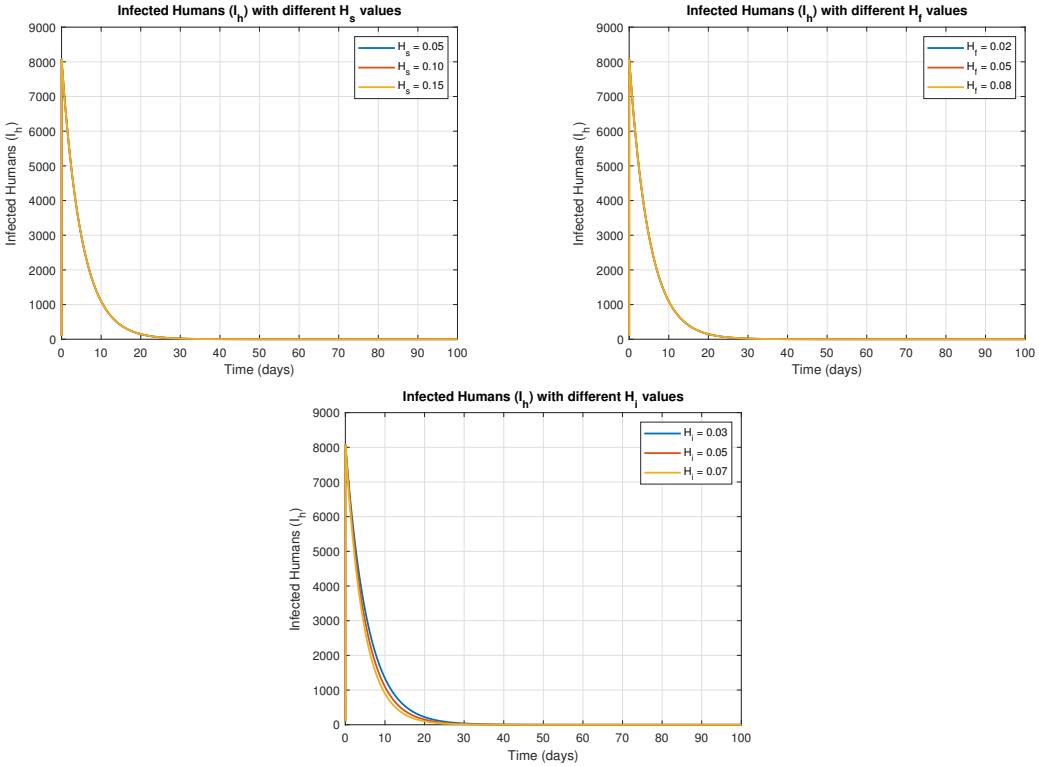

Figure 4: The effect of varying $H_s(t), H_f(t), H_i(t)$ on x_5.

- **(a):** Increasing H_s has a limited effect on x_5 since sterilization primarily targets baboons.
- **(b):** Increasing H_f significantly reduces x_5, as controlling food interactions limits exposure.
- **(c):** Higher H_i values effectively lower x_5, demonstrating that reduced human-baboon interactions are critical for controlling zoonotic transmission.

Interaction reduction (H_i) and food restriction (H_f) are particularly effective in reducing human infections. Figure 5 explores how H_s, H_f, and H_i influence the recovered baboon population: Subfigure (a) shows that increasing H_s leads to a steady rise in x_3 as more baboons recover. Subfigure (b) and (c) reveal similar trends for H_f and H_i, confirming that higher control measures indirectly promote recovery by reducing infection rates. Figure 5(a-c): Effect of Varying $H_s(t), H_f(t), H_i(t)$ on x_3 (Recovered Baboons).

- **(a):** Increasing H_s steadily increases x_3, showing more baboons recover when sterilization limits new infections.
- **(b):** Increasing H_f leads to a rise in x_3, as food restriction reduces x_2.
- **(c):** Higher H_i values also promote recovery (x_3) by preventing disease spread through reduced interactions.

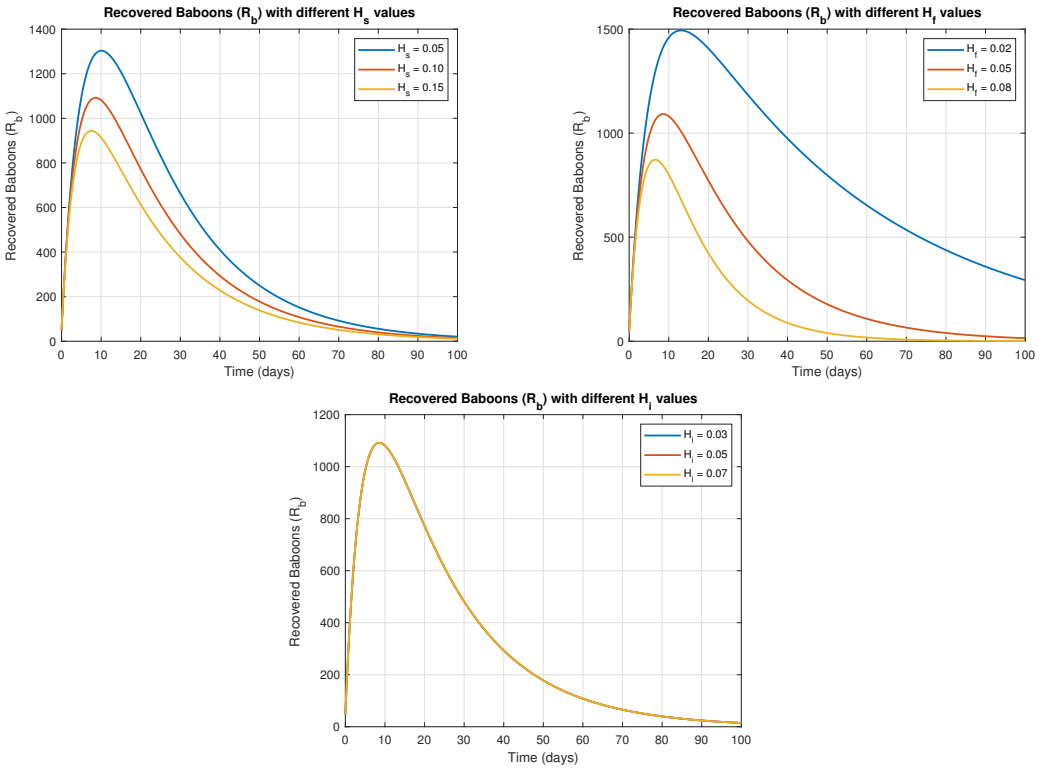

Figure 5: The effect of varying $H_s(t), H_f(t), H_i(t)$ on x_3.

Control measures indirectly promote recovery in baboons by reducing infection rates. Figure 6 illustrates the impact of control measures H_s, H_f, and H_i on the recovered human population (x_6): Subfigure (a) indicates minimal effect from H_s, as sterilization directly impacts baboons. Subfigures (b) and (c) show that higher H_f and H_i values promote recovery in humans, likely due to reduced exposure to infected baboons. Figure 6 (a-c) illustrate the effect of varying $H_s(t), H_f(t), H_i(t)$ on x_6 (Recovered Humans).

- **(a):** Minimal effect from H_s, as sterilization does not directly target humans.
- **(b):** Higher H_f values significantly increase x_6, as reduced exposure to infected baboons promotes recovery.
- **(c):** Higher H_i values also increase x_6, reflecting the importance of reducing human-baboon interactions.

Food restriction (H_f) and interaction reduction (H_i) have the most substantial impact on human recovery.

8.2. Application of the Milstein method

To study the dynamics of the model (2), we present numerical simulations by using the Milstein numerical approximation method for the SDE presented in [76]. Then, we can

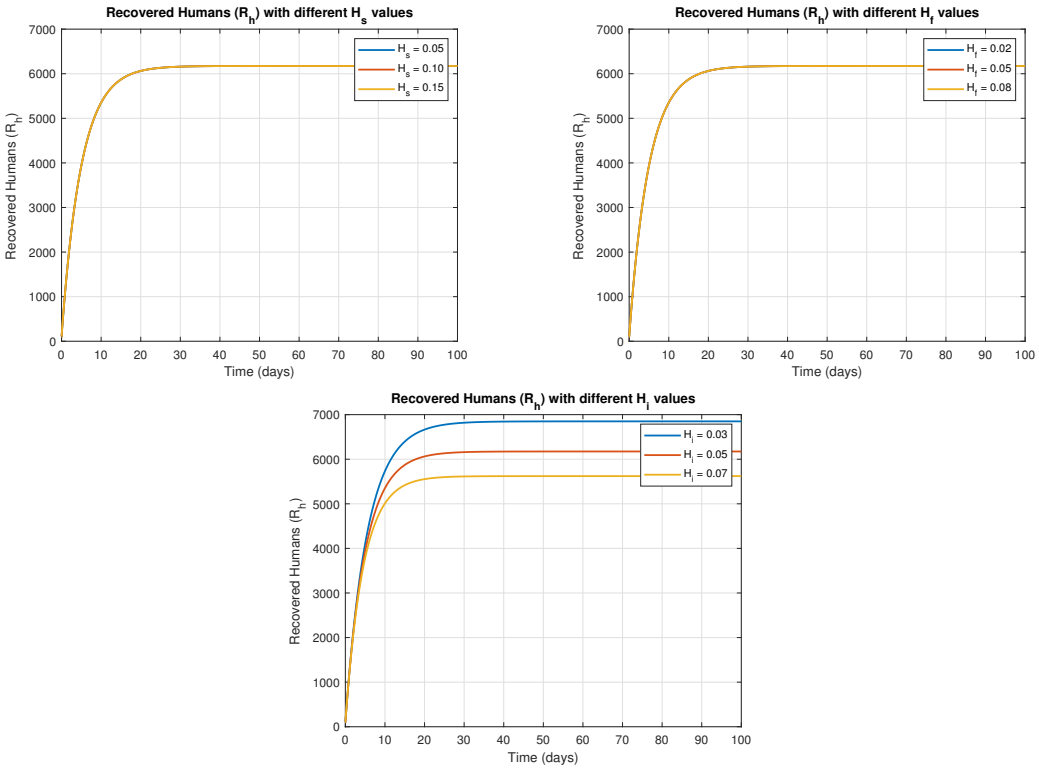

Figure 6: The effect of varying $H_s(t), H_f(t), H_i(t)$ on x_6.

write the system (2) by the following discretized form: The Milstein scheme for numerically approximating these equations is:

$$Y_{i+1} = Y_i + f(Y_i)\Delta t + g(Y_i)\sqrt{\Delta t}\xi_i + \frac{1}{2}g(Y_i)g'(Y_i)\left(\xi_i^2 - 1\right)\Delta t,$$

where:

- Y_i is the state at time step i,
- $f(Y_i)$ is the deterministic term (right-hand side of the ODE),
- $g(Y_i)$ is the diffusion coefficient representing noise intensity,
- $\xi_i \sim \mathcal{N}(0,1)$ is a Gaussian random variable,
- Δt is the time step.

The Milstein updates for each compartment of model (1) are:

$$\begin{aligned}
x_{1,i+1} &= x_{1,i} + \left[r x_{1,i} \left(1 - \frac{x_{1,i} + x_{2,i} + x_{3,i}}{K} \right) - \eta_b x_{1,i} x_{2,i} - H_s x_{1,i} - H_f x_{1,i} \right] \Delta t + \sigma_1 x_{1,i} \sqrt{\Delta t} \xi_{1,i} \\
&\quad + \frac{\sigma_1}{2} x_{1,i} \left(\xi_{1,i}^* 2 - 1 \right) \Delta t, \\
x_{2,i+1} &= x_{2,i} + \left[\eta_b x_{1,i} x_{2,i} - \kappa_b x_{2,i} - H_s x_{2,i} - H_f x_{2,i} \right] \Delta t + \sigma_2 x_{2,i} \sqrt{\Delta t} \xi_{2,i} + \frac{\sigma_2}{2} x_{2,i} \left(\xi_{2,i}^* 2 - 1 \right) \Delta t, \\
x_{3,i+1} &= x_{3,i} + \left[\kappa_b x_{2,i} - H_f x_{3,i} \right] \Delta t + \sigma_3 x_{3,i} \sqrt{\Delta t} \xi_{3,i} + \frac{\sigma_3}{2} x_{3,i} \left(\xi_{3,i}^* 2 - 1 \right) \Delta t, \\
x_{4,i+1} &= x_{4,i} + \left[-\eta_h x_{4,i} x_{2,i} - H_i x_{4,i} \right] \Delta t + \sigma_4 x_{4,i} \sqrt{\Delta t} \xi_{4,i} + \frac{\sigma_4}{2} x_{4,i} \left(\xi_{4,i}^* 2 - 1 \right) \Delta t, \\
x_{5,i+1} &= x_{5,i} + \left[\eta_h x_{4,i} x_{2,i} - \kappa_h x_{5,i} - H_i x_{5,i} \right] \Delta t + \sigma_5 x_{5,i} \sqrt{\Delta t} \xi_{5,i} + \frac{\sigma_5}{2} x_{5,i} \left(\xi_{5,i}^* 2 - 1 \right) \Delta t, \\
x_{6,i+1} &= x_{6,i} + \left[\kappa_h x_{5,i} \right] \Delta t + \sigma_6 x_{6,i} \sqrt{\Delta t} \xi_{6,i} + \frac{\sigma_6}{2} x_{6,i} \left(\xi_{6,i}^* 2 - 1 \right) \Delta t,
\end{aligned} \tag{5}$$

where $\{\xi_{u,i}, i \geq 0\}$ are sequences of random numbers uniformly distributed in $[0, 1]$ and Δt is a time step.

Now, we apply the Milstein method to the stochastic model (2): Figure 7 illustrates the comparison of the deterministic and stochastic model (2) for all compartments. Subfigures (a-f): These subfigures compare deterministic and stochastic dynamics for all compartments $(x_1, x_2, x_3, x_4, x_5, x_6)$ with varying levels of noise. The deterministic model (1) shows smooth trajectories, while the stochastic model (2) exhibits fluctuations due to random transmission dynamics. Larger noise levels amplify variability, highlighting the importance of the stochastic model (2) in capturing real-world uncertainty. Stochastic model (2) provides a more realistic representation of disease dynamics, especially under conditions of environmental and social variability.

8.3. Comparison of stochastic and deterministic model

Comparison of Deterministic and stochastic model (2) for $x_1(t)$ and $x_4(t)$. This figure highlights the dynamics of susceptible baboons (x_1) and humans (x_4) over time, comparing deterministic and stochastic model (2). Variability in the stochastic model (2) reflects the randomness in disease transmission dynamics due to environmental or social factors. The stochastic model (2) captures fluctuations in susceptible populations, particularly in human-baboon interactions. Compared to Runge-Kutta and Milstein methods, FDM is

Method	**Advantages**	**Disadvantages**
Runge-Kutta (RK4)	High accuracy, stable for stiff problems	Computationally expensive
Milstein Method	Captures stochastic effects well	Requires stochastic differential equation formulation

Table 4: Comparison of Numerical Methods

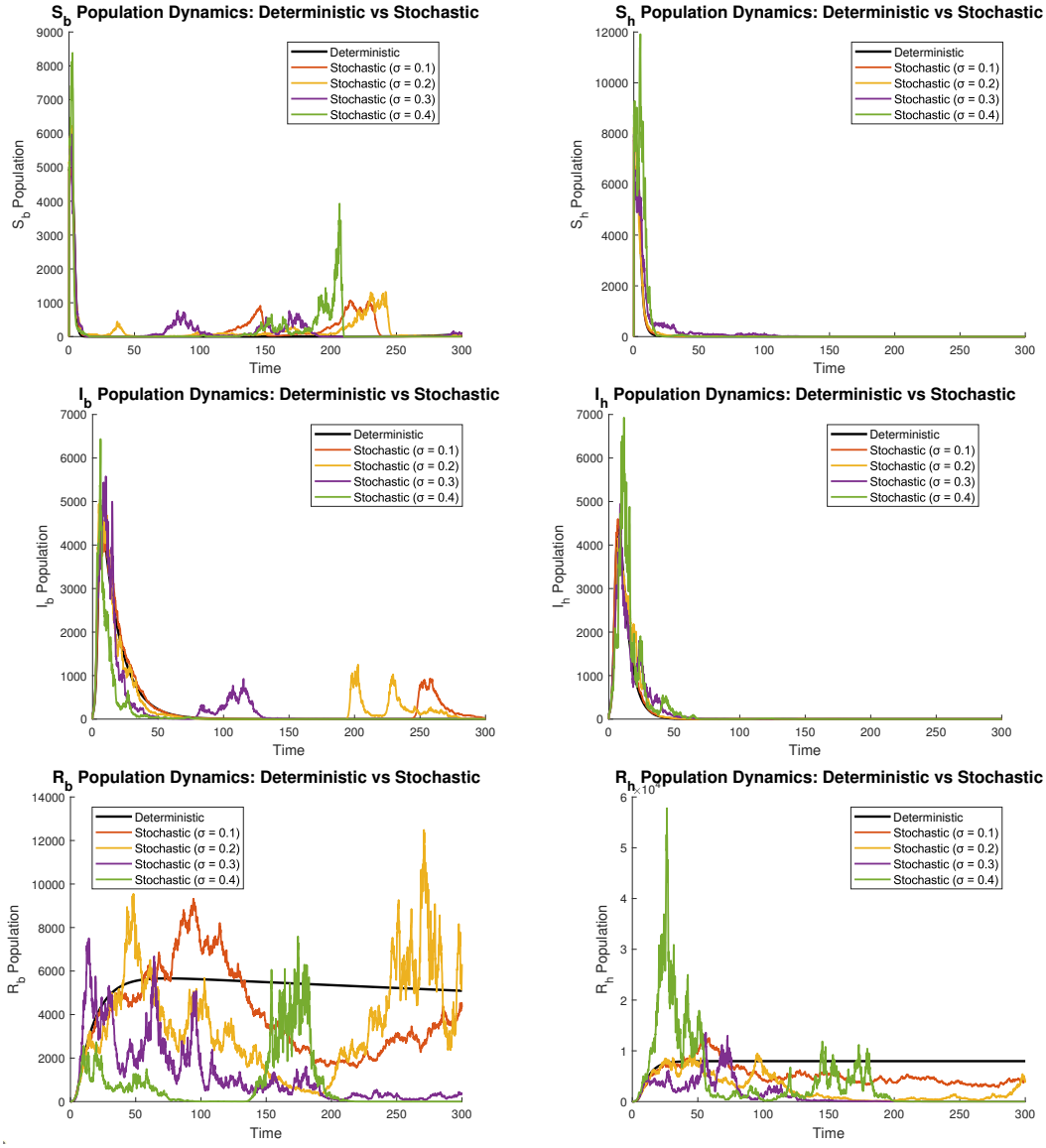

Figure 7: A comparison of deterministic and stochastic diabetes models with varying levels of noise=0.1, 0.2, 0.3, 0.4.

easier to implement but may require smaller time steps to maintain stability. In future studies, implicit FDM techniques could be explored for better stability in stiff systems.

9. Discussion

This study presents a mathematical model for zoonotic disease transmission between baboons and humans, incorporating both deterministic and stochastic frameworks. By integrating key control strategies such as sterilization, restricted food access, and reduced

human-baboon interactions, we provide a comprehensive analysis of disease dynamics at the human-wildlife interface. The deterministic model establishes fundamental properties such as solution existence, uniqueness, and boundedness, while the stochastic extension accounts for environmental and social variability. Theoretical and numerical analyses provide insights into disease persistence, eradication conditions, and optimal intervention strategies. The model (1) incorporates key intervention strategies such as sterilization, restricted food access, and reduced human-baboon interactions to mitigate disease spread. Analyzing both deterministic and stochastic versions of the model (1), we provide insights into how environmental and social variability influence disease dynamics. The deterministic model (1) establishes the theoretical foundations of the system, including solutions' existence, uniqueness, non-negativity, and boundlessness. Stability analysis of the infection-free and endemic equilibrium points reveals that the basic reproductive number, R_0, is critical for determining disease persistence or eradication. If $R_0 < 1$, the disease will eventually die out, while $R_0 > 1$ indicates sustained transmission within the population. Sensitivity analysis identifies key parameters, such as transmission rates (η_b, η_h) and recovery rates (κ_b, κ_h), as significant drivers of disease spread. In a stochastic version of the model, random fluctuations are incorporated into disease dynamics using Milstein's method. In this manner, real-world variables, such as population interactions and environmental conditions, are captured, which are not incorporated into the deterministic model (1). In stochastic simulations, higher noise levels increase the variability of population dynamics. Therefore, when designing robust interventions, randomness must be taken into account. Numerical simulations demonstrate the effectiveness of the proposed control measures. Sterilization (H_s) reduces the infected baboon population by limiting reproduction, while restricted food access (H_f) significantly decreases transmission rates by decreasing human-baboon interactions over shared food resources. Interaction reduction (H_i) is particularly effective at minimizing human infections. In both populations, a combination of these strategies results in a more comprehensive control strategy that results in fewer infections and higher recovery rates. A combination of environmental and behavioral factors affecting the transmission of disease should be addressed in integrated control strategies. Public health efforts should focus on reducing human-baboon interactions and improving recovery rates through medical interventions for humans. Wildlife management strategies, including sterilization and habitat control, are essential for mitigating risks at the source. To account for geographical factors influencing disease spread, future research should incorporate spatial dynamics. Additionally, collecting detailed empirical data on baboon-human interactions and disease prevalence in the Al-Baha region will enhance model (1) accuracy and provide better insights for intervention planning. By including migrations and seasonal variations in the stochastic framework, the model (1) could be further improved. Overall, this study highlights the value of combining deterministic and stochastic model (2) approaches to better understand and control zoonotic diseases. By identifying critical parameters and evaluating control strategies, this work contributes to the development of effective public health and wildlife management policies aimed at reducing zoonotic disease risks.

9.1. Key Findings and Contributions

One of the primary findings of this study is the role of the basic reproduction number, R_0, in determining disease dynamics. Stability analysis reveals that when $R_0 < 1$, the disease-free equilibrium is stable, implying that zoonotic disease transmission can be controlled or eradicated under appropriate intervention measures. Conversely, if $R_0 > 1$, the disease persists within the population, highlighting the need for sustained control efforts. Sensitivity analysis identifies transmission rates between baboons and humans as the most influential parameters affecting R_0. This underscores the importance of interventions targeting direct contact and environmental contamination pathways.

By incorporating stochasticity using the Milstein method, we demonstrate that environmental and behavioral fluctuations significantly impact disease transmission dynamics. The stochastic model shows higher variability in infection trends, particularly under conditions where control measures are inconsistently applied. This finding emphasizes the necessity of accounting for randomness in zoonotic disease modeling, as real-world transmission patterns rarely follow deterministic trajectories.

9.2. Novel Contributions and Medical Relevance of the Study

This study presents a novel mathematical model that integrates both deterministic and stochastic frameworks to analyze zoonotic disease transmission between baboons and humans. While classical compartmental models, such as the Susceptible-Infected-Recovered (SIR) and Susceptible-Exposed-Infected-Recovered (SEIR) frameworks, have been widely used to describe disease transmission, they often assume homogeneous mixing and overlook interspecies interactions, which are fundamental in zoonotic dynamics. Similarly, ecological models like the Lotka-Volterra framework, commonly applied to predator-prey interactions, have limited applicability in modeling human-animal disease transmission. By explicitly incorporating human-baboon interaction terms and control strategies—including sterilization, food restrictions, and reduced contact—this study extends classical epidemiological frameworks to better capture the complexities of zoonotic disease transmission.

Unlike prior deterministic approaches to wildlife-related disease modeling, such as those applied to rabies or avian influenza, this study employs a stochastic extension using the Milstein method. This accounts for environmental and social variability, making the results more reflective of real-world conditions. The region-specific application to Al-Baha, Saudi Arabia, where human-baboon interactions are prevalent due to food provisioning and shared resources, further highlights the necessity of tailoring intervention strategies to localized ecological and social conditions. The medical relevance of this study lies in its ability to inform public health officials and wildlife conservationists on designing data-driven intervention strategies to mitigate disease transmission risks from wildlife to humans. Through sensitivity analysis, this work systematically identifies the most influential transmission parameters, offering valuable insights for optimizing control measures and minimizing infection risks.

A key distinction of this study is its comprehensive parametric sensitivity analysis, which identifies the transmission rates among baboons (η_b) and from baboons to humans

(η_h) as the most critical factors influencing disease spread. Traditional models primarily establish threshold conditions for disease persistence but often neglect to quantify the relative importance of individual parameters. By highlighting the sensitivity of R_0 to η_b and η_h, this study emphasizes the need for targeted interventions focused on limiting direct human-baboon interactions and environmental contamination pathways.

Comparisons with established numerical methods, such as the Runge-Kutta method and Generalized Differential Transform Methods (GDTM), further demonstrate that the stochastic approach provides a more accurate representation of disease fluctuations under uncertain environmental conditions. Stochastic modeling better accounts for random perturbations in transmission dynamics, which are particularly relevant in ecological and wildlife disease settings where deterministic models may oversimplify real-world variability.

Furthermore, this study advances classical epidemiological modeling by integrating an optimal control framework, allowing for a quantitative assessment of intervention strategies in terms of both effectiveness and cost. While many existing studies analyze interventions qualitatively, they often lack a structured optimization approach to determine the most efficient long-term implementation of control measures. Our results demonstrate that combining sterilization and reduced human-baboon interaction strategies provides the most effective long-term control of zoonotic disease transmission. The numerical simulations confirm that a multi-pronged approach, incorporating sterilization, food access restrictions, and minimized human-baboon interactions, is essential for achieving sustained reductions in disease prevalence.

Unlike generalized epidemiological models, our framework is specifically designed to evaluate the impact of region-specific interventions, enhancing its real-world applicability. The findings of this study can directly inform public health policies and wildlife management strategies in regions with significant human-wildlife interactions. Moreover, the model can be extended to similar zoonotic transmission scenarios involving other wildlife species that frequently interact with human populations.

Overall, this study represents an original research contribution that advances the mathematical modeling of zoonotic diseases. The novelty of this work lies in the hybrid deterministic-stochastic approach, the detailed parametric sensitivity analysis, the region-specific application to Al-Baha, and the integration of an optimal control framework for intervention strategies. These elements distinguish this study from previous research and provide a comprehensive and practical framework for managing zoonotic disease transmission.

Future research directions could enhance this framework by incorporating vaccination strategies, spatial dynamics, or climate-driven seasonal variations to further refine disease control policies. Expanding the model to account for host-pathogen co-evolution and behavioral adaptations in both humans and wildlife could also provide deeper insights into long-term disease dynamics. As human-wildlife interactions continue to evolve due to environmental changes and urban expansion, mathematical models such as the one presented in this study will be increasingly crucial for guiding disease prevention and control efforts.

9.3. New Achievements in This Study

This study introduces several significant advancements in zoonotic disease modeling. First, explicit inter-species transmission is modeled by fully incorporating zoonotic disease dynamics between baboons and humans, accounting for both direct and indirect pathways of infection. Second, control strategies are integrated by introducing three intervention measures—sterilization, food control, and interaction reduction—each evaluated through numerical simulations to assess their effectiveness. Third, the Milstein method is employed to extend the deterministic model into a stochastic framework, allowing for the capture of random fluctuations in disease spread, thereby enhancing the model's realism. Fourth, a comprehensive sensitivity analysis identifies the most influential parameters affecting the transmission dynamics, providing insight for targeted interventions. Fifth, the model is applied to a real-world case study in the Al-Baha region, making the findings directly relevant to regional disease management efforts. Finally, the study expands to an optimal control framework, evaluating strategies to minimize infections while balancing the associated costs through various control scenarios. Overall, this study addresses previous limitations by developing a comprehensive, intervention-based, hybrid deterministic-stochastic model for zoonotic disease transmission, with real-world applicability, advanced numerical techniques, and an in-depth evaluation of control strategies, representing a significant advancement in epidemic modeling.

9.4. Modeling Zoonotic Disease Transmission Between Baboons and Humans in Al-Baha

This study presents a mathematical model aimed at analyzing and mitigating the transmission of zoonotic diseases between baboons and humans in the Al-Baha region. The model integrates both deterministic and stochastic approaches to evaluate disease dynamics under various environmental and social conditions. Key findings include:

The model incorporates three primary intervention measures—sterilization (H_s), food access restriction (H_f), and human-baboon interaction reduction (H_i)—which significantly reduce infection levels. Among these, food access restriction and interaction reduction have the strongest impact. The basic reproduction number, R_0, derived as

$$R_0 = \frac{\eta_b x_1^0}{\kappa_b + H_s} + \frac{\eta_h x_4^0}{\kappa_h + H_i},$$

determines disease persistence or eradication. If $R_0 < 1$, the disease dies out, whereas if $R_0 > 1$, long-term control measures are necessary. Increasing recovery rates (κ_b, κ_h) and enhancing control strategies (H_s, H_f, H_i) effectively reduce R_0.

Sensitivity analysis reveals that transmission rates (η_b, η_h) are the most influential parameters in increasing R_0, while increasing recovery rates and intervention measures reduce it. Among the interventions, food access restriction (H_f) is identified as the most effective in controlling disease spread. A comparison of deterministic and stochastic models highlights important differences. Deterministic models assume uniform transmission dynamics, while stochastic models, implemented using the Milstein method, account for

real-world fluctuations in disease spread. Stochastic fluctuations introduce variability in infection levels, with higher noise levels amplifying uncertainty and providing a more accurate representation of unpredictable transmission patterns. These findings emphasize the importance of using stochastic models to predict real-world outbreaks and evaluate control strategies. The implications for public health and wildlife management include designing more effective strategies to prevent zoonotic outbreaks, implementing targeted control measures such as sterilization campaigns and restricted food access, and using mathematical modeling as a predictive tool to assess intervention strategies before real-world implementation. Overall, this study provides actionable insights into controlling zoonotic disease transmission, emphasizing the need for data-driven public health measures and adaptive wildlife management strategies.

9.5. Previous Shortcomings in Existing Models

Several limitations exist in previous zoonotic disease models. First, limited consideration of inter-species transmission has been a key issue, as many past models primarily focused on human-to-human transmission or generalized animal-to-human interactions, without explicitly modeling baboon-human interactions. Some models also assumed simplified transmission pathways, failing to capture the complexity of zoonotic spillovers. Second, there has been a lack of integrated control strategies, as previous studies often neglected explicit intervention measures such as sterilization, food restriction, and interaction reduction. When control measures were included, they were not tested through optimal control theory to determine their effectiveness over time. Third, many models lacked a stochastic framework, relying solely on deterministic approaches that do not account for random environmental and social variations affecting disease spread. Stochasticity plays a critical role in zoonotic diseases, where unpredictable factors such as seasonal changes, human behavior, and ecological shifts can significantly influence transmission dynamics. Fourth, sensitivity analysis in existing models has been limited, with most studies conducting only basic threshold analyses without fully investigating the most influential parameters for disease control. Identifying key parameters is crucial for effective intervention, yet many models lacked detailed sensitivity analysis. Finally, a generalized application without regional context has reduced the practical impact of past studies, as many models were developed without applying them to specific real-world cases, making their conclusions harder to implement for policy-making. The absence of localized data has further limited their applicability in designing effective disease control strategies.

9.6. Study Limitations, Implications, and Future Research Recommendations

This study has certain limitations, including the assumption of homogeneous mixing of populations, which may not accurately represent real-world interactions, the lack of spatial considerations in disease spread, uncertainty in parameter estimations due to limited empirical data, and the exclusion of vaccination strategies as a potential intervention. Additionally, the model assumes a closed population with no migration effects, which may

not fully capture the complexity of disease dynamics in open ecosystems. While the model considers key control measures, other factors such as climate change, habitat fragmentation, and pathogen evolution may also influence zoonotic disease transmission. Expanding the model to include these elements could provide a more holistic understanding of disease spread. Moreover, the assumption of constant control parameters over time does not account for the potential variability in intervention strategies due to resource availability, policy changes, or behavioral adaptations among humans and baboons. Future research could develop adaptive control models that account for dynamic intervention strategies and improve model accuracy by incorporating migration patterns and seasonal variations. Empirical validation of the model through field data collection is also essential for refining parameter estimates and enhancing model predictions. Despite these limitations, the findings have important implications for public health and wildlife management, emphasizing the effectiveness of targeted control strategies such as restricting food access and reducing human-baboon interactions, the integration of stochastic modeling into policy decision-making, and the necessity for cross-disciplinary collaboration among epidemiologists, conservation biologists, and policymakers. Future research should focus on incorporating spatial and seasonal disease dynamics, collecting more empirical data on baboon-human interactions and disease prevalence in the Al-Baha region, evaluating the impact of vaccination strategies, and enhancing stochastic modeling by integrating additional sources of randomness to improve real-world applicability.

9.7. Comparison with Existing Studies and Justification for Variations

This study builds upon previous research on zoonotic disease transmission but introduces novel aspects that enhance its real-world applicability. Unlike traditional models that focus primarily on human-to-human disease spread or generalized animal-human interactions, this study explicitly models interspecies transmission between baboons and humans, incorporating region-specific control measures such as sterilization, restricted food access, and reduced human-baboon interactions. Studies conducted in other regions, such as Africa and Asia, have focused on zoonotic transmission from other wildlife species, often overlooking the unique dynamics of baboon-human interactions. Additionally, many previous models rely solely on deterministic approaches, whereas this work integrates a stochastic framework using the Milstein method, making it more suitable for capturing real-world variability in disease spread. The observed variations between this study and others may arise from differences in ecological settings, social behaviors, and intervention strategies. For instance, while some studies emphasize vaccination as a primary control measure, this research focuses on behavioral interventions due to the lack of existing vaccines for certain zoonotic diseases transmitted by baboons. The findings of this study are directly applicable to zoonotic disease control in the Al-Baha region but can also be adapted to similar wildlife-related transmission scenarios worldwide. Previous studies on zoonotic disease transmission have primarily focused on deterministic models [7, 8]. While these models provide valuable insights into disease spread, they often fail to capture the randomness inherent in real-world epidemiology. Our study builds upon this

work by introducing a stochastic framework, which better reflects the uncertainties in human-wildlife interactions and environmental factors. The use of the Milstein method enhances the model's accuracy in representing stochastic fluctuations, making it a more realistic tool for policy recommendations. Additionally, while traditional models have examined zoonotic transmission in broad ecological contexts, few studies have specifically analyzed human-baboon disease interactions. Our work fills this gap by focusing on the unique socio-environmental conditions of the Al-Baha region, where human-baboon encounters are frequent. This region-specific application provides a foundation for localized intervention strategies that can be adapted to similar ecological settings worldwide.

9.8. Comparative Analysis of Epidemic Models and Numerical Approaches for Zoonotic Disease Transmission

This study extends traditional epidemic models by incorporating zoonotic disease transmission between baboons and humans using a hybrid deterministic-stochastic framework. While classical compartmental models such as the Susceptible-Infected-Recovered (SIR) and Susceptible-Exposed-Infected-Recovered (SEIR) frameworks have been widely employed to describe disease transmission in human and animal populations, they often assume homogeneous mixing and overlook interspecies interactions, which are fundamental to zoonotic dynamics. Similarly, models like the Lotka-Volterra framework, commonly used in ecological systems to examine predator-prey dynamics, have limited applicability to human-animal disease interactions. By incorporating human-baboon interaction terms and control measures such as sterilization, food restrictions, and reduced contact, this study enhances classical frameworks to better capture the complexities of zoonotic disease transmission.

Unlike previous deterministic approaches for wildlife-related disease transmission, such as those applied to rabies or avian influenza, this work employs a stochastic extension using the Milstein method, which accounts for environmental and social variability, making the results more applicable to real-world scenarios. A region-specific application to the Al-Baha region of Saudi Arabia, where human-baboon interactions are particularly common due to food provisioning and shared resources, further underscores the necessity of tailoring intervention strategies to local conditions.

Numerical analysis, particularly sensitivity analysis, reveals that the transmission rates among baboons (η_b) and from baboons to humans (η_h) are the most influential parameters, highlighting the need for targeted interventions. Comparisons with numerical methods such as the Runge-Kutta method and Generalized Differential Transform Methods (GDTM) demonstrate that the stochastic approach provides a better representation of disease prevalence fluctuations under uncertain environmental conditions. Additionally, the study leverages optimal control theory to assess the cost-effectiveness of various interventions, drawing parallels with models used for malaria and COVID-19, where targeted control strategies such as sterilization and food restrictions significantly reduce infections.

The integration of deterministic and stochastic modeling, along with numerical methods and optimal control, offers a robust framework for understanding zoonotic disease

transmission. These insights provide valuable guidance for public health officials and wildlife managers seeking to mitigate disease risks at the human-wildlife interface. Future research could enhance this framework further by incorporating vaccination strategies or spatial dynamics to refine disease control policies.

9.9. Previous Shortcomings and New Achievements

Many previous studies on zoonotic disease modeling exhibit several limitations, including limited consideration of interspecies transmission, as most past models primarily focused on human-to-human transmission or generalized animal-to-human interactions without explicitly modeling baboon-human interactions. Additionally, few models have integrated targeted interventions such as sterilization, food restriction, and interaction reduction into a comprehensive framework. Many studies rely solely on deterministic models, which fail to capture environmental and social variability affecting disease transmission. Prior models often conduct only simple threshold analyses without investigating the most influential parameters governing disease dynamics, and most are developed without region-specific applications, reducing their practical value for policy implementation.

This study addresses these limitations by introducing several key advancements, including explicit interspecies transmission modeling, capturing both direct and indirect pathways of infection, and integrating three intervention strategies—sterilization, food access restriction, and reduced human-baboon interactions—evaluated through numerical simulations. A stochastic extension using the Milstein method is applied to account for random fluctuations in transmission, enhancing real-world applicability. Additionally, a detailed parametric sensitivity analysis identifies the most influential factors affecting disease spread, providing insights for optimal intervention strategies.

Unlike previous generalized models, this study is tailored to baboon-human interactions in the Al-Baha region, ensuring direct applicability to regional disease management. Furthermore, an optimal control framework is incorporated, evaluating strategies to minimize infection rates while balancing intervention costs. By addressing past limitations and introducing novel modeling techniques, this study significantly advances the field of zoonotic disease epidemiology. The integration of deterministic and stochastic approaches, coupled with real-world applications, contributes valuable insights to public health and wildlife management efforts.

9.10. Advantages of the Proposed Method Compared to Existing Approaches

The manuscript focuses on theoretical predictions for the epidemic using a mathematical modeling framework that integrates deterministic and stochastic elements. While various numerical solution algorithms exist for epidemiological models, the proposed approach offers several key advantages compared to traditional methods such as Runge-Kutta methods, which provide accurate numerical solutions but struggle with stochastic effects crucial for real-world disease modeling; finite difference methods, which are often used

for solving partial differential equations in spatially distributed models but may not effectively capture memory effects in fractional-order systems; and variational iteration and Adomian decomposition methods, which are suitable for solving nonlinear equations but can be computationally expensive and less efficient for large-scale epidemic simulations.

The proposed method improves upon these approaches by incorporating a stochastic extension through the Milstein numerical scheme, which accounts for random fluctuations in disease transmission, making predictions more realistic; fractional-order modeling, which captures memory effects and long-term epidemic behavior more accurately than Markovian dynamics; and comprehensive sensitivity analysis, which identifies key parameters affecting disease spread to optimize intervention strategies. Additionally, this hybrid deterministic-stochastic approach bridges the gap between theoretical predictions and real-world uncertainties, while the inclusion of an optimal control framework allows policymakers to evaluate and implement the most effective disease mitigation strategies.

Compared to traditional numerical methods, the proposed approach provides improved accuracy in modeling zoonotic disease dynamics by integrating stochasticity, fractional calculus, and sensitivity analysis, making it more applicable to real-world epidemic scenarios and offering valuable insights for public health interventions.

9.11. Implications for Public Health and Wildlife Management

The insights from this study have direct applications for public health policies and wildlife management strategies. Implementing a combination of sterilization, restricted food access, and reduced human-baboon interactions can significantly lower disease transmission risks. Among these measures, food restriction appears to have the greatest impact, as it reduces direct contact between baboons and humans while also limiting indirect transmission via contaminated food sources. Additionally, sterilization programs can help manage baboon population sizes, further reducing the likelihood of sustained disease transmission. From a public health perspective, these findings support policies aimed at regulating human behavior in areas with high wildlife interaction. Community education on the risks of feeding baboons and improved waste management strategies can minimize exposure to zoonotic pathogens. Furthermore, early disease surveillance programs that monitor baboon populations for potential outbreaks can enhance proactive response strategies.

10. Conclusion

This study develops a comprehensive mathematical model to analyze zoonotic disease transmission between baboons and humans in the Al-Baha region, integrating both deterministic and stochastic frameworks to assess the impact of key control measures—sterilization, food access restriction, and human-baboon interaction reduction—on mitigating disease spread. Through theoretical analysis, we established the existence, uniqueness, and boundedness of solutions, and stability analysis revealed that the basic reproduction number (R_0) is a critical threshold for disease persistence. The stochastic model, using the Mil-

stein method, provides a realistic representation of disease dynamics, capturing the effects of random environmental and social factors. Sensitivity analysis highlighted that transmission rates and control measures significantly affect disease spread, with food access restriction and interaction reduction being the most effective interventions. Key findings indicate that a strong intervention is needed when $R_0 > 1$ and that stochastic models offer a more accurate reflection of real-world disease dynamics compared to deterministic models. This study provides valuable insights for public health officials and wildlife managers, suggesting that future zoonotic disease control strategies should prioritize food access restrictions, human-baboon interaction reduction, and adopt stochastic models to account for real-world uncertainties. The research also lays the groundwork for future studies exploring seasonality, climate effects, vaccination strategies, and spatial dynamics in zoonotic disease transmission, offering practical recommendations for protecting both human and wildlife populations from emerging infectious diseases. This study highlights the importance of integrating deterministic and stochastic modeling approaches to understand and control zoonotic disease transmission. By identifying key transmission pathways, assessing control strategies, and incorporating stochastic fluctuations, our findings provide actionable insights for public health officials and wildlife managers. The results emphasize that a multi-faceted approach—combining sterilization, food restriction, and reduced human-baboon interactions—is essential for mitigating disease risks. Future research should focus on refining the model to include migration effects, dynamic control strategies, and empirical validation through real-world data. The integration of advanced computational techniques and field-based studies will further enhance our understanding of zoonotic disease dynamics, ultimately contributing to more effective disease prevention and wildlife management strategies.

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Data Availability

All data is included in the manuscript. https://doi.org/10.32604/cmc.2022.020732

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