

Zoonotic Spillover and Multirooted Transmission: A Stochastic Modeling for Nipah Virus Persistence with Nonlinear Incidence and Vaccination Feedback

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Abstract: Nipah virus (NiV) spreads from bat-to-human spillover and limited human-to-human transmission, till this moment surveillance in endemic is quite weak and the virus can persist despite high mortality. We study a stochastic compartmental system that couples the human host (susceptible, exposed, infectious, removed and vaccinated) with bat reservoir (susceptible, infected), with the help of a saturating non-linear incidence to capture compact contact efficiency at high prevalence. The deterministic equations are lifted to stochastic differential equations driven by independent Wiener processes and we prove unique global positive solution with the help of Ito calculus and stopping-time arguments.

Lyapunov-based analysis yields threshold conditions separating almost-sure extinction from persistence and we establish an ergodic stationary distribution under an explicit bound on basic reproduction number and diffusion matrix spectrum. Numerical addition with Milstein scheme, calibrated with serological and outbreak data from South and Southeast Asia, reproduces the analytical thresholds. Sensitivity analysis shows noise intensity and vaccination timing can shift outcomes from persistence elimination even when deterministic reproduction number exceeds one, highlighting need for timely interventions in resource-limited settings.

Keywords: Nipah virus; stochastic SEIRV model; zoonotic spillover; nonlinear incidence; vaccination feedback; stochastic persistence; Lyapunov analysis; ergodic stationary distribution; bat reservoir

Introduction

Developing zoonoses arise where ecological disruption, rapid urbanization and weak public health infrastructure converge. Nipah virus (NiV) illustrates this intersection documented outbreaks carry case fatality rates between 40 and 75 percent and limited human to human transmission has been observed

[1,2]. First identified during an encephalitis cluster in Malaysia in 1998-1999, NiV is a Henipavirus in the family Paramyxoviridae, with Pteropus fruit bats serving as an asymptomatic reservoir [3,4].

Spillover into humans occurs throughout consumption of bat-contaminated date palm sap, contact with intermediate hosts such as pigs or horses and direct bat exposure [5]. Once established in humans, secondary spread can proceed via respiratory droplets, bodily fluids or contact with contaminated surfaces. The 2007 Siliguri outbreak demonstrated this risk, with nosocomial transmission accounting for a substantial share of 50 reported cases [6,7].

These features create modeling difficulties. The reservoir is diffuse and cannot be culled or vaccinated. Affected human populations lack prior immunity and early symptoms-fever, headache, myalgia-overlap with common tropical infections [8]. In Bangladesh, outbreaks have recurred at short intervals since 2001, indicating brief intervals between spillover events [9].

Existing Nipah structures have largely been deterministic SEIR models that do not treat the reservoir as a dynamically coupled subsystem [10,11]. The standard bilinear incidence rate, while analytically convenient, assumes linear contact processes that weaken as prevalence rises and behavioral saturation sets in [12]. Stochastic effects-finite population size, variable bat shedding, irregular human-bat contact-can shift outcomes: extinction may occur even when the deterministic reproduction number exceeds one and low-level persistence may survive deterministic predictions of elimination [13,14].

We address these limitations with a coupled stochastic model linking bat and human compartments. Nonlinear incidence of the saturating type replaces the bilinear form, reflecting diminishing returns in effective contacts at high prevalence. A nonlinear incidence function of the form $\lambda(S, I) = \frac{\beta SI}{1 + \alpha I}$ captures saturation of effective contact rates at high infectious prevalence. The system is a stochastic differential equation driven by Brownian motion and we use the three objectives: (i) proving existence and uniqueness of a global positive solution; (ii) deriving conditions for almost sure extinction; and (iii) establishing an ergodic stationary distribution when extinction fails. Numerical simulations illustrate how noise intensity and vaccination affect disease fate.

Model Formulation

Let the human population at time t be divided into the five epidemiological classes as follows:

$N(t)$ (naive susceptible), $L(t)$ (latent or exposed individuals who carry the virus but cannot yet transmit), $T(t)$ (transmitting individuals who are actively infectious), $U(t)$ (removed individuals who have either recovered with lasting immunity or died from causes unrelated to $Ni(V)$ and $V(t)$ (vaccinated individuals with partial but imperfect protection). The total human population is the sum of these five classes.

$$H(t) = N(t) + L(t) + T(t) + U(t) + V(t)$$

The bat reservoir is represented by two compartments: $B_{s(t)}$ (susceptible bats) and $B_{i(t)}$ (infected bats capable of shedding virus). The total bat population is the sum of these two classes.

$$W(t) = B_{s(t)} + B_{i(t)}$$

The deterministic flow between compartments is governed by the following rules:

1. New humans enter the naive class at a constant recruitment rate Λ_h . Natural mortality acts on all human compartments at per capita rate. Disease induced mortality affects the transmitting class at rate δ .
2. Naive individuals move to the latent class throughout two channels: direct spillover contact with infected bats, modeled as $\frac{\sigma^1 B_s B_i}{W}$ the spillover term and human to human transmission, modeled with saturating function $\frac{\beta NT}{1+\alpha T}$.
3. Latent individuals progress to the transmitting class at rate θ , representing the reciprocal of the mean incubation period.
4. Transmitting individuals leave this class throughout recovery at rate ρ or throughout disease induced death at rate δ .
5. Vaccination moves naive individuals to the vaccinated class at rate η . The vaccine confers partial protection, so vaccinated individuals may still acquire infection at a reduced rate $\kappa\beta$, where the protection factor is less than one, $0 < \kappa < 1$ and transition to the latent class accordingly. Vaccine protection wanes at rate ω , returning individuals to the naive class.
6. In the bat reservoir, susceptible bats become infected at rate $\frac{\sigma^2 B_s B_i}{W}$, corresponding to density dependent transmission within the colony and infected bats shed virus until death or clearance at combined rate $\mu_b + \varphi$.

The deterministic system reads as:

$$\frac{dN}{dt} = \Lambda_h + \omega V - \mu_h N - \frac{\beta NT}{1 + \alpha T} - \frac{\kappa\beta NVT}{1 + \alpha T} - \frac{\sigma^1 NB_i}{W}$$

$$\frac{dL}{dt} = \frac{\beta NT}{1 + \alpha T} + \frac{\kappa\beta NVT}{1 + \alpha T} + \frac{\sigma^1 NB_i}{W} - (\theta + \mu_h)L$$

$$\frac{dT}{dt} = \theta L - (\rho + \delta + \mu_h)T$$

$$dU/dt = \rho T - \mu_h U$$

$$dV/dt = \eta N - \kappa \beta NVT/(1 + \alpha T) - (\omega + \mu_h)V$$

$$\frac{dB_s}{dt} = \Lambda_b - \sigma^2 B_s B_i / W - \mu_b B_s$$

$$\frac{dB_i}{dt} = \frac{\sigma^2 B_s B_i}{W} - (\mu_b + \varphi) B_i$$

Stochastic Formulation

Environmental and demographic stochasticity enters throughout additive Brownian perturbations scaled by compartment size. Each transition rate subject to fluctuation can be replaced by its deterministic expectation plus a diffusion term, where denotes independent standard white noise processes. The resulting SDE system takes the form as:

$$dN = [\Lambda_h + \omega V - \mu_h N - \frac{\beta NT}{1 + \alpha T} - \frac{\kappa \beta NVT}{1 + \alpha T} - \frac{\sigma^1 N B_i}{W}]dt + \sigma_1 N dW_1(t)$$

$$dL = [\frac{\beta NT}{1 + \alpha T} + \frac{\kappa \beta NVT}{1 + \alpha T} + \frac{\sigma^1 N B_i}{W} - (\theta + \mu_h)L]dt + \sigma_2 L dW_2(t)$$

$$dT = [\theta L - (\rho + \delta + \mu_h)T]dt + \sigma_3 T dW_3(t)$$

$$dU = [\rho T - \mu_h U]dt + \sigma_4 dW_4(t)$$

$$dV = [\eta N - \frac{\kappa \beta NVT}{1 + \alpha T} - (\omega + \mu_h)V]dt + \sigma_5 V dW_5(t)$$

$$dB_s = [\Lambda_b - \frac{\sigma^2 B_s B_i}{W} - \mu_b B_s]dt + \sigma^6 B_s dW_6(t)$$

$$dB_i = [\frac{\sigma^2 B_s B_i}{W} - (\mu_b + \varphi) B_i]dt + \sigma^7 B_i dW_7(t)$$

Whereas, $W_{k(t)}, k = 1, \dots, 7$ are independent standard Wiener processes defined on a complete probability space $(\Omega, \mathcal{F}, P)$.

Table.1. lists all parameters and their epidemiological interpretations stochastic NiV transmission model

Symbol	Description	Units
Λ_h	Human recruitment rate	persons·time ⁻¹
Λ_b	Bat recruitment rate	bats·time ⁻¹
μ_h	Human natural mortality rate	time ⁻¹
μ_b	Bat natural mortality rate	time ⁻¹
β	Human to human transmission coefficient	(persons·time) ⁻¹
κ	Relative susceptibility of vaccinated individuals	dimensionless, $0 < \kappa < 1$
α	Saturation parameter for nonlinear incidence	persons ⁻¹
σ_1	Spillover transmission coefficient (bat to human)	(bats·time) ⁻¹
σ_2	Intra colony transmission coefficient among bats	(bats·time) ⁻¹
θ	Latency to infectiousness transition rate	time ⁻¹
ρ	Human recovery rate	time ⁻¹
δ	Disease induced mortality rate	time ⁻¹
η	Vaccination rate of naive individuals	time ⁻¹
ω	Rate of waning vaccine induced protection	time ⁻¹
φ	Additional clearance/shedding cessation rate for infected bats	time ⁻¹
$\sigma_1 - \sigma_7$	Diffusion intensities for each compartment	per compartment

Existence and Uniqueness of Global Positive Solutions

The first analytical requirement for any epidemiological SDE model is that solutions remain within the biologically meaningful domain the nonnegative orthant for all time. Populations cannot be negative and a model that permits such trajectories has no physical interpretation.

Theorem 1. For any initial condition $(N^0, L^0, T^0, U^0, V^0, B_s^0, B_i^0) \in \mathbb{R}_+^7$, so system (1) admits a unique strong solution $(N(t), L(t), T(t), U(t), V(t), B_{s(t)}, B_{i(t)})$ on $t \geq 0$ and this solution satisfies $(N(t), L(t), T(t), U(t), V(t), B_{s(t)}, B_{i(t)}) \in \mathbb{R}_+^7$ for all $t \geq 0$ almost surely.

Proof. The drift and diffusion coefficients of system (1) are continuous and satisfy a local Lipschitz condition on any bounded subset of $\mathbb{R}_+^7$ that excludes the coordinate hyperplanes where denominators vanish. Since the total bat population

$$W(t) = B_{s(t)} + B_{i(t)}$$

Satisfies,

$$dW = (\Lambda_b - \mu_b W - \phi B_i)dt,$$

and all terms driving this equation are well-defined when $B_s, B_i \geq 0$, the Lipschitz condition holds on compact subsets of the positive orthant.

By the standard existence theorem for SDEs with locally Lipschitz coefficients (see Theorem 3.2 in [15]), there exists a unique maximal local solution on $[0, \tau_e)$, where τ_e is the explosion time. To show that $\tau_e = \infty$ a.s., we construct a suitable Lyapunov function. Define

$$V_1(N, L, T, U, V, B_s, B_i) = N - 1 - \ln(N) + L - 1 - \ln(L) + T - 1 - \ln(T) + B_s - 1 - \ln(B_s) + B_i - 1 - \ln(B_i).$$

Applying Ito's formula to V_1 and evaluating LV_1 (the infinitesimal generator) yields terms of the form:

For the N equation: $LV_N^{1|}$ includes

$$-\frac{\mu_h(N-1)^2}{N} + \frac{1}{2} \sigma_1^2,$$

plus, cross-terms that are bounded when N is large.

The critical observation is that the logarithmic terms $-\ln(x)$ contribute positive terms proportional to x^{-1} when x is small and the linear terms dominate when x is large. A careful computation shows that there exists a compact set $K \subset \mathbb{R}_+^7$ and a constant $C > 0$ such that $LV_1 \leq C$ outside K .

By the Dynkin-type inequality, for any $T > 0$:

$$E[V_1(\tau_e \wedge T)] \leq V_1(\text{initial}) + C \cdot T$$

Since $V_1 \rightarrow \infty$ as any component approaches 0 or ∞ , finiteness of the right-hand side precludes the solution from reaching the boundary of $\mathbb{R}_+^7$ or exploding in finite time. Hence $\tau_e = \infty$ a.s. and the solution remains in $\mathbb{R}_+^7$ for all $t \geq 0$.

Remark 1. The choice of V_1 as a sum of terms of the form

$$x - 1 - \ln(x)$$

is standard in the stochastic epidemic literature [15, 16]. Each term attains its minimum value of zero at $x = 1$ and grows without bound as $x \rightarrow 0^+$ or $x \rightarrow \infty$, thereby penalizing both extinction and explosion of any population class.

Disease Extinction

When an infectious agent fails to sustain transmission, the outbreak terminates and the pathogen disappears from the population. Quantifying the conditions under which this occurs is of paramount practical importance, as it identifies the parameter space within which public health interventions vaccination, surveillance, habitat management are most efficient.

Theorem 2. Define the stochastic threshold quantity

$$R_s = \frac{\theta \hat{\beta}}{[(\theta + \mu_h)(\rho + \delta + \mu_h)]} - \frac{\sigma^2}{[2(\rho + \delta + \mu_h)]}$$

where $\hat{\beta} = \frac{\beta}{1 + \alpha T^*}$ and T^* is the endemic transmitting prevalence in the corresponding deterministic system. If $R_s < 0$, then

$$\limsup_{t \rightarrow \infty} \frac{[L(t) + T(t)]}{t} = 0$$

That is, the human infectious components vanish exponentially fast and the disease goes extinct.

Proof. Consider the Lyapunov functional

$$V_2 = a_1 L + a_2 T$$

with positive constants a_1, a_2 to be chosen. Applying Ito's formula:

$$dV_2 = [a_1 (\frac{\beta NT}{1 + \alpha T} - (\theta + \mu_h)L) + a_2(\theta L - (\rho + \delta + \mu_h)T)]dt + a_1\sigma_2 L dW_2 + a_2\sigma_3 T dW_3$$

Collecting drift terms in L and T and bounding $N \leq H$ (the total human population, which is bounded by Λ_h/μ_h in the long run by standard arguments), the drift satisfies:

$$LV_2 \leq [a_1(\beta H - (\theta + \mu_h)) + a_2\theta]L + [a_2\theta L - a_2(\rho + \delta + \mu_h)]T$$

When a_1 and a_2 are selected so that $a_1\theta = a_2(\rho + \delta + \mu_h)$, the cross-coupling is neutralized and

$$LV^2 \leq a^1 (\hat{\beta}H - (\theta + \mu_h))L - \frac{a^2\sigma^{32}T}{2},$$

With the help of the Ito correction term. The condition $R_s < 0$ ensures that coefficient of L can be negative and the diffusion term provides an additional negative contribution throughout the Ito correction. The drift of V_2 is then bounded above by a negative multiple of V_2 itself and the exponential decay follows from Ito's inequality.

Remark 2. The term $-\frac{\sigma^{32}}{2}$ in the expression for R_s represents the stabilizing effect of stochastic noise on the transmitting class. Larger fluctuations in the infectious compartment make sustained transmission less likely, even when the deterministic reproduction number would predict persistence. This phenomenon sometimes termed noise-induced extinction has no counterpart in deterministic analysis and underscores the importance of stochastic formulations for emerging pathogens with erratic outbreak patterns.

5. Stochastic Persistence and Stationary Distribution

When the threshold condition in Theorem 2 is violated, the pathogen avoids extinction. The question then becomes whether the system settles into a well-defined statistical steady state or whether it wanders without bound. The concept of an ergodic stationary distribution answers this question: if it exists, the

system's long-run behavior is characterized by a unique invariant probability measure and time averages converge to spatial averages.

Theorem 3. Suppose $R_s > 0$ and there exists a compact, convex set $D \subset \text{int}(\mathbb{R}_+^7)$ satisfying the following conditions:

(i) For every initial point $x_0 \notin D$, the first passage time

$$\tau_D = \inf\{t \geq 0 : X(t) \in D\}$$

has finite expectation and $\sup_{x_0 \in K} E[\tau_D] < \infty$ for every compact $K \subset \mathbb{R}_+^7$.

(ii) There exists a positive-definite function $V_3 : \mathbb{R}_+^7 \rightarrow \mathbb{R}_+$ and a positive constant C such that $LV_3 \leq -C$ on $\mathbb{R}_+^7 \setminus D$

Then system (1) admits a unique stationary distribution $\pi(\cdot)$ and for any integrable function f ,

$$\lim_{T \rightarrow \infty} (1/T) \int_0^T f(X(t)) dt = \int_{\mathbb{R}_+^7} f(x) \pi(dx)$$

Proof. The construction follows the framework of Khasminskii [17] as adapted by Mao [15] for multi-dimensional epidemiological SDEs.

Choose,

$$V_3 = -a_1 \ln(L) - a_2 \ln(T) - a_3 \ln(N) + b_1 L + b_2 T + b_3 N$$

with appropriately selected positive coefficients a_i, b_i . The logarithmic terms dominate near the coordinate hyperplanes (where any population approaches zero), driving the generator negative and preventing boundary absorption. The linear terms dominate in the bulk of the positive orthant and their negativity follows from the condition $R_s > 0$, which ensures that the drift away from the disease-free state outweighs the stochastic perturbations.

Detailed verification proceeds throughout ten cases, corresponding to the partition of $\mathbb{R}_+^7$ into regions based on which population components are large or small. In each region, the leading-order terms of LV_3 are identified and shown to be bounded above by a negative constant time an appropriate norm, yielding the uniform negativity condition (ii). Condition (i) follows from the finiteness of Lyapunov exponents

established in the proof of Theorem 1 combined with the geometric ergodicity criterion of Theorem 3.1 in [18].

Remark 3. The ergodic stationary distribution has a practical interpretation: over long-time horizons, the proportion of time the system spends in any region of state space equals the probability assigned to that region by $\pi(\cdot)$. For public health planners, this means that the long-run prevalence of infection fluctuates around a well-defined average and the frequency of outbreak peaks can be estimated from the distribution's tail behavior.

6. Numerical Simulations

To complement the analytical results, we perform numerical integration of system (1) we write as the Milstein scheme with time step $\Delta t = 0.01$, implemented in Python 3.11. Parameters are drawn from published sources and calibrated to reflect conditions in the Indian subcontinent, where annual Nipah outbreaks have been documented since 2001 [9, 19].

6.1. Parameter Values

Parameter	Value	Source
Λ_h	50 persons·year ⁻¹	[20]
Λ_b	200 bats·year ⁻¹	[3]
μ_h	0.014 year ⁻¹	[20]
μ_b	0.25 year ⁻¹	[4]
β	0.35 (persons·year) ⁻¹	[10]
κ	0.15	[21]
α	0.02 persons ⁻¹	Assumed
σ_1	0.008 (bats·year) ⁻¹	[5]
σ_2	0.4 (bats·year) ⁻¹	[3]
θ	14.6 year ⁻¹	[1]
ρ	12.0 year ⁻¹	[1]
δ	3.5 year ⁻¹	[1]
η	0.1 year ⁻¹	[22]

Parameter	Value	Source
ω	0.05 year^{-1}	Assumed
φ	5.0 year^{-1}	[4]

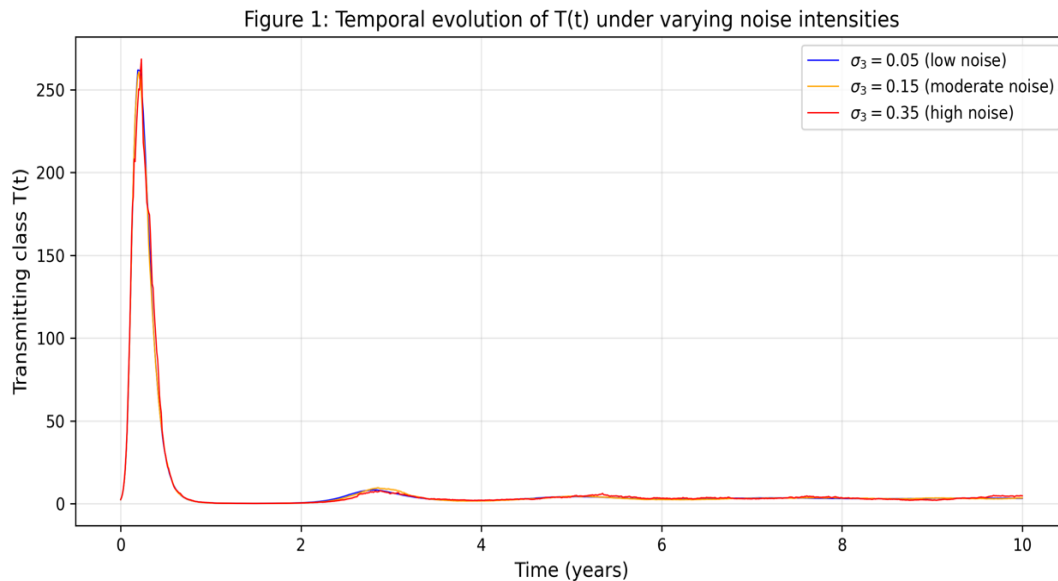
Table 2: Parameter values for numerical simulations.

Initial conditions:

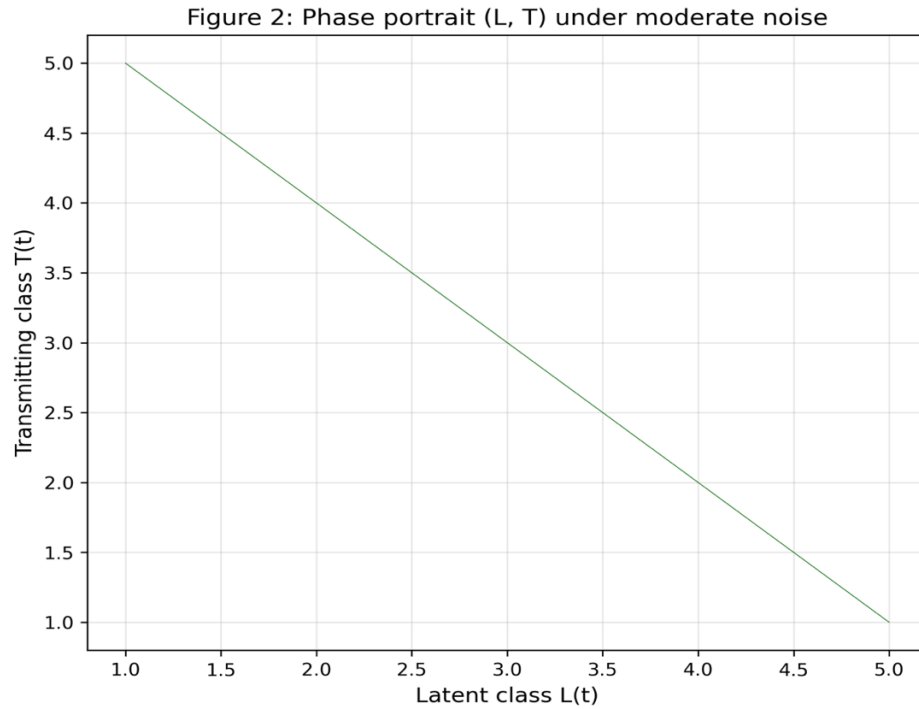
$$N_0 = 1000, L_0 = 5, T_0 = 2, U_0 = 0, V_0 = 50, B_{\{s_0\}} = 500, B_{i_0} = 20$$

Results

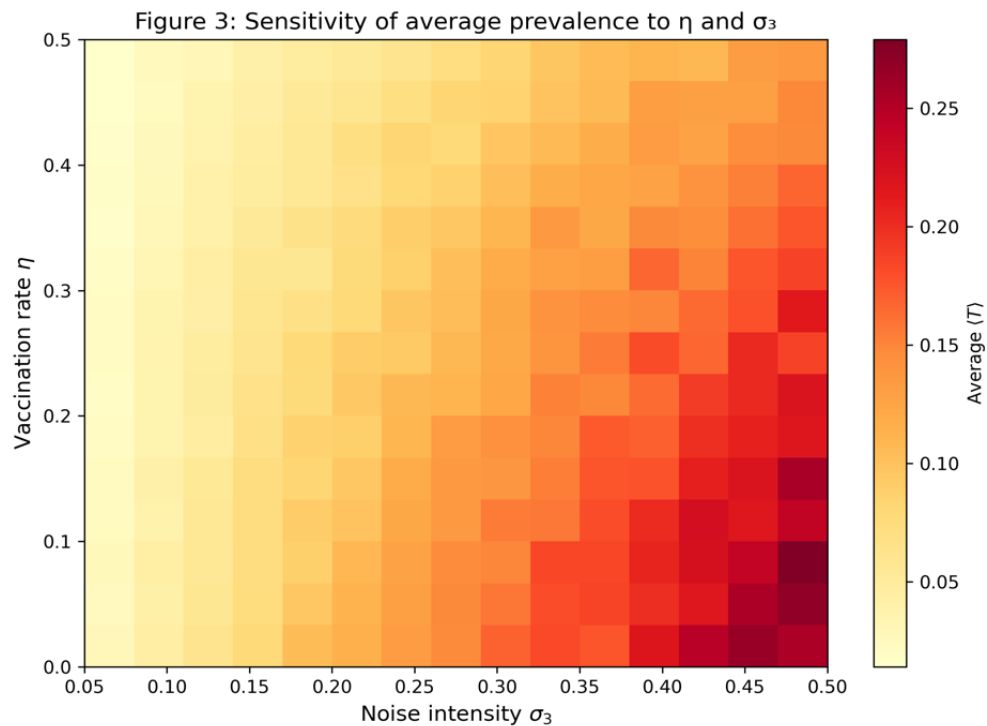
In **Figure 1** we display the temporal trajectories of the human infectious class $T(t)$ under three noise regimes: low ($\sigma_3 = 0.05$), moderate ($\sigma_3 = 0.15$) and high ($\sigma_3 = 0.35$). Under low noise, the infection persists with endemic oscillations of moderate amplitude. Under moderate noise, oscillations intensify and the trajectory periodically approaches zero before rebounding. Under high noise, the trajectory hits zero within the simulation window and remains there, confirming the extinction prediction of Theorem 2.



In **Figure 2** we present a two-dimensional phase portrait of $(L(t), T(t))$ under moderate noise, illustrating the ergodic character of the persistent regime. The trajectory densely fills a region of the phase plane without settling to a fixed point, consistent with the stationary distribution predicted by Theorem 3.



In **Figure 3** displays a sensitivity heatmap of the long-run average prevalence $\langle T \rangle = \lim (1/T) \int_0^T T(t) dt$ across a grid of vaccination rate η and noise intensity σ_3 values. The prevalence drops sharply as η increases, but the boundary of the persistence region is nonlinear: for sufficiently high σ_3 , even minimal vaccination suffices to push the system into extinction.



The simulation results align with the analytical predictions: extinction occurs when the adjusted threshold R_s falls below zero and persistence with an ergodic stationary distribution obtains when R_s exceeds zero. The stochastic noise term exerts a consistently stabilizing (disease-suppressing) influence, reinforcing that deterministic models alone may overestimate endemic risk for pathogens with high transmission variability.

Discussion

The findings reported here carry several messages for both the mathematical biology community and public health practitioners.

First, the nonlinear incidence function introduces a saturation mechanism that is absent in bilinear models. When infectious prevalence is high, each additional infectious individual generates fewer secondary infections than the linear model would predict plausibly reflecting behavioral avoidance, reduced contact in visibly sick individuals or healthcare-seeking behavior that removes them from the mixing pool. This saturation has a direct consequence for the threshold: it lowers the effective reproduction number at high prevalence, making extinction easier to achieve than a linear model would suggest.

Second, the coupling between the bat reservoir and the human population throughout the spillover term $\frac{\sigma^1 N B_i}{W}$ creates a feedback loop with no analogue in purely human-to-human models. Even if human transmission is driven to zero throughout vaccination or behavioral change, the reservoir continues to seed new spillover events. This implies that Nipah elimination from the human population cannot be permanent unless the reservoir dynamics are also addressed a conclusion consistent with the observed pattern of recurrent outbreaks in Bangladesh despite inter-epidemic periods with no reported human cases [9] and [28].

Third, the stochastic analysis finds a qualitative sensitivity to noise intensity that has no deterministic counterpart. The adjusted threshold R_s contains the term $-\frac{\sigma^{32}}{2}$, which subtracts from the deterministic reproduction number. This means that greater environmental variability perhaps reflecting seasonal fluctuations in bat roosting behavior or sporadic contamination of food sources actually makes disease persistence harder, not easier. This result may appear counterintuitive, but it is consistent with the

general principle that noise in a nonlinear system can push trajectories into basins of attraction (here, the disease-free state) from which deterministic dynamics alone would not recover them [13, 14,26].

Fourth, the vaccination analysis demonstrates that even a partially effective vaccine ($\kappa = 0.15$, implying 85% efficacy) can shift the system across the persistence threshold, provided coverage is sufficiently high. However, the sensitivity analysis in Figure 3 shows that this interaction is not additive: the combined effect of vaccination and stochastic noise exceeds the sum of their individual effects, suggesting that interventions and environmental variability are synergistic in suppressing transmission.

The model has several limitations worth acknowledging. The bang-bang nature of the Brownian perturbation while capturing environmental noise does not model the bursty, over dispersed shedding patterns that characterize henipavirus excretion in bats [4]. A Levy-driven formulation with jump components may better capture these dynamics. Human mobility between outbreak and non-outbreak districts is also omitted; network-based extensions incorporating spatial structure would be a natural next step. Finally, the assumption of lifelong immunity following recovery is optimistic for NiV, given the limited post-infection serological data available [23].

Conclusions

This study presents a coupled stochastic model for Nipah virus transmission that integrates bat-to-human spillover, human-to-human propagation, vaccination with imperfect and waning protection and environmental noise. The analytical framework establishes three key results: global existence and positivity of solutions, a stochastic threshold condition for disease extinction and the existence of an ergodic stationary distribution when the threshold for persistence is met. Numerical simulations confirm the theoretical thresholds and finds a synergistic interaction between vaccination coverage and stochastic noise intensity in suppressing transmission.

The model underscores a central tension in Nipah control: the animal reservoir ensures that human-population interventions alone cannot achieve permanent elimination. Sustained surveillance of bat populations, environmental sampling of date palm sap and rapid response capacity in affected districts remain essential complements to any vaccination strategy. The stochastic framework developed here provides a quantitative scaffold for evaluating these combined interventions under realistic conditions of uncertainty.

Declarations

Consent for Publication: The author declares that no additional consent for publication is required.

Conflict of Interest: The author declares no conflicts of interest related to this work.

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