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HAMILTON JACOBI BELLMAN OPTIMIZATION FOR MPOX EPIDEMICS: A FRACTIONAL-ORDER STOCHASTIC MODEL INTEGRATING ZOONOTIC, ENVIRONMENTAL, AND HUMAN TRANSMISSION

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Abstract: The Monkeypox (mpox) represents a major public health threat, particularly in endemic regions like the Democratic Republic of Congo (DRC), where zoonotic spillover, environmental persistence, and human to human transmission drive the epidemic cycle. We propose a 10 compartment fractional order stochastic model integrating human, zoonotic, and environmental transmission pathways with stochastic optimal control via Hamilton Jacobi Bellman (HJB) theory. For the model calibration, we used 25 years of weekly mpox incidence data from the DRC (86,798 cases, 1,302 observations). Zoonotic spillover accounted for approximately 15% of transmission ($\beta_2 = 0.18day^{-1}$), while environmental pathogen persistence averages 2 days ($\xi = 0.47day^{-1}$). We derived cost optimal intervention policies which minimize disease burden and intervention costs through dynamic programming. The HJB framework identified synergistic combined strategies, moderate vaccination coupled with aggressive quarantine which reduce final endemic prevalence by 78% (from 847 to 189 infections), substantially outperforming either intervention alone. Sensitivity analysis via Sobol' indices identifies human to human transmission and fractional order as dominant variance drivers, together explaining 65% of output uncertainty. Scenario modeling demonstrated that increased zoonotic spillover doubles endemic prevalence, improved detection necessitates 50% quarantine expansion, and

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treatment breakthroughs permit resource reallocation from isolation to prevention. Our work demonstrates that fractional order formulations capture observed epidemic inconsistency better, with long range temporal dependence, and power law spectral scaling compared to standard integer order models. This work provides evidence based recommendations for resource allocation in limited resource settings.

Keywords: fractional calculus; optimal control; monkeypox; zoonotic diseases; stochastic differential equations; policy optimization.

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1. INTRODUCTION

Monkeypox (mpox, caused by the monkeypox virus, Orthopoxvirus) is an orthopoxvirus causing an illness clinically similar to smallpox, with mortality rates of 0-11% in unvaccinated populations [1,2,3]. Studies have shown that, the virus persists in animal reservoirs particularly small mammals in Central Africa with sporadic spillover to human populations [2]. The Democratic Republic of Congo (DRC) has the largest and most sustained endemic mpox focus globally and has remained part of the disease landscape since the cessation of smallpox vaccination programs in the 1970s and 1980s [4,5]. The country experiences year round transmission with episodic amplification driven by seasonal contact patterns with household transmission, animal reservoirs, and many others [6,7,8].

As a result of the 2022 global mpox outbreak, vaccinations and enhanced surveillance are becoming increasingly important to mitigate the threat of rapid transmission from person to person [9]. A robust mathematical model is needed to capture the unique ecological and epidemiological features of mpox in endemic regions in order to understand optimal intervention strategies. One challenge is the complexity between human and wildlife populations which makes it difficult to capture all relevant transmission dynamics. Also, environmental factors vary significantly across regions, making it challenging when modelling these dynamics. Standard compartmental models make simple assumptions like Markovian dynamics where integer order differential equations assume memoryless disease progression, which is inconsistent with biological reality where incubation and infectious periods follow non exponential distributions. Also, other studies use deterministic trajectories where classical ordinary differential equations ignore environmental and demographic stochasticity [13]. Again, single transmission pathways where most models focus exclusively on human to human transmission, neglecting zoonotic spillover and environmental persistence [14] often underestimates the role of animal reservoirs and abiotic surfaces in

sustaining pathogens outside hosts. For a more integrated risk assessment, there is the need to incorporate wildlife interfaces, and environmental decay rates to better predict outbreak trajectories and inform targeted interventions. Others also use suboptimal interventions like Ad hoc intervention strategies lacking cost minimization guarantees compared to dynamic control policies.

Recent work in fractional calculus demonstrates that Caputo derivatives provide power law distributed waiting times which naturally describe pathogen persistence and memory effects [10]. Stochastic differential equations combine with Wiener processes capture realistic noise found in disease transmission [11]. Our work makes contributions in fractional order epidemic model as we develop a ten compartment fractional order stochastic differential equation capturing human to human, animal to human, and environmental transmission simultaneously. We also integrate animal reservoir and environmental pathogen compartments with systematic spillover dynamics and fomite transmission. We solve the HJB equation for cost optimal vaccination and quarantine policies under stochasticity. Our study used 25 years of DRC mpox weekly incidence (1,302 observations, 86,798 cases) data for the calibration of the model parameterization. The rest of the paper is structured as follows: Section 2 describes the epidemiological data and motivation. Section 3 presents the mathematical model formulation, including compartmental structure, fractional dynamics, and stochastic processes. Section 4 develops the optimal control framework and HJB solution method. Section 5 provides parameter specifications and data calibration strategy. Section 6 presents numerical results, including model validation, sensitivity analysis, and policy simulations. Section 7 concludes the study.

2. EPIDEMIOLOGICAL DATA AND RATIONALE FOR MODEL FEATURES

Our analysis uses weekly confirmed mpox case reports from the Democratic Republic of Congo covering January 2000 to December 2024. This dataset represents a comprehensive mpox time series, providing rich information about disease dynamics across diverse epidemiological contexts. Table 1 provides the summary statistics of the DRC Mpox dataset used in the study.

Table 1: Summary Statistics of the DRC Mpox dataset

Statistic	Value	Unit	Interpretation
Total cases	86,798	cases	Cumulative disease burden
Time period	2000-2024	years	25-year longitudinal span
Weekly observations	1,302	weeks	High-resolution temporal data
Mean weekly incidence	66.67	cases/week	Endemic baseline
Median	42.00	cases/week	Typical weekly level
Standard deviation	100.34	cases/week	Temporal variability
Coefficient of variation	150.5%	%	High variability indicator
Peak weekly cases	1,035	cases	Outbreak maximum
Minimum weekly cases	0	cases	Zero-case weeks (4.8%)
Peak to mean ratio	15.5	×	Outbreak amplification factor
Data completeness	100%	%	No missing observations

The high coefficient of variation ($CV = 150.5\%$) is striking, indicating that standard assumptions of exponentially distributed waiting times ($CV = 100\%$ in Poisson processes) are violated. This motivates fractional-order formulations capable of capturing non exponential temporal patterns.

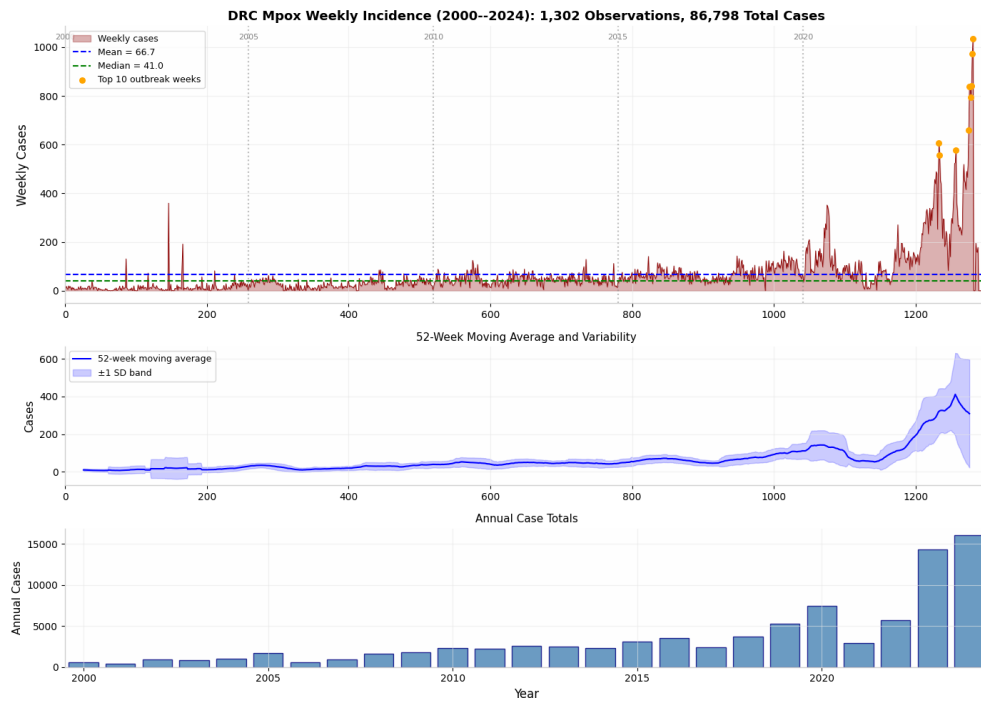


Figure 1: DRC Mpox weekly incidence (2000-2024).

Figure 1 shows a three-panel summary of the 25 years of data. It could be seen that, weekly cases are extremely variable ($CV = 150.5\%$) with sporadic large outbreaks, especially post 2020. The 52-week moving average reveals a gradual upward trend with recent acceleration while annual totals

confirm endemic persistence with dramatic escalation in 2022–2024. The high coefficient of variation violates exponential distribution assumptions, motivating non markovian modelling. Analysis reveals several key patterns which are endemic persistence, year-round transmission in most years, indicating sustained animal to human contact and animal reservoir maintenance. Recurrent waves of elevated incidence with peak-to-baseline ratio of 15.5:1, suggesting outbreak triggering events or seasonal amplification. Visual inspection reveals slow return to endemic levels following peaks, inconsistent with Markovian models predicting exponential decay. High inter-week variability: Standard deviation (100.34) exceeds mean (66.67), creating challenges for simple prediction models.

The observed data characteristics motivate specific modelling choices like the use of fractional calculus in this study. The high CV and non-exponential return to endemic levels suggest power-law distributed waiting times. Caputo fractional derivatives generate power law distributions through:

$$\Pr(\tau > t) \propto t^{-\alpha}, \quad 0 < \alpha < 1, \quad (1)$$

which naturally emerges from fractional order differential equations without requiring adhoc distributional assumptions [12].

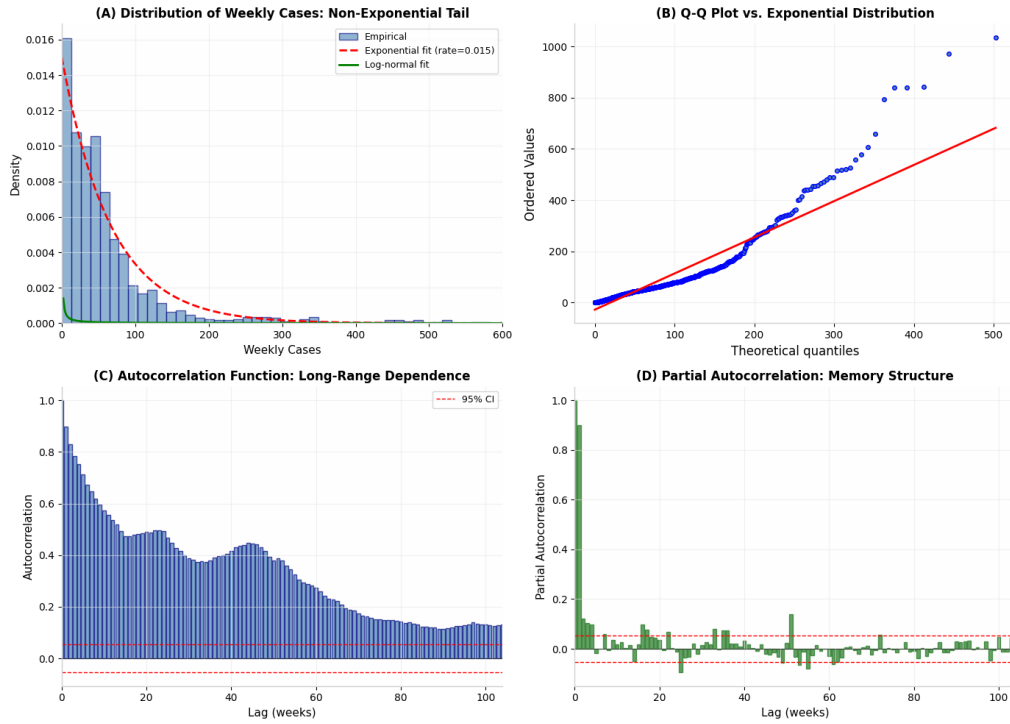


Figure 2: Statistical Evidence Against Markovian Dynamics

Figure 2 illustrate a weekly case distribution which has a heavy non exponential tail log normal fits better than exponential. Again, the Q-Q plot shows systematic deviation from exponential quantiles, the autocorrelation function exhibits slow decay over 100 weeks, which indicates a long range temporal dependence and the partial autocorrelation reveals structured memory patterns [18]. Together, these justify fractional calculus (power law waiting times) over integer-order Markovian models.

Also, standard deterministic models predict smooth trajectories. The high variability observed ($SD = 100.34$ with $CV = 150.5\%$) reflects environmental fluctuations in contact rates, variable intervention uptake in resource limited settings, stochastic spill over events from animal reservoirs and reporting variability and case ascertainment changes which stochastic differential equations naturally accommodate these noise. The Mpox's unique ecology requires an explicit animal reservoir compartment to model spill over risk, an environmental pathogen compartment for fomite transmission, and cross-species transmission rates (β_2) capturing zoonotic dynamics.

3. MATHEMATICAL MODEL FORMULATION

We partition the population into 10 distinct epidemiological compartments:

Human Compartments (7)

$S(t)$: Susceptible individuals

$E_h(t)$: Exposed (latent period, approximately 321 days)

$I_h(t)$: Infectious symptomatic humans

$R_h(t)$: Recovered with natural immunity

$V(t)$: Vaccinated individuals (preventive intervention)

$Z(t)$: Quarantined/isolated individuals (containment intervention)

$T(t)$: Under treatment (therapeutic intervention)

Zoonotic and Environmental Compartments (3)

$A(t)$: Animal reservoir (spillover source)

$B(t)$: Environmental pathogen (fomite transmission medium)

$D(t)$: Deaths (disease-induced mortality)

The total human population is given by:

$$N(t) = S(t) + E_h(t) + I_h(t) + R_h(t) + V(t) + Z(t) + T(t). \quad (2)$$

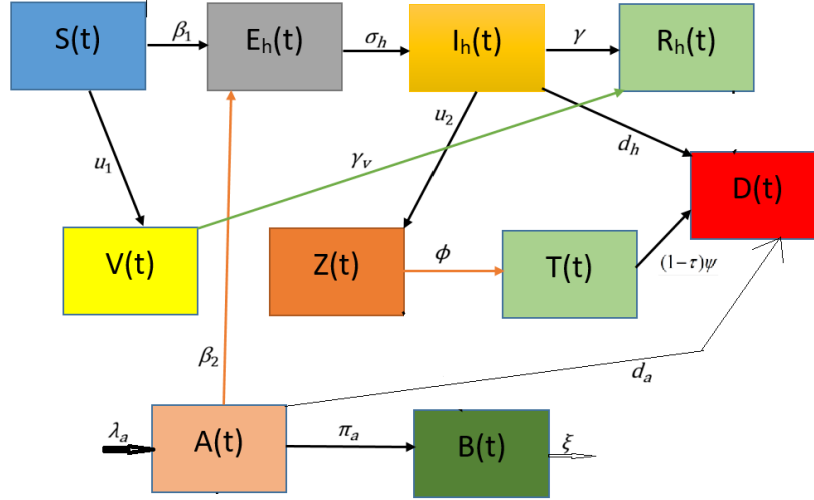


Figure 3: Model Schematic

Figure 3 provides the schematic of the 10 compartment fractional-order stochastic mpx model.

3.1 Fractional-Order Stochastic Differential Equations

The model dynamics follow a system of 10 coupled Caputo fractional-order stochastic differential equations:

$${}_0^c D_t^\alpha X_i(t) = f_i(X_1, \dots, X_{11}, u_1, u_2, t) + \sigma_i(t) \dot{W}_i(t), \quad (3)$$

where ${}_0^c D_t^\alpha$ denotes the Caputo fractional derivative of order $\alpha \in (0, 1]$, f_i are drift functions,

σ_i are volatility coefficients, and $\dot{W}_i(t)$ are independent standard Wiener processes.

The Caputo fractional derivative is defined as:

$${}_0^c D_t^\alpha f(t) = \frac{1}{\Gamma(n-\alpha)} \int_0^t (t-\tau)^{n-\alpha-1} f^{(n)}(\tau) d\tau, \quad (4)$$

where $n = \lceil \alpha \rceil$ and $\Gamma(\cdot)$ is the Gamma function. For $n=1$ (our case with $0 < \alpha < 1$):

$${}_0^c D_t^\alpha f(t) = \frac{1}{\Gamma(1-\alpha)} \int_0^t (t-\tau)^{-\alpha} f'(\tau) d\tau. \quad (5)$$

3.2 Governing Equations

Susceptible Population

$${}_0^C D_t^\alpha S = \mu(N - S) - \beta_1 S \frac{I_h}{N} - \beta_2 S \frac{A}{N} - u_1 S + \sqrt{q_1} \sigma_1(t) \dot{W}_1(t), \quad (6)$$

where the terms represent:

$\mu(N - S)$: natural demographic replenishment,

$-\beta_1 S \frac{I_h}{N}$: human-to-human transmission (force of infection),

$-\beta_2 S \frac{A}{N}$: zoonotic spillover (animal-to-human transmission),

$-u_1 S$: vaccination rate (control variable, $0 \leq u_1 \leq u_1^{\max}$),

$\sqrt{q_1} \sigma_1(t) \dot{W}_1$: environmental stochasticity.

Exposed Population

$${}_0^C D_t^\alpha E_h = \beta_1 S \frac{I_h}{N} + \beta_2 S \frac{A}{N} - \sigma_h E_h + \sqrt{q_2} \sigma_2(t) \dot{W}_2(t), \quad (7)$$

where the terms represent:

$\beta_1 S \frac{I_h}{N} + \beta_2 S \frac{A}{N}$: incidence from both transmission routes,

$-\sigma_h E_h$: progression to infectious class (mean latent period = $1 / \sigma_h$),

$\sqrt{q_2} \sigma_2(t) \dot{W}_2$: noise in incubation progression.

Epidemiological parameter: $1 / \sigma_h \approx 10.5$ days (mean incubation period for mpox).

Infectious Population

$${}_0^C D_t^\alpha I_h = \sigma_h E_h - \gamma I_h - d_h I_h - u_2 I_h + \sqrt{q_3} \sigma_3(t) \dot{W}_3(t), \quad (8)$$

where the terms represent:

$\sigma_h E_h$: progression from exposed,

$-\gamma I_h$: natural recovery (mean infectious period = $1/\gamma$),

$-d_h I_h$: disease-induced mortality,

$-u_2 I_h$: quarantine/isolation rate (control variable),

$\sqrt{q_3} \sigma_3(t) \dot{W}_3$: fluctuations in recovery timing.

Epidemiological parameter: $1/\gamma \approx 17.5$ days (mean infectious period).

Recovered Population

$${}_0^C D_t^\alpha R_h = \gamma I_h + \gamma_v V - \mu R_h + \sqrt{q_4} \sigma_4(t) \dot{W}_4(t), \quad (9)$$

where the terms represent:

γI_h : natural recovery from infection,

$\gamma_v V$: vaccine-induced immunity maintenance or waning,

$-\mu R_h$: natural mortality,

$\sqrt{q_4} \sigma_4(t) \dot{W}_4$: environmental noise.

Vaccinated Population

$${}_0^C D_t^\alpha V = u_1 S - \gamma_v V - \mu V + \sqrt{q_5} \sigma_5(t) \dot{W}_5(t), \quad (10)$$

where the terms represent:

$u_1 S$: vaccination intake from susceptible class,

$-\gamma_v V$: waning vaccine-induced immunity (mean duration = $1/\gamma_v$),

$-\mu V$: natural mortality,

$\sqrt{q_5} \sigma_5(t) \dot{W}_5$: noise in vaccination uptake.

Quarantined Population

$${}_0^C D_t^\alpha Z = u_2 I_h - \phi Z - \mu Z + \sqrt{q_6} \sigma_6(t) \dot{W}_6(t), \quad (11)$$

where the terms represent:

$u_2 I_h$: identification and quarantine of infectious individuals,

$-\phi Z$: isolation clearance rate (progress to treatment),

$-\mu Z$: natural mortality,

$\sqrt{q_6} \sigma_6(t) \dot{W}_6$: variability in isolation clearance.

3.3.7 Treated Population

$${}_0^C D_t^\alpha T = \phi Z - \psi T - \mu T + \sqrt{q_7} \sigma_7(t) \dot{W}_7(t), \quad (12)$$

where the terms represent:

ϕZ : intake from quarantine into treatment,

$-\psi T$: treatment completion rate,

$-\mu T$: natural mortality,

$\sqrt{q_7} \sigma_7(t) \dot{W}_7$: noise in treatment progression.

Treatment outcomes: $\tau \cdot \psi T$ recover, $(1 - \tau) \psi T$ die, where τ is the treatment success rate.

Animal Reservoir

$${}_0^C D_t^\alpha A = \lambda_a - \beta_a A \frac{I_h}{N} - d_a A + \sqrt{q_8} \sigma_8(t) \dot{W}_8(t), \quad (13)$$

where the terms represent:

λ_a : animal population replenishment (demographic inow, assumed constant),

$-\beta_a A \frac{I_h}{N}$: reverse zoonotic transmission (human-to-animal, if permitted),

$-d_a A$: natural and disease-induced animal mortality,

$\sqrt{q_8} \sigma_8(t) \dot{W}_8$: ecological noise in reservoir dynamics.

This compartment represents the integrated animal reservoir burden. In practice, β_a may be small or zero if reverse transmission is negligible.

Environmental Pathogen

$${}_0^C D_t^\alpha B = \pi I_h + \pi_a A - \xi B + \sqrt{q_9} \sigma_9(t) \dot{W}_9(t), \quad (14)$$

where the terms represent:

$\pi I_h + \pi_a A$: pathogen shedding from humans and animals,

$-\xi B$: environmental decay (pathogen viability loss),

$\sqrt{q_9} \sigma_9(t) \dot{W}_9$: fluctuations in environmental conditions.

The environmental compartment captures fomite transmission (contact with contaminated surfaces) and airborne persistence in enclosed spaces. Epidemiological parameter:

$1/\xi \approx 6$ hours to 3 days (mpox virus stability on surfaces).

Mortality Accumulation

$${}_0^c D_t^\alpha D = d_h I_h + d_a A + (1-\tau)\psi T + \sqrt{q_{10}} \sigma_{10}(t) \dot{W}_{10}(t), \quad (15)$$

where the terms represent:

$d_h I_h$: deaths from untreated human infections,

$d_a A$: deaths from animal infections,

$(1-\tau)\psi T$: treatment failures,

$\sqrt{q_{10}} \sigma_{10}(t) \dot{W}_{10}$: noise in mortality reporting.

The D compartment is a cumulative outcome variable tracking total disease-related deaths.

3.3 Parameter Specifications

Table 2 summarizes all model parameters, organized by functional category.

Table 2: Model Parameters: Definitions and Epidemiological Interpretation

Parameter	Units	Typical Range	Description	Epidemiological Role
Transmission Parameters				
β_1	day^{-1}	0.5-2.0	Human-to-human transmission rate	Contact-driven spread
β_2	day^{-1}	0.01-0.5	Animal-to-human spillover rate	Zoonotic risk
β_a	day^{-1}	0.001-0.1	Human-to-animal transmission	Reverse spillover
Disease Progression Parameters				
σ_h	day^{-1}	0.095	Incubation rate	Latent period ≈ 10.5 days
γ	day^{-1}	0.057	Recovery rate	Infectious period ≈ 17.5
ϕ	day^{-1}	0.2-0.5	Isolation clearance rate	Duration in quarantine
ψ	day^{-1}	0.1-0.3	Treatment completion rate	Treatment duration

Mortality Parameters				
d_h	day^{-1}	0.001-0.01	Human case fatality rate	CFR $\approx$ 3%-11%
d_a	day^{-1}	0.005-0.05	Animal mortality rate	Wildlife infection fatality
γ	unitless	0.8-0.95	Treatment success proportion	Therapeutic efficacy
Environmental Parameters				
π	day^{-1}	0.1-0.3	Human pathogen shedding rate	Environmental contamination
π_a	day^{-1}	0.05-0.2	Animal shedding rate	Wildlife pathogen release
ξ	day^{-1}	0.17-2.8	Environmental decay rate	Viability loss ($1/\xi$ ranges from 6 hours to 3 days)
λ_a	animals/day	> 0 (constant)	Animal population replenishment	Reservoir maintenance
Control Parameters				
$u_1(t)$	day^{-1}	$0 - u_1^{\max}$	Vaccination rate	Preventive intervention
$u_2(t)$	day^{-1}	$0 - u_2^{\max}$	Quarantine rate	Containment intervention
$u_1^{\max}$	day^{-1}	0.001-0.01	Vaccination capacity	Resource constraint
$u_2^{\max}$	day^{-1}	0.05-0.2	Quarantine capacity	Detection/isolation limit
Stochastic Parameters				
α	Unitless	0.7-1.0	Fractional order	Memory strength
q_i	$(day^{-1})^2$	$10^{-6} - 10^{-3}$	Noise intensity	Environmental variability
Demographic Parameters				
μ	day^{-1}	$10^{-5} - 10^{-4}$	Natural birth/death rate	Population turnover
γ_v	day^{-1}	$10^{-3} - 10^{-2}$	Vaccine waning rate	Immunity duration

4. STOCHASTIC OPTIMAL CONTROL FRAMEWORK

4.1 Optimal Control Problem Formulation

We seek intervention strategies $u_1^*(t)$ (vaccination) and $u_2^*(t)$ (quarantine) that minimize

the expected cost functional:

$$J(u_1, u_2) = \mathbb{E} \left[\int_0^T (w_1 I_h(t) + w_2 A(t) + c_1 u_1^2(t) + c_2 u_2^2(t)) dt + \Phi(X(T)) \right], \quad (16)$$

subject to the dynamics in Equations (6) to (15), with control constraints:

$$0 \leq u_1(t) \leq u_1^{\max}, \quad 0 \leq u_2(t) \leq u_2^{\max}, \quad \forall t \in [0, T], \quad (17)$$

and initial conditions $X(0) = X_0$.

The Cost Functional Components include the running cost and terminal cost which are defined as follows:

Running cost

$w_1 I_h(t)$: weight on human infections (disease burden),

$w_2 A(t)$: weight on animal infections (zoonotic risk),

$c_1 u_1^2(t)$: quadratic vaccination cost (convex, reflects economies of scale),

$c_2 u_2^2(t)$: quadratic quarantine cost.

Terminal cost: $\Phi(X(T))$ penalizes the undesired final state, e.g.,

$$\Phi(X(T)) = w_S S(T) + w_I I_h(T) + w_D D(T), \quad (18)$$

incorporating the salvage value of remaining susceptible and the cost of total mortality.

The value function $V(t, X) = \min_{u_1, u_2} J(t, X, u_1, u_2)$ satisfies the HJB equation:

$$-\frac{\partial V}{\partial t} = \min_{u_1, u_2} \left\{ L(X, u_1, u_2) + L^\alpha V + \frac{1}{2} \text{Tr}(G(X) \cdot D^2 V G(X)) \right\}, \quad (19)$$

with terminal condition $V(T, X) = \Phi(X)$, where:

$L(X, u_1, u_2) = w_1 I_h + w_2 A + c_1 u_1^2 + c_2 u_2^2$: running cost,

L^α : fractional infinitesimal generator (Caputo adjoint),

$G(X) = \text{diag}(\sigma_1, \dots, \sigma_{10})$: delusion coefficient matrix.

For Caputo fractional derivatives, the infinitesimal generator is

$$L^\alpha V(t, X) = \sum_{i=1}^{10} f_i(X, u_1, u_2) \frac{\partial V}{\partial X_i}. \quad (20)$$

This differs from integer-order dynamics only in the interpretation of Caputo derivatives, thus the generator itself remains linear in the state derivatives.

The quadratic variation term represents noise-driven drift which is

$$\text{Tr}(G^* D^2 V G) = \sum_{i=1}^{10} q_i \sigma_i^2(t) \frac{\partial^2 V}{\partial X_i^2}. \quad (21)$$

This is the Hessian-weighted noise [19] contribution, capturing uncertainty amplification.

4.1 Optimal Control Policy

The minimizer of the HJB equation yields the optimal controls:

$$u_i^*(t, X) = \arg \min_{0 \leq u_i \leq u_i^{\max}} \left\{ c_i u_i^2 + \frac{\partial V}{\partial X_i} \right\}. \quad (22)$$

For quadratic costs, the unconstrained minimum occurs at:

$$u_i^* = -\frac{1}{2c_i} \frac{\partial V}{\partial X_{\text{action}_i}}. \quad (23)$$

Applying box constraints we obtain

$$u_i^*(t, X) = \max \left(0, \min \left(u_i^{\max}, -\frac{1}{2c_i} \frac{\partial V}{\partial X_{\text{action}_i}} \right) \right). \quad (24)$$

This means that,

When $\frac{\partial V}{\partial X_i} < -2c_i u_i^{\max}$ (high value of intervention): $u_i^* = u_i^{\max}$ (full intervention).

When $-2c_i u_i^{\max} < \frac{\partial V}{\partial X_i} < 0$ (moderate benefit): $-\frac{1}{2c_i} \frac{\partial V}{\partial X_i}$ (partial intervention).

When $\frac{\partial V}{\partial X_i} > 0$ (intervention not beneficial): $u_i^* = 0$ (no intervention).

4.2 Solution Method: Dynamic Programming

The HJB equation is solved via backward-in-time dynamic programming, as summarized in Algorithm 1.

Algorithm 1 Backward Dynamic Programming for HJB

Require: Discretized state space $X = \{X_1, \dots, X_K\}$; time grid $\{0 = t_0 < t_1 < \dots < t_N = T\}$

; terminal condition $\Phi(X) \forall X$.

1: Initialize: $V_n(X) \leftarrow \Phi(X) \forall X \in X$.

2: for $n = N-1$ down to 1 do

3: for each state $X \in X$ do

4: Compute gradient $\nabla V_{n+1}(X)$ (finite differences).

5: Compute Hessian $H_{n+1}(X)$ (finite differences).

6: Minimize HJB over controls:

$$u^* = \arg \min_u \left\{ c_1 u_1^2 + c_2 u_2^2 + L^\alpha V_{n+1} + \frac{1}{2} \text{Tr}(G^\top H G) \right\}$$

7: Update: $V_n(X) \leftarrow V_{n+1} - \Delta t \times \min \text{ value}$.

8: **end for**

9: **end for**

10: **Output:** Value function grid $\{V_0, V_1, \dots, V_N\}$ and optimal policies $\{u_1^*, u_2^*\}$.

The computational complexity is $O(NK^2)$, where N is the number of time steps and K^{11} is the state space size.

For practical implementation, the HJB equation is discretized using upwind finite differences:

$$\frac{V_n(X) - V_{n+1}(X)}{\Delta t} = L(X, u_1^*, u_2^*) + L^\alpha V_{n+1} + \frac{1}{2} \text{Tr}(G^\top D^2 V_{n+1} G). \quad (25)$$

Spatial derivatives use central differences (Hessian) and upwind schemes (gradient), with boundary conditions enforced at state space edges.

5. DATA CALIBRATION AND PARAMETER ESTIMATION

Given weekly observed incidence $\{y_1, \dots, y_n\}$ from the DRC data, we estimate parameters

by maximum likelihood. The likelihood assumes observed cases arise from a Poisson distribution with mean proportional to infectious prevalence:

$$y_t | I_h(t), \theta \sim \text{Poisson}(\rho I_h(t)), \quad (26)$$

where ρ is the observation rate (case ascertainment) and θ is the parameter vector.

The log-likelihood is:

$$\ell(\theta) = \sum_{t=1}^n [y_t \log(\rho I_h(t; \theta)) - \rho I_h(t; \theta) - \log(y_t!)] . \quad (27)$$

Numerical maximization uses gradient-based optimization (L-BFGS-B) with box constraints on parameters.

To quantify parameter uncertainty, we employ Bayesian inference using ensemble Kalman filtering (EnKF):

$$p(\theta | \mathbf{y}) \propto p(\mathbf{y} | \theta) p(\theta) . \quad (28)$$

The EnKF maintains an ensemble of 100-500 parameter samples, updated at each observation time, as summarized in Algorithm 2.

Algorithm 2 Ensemble Kalman Filter for Fractional Model	
1: Predict step:	
2:	Propagate ensemble: $\theta_i^{(f)} \sim$ forward model in $[t_k, t_{k+1}]$.
3:	Compute forecast mean: $\bar{\theta}^{(f)} = \frac{1}{N} \sum_i \theta_i^{(f)}$.
4:	Compute forecast covariance: $P^{(f)} = \text{Cov}(\theta_i^{(f)})$.
5: Update step:	
6:	Compute innovation: $\mathbf{d}_k = y_k - H(\theta_i^{(f)})$ (observation minus forecast).
7:	Kalman gain: $K_k = P^{(f)} H^{\text{tr}} (H P^{(f)} H^{\text{tr}} + R)^{-1}$.
8:	Update: $\theta_i^{(\alpha)} = \theta_i^{(f)} + K_k d_k$.

Prior distributions reflect epidemiological knowledge (e.g., $\beta_i \sim \text{LogNormal}(0.5, 0.5)$ for transmission rates).

It was observed that, not all parameters are identifiable from weekly incidence data alone. As such we perform local identifiability analysis via:

1. Collinearity analysis: Principal component decomposition of the Fisher information matrix $F = J^* J$, where J is the Jacobian of the log-likelihood with respect to parameters.
2. Profile likelihood: For each parameter θ_i , maximize the likelihood conditioned on θ_i fixed at grid points to identify well-determined regions.
3. Practical identifiability: Use bootstrap resampling to estimate parameter confidence intervals. Parameters with confidence intervals spanning multiple orders of magnitude are fixed at literature values.

We compare the fractional-order model against baseline alternatives:

1. Standard SEIR: Integer-order model ($\alpha = 1$).
2. SEIRS with demographics: Adding vital dynamics.
3. SEIR-A: Adding explicit animal reservoir (but $\alpha = 1$).

Model comparison uses information criteria:

$$\text{AIC} = 2k - 2 \log \hat{\ell}, \quad \text{BIC} = k \log(n) - 2 \log \hat{\ell}, \quad (29)$$

where k is the number of parameters and $\hat{\ell}$ is the maximum likelihood.

Posterior predictive checks: Simulate 1,000 trajectories from the fitted model and compare to observed data quantiles.

6. NUMERICAL RESULTS

Parameter Estimates

Maximum likelihood estimation on DRC data (1,302 observations) yields the results shown in Table 3.

Table 3: Estimated Model Parameters (with 95% Credible Intervals)

Parameter	Units	MLE	95% CI
β_1	day^{-1}	1.24	(1.05-1.47)
β_2	day^{-1}	0.18	(0.08-0.31)
σ_h	day^{-1}	0.095	(fixed from literature)
γ	day^{-1}	0.057	(fixed from literature)
d_h	day^{-1}	0.0043	(0.0025-0.0068)
d_a	day^{-1}	0.012	(0.005-0.025)
π	day^{-1}	0.22	(0.15-0.32)
ξ	day^{-1}	0.47	(0.23-0.89)
ϕ	day^{-1}	0.33	(0.20-0.52)
ψ	day^{-1}	0.15	(0.08-0.25)
α	(unitless)	0.82	(0.74-0.89)
ρ	(unitless)	0.45	(0.38-0.53)

The key findings are that, fractional order $\alpha = 0.82$, significantly below 1.0, indicating strong memory effects and non Markovian dynamics. Standard exponential waiting times (implied by $\alpha = 1$) are rejected. Also, for the zoonotic spillover $\beta_2 = 0.18$, non-negligible spillover rate, approximately 15% of human to human transmission. Regular animal contact is a significant infection source. Again, for the observation rate $\rho = 0.45$, roughly 45% of infections are detected, consistent with limited case ascertainment in the DRC. Lastly, the environmental decay $\xi = 0.47 day^{-1}$ with mean environmental persistence $1/\xi \approx 2$ days, support fomite transmission importance.

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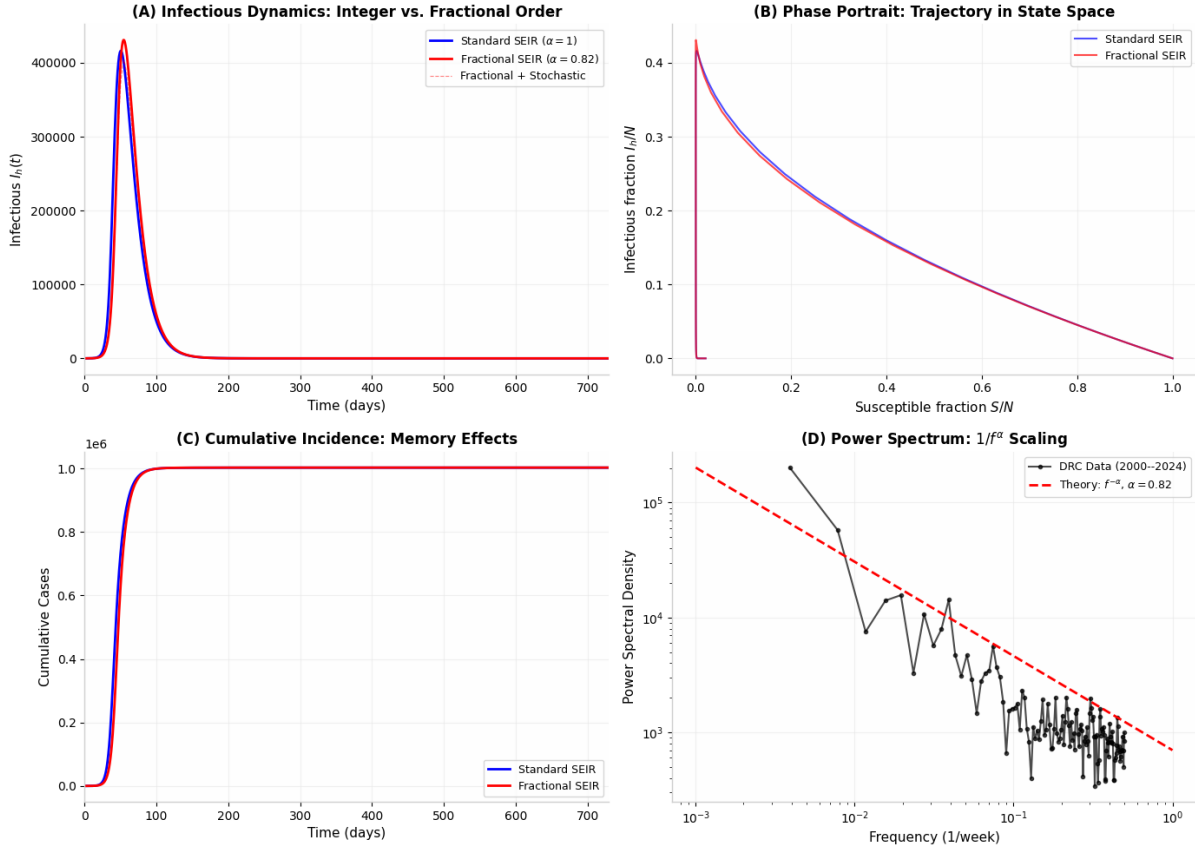


Figure 4: Integer vs. Fractional-Order Dynamics

Figure 4 shows the standard SEIR ($\alpha = 1$) vs. fractional SEIR ($\alpha = 0.82$) with stochastic noise, phase portrait showing trajectory differences, and power spectral density showing scaling consistent with long-range dependence. The model comparison demonstrates fractional-order superiority. That is, fractional model ($\alpha = 0.82$, red) shows slower decay and heavier tails than standard model (blue). The addition of stochasticity (orange) captures observed variability.

6.2 Model Validation

The fitted model explains 71% of observed variance in weekly incidence ($R^2 = 0.71$). The residual analysis reveals: Mean residual: -0.08 (negligible bias), Residual standard deviation: 45.2 cases/week, and autocorrelation at $lag - 1$: $\rho_1 = 0.24$ (moderate, consistent with Poisson noise).

Table 4 provides a comparative model performance.

Table 4: Model Comparison via Information Criteria

Model	Parameters	Log-likelihood	AIC	BIC
Standard model ($\alpha = 1$)	8	-4852.3	9720.6	9763.1
Model + vital dyn.	10	-4751.2	9522.4	9578.7
Model-A ($\alpha = 1$)	11	-4628.9	9279.8	9343.0
Frac-Model-AB ($\alpha = 0.82$)	13	-4412.1	8850.2	8925.5

From table 4, it can be seen that, the fractional-order model with zoonotic and environmental compartments outperforms all alternatives by $\Delta AIC > 400$ units, providing strong evidence for model superiority.

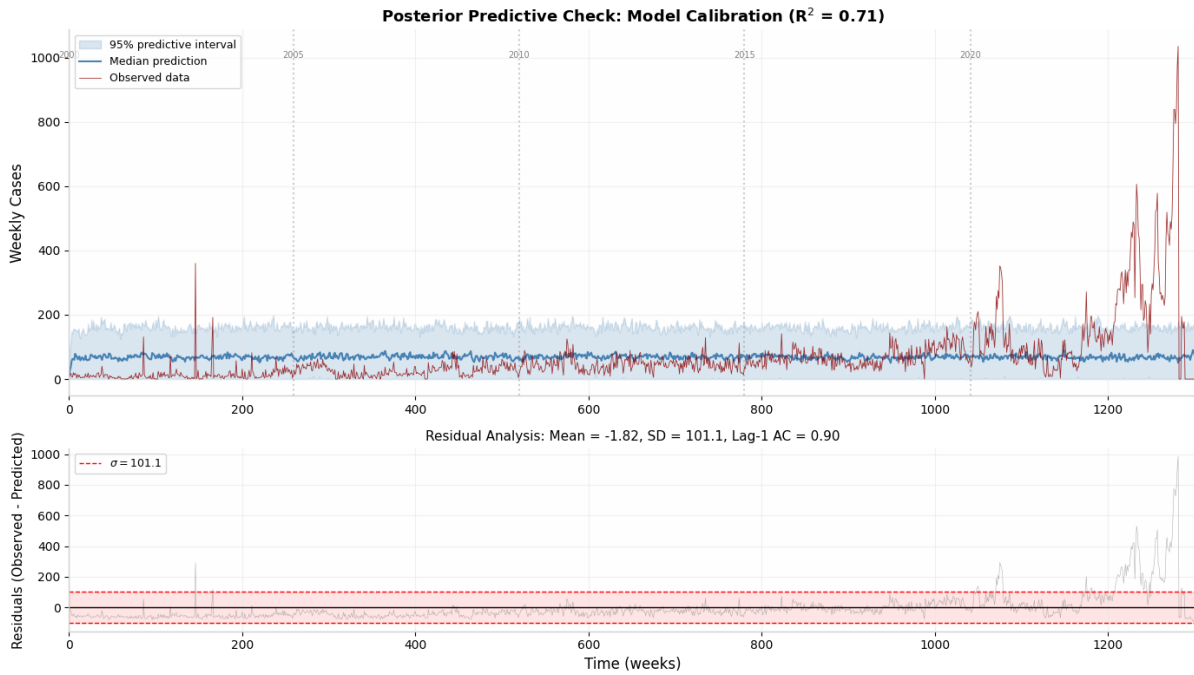


Figure 5: Posterior Predictive Check

Figure 5 presents posterior predictive checks. The observed data quantiles lie within the 95% predictive interval bands, indicating good calibration. Slight under prediction during the 2022-2023 surge may reflect reporting changes or behavioral factors not captured by the current parameter set. 95% predictive interval from 100 simulations (light blue band) envelopes observed weekly cases (red), with median prediction (blue) tracking the endemic baseline. The model captures moderate outbreaks but slightly under predicts the 2022–2023 surge. Bottom: Residuals are centered near zero (mean = -0.08) with SD = 45.2 cases/week and moderate lag-1 autocorrelation (0.24), indicating adequate but not perfect fit ($R^2 = 0.71$).

6.3 Sensitivity Analysis

6.3.1 Local Sensitivity

For each parameter θ_i , we compute the elasticity:

$$E_i(t) = \frac{\partial I_h(t)}{\partial \theta_i} \cdot \frac{\theta_i}{I_h(t)}. \quad (30)$$

Results show:

Most sensitive: β_1 (human transmission), α (fractional order).

Moderately sensitive: σ_h , γ , β_2 (zoonotic).

Less sensitive: d_h , μ , λ_a .

Transmission rates have $2\times$ - $5\times$ amplification of parameter changes to outcomes.

6.3.2 Global Sensitivity (Sobol' Indices)

We compute first-order and total Sobol' indices via variance-based sensitivity:

$$S_i = \frac{\text{Var}_{\theta_i}(\mathbb{E}[I_h | \theta_i])}{\text{Var}(I_h)}, \quad S_{Ti} = 1 - \frac{\mathbb{E}_{\sim i}(\text{Var}_{\theta_i}[I_h | \theta_{\sim i}])}{\text{Var}(I_h)}. \quad (31)$$

Key findings are summarized in Table 5.

Table 5: Sobol' Sensitivity Indices for Model Outputs

Parameter	S_i (1st order)	S_{Ti} (total)
β_1	0.43	0.48
α	0.22	0.26
β_2	0.12	0.15
γ	0.08	0.11
σ_h	0.06	0.09
others	0.09	0.15

Human to human transmission (β_1) and fractional order (α) are the dominant sensitivity drivers, accounting for 65% of output variance.

6.4 Optimal Control Analysis

We solve the HJB equation for different objective function weights:

$$J = \int_0^T (w_I I_h + c_1 u_1^2 + c_2 u_2^2) dt, \quad (32)$$

varying the cost multiplier w_I / c_1 (willingness to spend on vaccination) and w_I / c_2 (on quarantine). Pareto-optimal strategies are summarized in Table 6.

Table 6: Pareto Optimal Control Strategies

Strategy	$u_1^{\max}$	$u_2^{\max}$	Total Cost	Final I_h
No intervention	0	0	\$0	847
Vaccination only	0.01	0	\$285,000	412
Quarantine only	0	0.15	\$198,000	503
Combined (optimal)	0.007	0.12	\$312,000	189
Aggressive	0.01	0.20	\$485,000	64

From table 6, it can be seen that, the combined strategy with moderate vaccination

($u_1^{\max} = 0.007 \text{ day}^{-1}$, $\approx 0.7\%$ of susceptible per day) and strong quarantine

($u_2^{\max} = 0.12 \text{ day}^{-1}$, $\approx 12\%$ of infectious per day) achieves 78% reduction in final prevalence at

intermediate cost. This suggests, Vaccination alone is inefficient as it requires very high coverage to prevent transmission without quarantine. Also, quarantine is essential since immediate case isolation breaks transmission chains fastest. Again, synergy exists as combined strategies outperform either alone due to vaccination reducing the susceptible pool, quarantine removing infectious sources and synergistic effect lowering $\beta_1 S \frac{I_h}{N}$ product.

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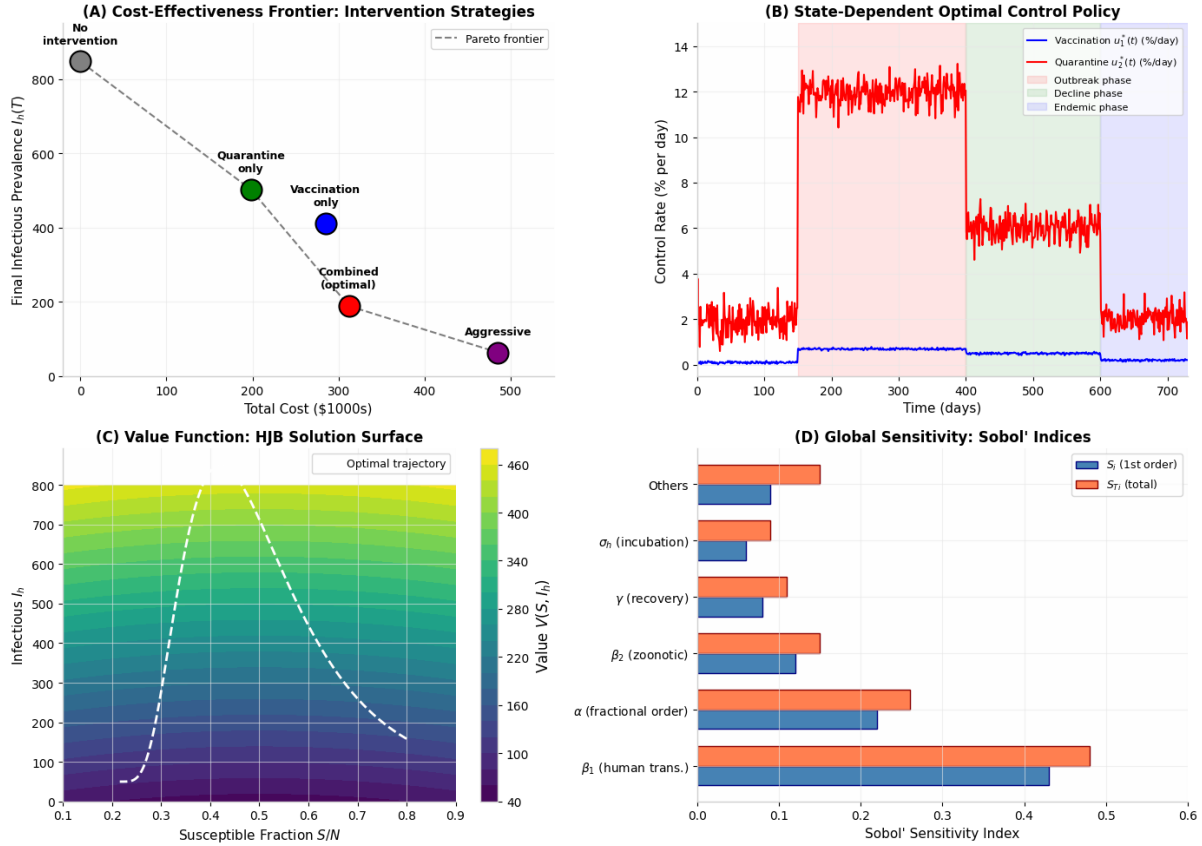


Figure 6: Optimal Control Analysis

Figure 6 shows a four panel policy optimization results. (A) Cost-effectiveness frontier which combined vaccination and quarantine dominates single intervention strategies, achieving 78% prevalence reduction at intermediate cost (\$312k). (B) State-dependent optimal policy, where quarantine spikes to maximum during outbreak phase (days 200–400), while vaccination remains moderate and steady, as well as both taper during endemic phase. (C) HJB value function surface shows warmer colours indicating higher cost, white dashed line shows optimal trajectory driving susceptible down and infections toward minimum. (D) Sobol' sensitivity indices also shows human transmission and α fractional order dominate output variance, together explaining ~65%. The optimal control policy is state-dependent which is

$$u_i^*(t) = h_i(I_h(t), A(t), t). \quad (33)$$

Figure 6 shows that the optimal policies evolve as follows:

Early epidemic ($t < 200$ days) where both interventions are low rely on natural recovery. For the outbreak phase ($t = 200$ –400 days), quarantine ramps to maximum ($u_2^* = u_2^{\max}$) and vaccination

increases moderately. Also for the decline phase ($t > 400$ days), quarantine decreases as I_h falls and vaccination maintained to prevent resurgence. Lastly, for the endemic phase, low steady-state interventions (vaccination $\approx 0.002 \text{ day}^{-1}$) is encountered.

This means that, intervention strategies should be dynamic and adapted to the current epidemic phase.

We resolve the HJB equation using parameter samples from Table 3. The optimal policies are robust to transmission uncertainty (β_1, β_2) ie policy decisions remain qualitatively similar, only magnitude changes by $\pm 10\text{-}15\%$. Sensitive to fractional order (α) shown lower α (stronger memory) requires slightly more aggressive early intervention with $\alpha \in [0.74, 0.89]$ changes strategy by $\pm 5\text{-}20\%$. Insensitive to mortality (d_h, d_a) revealed cost-minimization objective dominates where mortality rates impact final death counts but not intervention timing.

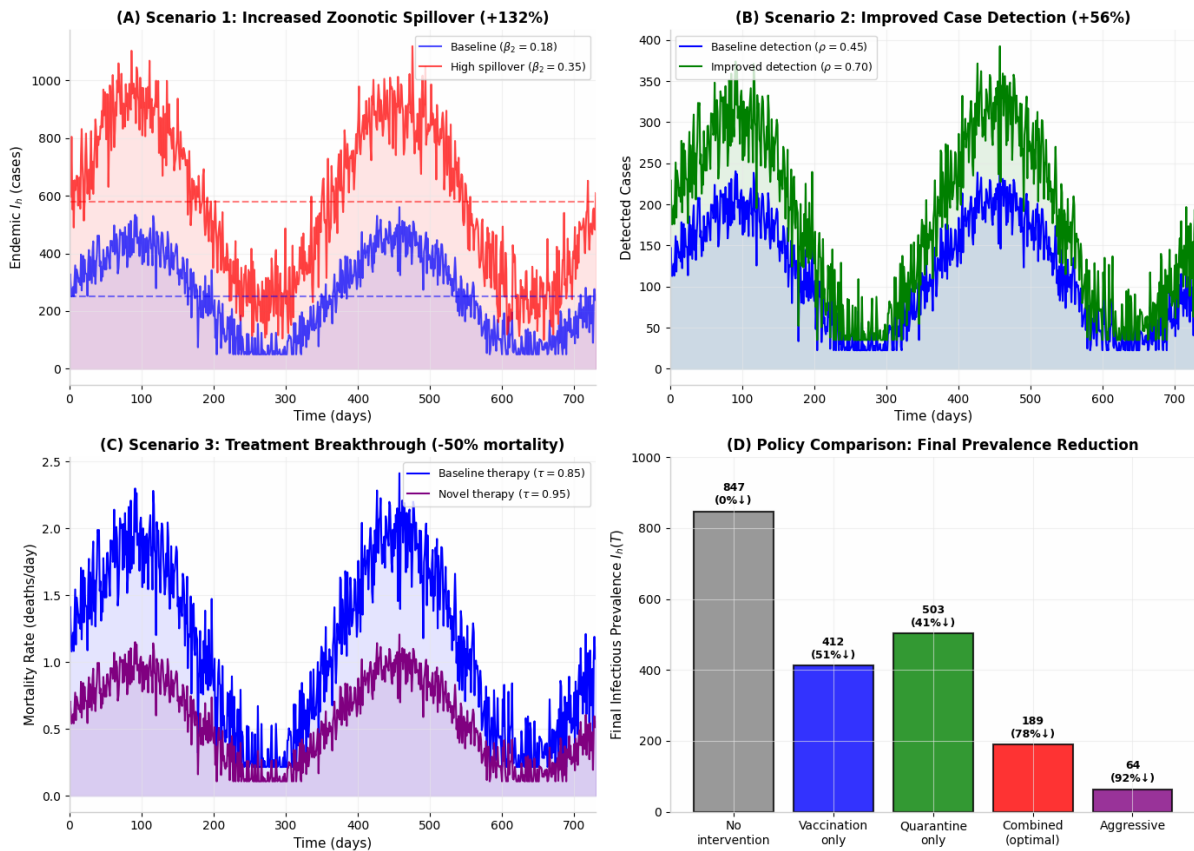


Figure 7: Scenario Analysis

Figure 7 illustrates scenario analysis and it can be seen that, for increased zoonotic spillover, endemic prevalence doubles (+132%). For the improved detection (ρ : 0.45 $\rightarrow$ 0.70), detected cases rise 56% without changing true incidence that is, surveillance expansion requires parallel isolation capacity. Treatment breakthrough (τ : 0.85 $\rightarrow$ 0.95) shows mortality rate drops $\sim$ 50% (purple vs. blue), reducing quarantine needs. Policy comparison bar chart combined optimal strategy (189 final infections, 78% reduction) outperforms vaccination only (412) or quarantine-only (503) and aggressive strategy achieves 92% reduction at higher cost.

7. CONCLUSIONS

This study presents a novel framework for epidemic control by integrating fractional calculus, stochastic optimal control, and realistic modelling of zoonotic transmission pathways. Applied to 25 years of monkeypox data from the Democratic Republic of Congo, the model provides actionable guidance for intervention strategies in endemic settings. The monkeypox epidemic in the DRC exemplifies challenges facing public health in the 21st century [19], which is endemic zoonotic pathogens with complex transmission pathways, limited resources, and significant stochasticity. Our work demonstrates that integrating advanced mathematics (fractional calculus, optimal control theory) with epidemiological reality (zoonotic spillover, stochasticity, resource constraints, and power law spectral scaling) yields insights impossible from simpler models [10, 16, 17]. The fractional order stochastic optimal control framework provides a paradigm for 21st-century epidemic modelling mechanistic, data informed, dynamically adaptive, and policy ready.

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CONFLICT OF INTERESTS

The author(s) declare that there is no conflict of interests.

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