AIMS Mathematics, 11(8): 24490–24524.
DOI: 10.3934/math.2026988
Received: 17 May 2026
Revised: 16 July 2026
Accepted: 20 July 2026
Published: 11 August 2026

http://www.aimspress.com/journal/Math

Research article

Modeling environmental and memory effects in epidemic transmission: Deterministic, stochastic, and fractional perspectives

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Abstract: In this work, we create and analyze a new model of an epidemic that incorporates indirect environmental transmission through a contaminated reservoir as well as direct human-to-human transmission. The environmental pathogen concentration is precisely predicted, and the population is separated into susceptible, exposed, infected, hospitalized, and recovered compartments. The transmission processes are described by saturated incidence functions to account for behavioral and environmental limitations. The model is investigated under three complementary mathematical frameworks. First, the deterministic integer-order system is analyzed to ascertain the global stability, boundedness, threshold dynamics, and positivity of the endemic and disease-free equilibria. Second, adequate conditions for disease extinction and persistence are determined by introducing stochastic perturbations to reflect random fluctuations resulting from environmental variability and unclear contact patterns. Third, the Caputo derivative is used to extend the model to a fractional-order formulation that takes into consideration memory effects found in biological systems. Volterra-type Lyapunov functionals are used to produce global stability results for the fractional system and to establish the existence and uniqueness of solutions. Numerical simulations are provided to illustrate the theoretical findings and to compare the effects of stochasticity and memory on disease dynamics. The results demonstrate that random perturbations can suppress outbreaks even when the basic reproduction number exceeds unity, while fractional-order dynamics significantly alter the speed and persistence of disease transmission.

Keywords: epidemic model; environmental transmission; Caputo fractional derivative; stochastic stability; global stability; Lyapunov function
Mathematics Subject Classification: 34D20, 34D23

1. Introduction

Infectious diseases still provide serious obstacles to sustainable growth, socioeconomic stability, and international public health systems. Cholera, influenza, Ebola, and COVID-19 are just a few of the infectious diseases that have frequently shown the intricacy of epidemic propagation and the shortcomings of traditional modeling techniques. The COVID-19 pandemic, in particular, revealed the critical role of asymptomatic transmission, environmental contamination, and stochastic effects in shaping epidemic trajectories. A wide range of deterministic and stochastic compartmental models were developed to find out the transmission dynamics of COVID-19 and to address the effect of non-pharmaceutical interventions [1–3]. These studies highlighted that environmental persistence of viral particles and random fluctuations in contact patterns can significantly affect outbreak severity and duration. Ebola virus disease represents another class of epidemics where environmental exposure and hospitalization dynamics play a key role. To explain frequent outbreaks and high case fatality rates, mathematical models of Ebola transmission have included hospital based transmission, delayed isolation, and environmental contamination [4, 5].

Stochastic formulations of Ebola models have further demonstrated that random environmental and behavioral factors can trigger out-break resurgence even after apparent disease control [6]. Cholera is a paradigmatic example of an environmentally mediated infectious disease, where pathogen concentration in water reservoirs directly influences transmission. The coupled human-environment dynamics of cholera transmission have been captured by a number of deterministic and stochastic models since the groundbreaking work of Codeço [7–9]. Fractional-order models for cholera have been introduced to account for memory effects related to pathogen persistence and the gradual loss of immunity, demonstrating better consistency with epidemiological data [10]. Influenza epidemics are characterized by strong seasonality, stochastic variability, and rapid mutation. Mathematical models of influenza transmission have emphasized the role of environmental persistence, random contact rates, and partial immunity in driving recurrent outbreaks [11, 12]. Recent stochastic and fractional-order influenza models have shown that memory effects and noise can substantially alter epidemic peaks and persistence patterns [13]. These disease-specific studies clearly demonstrate that realistic epidemic modeling requires the integration of environmental transmission pathways, stochastic perturbations, and memory effects. However, most existing works focus on a single disease or modeling framework, and few studies provide a unified analysis that simultaneously addresses deterministic, stochastic, and fractional-order dynamics within an environmentally mediated epidemic model. The rise and recurrence of infectious diseases including influenza, cholera, Ebola, and COVID-19 have brought attention to the critical need for strong mathematical tools that can accurately depict intricate transmission mechanisms and direct successful intervention tactics. Mathematical modeling has therefore become an essential component of modern epidemiology, providing quantitative insights into disease transmission, control, and long-term behavior. Traditional

deterministic compartmental models formulated through systems of ordinary differential equations have been widely employed to investigate the dynamics of infectious diseases. Foundational works, such as those by Hethcote [14] and Keeling and Rohani [15], established the theoretical basis for threshold dynamics, equilibrium analysis, and stability properties of epidemic models. These frameworks, important to mathematical epidemiology, have been effectively used for the analysis of different diseases. However, growing empirical evidence proposes that disease transmission is mostly impacted by indirect pathways, specially through contaminated environments. Environmental reservoirs are important in the transmission of waterborne and airborne diseases, such as respiratory infections, and cholera. Mathematical models incorporating environmental transmission have been shown to results in enforcement persistence and recurrent out-breaks [11, 12]. Such models stress the important of pathogen survival and decline in the environment as main drivers of disease dynamics. In spite of their mathematical tractability, deterministic models ignore the inherent randomness that exists in real epidemics. Environmental noise, demographic stochasticity, and random contact shapes can significantly change epidemic results, especially in small or heterogeneous populations. To handle these gapes, stochastic epidemic models have been modeled by introducing random perturbations into deterministic systems. These models allow the analysis of extinction probabilities, stochastic persistence, and noise-induced transitions [16, 17]. Recent studies have shown that stochastic impacts can either suppress or amplify disease transmission, even when the deterministic basic reproduction number remains unchanged [18–20]. The capacity of standard integer-order models to account for memory and genetic effects, which are fundamental to many biological processes, is another significant drawback. Fractional-order epidemic models, formulated using non-integer order derivatives, have emerged as a powerful framework for incorporating long-term memory and anomalous diffusion into disease dynamics. Among the different formulations, the Caputo fractional derivative is especially appealing because it accommodates classical initial conditions and offers a clear physical interpretation [21]. Fractional epidemic models have been shown to exhibit slower convergence rates, richer transient dynamics, and improved agreement with empirical data [22–24]. Despite the extensive literature on environmental transmission [7, 8], stochastic epidemic models [2, 25], and fractional-order formulations [22–24], no existing study combines all three aspects within a unified SEIHRM structure with saturated incidence. Specifically, most environmental models ignore stochastic noise or memory, stochastic models rarely include fractional dynamics, and fractional models seldom treat environmental contamination together with hospitalization. Our work fills this gap by providing a consistent mathematical framework where the same incidence functions, parameter definitions, and Lyapunov function techniques are applied across deterministic, stochastic, and fractional domains. The novelty lies in the simultaneous global stability analysis for all three frameworks and the derivation of explicit noise thresholds for disease extinction. In recent years, considerable attention has been devoted to epidemic models that combine stochasticity and fractional-order dynamics. Such hybrid frameworks provide a more realistic description of disease spread by accounting simultaneously for random perturbations and memory effects [26, 27]. Nevertheless, most existing studies focus on simplified transmission mechanisms or consider only one modeling framework at a time. Comprehensive investigations integrating environmental transmission, stochastic perturbations, and fractional-order dynamics within a unified model remain relatively scarce. Motivated by these gaps, this paper proposes and analyzes a novel epidemic model incorporating direct transmission, environmental contamination, hospitalization, and recovery. Deterministic, stochastic, and fractional-order formulations are the three

complimentary frameworks under which the model is examined. The deterministic model is employed to compute the basic reproduction number and to establish global stability results for both the disease-free and endemic equilibria. To evaluate illness persistence under random perturbations, the stochastic model is numerically examined, taking demographic and environmental noise into consideration. The Caputo fractional derivative is used to build the fractional-order model, which captures memory effects and offers a more profound comprehension of the dynamics of chronic disease. The main contributions of this work are summarized as follows:

- We propose a comprehensive epidemic model incorporating environmental transmission and hospitalization.
- The global stability of the endemic and disease-free equilibria is systematically analyzed, and the basic reproduction number is determined.
- We perform deterministic and stochastic sensitivity analyses to identify key parameters influencing disease transmission.
- We look into how noise level affects the persistence of sickness.
- We examine the impact of fractional order on convergence dynamics and memory effects.

The rest of this paper is organized as follows: Section 2 introduces the fundamental concepts applied in this study. In Section 3, model formulation are conducted. Sections 4–6, presents the basic mathematical properties of deterministic, fractional, stochastic models, respectively. Numerical simulations, and sensitivity analyses are provided in Section 7. Finally, Section 8 concludes the paper and provides directions for future work.

2. Preliminaries and important definitions

The definitions and mathematical techniques used throughout the research to analyze the deterministic, stochastic, and fractional-order epidemic models are provided in this subsection.

2.1. Deterministic dynamical systems

Consider an autonomous system of ordinary differential equations (ODEs)

$$\frac{d\tilde{X}(t)}{dt} = F(\tilde{X}(t)), \qquad \tilde{X}(0) = \tilde{X}_0 \in \mathbb{R}^n, \tag{2.1}$$

where $F : \mathbb{R}^n \to \mathbb{R}^n$ is a continuously differentiable vector field.

Definition 2.1 (Equilibrium point)**.** A point $\tilde{X}^* \in \mathbb{R}^n$ is called an equilibrium (or steady state) of the system if

$$F(\tilde{X}^*) = 0.$$

Definition 2.2 (Global asymptotic stability (GAS))**.** An equilibrium point $\tilde{X}^*$ is said to be GAS in a region Ω if and only if

$$\lim_{t\to\infty} \tilde{X}(t) = \tilde{X}^*, \qquad \forall \tilde{X}(0) \in \Omega.$$

Definition 2.3 (Local asymptotic stability (LAS))**.** An equilibrium point $\tilde{X}^*$ is said to be LAS if it is stable and

$$\lim_{t\to\infty} \tilde{X}(t) = \tilde{X}^*$$

for all initial conditions $\tilde{X}(0)$ sufficiently close to $\tilde{X}^*$.

The above concepts are standard in dynamical systems theory and epidemic modeling [14, 28].

2.2. Lyapunov functions

Definition 2.4 (Lyapunov function)**.** Let $\tilde{X}^*$ be a fixed point of the system. $V : \Omega \to \mathbb{R}$ is a continuously differentiable function, then it is called a Lyapunov function if

$$V(\tilde{X}^*) = 0, \quad V(\tilde{X}) > 0 \text{ for } \tilde{X} \neq \tilde{X}^*,$$

and its differentiation along solutions satisfies

$$\dot{V}(\tilde{X}) \leq 0 \quad \text{in } \Omega.$$

If $\dot{V}(\tilde{X}) < 0$ for all $\tilde{X} \neq \tilde{X}^*$, then $\tilde{X}^*$ is globally asymptotically stable in Ω [28, 29].

2.3. Basic reproduction number

Definition 2.5 (Basic reproduction number)**.** The estimated number of secondary cases produced by introducing a single sick person into a community that is completely susceptible is represented by the basic reproduction number R_0.

The next-generation matrix approach is frequently used to calculate R_0 in compartmental epidemic models [15, 30]. A key factor in deciding whether a disease persists or goes extinct is the threshold property of R_0.

2.4. Stochastic differential equations

We take into consideration stochastic differential equations (SDEs) of the type

$$d\tilde{X}(t) = F(\tilde{X}(t))\,dt + G(\tilde{X}(t))\,dW(t), \tag{2.2}$$

where $W(t)$ is a conventional Brownian motion, in order to account for random perturbations.

Definition 2.6 (Itô solution)**.** A stochastic process $\tilde{X}(t)$ is said to be a solution of the above SDE if it satisfies the equation almost surely for all $t \geq 0$.

Definition 2.7 (Stochastic stability)**.** An equilibrium point $\tilde{X}^*$ is called stochastically stable if, for every $\varepsilon > 0$,

$$\lim_{\tilde{X}(0)\to\tilde{X}^*} \mathbb{P}\left(\sup_{t\geq 0} \|\tilde{X}(t) - \tilde{X}^*\| < \varepsilon\right) = 1.$$

Stochastic epidemic models and their stability properties are well documented in [16, 20].

2.5. Fractional calculus

Fractional calculus generalizes classical differentiation and integration to non-integer orders and is particularly suitable for modeling memory and hereditary effects.

Definition 2.8 (Caputo fractional derivative)**.** Let $q \in (0,1)$ and $f \in C^1([0,T])$. The Caputo fractional derivative of order q is given by

$$ {}^{C}D_t^q f(t) = \frac{1}{\Gamma(1-q)} \int_0^t (t-s)^{-q} f'(s)\, ds. $$

The Caputo derivative is widely favored in physical and biological modeling because it is consistent with classical initial conditions [21, 31].

Definition 2.9 (Fractional-order dynamical system)**.** A fractional-order system is given by

$$ {}^{C}D_t^q \tilde{X}(t) = F(\tilde{X}(t)), \qquad 0 < q < 1. $$

Fractional-order epidemic models have been shown to exhibit slower convergence and richer transient behavior than integer-order models [22, 23].

2.6. Threshold dynamics

Lemma 2.1 (LaSalle's invariance principle)**.** *[29] Let $V : \Omega \to \mathbb{R}_+$ be a continuously differentiable function satisfying $\dot{V} \leq 0$ in Ω. Then any trajectory that starts in Ω converges to the largest invariant subset of $\{\tilde{X} \in \Omega : \dot{V} = 0\}$.*

Lemma 2.2 (Moment boundedness)**.** *[16, 25] Let $X(t)$ denote the solution to the stochastic epidemic model. Then, for every $p \geq 2$, there exists a positive constant C_p such that*

$$ \sup_{t \geq 0} \mathbb{E}\|\tilde{X}(t)\|^p \leq C_p. $$

Lemma 2.3 (Caputo chain-rule inequality)**.** *Let $q \in (0,1)$ and let $X : [0,T] \to \mathbb{R}^n$ be a continuously differentiable function. Suppose that $V : \mathbb{R}^n \to \mathbb{R}$ is a convex function of class C^1. Then*

$$ {}^{C}D_t^q V(X(t)) \leq \nabla V(X(t))^{\top}\, {}^{C}D_t^q X(t), $$

where ${}^{C}D_t^q$ denotes the Caputo fractional derivative and

$$ \nabla V(X) = \left(\frac{\partial V}{\partial x_1}, \frac{\partial V}{\partial x_2}, \ldots, \frac{\partial V}{\partial x_n} \right)^{\top}. $$

3. Model formulation

In this section, we create a deterministic epidemic model that incorporates direct human-to-human transmission as well as indirect environmental transmission. The model accounts for the duration of illness, hospitalization, and recovery in addition to correcting for saturation effects in the transmission pathways.

3.1. Deterministic model

The following system of ordinary differential equations governs the deterministic dynamics of the epidemic under the above-mentioned assumptions:

$$\begin{cases} \dfrac{dS_a}{dt} = \Lambda - \dfrac{\beta_1 S_a M_a}{1+\alpha_1 M_a} - \dfrac{\beta_2 S_a I_a}{1+\alpha_2 I_a} - (\mu+\nu)S_a, \\ \dfrac{dE_a}{dt} = \dfrac{\beta_1 S_a M_a}{1+\alpha_1 M_a} + \dfrac{\beta_2 S_a I_a}{1+\alpha_2 I_a} - (\mu+\kappa)E_a, \\ \dfrac{dI_a}{dt} = \kappa E_a - (\mu+\delta_1+\tau)I_a, \\ \dfrac{dH_a}{dt} = \tau I_a - (\mu+\delta_2+\rho)H_a, \\ \dfrac{dR_a}{dt} = \rho H_a - (\mu+\phi_1)R_a, \\ \dfrac{dM_a}{dt} = \eta I_a - dM_a. \end{cases} \tag{3.1}$$

The initial conditions are given by

$$S_a(0) > 0, \quad I_a(0) \geq 0, \quad E_a(0) \geq 0, \quad R_a(0) \geq 0, \quad H_a(0) \geq 0, \quad M_a(0) \geq 0. \tag{3.2}$$

The total human population at time t, denoted by $N_a(t)$, is divided into five epidemiological compartments: Susceptible individuals $S_a(t)$, Exposed individuals $E_a(t)$, Infectious individuals $I_a(t)$, Hospitalized individuals $H_a(t)$, and Recovered individuals $R_a(t)$. In addition, the concentration of pathogens in the environment is represented by $M_a(t)$. The parameters used in the above system, are presented in Table 1.

Table 1. Description and values of the parameters for the SEIHRM model.

Parameter	Biological description
Λ	Recruitment rate of susceptible population
β_1	Transmission rate from environmental pathogen (M_a)
β_2	Direct transmission rate from infectious individuals (I_a)
α_1	Saturation constant for environmental transmission
κ	Progression rate from Exposed (E_a) to Infected (I_a)
δ_2	Disease-induced death rate for Hospitalized individuals (H_a)
α_2	Saturation constant for human-to-human transmission
μ	Natural death rate
δ_1	Disease-induced death rate for Infected individuals (I_a)
ν	Vaccination or natural immunity rate
τ	Rate of hospitalization for infected individuals
d	Natural decay rate of the pathogen in the environment
ϕ_1	Rate of loss of immunity in recovered individuals
ρ	Recovery rate for hospitalized individuals
η	Pathogen shedding rate into the environment

3.2. Fractional-order model

To incorporate memory effects inherent in biological systems, we generalize model (3.1) to a fractional-order formulation by employing the Caputo fractional derivative. For a sufficiently smooth function $X(t)$ and $0 < q < 1$, the Caputo derivative is given by

$$^{C}D_t^q X(t) = \frac{1}{\Gamma(1-q)} \int_0^t \frac{X'(s)}{(t-s)^q}\, ds.$$

The fractional-order epidemic model is then given by

$$\begin{cases} ^{C}D_t^q S_a = \Lambda - \dfrac{\beta_1 S_a M_a}{1+\alpha_1 M_a} - \dfrac{\beta_2 S_a I_a}{1+\alpha_2 I_a} - (\mu+\nu) S_a, \\ ^{C}D_t^q E_a = \dfrac{\beta_1 S_a M_a}{1+\alpha_1 M_a} + \dfrac{\beta_2 S_a I_a}{1+\alpha_2 I_a} - (\mu+\kappa) E_a, \\ ^{C}D_t^q I_a = \kappa E_a - (\mu+\delta_1+\tau) I_a, \\ ^{C}D_t^q H_a = \tau I_a - (\mu+\delta_2+\rho) H_a, \\ ^{C}D_t^q R_a = \rho H_a - (\mu+\phi_1) R_a, \\ ^{C}D_t^q M_a = \eta I_a - d M_a. \end{cases} \tag{3.3}$$

When $q = 1$, system (3.3) reduces to the classical deterministic model (3.1). We may examine how memory effects influence the disease's persistence and spread using the fractional-order model.

3.3. Stochastic model formulation

Deterministic epidemic models assume that all parameters and interactions evolve smoothly in time. However, in real biological systems, disease transmission and environmental contamination are often subject to random fluctuations caused by climatic variability, behavioral changes, and unpredictable contact patterns. We expand the deterministic model (3.1) to a stochastic framework in order to capture these effects.

3.4. Incorporation of stochastic perturbations

We assume that random perturbations affect the transmission process and the environmental pathogen dynamics. In particular, we consider that the effective contact rate and the pathogen shedding process fluctuate randomly around their mean values. These fluctuations are modeled by independent standard Brownian motions. Let $(\Omega, \mathcal{F}, \{\mathcal{F}_t\}_{t\geq 0}, \mathbb{P})$ be a complete filtered probability space that meets the standard requirements and let

$$B_i(t), \quad 1 \leq i \leq 3,$$

be independent standard Brownian motions defined on this space.

3.5. Stochastic epidemic model

The following system of stochastic differential equations in the Itô sense is the stochastic counterpart of system (3.1):

$$\begin{cases} dS_a = \Big[\Lambda - \dfrac{\beta_1 S_a M}{1+\alpha_1 M} - \dfrac{\beta_2 S_a I}{1+\alpha_2 I} - (\mu+\nu)S_a\Big]dt + \sigma_1 S\, dB_1(t), \\ dE = \Big[\dfrac{\beta_1 S_a M}{1+\alpha_1 M} + \dfrac{\beta_2 S_a I}{1+\alpha_2 I} - (\mu+\kappa)E\Big]dt + \sigma_2 E\, dB_2(t), \\ dI = \Big[\kappa E - (\mu+\delta_1+\tau)I\Big]dt + \sigma_3 I\, dB_3(t), \\ dH = \Big[\tau I - (\mu+\delta_2+\rho)H\Big]dt, \\ dR = \Big[\rho H - (\mu+\phi_1)R\Big]dt, \\ dM = \Big[\eta I - dM\Big]dt + \sigma_4 M\, dB_4(t). \end{cases} \tag{3.4}$$

Where $\sigma_i > 0$ $(i = 1, 2, 3, 4)$ the intensities of the stochastic perturbations are represented. The noise terms are assumed to be proportional to the state variables, ensuring that the stochastic perturbations vanish when the corresponding compartments are empty. This modeling choice preserves biological realism and guarantees the non-negativity of solutions. The initial history for model (3.4) is as follow:

$$S_a(0) > 0, \quad E_a(0) \geq 0, \quad I_a(0) \geq 0, \quad H_a(0) \geq 0, \quad R_a(0) \geq 0, \quad M_a(0) \geq 0. \tag{3.5}$$

Which are assumed to be $\mathcal{F}_0$-measurable random variables.
Remark: When noise intensities σ_i vanish, system (3.4) reduces to the deterministic model (3.1). The stochastic formulation thus provides a natural extension of the deterministic framework and allows us to investigate the impact of random perturbations on disease extinction and persistence.

4. Basic properties of deterministic model

Theorem 4.1. *For any initial conditions given in Eq* (3.2)*, the solution of system* (3.1) *remains nonnegative for all* $t \geq 0$*.*

Proof. We verify the positivity of each state variable of the proposed model (3.1).

From the first equation of (3.1), we have

$$\frac{dS_a}{dt} = \Lambda - \frac{\beta_1 S_a M_a}{1+\alpha_1 M_a} - \frac{\beta_2 S_a I_a}{1+\alpha_2 I_a} - (\mu+\nu)S_a.$$

Since $M_a, I_a \geq 0$ and all parameters are positive, we use the estimate

$$\frac{\beta_i S_a M_a}{1+\alpha_i M_a} \leq \frac{\beta_i}{\alpha_i} S_a, \qquad i = 1, 2,$$

which follows from the fact that

$$\frac{M_a}{1+\alpha_i M_a} \leq \frac{1}{\alpha_i}, \qquad i = 1, 2.$$

Therefore,

$$\frac{dS_a}{dt} \geq \Lambda - \left(\frac{\beta_1}{\alpha_1} + \frac{\beta_2}{\alpha_2} + \mu + \nu\right) S_a.$$

Let

$$\gamma = \mu + \nu + \frac{\beta_1}{\alpha_1} + \frac{\beta_2}{\alpha_2}.$$

Then

$$\frac{dS_a}{dt} \geq \Lambda - \gamma S_a \geq -\gamma S_a.$$

Solving the differential inequality yields

$$S_a(t) \geq S_a(0)e^{-\gamma t} \geq 0.$$

Similarly, from the second equation of (3.1), we have

$$\frac{dE_a}{dt} = \frac{\beta_1 S_a M_a}{1 + \alpha_1 M_a} + \frac{\beta_2 S_a I_a}{1 + \alpha_2 I_a} - (\mu + \kappa)E_a \geq -(\mu + \kappa)E_a,$$

which implies

$$E_a(t) \geq E_a(0)e^{-(\mu+\kappa)t} \geq 0.$$

From the third equation,

$$\frac{dI_a}{dt} = \kappa E_a - (\mu + \delta_1 + \tau)I_a \geq -(\mu + \delta_1 + \tau)I_a,$$

yielding

$$I_a(t) \geq I_a(0)e^{-(\mu+\delta_1+\tau)t} \geq 0.$$

From the fourth and fifth equations of model (3.1), we obtain

$$H_a(t) \geq H_a(0)e^{-(\mu+\delta_2+\rho)t} \geq 0,$$

and

$$R_a(t) \geq R_a(0)e^{-(\mu+\phi_1)t} \geq 0.$$

Finally, from the last equation,

$$\frac{dM_a}{dt} = \eta I_a - dM_a \geq -dM_a,$$

which gives

$$M_a(t) \geq M_a(0)e^{-dt} \geq 0.$$

Therefore, all solutions of system (3.1) remain nonnegative for all $t \geq 0$. This completes the proof. □

4.1. Boundedness and invariant region

Lemma 4.1. *To show that the total population $N_a(t)$ satisfies the condition of*

$$\frac{dN_a}{dt} \leq \Lambda - \mu N_a(t).$$

Proof. Let us define the total human population as

$$N_a(t) = S_a(t) + E_a(t) + I_a(t) + H_a(t) + R_a(t). \tag{4.1}$$

Using the first five equations of system (3.1) in Eq (4.1) and after simplification, we get

$$\begin{aligned}\frac{dN_a}{dt} &= \Lambda - \mu N_a - \nu S_a - \delta_1 I_a - \delta_2 H_a - \phi_1 R_a,\\ &\leq \Lambda - \mu N_a.\end{aligned}$$

□

Theorem 4.2. *Show that our proposed model (3.1) is bounded for all $t \geq 0$.*

Proof. First, let us prove the boundedness of the human population of our proposed model (3.1). Solving the differential inequality in Lemma 4.1, we obtain

$$N_a(t) \leq \frac{\Lambda}{\mu} + \left(N_a(0) - \frac{\Lambda}{\mu}\right)e^{-\mu t} \quad \text{for all } t \geq 0.$$

Thus we get

$$\limsup_{t\to\infty} N_a(t) \leq \frac{\Lambda}{\mu}. \tag{4.2}$$

Using the bound $I_a(t) \leq N_a(t) \leq \Lambda/\mu$ in the last equation of (3.1), we obtain

$$\frac{dM_a}{dt} \leq \eta\frac{\Lambda}{\mu} - dM_a.$$

Solving this inequality yields

$$M_a(t) \leq \frac{\eta\Lambda}{\mu d} + \left(M_a(0) - \frac{\eta\Lambda}{\mu d}\right)e^{-dt},$$

or

$$\limsup_{t\to\infty} M_a(t) \leq \frac{\eta\Lambda}{\mu d}. \tag{4.3}$$

From Eqs (4.2) and (4.3), we get that our proposed model (3.1) is bounded for all $t \geq 0$. □

Theorem 4.3. *System* (3.1) *is positively invariant in the region*

$$\Omega = \left\{(S_a, E_a, I_a, H_a, R_a, M_a) \in \mathbb{R}^6_+ : N_a(t) \leq \frac{\Lambda}{\mu},\ M_a(t) \leq \frac{\eta\Lambda}{\mu d}\right\}.$$

Proof. According to Lemma 4.1, for any $t \geq 0$ that yielded $N_a(0) \leq \Lambda/\mu$, $N_a(t) \leq \Lambda/\mu$. Moreover, using the bound $I(t) \leq N_a(t) \leq \Lambda/\mu$ in the last equation of (3.1), we obtain

$$\frac{dM_a}{dt} \leq \eta\frac{\Lambda}{\mu} - dM_a.$$

Solving this inequality yields

$$M_a(t) \leq \frac{\eta\Lambda}{\mu d} + \left(M_a(0) - \frac{\eta\Lambda}{\mu d}\right)e^{-dt}.$$

Hence, $M_a(t)$ is bounded for all $t \geq 0$, and the region Ω is positively invariant. □

The results of this section ensure that model (3.1) is mathematically well-posed and biologically meaningful. All subsequent analyses are therefore conducted within the invariant region Ω.

4.2. Equilibria and threshold dynamics

In this subsection, we derive the basic reproduction number, which governs the threshold dynamics of the disease, along with the equilibrium points of system (3.1).

4.3. Disease-free equilibrium

In this subsection, we determine the equilibrium points of system (3.1) and calculate the fundamental reproduction number, which describes the threshold dynamics of the disease

$$E_a = I_a = H_a = R_a = M_a = 0.$$

We obtain the disease-free equilibrium (DFE)

$$\tilde{\mathbf{X}}_0 = ({S_a}^0, 0, 0, 0, 0, 0), \text{where} \quad {S_a}^0 = \frac{\Lambda}{\mu + \nu}.$$

4.4. Endemic equilibrium and R_0

A steady-state solution of system (3.1) in which all components are strictly positive is referred to as an endemic equilibrium,

$$\tilde{\mathbf{X}}^* = (S_a^*, E_a^*, I_a^*, H_a^*, R_a^*, M_a^*).$$

Explicit formulas are omitted since they are not needed for the global stability analysis. Such an equilibrium exists whenever $R_0 > 1$.

The basic reproduction number, denoted by R_0, represents the expected number of secondary infections generated by a single infectious individual introduced into a wholly susceptible population. To compute R_0, we apply the next-generation matrix method. Let

$$\tilde{\mathbf{X}} = (E_a, I_a, M_a)^T,$$

denote the vector of infected compartments. The dynamics of $\tilde{\mathbf{X}}$ can be written as:

$$\frac{d\tilde{\mathbf{X}}}{dt} = \mathcal{F}(\tilde{\mathbf{X}}) - \mathcal{V}(\tilde{\mathbf{X}}),$$

where $\mathcal{V}$ stands for the transfer across compartments and $\mathcal{F}$ for the rate at which new infections arise. At the DFE $\tilde{\mathbf{X}}_0$, we have

$$\mathcal{F} = \begin{pmatrix} \dfrac{\beta_1 {S_a}^0 M_a}{1+\alpha_1 M_a} + \dfrac{\beta_2 {S_a}^0 I_a}{1+\alpha_2 I_a} \\ 0 \\ 0 \end{pmatrix}, \qquad \mathcal{V} = \begin{pmatrix} (\mu+\kappa)E_a \\ (\mu+\delta_1+\tau)I_a - \kappa E_a \\ dM_a - \eta I_a \end{pmatrix}.$$

The Jacobian matrices of $\mathcal{F}$ and $\mathcal{V}$ that were assessed at the DFE are provided as

$$F = \begin{pmatrix} 0 & \beta_2 {S_a}^0 & \beta_1 {S_a}^0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}, \qquad V = \begin{pmatrix} \mu+\kappa & 0 & 0 \\ -\kappa & \mu+\delta_1+\tau & 0 \\ 0 & -\eta & d \end{pmatrix}.$$

We have

$$\mathcal{V}^{-1} = \begin{bmatrix} \frac{1}{\mu+\kappa} & 0 & 0 \\ \frac{\kappa}{(\mu+\kappa)(\mu+\delta_1+\tau)} & \frac{1}{\mu+\delta_1+\tau} & 0 \\ \frac{\kappa\eta}{(\mu+\kappa)(\mu+\delta_1+\tau)d} & \frac{\eta}{d(\mu+\delta_1+\tau)} & \frac{1}{d} \end{bmatrix}.$$

The next-generation matrix is defined as

$$K = \mathcal{F}\mathcal{V}^{-1} = \begin{pmatrix} \frac{\beta_2 S_a^0 \kappa}{(\mu+\kappa)(\mu+\delta_1+\tau)} + \frac{\beta_1 S_a^0 \kappa\eta}{(\mu+\kappa)(\mu+\delta_1+\tau)d} & \frac{\beta_2 S_a^0}{\mu+\delta_1+\tau} + \frac{\beta_1 S_a^0 \eta}{d(\mu+\delta_1+\tau)} & \frac{\beta_1 S_a^0}{d} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}.$$

The basic reproduction number R_0 is the spectral radius of K, which equals the only non-zero entry

$$R_0 = \frac{\kappa S_a^0}{\mu+\kappa}\left(\frac{\beta_2}{\mu+\delta_1+\tau} + \frac{\beta_1\eta}{d(\mu+\delta_1+\tau)}\right).$$

where $\rho(K)$ represents K's spectral radius.

Substituting $S_0 = \Lambda/(\mu+\nu)$ yields

$$R_0 = \frac{\kappa\Lambda}{(\mu+\nu)(\mu+\kappa)(\mu+\delta_1+\tau)}\left(\beta_2 + \frac{\beta_1\eta}{d}\right). \tag{4.4}$$

4.5. Biological interpretation of R_0

The basic reproduction number consists of two additive components. The term involving β_2 corresponds to direct human-to-human transmission, while the term involving $\beta_1\eta/d$ represents indirect transmission mediated by environmental contamination. The parameter τ reflects the impact of hospitalization, which reduces the infectious period and hence decreases R_0.

Therefore, disease elimination is possible if $R_0 < 1$, while disease persistence occurs when $R_0 > 1$.

4.6. Stability analysis of the deterministic model

In this subsection, we examine the global asymptotic stability of the disease-free equilibrium of system (3.1). The analysis reveals that the basic reproduction number R_0 serves as a crucial threshold parameter determining whether the disease dies out or persists.

Theorem 4.4. *Within the biologically feasible region* Ω*, the disease-free equilibrium* $\tilde{\mathbf{X}}_0$ *of system* (3.1) *is globally asymptotically stable whenever* $R_0 < 1$.

Proof. Consider the Lyapunov function as prescribed in [32].

$$\mathcal{L}(t) = E_a(t) + a_1 I_a(t) + a_2 M_a(t), \tag{4.5}$$

where $a_1 > 0$ and $a_2 > 0$ are constants to be determined. For all $t \geq 0$, the function $\mathcal{L}(t)$ is nonnegative and

$$\mathcal{L}(t) = 0 \quad \text{if and only if} \quad E_a(t) = I_a(t) = M_a(t) = 0.$$

Following the solutions of system (3.1), the time derivative of (4.5) yields

$$\begin{aligned}\frac{d\mathcal{L}}{dt} &= \frac{dE_a}{dt} + a_1\frac{dI_a}{dt} + a_2\frac{dM_a}{dt}\\ &= \frac{\beta_1 S_a M_a}{1+\alpha_1 M_a} + \frac{\beta_2 S_a I_a}{1+\alpha_2 I_a} - (\mu+\kappa)E_a\\ &\quad + a_1(\kappa E_a - (\mu+\delta_1+\tau)I_a) + a_2(\eta I_a - dM_a).\end{aligned}$$

Since $S_a(t) \leq {S_a}^0 = \Lambda/(\mu+\nu)$ for all $t \geq 0$ in Ω, we obtained

$$\frac{\beta_1 S_a M_a}{1+\alpha_1 M_a} \leq \beta_1 {S_a}^0 M_a, \quad \frac{\beta_2 S_a I_a}{1+\alpha_2 I_a} \leq \beta_2 {S_a}^0 I_a.$$

Thus

$$\begin{aligned}\frac{d\mathcal{L}}{dt} &\leq -(\mu+\kappa)E_a + a_1\kappa E_a\\ &\quad + (\beta_2 {S_a}^0 - a_1(\mu+\delta_1+\tau) + a_2\eta)I_a + (\beta_1 {S_a}^0 - a_2 d)M_a.\end{aligned}$$

We now choose the constants

$$a_1 = \frac{\mu+\kappa}{\kappa}, \qquad a_2 = \frac{\beta_1 {S_a}^0}{d}.$$

With this choice, the E_a and M_a terms vanish, and we obtain

$$\frac{d\mathcal{L}}{dt} \leq (\mu+\kappa)(R_0 - 1) I_a.$$

If $R_0 < 1$, then we get

$$\frac{d\mathcal{L}}{dt} \leq 0 \quad \text{for all } t \geq 0.$$

Moreover,

$$\frac{d\mathcal{L}}{dt} = 0 \quad \text{if and only if} \quad E_a = I_a = M_a = 0.$$

From system (3.1), it follows that $H_a(t)$ and $R_a(t)$ also converge to zero, while $S_a(t) \to {S_a}^0$ as $t \to \infty$.

By LaSalle's invariance principle (Lemma 2.1), all solutions of system (3.1) starting in Ω approach the disease-free equilibrium $\tilde{\mathbf{X}}_0$ as $t \to \infty$. □

4.7. Global stability of the endemic equilibrium (EE)

In this subsection, we prove that when the fundamental reproduction number is more than unity, the EE of system (3.1) is globally asymptotically stable.

Theorem 4.5. *If $R_0 > 1$, then system* (3.1) *admits an EE* $\tilde{\mathbf{X}}^*$ *that is globally asymptotically stable.*

Proof. Assume that $R_0 > 1$, so that system (3.1) has a unique endemic equilibrium $\tilde{\mathbf{X}}^*$ with strictly positive components. The Volterra-type Lyapunov functional is defined as follows:

$$\begin{aligned}\mathcal{V}(t) = {} & S_a^*\left(\frac{S_a}{S_a^*} - 1 - \ln\frac{S_a}{S_a^*}\right) + E_a^*\left(\frac{E}{E_a^*} - 1 - \ln\frac{E_a}{E^*}\right) \\ & + c_1 I_a^*\left(\frac{I_a}{I_a^*} - 1 - \ln\frac{I}{I^*}\right) + c_2 H_a^*\left(\frac{H_a}{H_a^*} - 1 - \ln\frac{H_a}{H_a^*}\right) \\ & + c_3 M^*\left(\frac{M_a}{M_a^*} - 1 - \ln\frac{M_a}{M_a^*}\right),\end{aligned} \tag{4.6}$$

where the positive constants c_1, c_2, and c_3 need to be found.

The functional $\mathcal{V}(t)$ is nonnegative for all $t \geq 0$, and

$$\mathcal{V}(t) = 0 \quad \text{if and only if} \quad (S_a, E_a, I_a, H_a, M_a) = (S_a^*, E_a^*, I_a^*, H_a^*, M_a^*).$$

Using the identity

$$\frac{d}{dt}\left[\tilde{X}^*\left(\frac{\tilde{X}}{\tilde{X}^*} - 1 - \ln\frac{\tilde{X}}{\tilde{X}^*}\right)\right] = \left(1 - \frac{\tilde{X}^*}{\tilde{X}}\right)\frac{dx}{dt}. \tag{4.7}$$

We compute the time derivative of $\mathcal{V}$ along the solutions of system (3.1) and using of formula (4.7).

$$\begin{aligned}\frac{d\mathcal{V}}{dt} = {} & \left(1 - \frac{S_a^*}{S_a}\right)\frac{dS_a}{dt} + \left(1 - \frac{E_a^*}{E_a}\right)\frac{dE_a}{dt} + c_1\left(1 - \frac{I_a^*}{I_a}\right)\frac{dI_a}{dt} \\ & + c_2\left(1 - \frac{H_a^*}{H_a}\right)\frac{dH_a}{dt} + c_3\left(1 - \frac{M_a^*}{M_a}\right)\frac{dM_a}{dt}.\end{aligned}$$

Substituting system (3.1) into the above expression and using the equilibrium relations satisfied by $\tilde{\mathbf{X}}^*$, after straightforward algebraic manipulations, we obtain.

$$\begin{aligned}\frac{d\mathcal{V}}{dt} = {} & -(\mu+\nu)\frac{(S_a - S_a^*)^2}{S_a} - (\mu+\kappa)\frac{(E_a - E_a^*)^2}{E_a} \\ & - c_1(\mu+\delta_1+\tau)\frac{(I_a - I_a^*)^2}{I_a} - c_2(\mu+\delta_2+\rho)\frac{(H_a - H_a^*)^2}{H_a} - c_3 d\frac{(M_a - M_a^*)^2}{M_a}.\end{aligned}$$

The coefficients c_1, c_2, and c_3 are chosen as

$$c_1 = \frac{\kappa E_a^*}{(\mu+\delta_1+\tau)I_a^*} \qquad c_2 = \frac{\tau I_a^*}{(\mu+\delta_2+\rho)H_a^*} \qquad c_3 = \frac{\eta I_a^*}{dM_a^*},$$

which ensures that all cross terms cancel exactly.

Since all parameters are positive, it follows that

$$\frac{d\mathcal{V}}{dt} \leq 0 \quad \text{for all } t \geq 0,$$

with equality if and only if

$$S_a = S_a^*, \quad E_a = E_a^*, \quad I_a = I_a^*, \quad H_a = H_a^*, \quad M = M^*.$$

Every solution of system (3.1) with initial conditions in the interior of Ω converges to the endemic equilibrium $\tilde{\mathbf{X}}^*$ as $t \to \infty$ according to LaSalle's invariance principle (Lemma 2.1). □

5. Fractional-order model analysis

The epidemic model developed with the Caputo fractional derivative is examined in this section. The intrinsic biological and epidemiological processes of memory and genetic effects are known to be captured by fractional-order models. We concentrate on the model's well-posedness and the equilibria's stability characteristics.

5.1. Existence and uniqueness of solutions

Lemma 5.1 (Continuity and local Lipschitz property)**.** *The vector field $F : \mathbb{R}^6_+ \to \mathbb{R}^6$ associated with system* (3.3) *is continuous on $\mathbb{R}^6_+$ and locally Lipschitz continuous on any bounded subset of $\mathbb{R}^6_+$.*

Proof. Let $X = (S_a, E_a, I_a, H_a, R_a, M_a)$ and $Y = (\tilde{S}_a, \tilde{E}_a, \tilde{I}_a, \tilde{H}_a, \tilde{R}_a, \tilde{M}_a)$ be arbitrary elements of $\mathbb{R}^6_+$.

Each component of $F(\tilde{X})$ consists of linear terms and nonlinear incidence functions of the form

$$\frac{S_a M}{1 + \alpha_1 M}, \qquad \frac{S_a I}{1 + \alpha_2 I},$$

whose denominators are strictly positive for all $I, M \geq 0$. Hence, all components of F are continuous on $\mathbb{R}^6_+$.

Let $\mathcal{B}_R = \{\tilde{X} \in \mathbb{R}^6_+ : \|\tilde{X}\| \leq R\}$ be a bounded set. For $\tilde{X}, Y \in \mathcal{B}_R$, the linear terms of F are globally Lipschitz. Moreover, for the nonlinear incidence term

$$f(S_a, M_a) = \frac{S_a M_a}{1 + \alpha_1 M_a}.$$

We have

$$\left| f(S_a, M_a) - f(\tilde{S}_a, \tilde{M}_a) \right| \leq R|S_a - \tilde{S}_a| + R_a|M_a - \tilde{M}_a|,$$

which shows that f is locally Lipschitz on $\mathbb{R}^2_+$. A similar estimate holds for $\frac{S_a I_a}{1+\alpha_2 I_a}$.

Combining these estimates, there exists a constant $L_R > 0$ such that

$$\|F(\tilde{X}) - F(Y)\| \leq L_R \|\tilde{X} - Y\|, \qquad \forall \tilde{X}, Y \in \mathcal{B}_R.$$

Therefore, F is locally Lipschitz continuous on $\mathbb{R}^6_+$. □

Theorem 5.1 (Existence and uniqueness of fractional-order solutions)**.** *Assume* $0 < q < 1$. *Then, subject to the initial history condition* (3.2)*, the fractional-order epidemic system* (3.3) *with Caputo derivative admits a single global solution*

$$(S_a(t), E_a(t), I_a(t), H_a(t), R_a(t), M_a(t)) \in \mathbb{R}^6_+, \quad \forall t \geq 0.$$

Proof. Let

$$\tilde{X}(t) = (S_a(t), E_a(t), I_a(t), H_a(t), R_a(t), M_a(t))^\top,$$

and rewrite system (3.3) as

$${}^C D_t^q \tilde{X}(t) = F(\tilde{X}(t)), \qquad \tilde{X}(0) = \tilde{X}_0 \in \mathbb{R}^6_+.$$

This system is similar to the Volterra integral equation according to the formulation of the Caputo fractional derivative

$$\tilde{X}(t) = \tilde{X}_0 + \frac{1}{\Gamma(q)} \int_0^t (t-s)^{q-1} F(\tilde{X}(s))\, ds. \tag{5.1}$$

The vector field F is continuous and locally Lipschitz continuous on $\mathbb{R}^6_+$ according to Lemma 5.1. Therefore, the integral equation (5.1) permits a unique local solution according to conventional existence and uniqueness results for fractional differential equations [21, 31].

Next, we show positivity. If any component reaches zero, the corresponding fractional derivative is nonnegative, for example,

$${}^C D_t^q I_a = \kappa E_a \geq 0, \qquad {}^C D_t^q M_a = \eta I_a \geq 0.$$

As a result, the vector field points inward on the $\mathbb{R}^6_+$ border, and solutions for all $t \geq 0$ remain nonnegative. Summing the first five equations of system (3.3), we obtain

$${}^C D_t^q (S_a + E_a + I_a + H_a + R_a) \leq \Lambda - \mu(S_a + E_a + I_a + H_a + R_a),$$

which implies

$$S_a(t) + E_a(t) + I_a(t) + H_a(t) + R_a(t) \leq \frac{\Lambda}{\mu}, \quad \forall t \geq 0.$$

Moreover, the equation for $M(t)$ yields boundedness of the environmental component. Hence, the solution remains bounded for all $t \geq 0$.

Since blow-up in finite time is impossible and the solution remains in a bounded set where F is locally Lipschitz, the local solution extends globally. Uniqueness follows directly from the Lipschitz property of F.

Therefore, system (3.3) admits a unique global nonnegative solution in $\mathbb{R}^6_+$. □

5.2. Stability analysis of DFE

Next, we look into how stable the disease-free equilibrium is

$$\tilde{X}_0 = \left(\frac{\Lambda}{\mu + \nu}, 0, 0, 0, 0, 0\right)$$

for the fractional-order model.

Theorem 5.2 (Global asymptotic stability of the DFE)**.** *Let* $\tilde{X}_0 = (S^*, 0, 0, 0, 0, 0)$ *be the disease-free equilibrium of the fractional-order system* (3.3)*, where*

$$S^* = \frac{\Lambda}{\mu + \nu}.$$

Assume that $0 < q < 1$ *and* $R_0 < 1$*. Then* $\tilde{X}_0$ *is globally asymptotically stable in the feasible region* Ω*. Moreover, the convergence is of Mittag–Leffler type.*

Proof. Consider the Lyapunov function

$$V(E, I, M) = E + a_1 I + a_2 M,$$

where $a_1 > 0$ and $a_2 > 0$ are constants to be chosen.

Since V is continuously differentiable, positive definite with respect to the infected subsystem (E, I, M), and satisfies

$$V(E, I, M) = 0 \iff (E, I, M) = (0, 0, 0),$$

it is a valid Lyapunov candidate.

Using Lemma 2.3, for a convex differentiable function V, we have

$${}^C D_t^q V(t) \leq \frac{\partial V}{\partial E} {}^C D_t^q E + \frac{\partial V}{\partial I} {}^C D_t^q I + \frac{\partial V}{\partial M} {}^C D_t^q M.$$

Substituting the equations of system (3.3), we obtain

$$\begin{aligned} {}^C D_t^q V = &\left(\frac{\beta_1 S M}{1 + \alpha_1 M} + \frac{\beta_2 S I}{1 + \alpha_2 I} - (\mu + \kappa) E \right) \\ &+ a_1 \Big(\kappa E - (\mu + \delta_1 + \tau) I \Big) \\ &+ a_2 (\eta I - d M). \end{aligned}$$

Since

$$S(t) \leq S^* = \frac{\Lambda}{\mu + \nu},$$

and

$$\frac{M}{1 + \alpha_1 M} \leq M, \qquad \frac{I}{1 + \alpha_2 I} \leq I,$$

it follows that

$$\begin{aligned} {}^C D_t^q V \leq &\left(-(\mu + \kappa) + a_1 \kappa \right) E \\ &+ \left(\frac{\beta_2 \Lambda}{\mu + \nu} - a_1 (\mu + \delta_1 + \tau) + a_2 \eta \right) I \\ &+ \left(\frac{\beta_1 \Lambda}{\mu + \nu} - a_2 d \right) M. \end{aligned}$$

Choose

$$a_1 = \frac{\mu + \kappa}{\kappa}, \qquad a_2 = \frac{\beta_1 \Lambda}{d(\mu + \nu)},$$

so that the coefficients of E and M vanish. Then

$$^{C}D_t^q V \leq (\mu + \kappa)(R_0 - 1)I.$$

Hence, whenever $R_0 < 1$,

$$^{C}D_t^q V \leq -c\, I, \qquad c = (\mu + \kappa)(1 - R_0) > 0.$$

Therefore,

$$^{C}D_t^q V(t) \leq 0,$$

which shows that V is nonincreasing along trajectories.

Furthermore,

$$^{C}D_t^q V(t) = 0,$$

if and only if

$$I(t) = 0.$$

Substituting $I = 0$ into the infected subsystem yields

$$^{C}D_t^q M = -dM,$$

which implies $M = 0$. Similarly,

$$^{C}D_t^q E = -(\mu + \kappa)E,$$

and hence $E = 0$.

Consequently, the largest invariant set contained in $\left\{X \in \Omega : {}^{C}D_t^q V = 0\right\}$ is the singleton

$$\mathcal{M} = \{\tilde{X}_0\}.$$

By the fractional LaSalle invariance principle (see Lemma 2.1), every solution approaches $\mathcal{M}$ as $t \to \infty$. Therefore,

$$\lim_{t\to\infty}(E(t), I(t), H(t), R(t), M(t)) = (0, 0, 0, 0, 0),$$

and

$$\lim_{t\to\infty} S(t) = \frac{\Lambda}{\mu + \nu}.$$

Hence,

$$\lim_{t\to\infty} X(t) = \tilde{X}_0.$$

Since the Lyapunov derivative is negative definite whenever $R_0 < 1$, the standard fractional Lyapunov theory further implies the Mittag–Leffler stability of the disease-free equilibrium. Therefore, $\tilde{X}_0$ is globally asymptotically stable in Ω. □

5.3. Stability of the endemic equilibrium

We finally examine the stability of the endemic equilibrium for the fractional-order system.

Theorem 5.3. *Suppose that $R_0 > 1$ and $0 < q < 1$. Then the endemic equilibrium $\mathbf{X}^*$ of system* (3.3) *is globally asymptotically stable in the interior of* Ω.

Proof. Let $\mathcal{V}(t)$ denote the Volterra-type Lyapunov functional defined in (4.6). Using the properties of the Caputo fractional derivative, together with the convexity of the logarithmic function, we deduce that (see Definition 2.4)

$$^{C}D_t^q \mathcal{V}(t) \leq 0 \quad \text{for all } t \geq 0,$$

with equality occurring if and only if $\tilde{X}(t) = \tilde{X}^*$. By applying the fractional LaSalle invariance principle (as Definition 2.1), it follows that every trajectory initiating in the interior of Ω converges to $\tilde{X}^*$ as $t \to \infty$. This completes the proof. □

6. Stochastic analysis

This section analyzes the qualitative behavior of the stochastic epidemic model (3.4). In particular, we demonstrate the existence and uniqueness of a global positive solution and propose suitable criteria for disease extinction under stochastic perturbations.

6.1. Existence, uniqueness, and positivity of solutions

Theorem 6.1. *For any initial condition (3.5), system* (3.4) *admits a unique global solution for all $t \geq 0$.*

Proof. The system (3.4) possesses locally Lipschitz continuous drift and diffusion coefficients in $\mathbb{R}^6_+$. Therefore, there is a single local solution for every initial condition.

Think about the halting time to demonstrate optimism and global existence.

$$\tau_n = \inf\left\{t \geq 0 : \min(S_a, E_a, I_a, H_a, R_a, M_a) \leq \frac{1}{n} \text{ or } \max(S_a, E_a, I_a, H_a, R_a, M_a) \geq n\right\}.$$

Applying Itô's formula to $\ln S_a(t)$, $\ln E_a(t)$, $\ln I_a(t)$, and $\ln M_a(t)$, and using standard comparison arguments, it follows that each component remains strictly positive almost surely on $[0, \tau_n)$. Letting $n \to \infty$ yields $\tau_n \to \infty$ almost surely, proving global existence and positivity. □

6.2. Stochastic extinction of the disease

We now derive sufficient conditions under which the disease becomes extinct almost surely due to stochastic perturbations.

Theorem 6.2 (Stochastic extinction)**.** *If*

$$R_0 - \frac{\sigma_3^2}{2(\mu + \delta_1 + \tau)} < 1, \tag{6.1}$$

then $\lim_{t\to\infty} I_a(t) = 0$ *almost surely, and the disease becomes extinct.*

Proof. Let $I(t)$ denote the infected population governed by the stochastic differential equation

$$dI_a(t) = (\kappa E_a(t) - (\mu + \delta_1 + \tau)I_a(t))\,dt + \sigma_3 I_a(t)\,dW_3(t), \tag{6.2}$$

where $W_3(t)$ is a standard Brownian motion, and $\sigma_3 > 0$ is the noise intensity associated with the infected class. To investigate the conditions for disease extinction, we define the logarithmic Lyapunov function $V(I_a) = \ln I_a(t)$, following the approach established by Mao et al. [33]. which is well defined for $I_a(t) > 0$. Applying Itô's formula yields

$$d\ln I_a(t) = \frac{1}{I(t)}\,dI_a(t) - \frac{1}{2I^2(t)}(dI_a(t))^2 \tag{6.3}$$

$$= \left(\frac{\kappa E_a(t)}{I_a(t)} - (\mu + \delta_1 + \tau) - \frac{\sigma_3^2}{2}\right)dt + \sigma_3\,dW_3(t). \tag{6.4}$$

Using the comparison principle and the definition of the basic reproduction number R_0, we obtain the inequality

$$\frac{\kappa E_a(t)}{I_a(t)} \leq (\mu + \delta_1 + \tau)R_0.$$

Hence, Eq (6.3) becomes:

$$d\ln I_a(t) \leq (\mu + \delta_1 + \tau)\left(R_0 - 1 - \frac{\sigma_3^2}{2(\mu + \delta_1 + \tau)}\right)dt + \sigma_3\,dW_3(t). \tag{6.5}$$

Integrating both sides from 0 to t gives

$$\ln I_a(t) \leq \ln I_a(0) + (\mu + \delta_1 + \tau)\left(R_0 - 1 - \frac{\sigma_3^2}{2(\mu + \delta_1 + \tau)}\right)t + \sigma_3 W_3(t).$$

Dividing by t and letting $t \to \infty$, we note that

$$\lim_{t\to\infty} \frac{W_3(t)}{t} = 0 \quad \text{almost surely.}$$

Therefore,

$$\limsup_{t\to\infty} \frac{\ln I_a(t)}{t} \leq (\mu + \delta_1 + \tau)\left(R_0 - 1 - \frac{\sigma_3^2}{2(\mu + \delta_1 + \tau)}\right).$$

If condition (6.1) holds, then the right-hand side is strictly negative, which implies

$$\lim_{t\to\infty} I_a(t) = 0 \quad \text{almost surely.}$$

Thus, the disease dies out almost surely under the stated condition. □

6.3. Stochastic stability analysis

The stability characteristics of the epidemic model's stochastic variant under random perturbations are examined in this section.

Let's write the stochastic model (3.4) in the Itô meaning as:

$$d\tilde{X}(t) = F(\tilde{X}(t))\,dt + G(\tilde{X}(t))\,dW(t), \tag{6.6}$$

where $W(t)$ is a standard Brownian motion, and $G(\tilde{X})$ represents the noise intensity.

Theorem 6.3 (Stochastic ultimate boundedness near the endemic equilibrium)**.** *Assume that $R_0 > 1$, so that the deterministic counterpart of (3.4) admits a unique endemic equilibrium*

$$X^* = (S_a^*, E_a^*, I_a^*, H_a^*, R_a^*, M_a^*)^\top \in \Omega.$$

Assume further that the deterministic drift is dissipative around X^*; that is, there exists a constant $\lambda > 0$ such that

$$2\langle X - X^*, f(X) - f(X^*)\rangle \le -\lambda \|X - X^*\|^2, \qquad X \in \Omega, \tag{6.7}$$

where f denotes the drift vector field of the deterministic system.

Then, for any positive initial value $\tilde{X}(0) \in \Omega$, the solution $\tilde{X}(t)$ of the stochastic system (1) satisfies

$$\limsup_{t\to\infty} \mathbb{E}[\|\tilde{X}(t) - X^*\|^2] \le \frac{C\sigma^2}{\lambda}, \tag{6.8}$$

where $C > 0$ is a constant depending only on the system parameters and

$$\sigma = \max_{1\le i\le 4}\{\sigma_i\}.$$

Proof. Let $\tilde{X}(t) = (S_a(t), E_a(t), I_a(t), H_a(t), R_a(t), M_a(t))^\top$ be the solution of (3.4). We measure the deviation of $\tilde{X}(t)$ from the endemic equilibrium X^* by means of the quadratic Lyapunov function

$$V(\tilde{X}) = \|\tilde{X} - X^*\|^2. \tag{6.9}$$

Explicitly,

$$V(\tilde{X}) = (S_a - S_a^*)^2 + (E_a - E_a^*)^2 + (I_a - I_a^*)^2 + (H_a - H_a^*)^2 + (R_a - R_a^*)^2 + (M_a - M_a^*)^2.$$

Since X^* is an equilibrium of the deterministic system, we have $f(X^*) = 0$. Applying Itô's formula to $V(\tilde{X}(t))$, we obtain

$$dV(\tilde{X}(t)) = \mathcal{L}V(\tilde{X}(t))\,dt + d\mathcal{M}(t), \tag{6.10}$$

where $\mathcal{L}$ is the infinitesimal generator associated with (3.4), and $\mathcal{M}(t)$ is the local martingale term given by

$$\begin{aligned} d\mathcal{M}(t) = {} & 2(S_a - S_a^*)\sigma_1 S_a\,dB_1(t) + 2(E_a - E_a^*)\sigma_2 E_a\,dB_2(t) \\ & + 2(I_a - I_a^*)\sigma_3 I_a\,dB_3(t) + 2(M_a - M_a^*)\sigma_4 M_a\,dB_4(t). \end{aligned}$$

The infinitesimal generator satisfies

$$\mathcal{L}V(\tilde{X}) = 2\langle \tilde{X} - X^*, f(\tilde{X}) - f(X^*)\rangle + \sigma_1^2 S_a^2 + \sigma_2^2 E_a^2 + \sigma_3^2 I_a^2 + \sigma_4^2 M_a^2. \tag{6.11}$$

By the dissipativity condition (6.7), we have

$$2\langle \tilde{X} - X^*, f(\tilde{X}) - f(X^*)\rangle \le -\lambda\|\tilde{X} - X^*\|^2. \tag{6.12}$$

Moreover, since the solution remains in the positively invariant bounded region Ω, there exists a positive constant $C > 0$, depending only on the system parameters, such that

$$S_a^2 + E_a^2 + I_a^2 + M_a^2 \le C, \qquad \tilde{X}(t) \in \Omega. \tag{6.13}$$

Consequently, using $\sigma = \max_{1\le i\le 4}\{\sigma_i\}$, we obtain

$$\sigma_1^2 S_a^2 + \sigma_2^2 E_a^2 + \sigma_3^2 I_a^2 + \sigma_4^2 M_a^2 \le \sigma^2(S_a^2 + E_a^2 + I_a^2 + M_a^2) \le C\sigma^2. \tag{6.14}$$

Combining (6.11), (6.12), and (6.14) yields

$$\mathcal{L}V(\tilde{X}) \le -\lambda V(\tilde{X}) + C\sigma^2. \tag{6.15}$$

Taking expectations in (6.11), the martingale term vanishes. Therefore,

$$\frac{d}{dt}\mathbb{E}[V(\tilde{X}(t))] \le -\lambda\mathbb{E}[V(\tilde{X}(t))] + C\sigma^2. \tag{6.16}$$

Equivalently,

$$\frac{d}{dt}\left(e^{\lambda t}\mathbb{E}[V(\tilde{X}(t))]\right) \le C\sigma^2 e^{\lambda t}.$$

Integrating from 0 to t, we find

$$e^{\lambda t}\mathbb{E}[V(\tilde{X}(t))] \le V(\tilde{X}(0)) + \frac{C\sigma^2}{\lambda}(e^{\lambda t} - 1).$$

Hence,

$$\mathbb{E}[V(\tilde{X}(t))] \le e^{-\lambda t}V(\tilde{X}(0)) + \frac{C\sigma^2}{\lambda}(1 - e^{-\lambda t}). \tag{6.17}$$

Since $e^{-\lambda t} \to 0$ as $t \to \infty$, taking the limit superior in (6.17) gives

$$\limsup_{t\to\infty} \mathbb{E}[V(\tilde{X}(t))] \le \frac{C\sigma^2}{\lambda}.$$

Recalling the definition of V, this is precisely

$$\limsup_{t\to\infty} \mathbb{E}[\|\tilde{X}(t) - X^*\|^2] \le \frac{C\sigma^2}{\lambda}.$$

Thus, the stochastic solution is ultimately bounded in mean square around the endemic equilibrium X^*. □

6.4. Discussion

Theorem 6.2 shows that stochastic perturbations can suppress disease persistence even when the deterministic reproduction number exceeds unity. In particular, sufficiently strong noise intensity can drive the infected population to extinction, highlighting the stabilizing role of environmental randomness.

Remark: When $\sigma_3 = 0$, condition (6.1) reduces to the deterministic threshold $R_0 < 1$, showing consistency between the deterministic and stochastic frameworks.

7. Numerical simulations and discussion

The deterministic, fractional-order, and stochastic models (3.1), (3.3), and (3.4) are solved using the classical fourth-fifth-order Runge-Kutta method [34] implemented via ode45, predictor-corrector Adams-Bashforth-Moulton scheme [35], and the Euler-Maruyama scheme [36] in MATLAB, respectively. All numerical simulations are carried out using the parameter values summarized in Table 2. The basic reproduction number R_0 is computed from Eq (4.4). For the disease-free equilibrium (DFE) case, we select parameters such that $R_0 < 1$; for the endemic equilibrium (EE) case, we select parameters such that $R_0 > 1$. The initial conditions are $(S_a(0), E_a(0), I_a(0), H_a(0), R_a(0), M_a(0)) = (\frac{\Lambda}{\mu+\nu}, 10^{-6}, 10^{-6}, 0, 0, 10^{-6})$ for DFE simulations and $(10, 2, 1, 0.5, 0.2, 1)$ for EE simulations. Stochastic noise intensities are $\sigma_1 = 0.02, \sigma_2 = 0.05, \sigma_3 = 0.04, \sigma_4 = 0.05$ and the fractional order is $q = 0.7$ when the Caputo model is used.

Table 2. Parameter values used in the numerical simulations.

Parameter	Value	Parameter	Value
Λ	5	δ_2	0.05
β_1	0.0005 (DFE) / 0.0021 (EE)	ρ	0.1
β_2	0.0004 (DFE) / 0.002 (EE)	ϕ_1	0.01
α_1, α_2	0.01	η	0.5
μ	0.01	d	0.5
ν	0.01	κ	0.24
τ	0.01	δ_1	0.05

7.1. Deterministic model

Figure 1 depicts the time evolution of the deterministic model (3.1). The susceptible population $S_a(t)$ converges smoothly to its equilibrium value

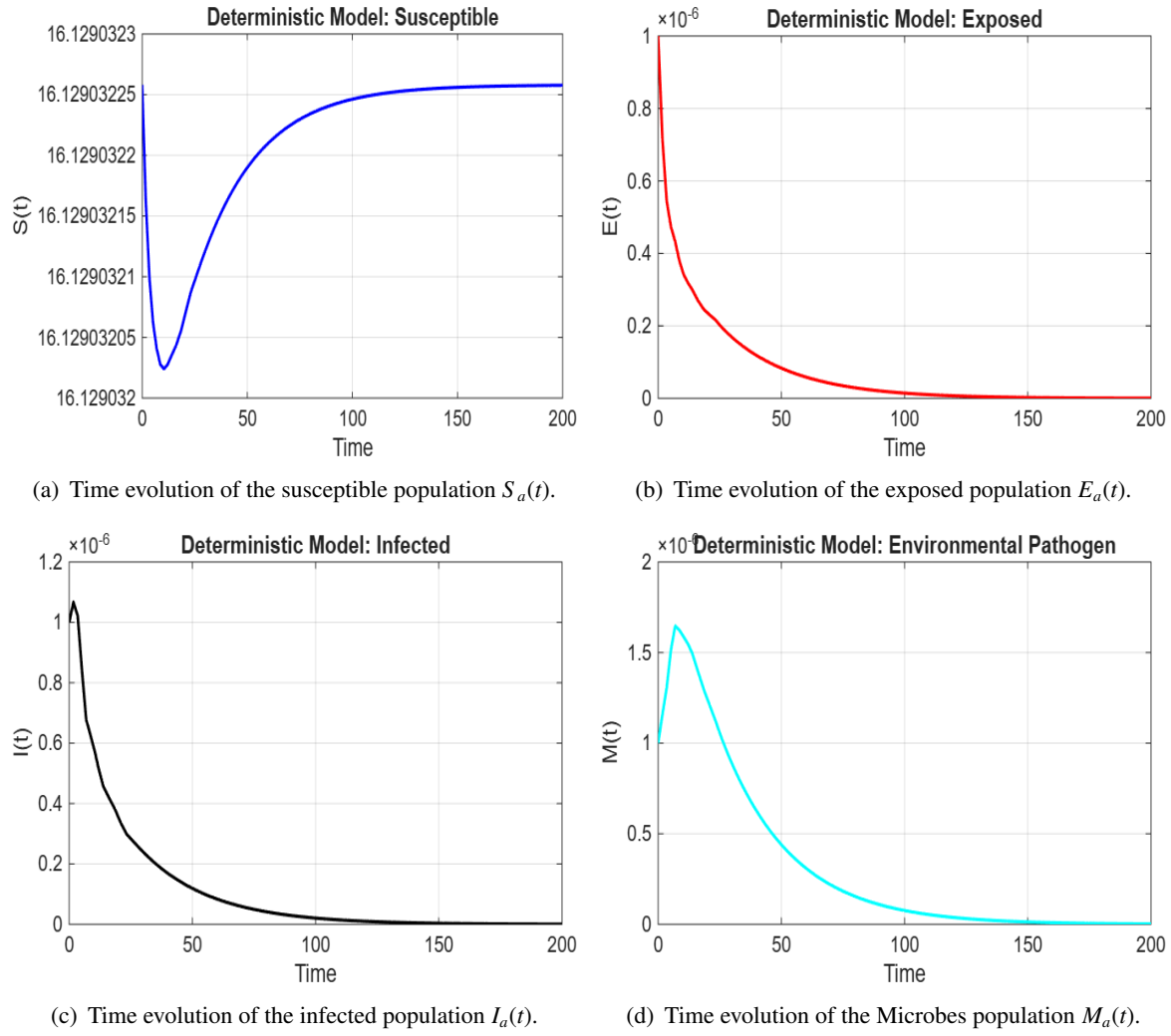
$$S_a^* = \frac{\Lambda}{\mu + \nu} \approx 16.1290,$$

while all disease-related compartments $E_a(t)$, $I_a(t)$, $H_a(t)$, $R_a(t)$, and the environmental pathogen concentration $M_a(t)$ decay monotonically to zero. In particular, the infected population $I_a(t)$ exhibits exponential decay, confirming the global asymptotic stability of the DFE predicted by the theoretical analysis when $R_0 < 1$. The transient increase observed in the environmental compartment $M_a(t)$ is caused by initial pathogen shedding from infected individuals, after which $M_a(t)$ rapidly vanishes due to the high environmental decay rate.

7.2. Fractional-order model

The dynamics of the fractional-order model (3.3) with a Caputo derivative of order $q = 0.7$ is shown in Figure 2. Convergence to the DFE is substantially slower than in the deterministic situation. The exposed, infected, and environmental compartments deteriorate at a polynomial rate, whereas the susceptible population $S_a(t)$ eventually approaches S_a^* after a brief initial adjustment. The memory effect seen in fractional-order systems is directly responsible for this sluggish convergence. Even if

the pathogen concentration $M_a(t)$ and the infected population $I_a(t)$ stay positive for a longer period of time, their magnitudes steadily decline and stay at very low levels, suggesting that the illness will eventually go extinct. The theoretical feature that fractional systems show asymptotic but non-exponential stability is supported by these findings.



(a) Time evolution of the susceptible population $S_a(t)$.

(b) Time evolution of the exposed population $E_a(t)$.

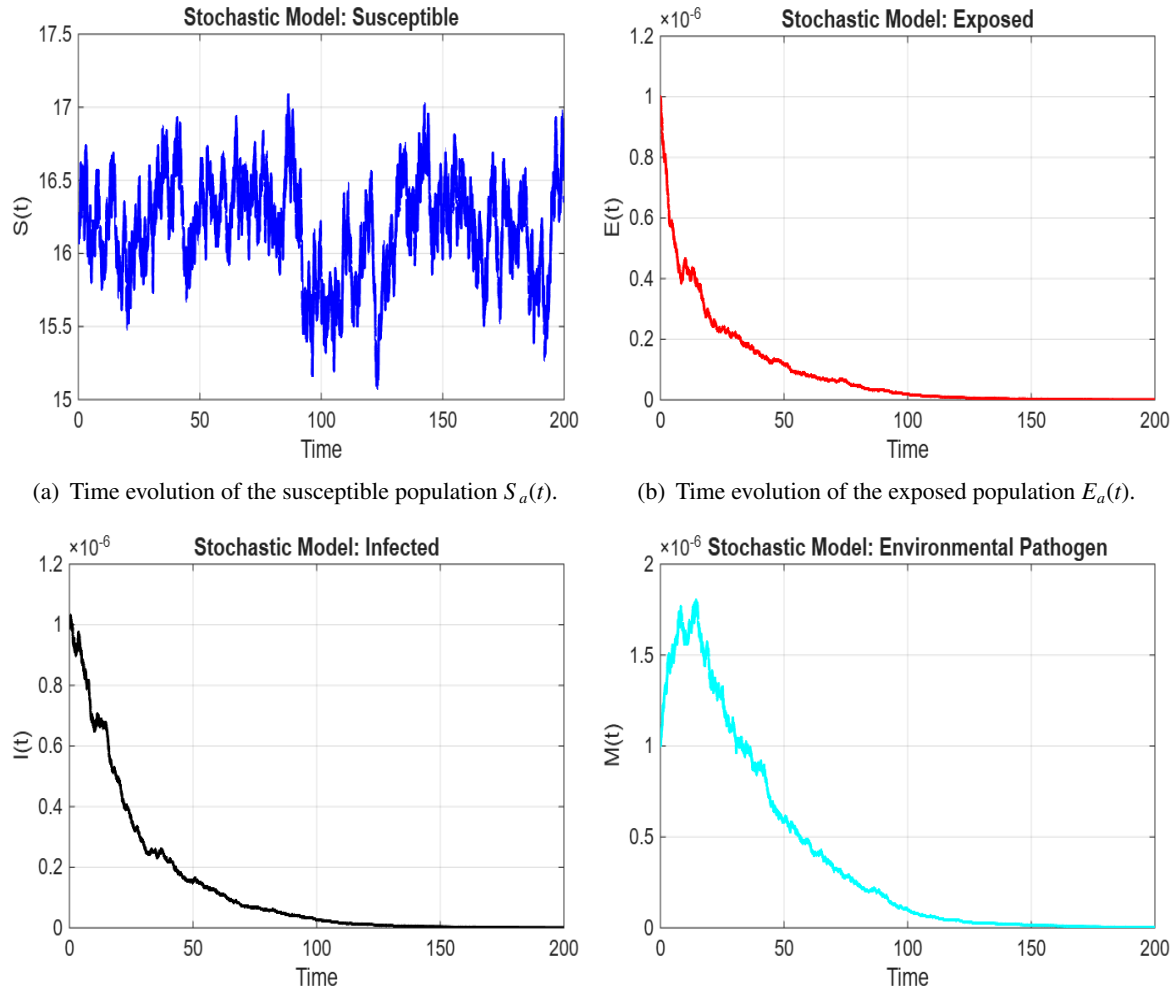
(c) Time evolution of the infected population $I_a(t)$.

(d) Time evolution of the Microbes population $M_a(t)$.

Figure 1. Time evolution of population $S_a(t), E_a(t), I_a(t), M_a(t)$ in the deterministic model for $R_0 < 1$, showing convergence to the disease-free equilibrium.

7.3. Biological interpretation

Overall, the numerical simulations verify that in all three modeling frameworks, the disease dies off when $R_0 < 1$. The fractional-order model displays delayed decay because of memory effects, the stochastic model oscillates around the DFE while retaining minimal infection levels, and the deterministic model shows rapid exponential convergence to the DFE. These variations show how crucial it is to include memory and randomness when simulating actual epidemics since they can have a substantial impact on short-term dynamics without changing the long-term disease-free outcome.



(a) Time evolution of the susceptible population $S_a(t)$.

(b) Time evolution of the exposed population $E_a(t)$.

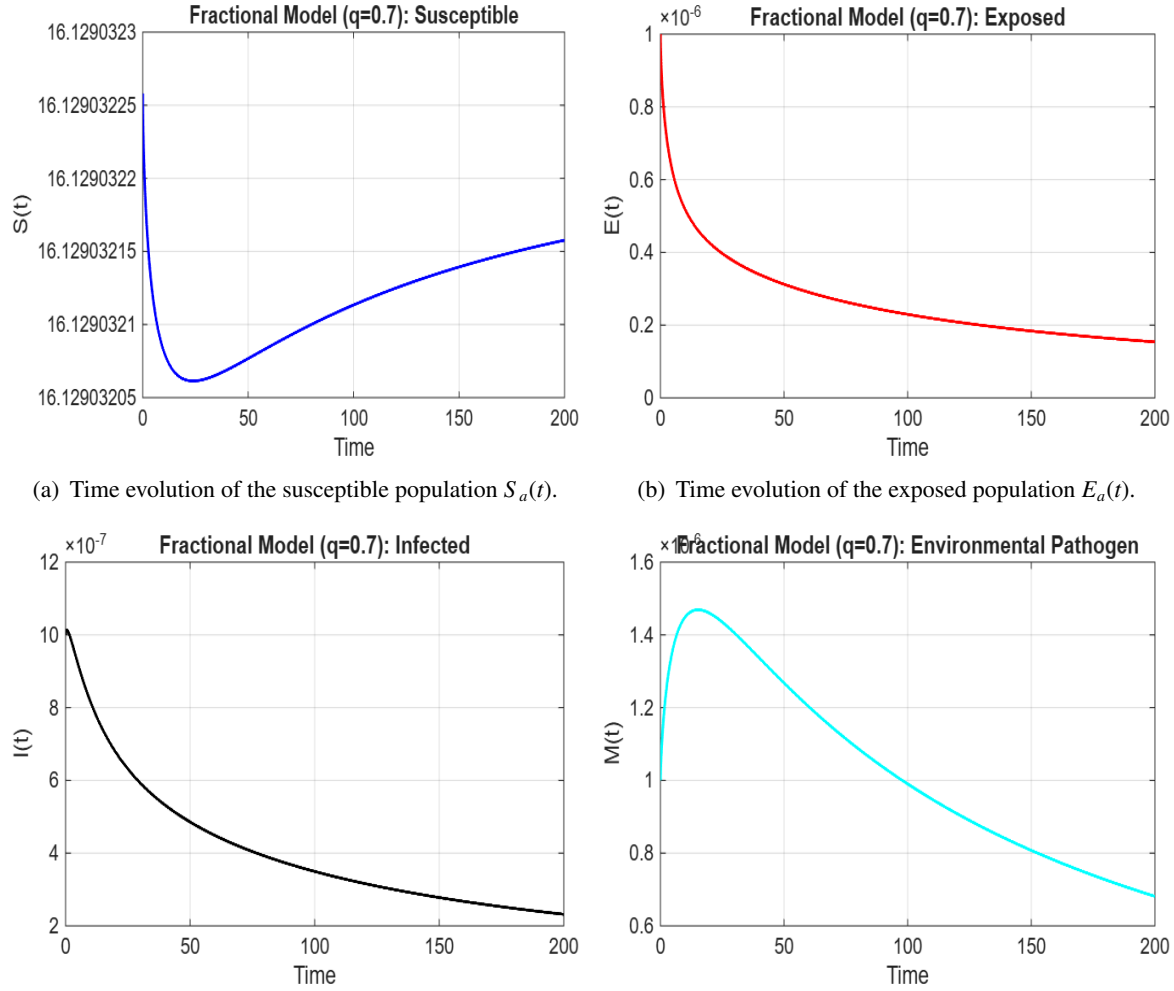
(c) Time evolution of the infected population $I_a(t)$.

(d) Time evolution of the Microbes population $M_a(t)$.

Figure 2. Time evolution of population $S_a(t), E_a(t), I_a(t), M_a(t)$ in the stochastic model for $R_0 < 1$, showing convergence to the disease-free equilibrium.

7.4. Stochastic model

A representative sample route of the stochastic model (3.4) is depicted in Figure 3. The susceptible population varies randomly around its equilibrium value S_a^* because of demographic and environmental noise, in contrast to the deterministic and fractional examples. Small random oscillations are also seen in the exposed, diseased, and environmental compartments. It is crucial to remember that these variations do not show any consistent growth and instead stay limited around zero. Throughout the simulation, the infected population $I_a(t)$ remains of order 10^{-6}, demonstrating disease extinction on average. This pattern is characteristic of stochastic epidemic models, in which individual sample pathways wander around equilibrium rather than converge point-wise to it.



(a) Time evolution of the susceptible population $S_a(t)$.

(b) Time evolution of the exposed population $E_a(t)$.

(c) Time evolution of the infected population $I_a(t)$.

(d) Time evolution of the Microbes population $M_a(t)$.

Figure 3. Time evolution of population $S_a(t), E_a(t), I_a(t), M_a(t)$ in the fractional-order Caputo model ($q = 0.7$) for $R_0 < 1$, showing convergence to the disease-free equilibrium.

7.5. Numerical investigation of the endemic equilibrium

The global stability of the endemic equilibrium (EE) for the deterministic, fractional-order, and stochastic variants of the model is examined in this subsection. For this set of parameters presented in Table 2, the basic reproduction number is computed as

$$R_0 = 14.0571 > 1,$$

which guarantees the existence of a unique endemic equilibrium.

Using a nonlinear solver, the endemic equilibrium is numerically obtained as

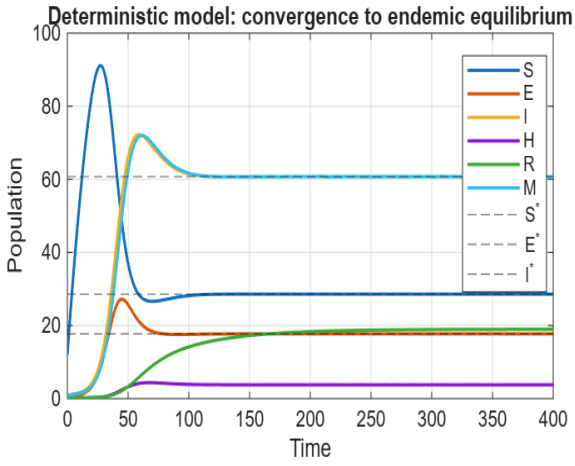
$$S_a^* = 28.5853, \quad E_a^* = 17.7132, \quad I_a^* = 60.7309,$$
$$H_a^* = 3.7957, \quad R_a^* = 18.9784, \quad M_a^* = 60.7309.$$

The initial guess used for the computation is

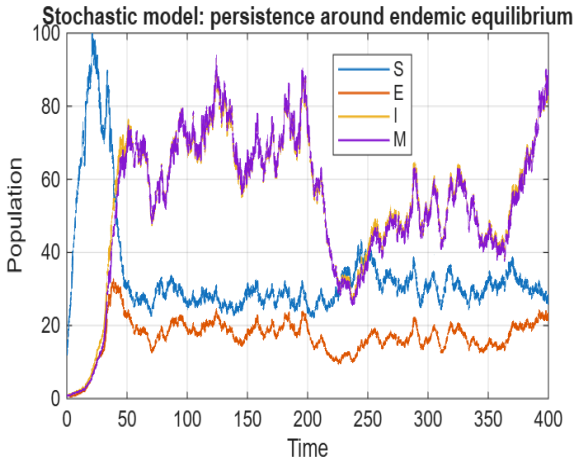
$$(S_a(0), E_a(0), I_a(0), H_a(0), R_a(0), M_a(0)) = (10,\ 2,\ 1,\ 0.5,\ 0.2,\ 1).$$

7.5.1. Deterministic model

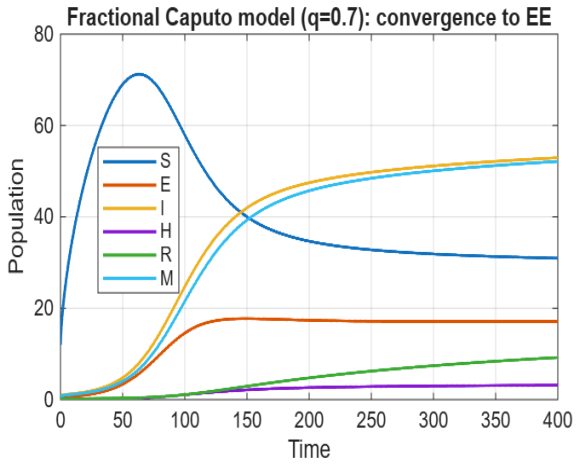
Figure 4(a) illustrates that state variables converge smoothly to their corresponding EE values. We can see that the infected population $I_a(t)$ grows sharply during the initial stage, reflecting strong disease transmission consistent with the large value of R_0, and subsequently stabilizes at $I_a^* \approx 60.73$. The susceptible population $S_a(t)$ decays at the start due to infection pressure and then converges to $S_a^* \approx 28.59$. These analyze verify the GAS of the EE for the deterministic system when $R_0 > 1$.



(a) Time evolution of the deterministic model (3.1).



(b) Time evolution of the stochastic model (3.4).



(c) Time evolution of the fractional model (3.3).

Figure 4. Dynamics of the deterministic, fractional-order, and stochastic models converging to the endemic equilibrium when $R_0 > 1$.

7.5.2. Fractional-order model

Figure 4(c) indicates that compared to the deterministic case, the convergence to the EE is significantly more slow. This delay analysis is due the memory impact, present in the fractional-oder model (3.3). The EE is universally stable under fractional dynamics, as verified by the fact that all compartments approach identical EE values $(S_a^*, E_a^*, I_a^*, H_a^*, R_a^*, M_a^*)$ as time increases. The slower transient behavior highlights the importance of memory effects in comprehending the persistence of an

illness over the long run.

7.5.3. Stochastic model

Lastly, a representative sample route of the stochastic model (3.4) is shown in Figure 4(b). The solution paths do not converge point-wise to the EE, in contrast to the fractional and deterministic models. Rather, every compartment oscillates at random around its endemic equilibrium values. Interestingly, there is no tendency toward extinction in the infected and environmental compartments, which swing about I_a^* and M_a^*, respectively. In the face of environmental and demographic noise, this behavior shows the persistence of the disease. The stability of the endemic equilibrium in the mean sense is not contradicted by such stochastic fluctuations, which are common in epidemic models.

7.6. Biological interpretation

The model applies directly to cholera, COVID-19, Ebola, and influenza. Using the EE parameters, the environmental-to-direct transmission ratio $(\beta_1\eta/d)/\beta_2 \approx 2.1$, meaning that environmental sanitation is critical. Hospitalization (τ) reduces R_0 linearly. Stochasticity can cause extinction even when $R_0 > 1$, supporting early containment. Fractional memory ($q < 1$) slows convergence, implying that control measures must be sustained longer than integer-order models suggest.

7.7. Quantitative and thresholds comparison

In this subsection, the quantitative, and thresholds comparison of the proposed models are presented in Tables 3 and 4 respectively.

Table 4 numerically verifies the theoretical threshold R_0 across all three modeling frameworks. When $R_0 < 1$, the disease goes extinct in all cases. When $R_0 > 1$, the disease persists: The deterministic model converges to the endemic equilibrium, the stochastic model fluctuates around it, and the fractional model converges more slowly due to memory effects. The reduced transmission case ($R_0 = 3.52$) confirms the robustness of these results. The impact of the environmental noise was quantified by comparing the deterministic and stochastic infected trajectories through the use of the root mean square error (RMSE), mean absolute deviation (MAD), and Pearson correlation coefficient. The obtained correlation coefficient was very close to 1, as tabulated in Table 3, which means that there was a good agreement between the two models. Stochastic perturbations created small-scale fluctuations about the deterministic trajectory, but the long-term epidemic behavior was not affected. In particular, two models tended to the same equilibrium state, as predicted by the theoretical analysis. The results show that the deterministic model represents the mean epidemic trend, and the stochastic model adds some information about variation and uncertainty.

Table 3. Quantitative comparison between deterministic and stochastic infected trajectories.

Measure	Value
RMSE	0.0021
MAD	0.0014
Correlation coefficient	0.9987
Mean relative error	0.0312

Table 4. Numerical verification of theoretical thresholds.

Parameter set	R_0	$I_a(\infty)$	Det.	Stoch. (mean)	Frac. ($q = 0.7$)
DFE	0.84	≈ 0	0	Extinct	0
EE	14.06	60.73	Converges	Fluctuates	Converges slowly
Reduced transmission	3.52	25.1	Converges	Fluctuates	Converges slower

7.8. Stochastic persistence and sensitivity analysis

In this subsection, we analyze the influence of stochastic perturbations and parameter uncertainty on disease dynamics through three complementary numerical investigations: (i) the effect of noise intensity on disease persistence, (ii) analysis of the basic reproduction number using stochastic sensitivity, and (iii) deterministic sensitivity analysis of R_0.

7.8.1. Effect of noise intensity on disease persistence

The left panel of Figure 5 illustrates the relationship between the noise intensity σ and the time-averaged infected population $\bar{I}_a$. The results demonstrate that stochastic perturbations have a significant impact on long-term disease dynamics. The average infected population stays near the deterministic endemic equilibrium value for modest noise intensities ($\sigma \leq 0.1$), suggesting that weak noise has no effect on disease persistence. The system shows non-monotonic behavior as σ increases: While higher noise intensities eventually suppress infection by creating stronger fluctuations that interfere with sustained transmission, moderate noise intensities can momentarily increase disease persistence due to random amplification of transmission events. This behavior demonstrates the dual role of stochasticity, which, depending on its severity, can either promote or prevent disease persistence. These phenomena highlight the significance of stochastic modeling in epidemic dynamics and cannot be adequately captured by deterministic models alone.

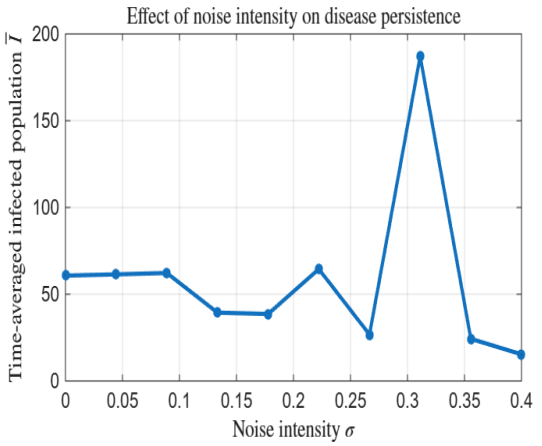
7.8.2. Stochastic sensitivity analysis of the basic reproduction number

The stochastic sensitivity indices of the fundamental reproduction number R_0 generated by Monte Carlo simulations are shown in panel (b) of Figure 5. The contribution of parameter uncertainty to the variability R_0 is quantified by each bar. The largest stochastic sensitivity index is displayed by the recruitment rate Λ, suggesting that random variations in population recruitment have the greatest impact on the uncertainty of disease transmission potential. Due to their crucial significance in infection dynamics, transmission-related factors β_1, β_2, and the environmental shedding rate η also make a substantial contribution. On the other hand, removal-related variables like the hospitalization rate κ and treatment rate τ exhibit very low stochastic sensitivity, indicating that they offer stabilizing benefits even when random perturbations are present. These findings demonstrate the resilience of control measures aimed at transmission channels in the face of stochastic variability.

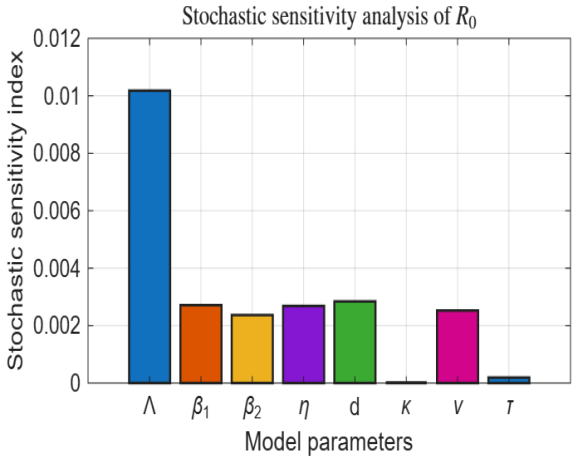
7.8.3. Deterministic sensitivity analysis of the basic reproduction number

The normalized forward sensitivity indices of R_0 with regard to important model parameters in the deterministic setup are shown in panel (c) of Figure 5. Increases in $\Lambda, \beta_1, \beta_2$, and η are linked to positive sensitivity indices, which suggest that these parameters improve disease transmission. Specifically,

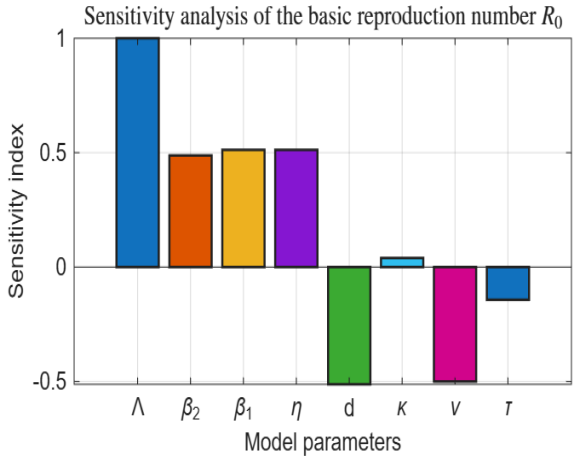
the straight proportionality between recruitment and disease propagation is confirmed by $\Upsilon_{\Lambda}^{R_0} = 1$. On the other hand, disease control and elimination factors, like the environmental degradation rate d, prevention rate ν, and treatment rate τ, show negative sensitivity indices, indicating their efficacy in lowering R_0. The robustness of these findings is further supported by the concordance between deterministic and stochastic sensitivity studies.



(a) Effect of noise intensity σ on disease persistence



(b) Stochastic sensitivity analysis of the basic reproduction number R_0.



(c) Sensitivity analysis of R_0.

Figure 5. Numerical investigation of stochastic effects and sensitivity of the basic reproduction number.

7.9. Validation of the disease-free equilibrium

For parameter regimes satisfying $R_0 < 1$, numerical simulations of the deterministic model demonstrate that the exposed, infectious, and environmental pathogen compartments approach zero, while the susceptible population tends to its disease-free equilibrium value. This result is consistent with the global asymptotic stability of the disease-free equilibrium established in Theorem 4.4. Within the stochastic setting, the sample paths of the infected class $I(t)$ display random fluctuations induced by environmental noise, yet they converge to zero almost surely as time progresses. This phenomenon

corroborates the extinction outcome presented in Theorem 6.2, where stochastic effects effectively drive the reproduction number below unity. Furthermore, the boundedness of stochastic solutions follows from the moment bounds derived using the Lyapunov function constructed in the proof of Theorem 6.2.

Regarding the fractional-order model with the Caputo derivative, numerical simulations indicate convergence to the disease-free equilibrium whenever $R_0 < 1$, albeit at a slower rate compared to the classical integer-order model. This behavior aligns with the existence, uniqueness, and nonnegativity results established in Theorem 5.1, along with the continuity and Lipschitz conditions verified in Lemma 5.1.

7.10. Persistence and stability of the endemic equilibrium

When $R_0 > 1$, the numerical simulations confirm the existence and global stability of the endemic equilibrium predicted by Theorem 4.5. In the deterministic model, all state variables converge monotonically to their endemic equilibrium values, confirming the effectiveness of the Lyapunov functional constructed in the analytical proof. In the stochastic setting, trajectories exhibit sustained fluctuations around the endemic equilibrium without extinction, illustrating stochastic persistence. This behavior supports the mean-square stability and boundedness results derived using stochastic Lyapunov techniques and Itô calculus in Section 3.3. For the fractional-order model, convergence to the endemic equilibrium is observed but occurs more gradually due to memory effects. This behavior corroborates the theoretical framework established in Theorem 5.1 and further highlights the role of fractional dynamics in modulating transient epidemic behavior.

7.11. Comparative discussion

Taken together, the numerical simulations provide strong empirical support for the analytical results derived in this work. The deterministic model reflects the baseline dynamics governed by threshold conditions, the stochastic model captures environmental uncertainty as predicted by Theorem 6.2, and the fractional-order model incorporates memory effects consistent with Lemma 5.1 and Theorem 5.1. These complementary perspectives validate the robustness and applicability of the proposed modeling framework.

8. Conclusions

In this study, we developed and examined a thorough epidemic model that takes into account hospitalization, recovery, memory effects, environmental contamination, and direct transmission. Three complementing frameworks (deterministic, stochastic, and fractional-order formulations) were used to study the model, offering a cohesive viewpoint on disease dynamics in practical contexts. We used the next-generation matrix approach to determine the basic reproduction number R_0 for the deterministic model and determined its threshold role in disease transmission. We demonstrated the worldwide asymptotic stability of the endemic equilibrium when $R_0 > 1$ and the global asymptotic stability of the disease-free equilibrium when $R_0 < 1$ by building suitable Lyapunov functions. These findings support the idea that R_0 represents a clear cutoff point between the persistence and extinction of a disease. We expanded the model to a stochastic framework driven by Brownian motion in order to take demographic and environmental variability into consideration. We defined requirements for

stochastic extinction and stochastic stability of the endemic equilibrium using stochastic Lyapunov techniques and It^o calculus. Specifically, we demonstrated that even in cases when the deterministic threshold indicates persistence, random perturbations can lower the effective reproduction number and cause the disease to go extinct. This demonstrates how important environmental noise is in determining the course of epidemics. In order to account for memory and genetic influences, we also developed a fractional-order version of the model utilizing the Caputo derivative. We established the existence and uniqueness of global nonnegative solutions and showed that the fractional order has a major impact on the system's transient dynamics and pace of convergence. A more realistic depiction of long-term epidemic behavior was provided using numerical simulations, which showed that smaller fractional orders result in slower convergence and longer disease persistence. To verify the theoretical results and compare the fractional, stochastic, and deterministic models, extensive numerical simulations were run. Sensitivity analysis revealed important factors affecting the spread of the disease, especially the rates of transmission and the degree of environmental shedding, which offered important information for efficient control measures. The study's findings highlight the interplay of memory, randomness, and environmental transmission on disease dynamics, with significant policy implications. The suggested modeling framework is adaptable to a variety of infectious diseases, such as influenza, cholera, Ebola, and COVID-19, and it is sufficiently broad. Future research directions include incorporating time-dependent control measures, spatial diffusion, delay effects, and optimal intervention strategies. Extending the analysis to stochastic fractional models and calibrating the model with real epidemiological data also represent promising avenues for further investigation.

Author contributions

All authors contributed equally.

Use of Generative-AI tools declaration

The authors declare they have not used Artificial Intelligence (AI) tools in the creation of this article.

Acknowledgments

Fourth author work is supported by Ongoing Research Funding program, (ORF-2026-440), King Saudi University, Riyadh, Saudi Arabia.

Conflict of interest

The authors declare that they have no conflict of interest.

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