



RESEARCH PAPER

Asymptotic extinction and persistence of a perturbed epidemic model with different intervention measures and standard Lévy jumps

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Abstract

Controlling an outbreak through response measures is critical to saving lives and protecting vulnerable populations. This article proposes an epidemic model with three intervention measures: media coverage, isolation, and medical therapy. Since randomness plays an important role in biology, from the molecular level to the organismal level, we extend our system to a more realistic framework, which then takes into account the effect of standard jumps due to some sudden environmental changes. After providing the associated framework, the sharp criteria for asymptotic extinction and persistence of illness are derived. To check the accuracy of our results, we perform two numerical examples.

Keywords: Epidemic model; isolation; therapy; coverage media; asymptotic analysis; Lévy jumps

AMS 2020 Classification: 37A50; 37C10; 92D30

1 Introduction

Mathematical modeling is a robust tool for understanding the attitude of infections and assessing the influence of various intervention strategies. In the context of biological modeling, "intervention measures" refer to the implementation of measures to reduce the spread of disease [1]. The most commonly used interventions include social distancing, wearing masks, contact tracing, isolation or quarantine, and hospitalization. To simulate the impact of these interventions, one can use a compartmental model, which divides the population into different compartments based on their infection status [2]. Also, it is possible to introduce parameters that represent the effectiveness of these interventions, such as the reduction in transmission due to social distancing or the effectiveness of a vaccine. By adjusting these parameters, one can simulate the impact of different intervention strategies on the evolution of the illness [3].

Media intervention can play a critical role in shaping public perception, awareness, and behavior during an epidemic. Effective media interventions can help to disseminate accurate information about the disease, promote healthy behaviors, and counter misinformation and rumors [4]. One way that media intervention can have an impact is by increasing knowledge and awareness about the disease. This can be done through the dissemination of accurate and up-to-date information about the disease, its transmission, symptoms, and prevention [5]. By providing clear and concise information, media intervention can help to increase public understanding of the disease and the need for preventive measures. Another way that media intervention can have an impact is by promoting healthy behaviors. During an epidemic, media intervention can be used to encourage individuals to adopt protective behaviors such as hand washing, wearing masks, and social distancing. By promoting these behaviors, media intervention can help to reduce the transmission of the disease and slow the spread of the epidemic. Media intrusion can also help to counter misinformation and rumors that may be circulating during an epidemic. Misinformation can lead to fear, panic, and irrational behavior, which can exacerbate the spread of the disease [6]. By providing accurate information and dispelling rumors, media intervention can help to reduce fear and promote rational decision-making.

Isolation is one of the key measures used to control the spread of an epidemic. It involves separating individuals who are infected with the disease from those who are not infected [7]. Generally, isolation can take different forms, depending on the severity of the epidemic and the resources available. In some cases, isolation may involve self-isolation at home for individuals who have mild symptoms or who have been exposed to the disease. In more severe cases, isolation may involve hospitalization of individuals who are severely ill or at high risk of complications. Isolation is effective for controlling the spread of an epidemic for several reasons [8]. First, it can prevent infected individuals from coming into contact with uninfected individuals, which can reduce the transmission of the disease. By separating infected individuals from others, isolation can help to break the chain of transmission and slow the spread of the disease [9]. Second, isolation can provide medical care and support for individuals who are infected. In some cases, infected individuals may require hospitalization and medical treatment to manage their symptoms and prevent complications. Isolation in a hospital setting can ensure that infected individuals receive the care and treatment they need. Third, isolation can provide time for public health officials to track and monitor the spread of the disease. By isolating infected individuals and tracing their contacts, public health officials can identify and isolate additional cases, which can further reduce the spread of the disease [10].

A hospitalization intervention is a strategy implemented during an epidemic to reduce the number of hospitalizations due to the illness [11]. This can involve various measures, such as increasing hospital capacity, improving triage processes to identify and prioritize the most severe cases, and implementing effective treatments. During an epidemic, hospitalizations can quickly overwhelm healthcare systems, leading to shortages of beds, equipment, and staff [12]. By implementing hospitalization interventions, health-care providers can work to ensure that those most in need receive the care they require, while also preventing the spread of the illness to others. Some examples of hospitalization interventions that may be used during an epidemic include setting up temporary field hospitals to increase capacity, using telemedicine to reduce in-person visits and decrease the risk of transmission, and developing effective treatments and therapies to help patients recover more quickly and avoid hospitalization altogether [13].

The incubation period of an epidemic is the time period between the initial infection with a pathogen and the onset of symptoms of the disease [14]. During this period, the infected individual may be asymptomatic, meaning that they are not yet showing any symptoms of the disease, but they may still be able to transmit the pathogen to others. The length of the incubation period

can vary depending on the pathogen and the individual's immune system [15]. For example, the incubation period for influenza is typically between 1 – 4 days, while the incubation period for COVID-19 can range from 2 – 14 days, with an average of 5 – 6 days. Understanding the incubation period of an epidemic is important for several reasons. First, it can help public health officials to identify and isolate infected individuals before they become symptomatic, which can help to prevent the spread of the disease [16]. Second, it can help to determine the length of time that exposed individuals need to be monitored for symptoms and potential infection. It is important to note that the incubation period is not the same as the infectious period, which is the length of time during which an infected individual can transmit the disease to others. The infectious period can be shorter or longer than the incubation period, depending on the pathogen and the individual's immune system [17].

In order to build a mathematical model that takes into consideration the above interventions and the different types of immunities, we assume that the total population is divided into seven groups of susceptible, exposed, infectious persons with actual viral symptoms, individuals asymptotically infected, isolated, individuals under treatment and persons with full cure, with concentrations expressed respectively by $S(t)$, $E(t)$, $C(t)$, $I(t)$, $Q(t)$, $Z(t)$ and $P(t)$. The epidemiological exchanges between these groups are depicted through the following dynamical system (denoted by SECIQZP):

$$\begin{aligned}
 dS(t) &= \left(\Pi - (m + a)S(t) - S(t)(I(t) + gC(t)) \left(b_1 - \frac{b_2 I(t)}{p + I(t)} \right) + \varepsilon Q(t) \right) dt, \\
 dE(t) &= \left(S(t)(I(t) + gC(t)) \left(b_1 - \frac{b_2 I(t)}{p + I(t)} \right) - (m + \beta)E(t) \right) dt, \\
 dC(t) &= \left((1 - \vartheta) \beta E(t) - (m + \omega_C + s_C + h_C) C(t) \right) dt, \\
 dI(t) &= \left(\beta \vartheta E(t) - (m + \omega_I + s_I + h_I) I(t) \right) dt, \\
 dQ(t) &= \left(aS(t) - (m + \varepsilon) Q(t) \right) dt, \\
 dZ(t) &= \left(\omega_I I(t) + \omega_C C(t) - (m + s_Z + h_Z) Z(t) \right) dt, \\
 dP(t) &= \left(s_Z Z(t) + s_I I(t) + s_C C(t) - mP(t) \right) dt.
 \end{aligned} \tag{1}$$

The positive parameters of this model are defined as follows:

- Π is the recruitment rate of the uninfected (but susceptible) persons that corresponds to normal births and immigration.
- m , h_C , h_I and h_Z are denoting, in this order, the normal mortality rate of all individuals and the infection-induced mortality rates affecting only from groups C , I and Z .
- a and ε represent the exchange rates between S and Q classes.
- b_1 is the standard contamination rate before applying media intervention. b_2 is the extra reduced contact rate under the application of media intrusion such that $b_1 - b_2$ is positive. p is the saturation coefficient.
- $0 < g < 1$ is the parameter that ensures the high infectivity of infected individuals.
- β is the transfer rate from E group to I population with the probability $0 < \vartheta < 1$ of becoming infectious and $(1 - \vartheta)$ for entering C class.
- ω_I and ω_C are respectively the treatment rates I and C people.
- s_Z , s_I and s_C are the total cure rates of Z , I and C people.

To better understand the different transfer rates between classes, we present the diagram shown in Figure 1.

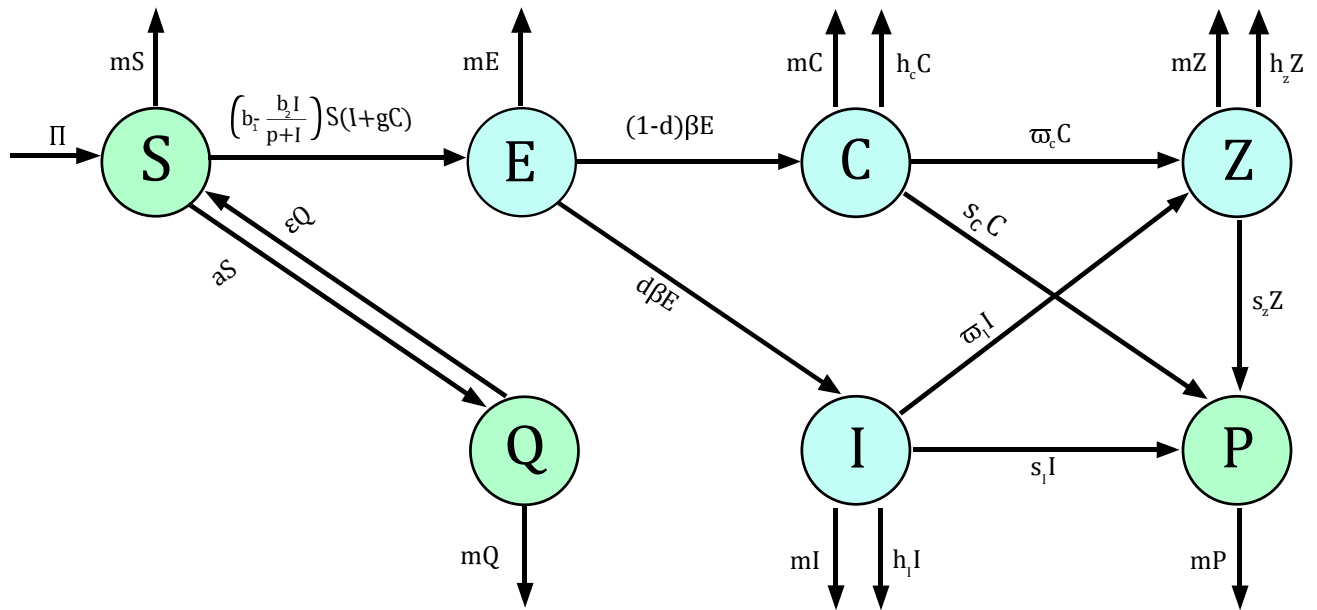


Figure 1. Illustrative diagram of SECIQZP epidemic model transfer rates

Fluctuations are a common feature of epidemic outbreaks, where the number of cases or the transmission rate can vary over time due to various factors such as changes in behavior, public health interventions, or the emergence of new variants of the pathogen [18]. The magnitude and frequency of fluctuations in an epidemic outbreak can depend on a variety of factors, including the mode of transmission, the population demographics, and the effectiveness of control measures. For example, in an outbreak of a highly infectious disease with a short incubation period, fluctuations may be more pronounced due to the rapid spread of the pathogen [19]. To account for fluctuations in epidemic outbreaks, mathematical models are often used to simulate the spread of the disease over time and evaluate the impact of different control measures. These models can help public health officials make informed decisions about when to implement or relax interventions to achieve the optimal balance between controlling the outbreak and minimizing the economic and social costs of control measures [20].

Random jumps are a type of stochastic process where the magnitude of changes in a variable occurs randomly over time [21]. In the context of an epidemic situation, random jumps could refer to sudden and significant increases in the number of cases or spread of the disease that occur unpredictably [22]. In epidemiology, random jumps have been used to model the spread of infectious diseases in populations with complex social structures or mobility patterns, where outbreaks can occur in unpredictable locations or at unpredictable times. Random jumps can also capture the behavior of disease outbreaks that exhibit sudden bursts of activity due to super-spreader events or other factors [23].

The novelty of this study is to probe the impact of jumps on the dynamics of system (1). The pivotal objective of this article is to provide sufficient criteria for asymptotic extinction and persistence. The global threshold is difficult to derive in this model due to its complexity, but we will do our best to offer precise conditions and this is the strength of our study. By considering the standard Lévy jumps associated with the compensated Poisson measure defined on a probabilistic basis

$(\Omega, \mathcal{F}, \{\mathcal{F}_t\}_{t \geq 0}, \mathbb{P})$, our model takes the following general form:

$$\begin{aligned}
 dS(t) &= \left(\Pi - (m + a)S(t) - S(t) \left(I(t) + gC(t) \right) \left(b_1 - \frac{b_2 I(t)}{p + I(t)} \right) + \varepsilon Q(t) \right) dt + dL_1(t), \\
 dE(t) &= \left(S(t) \left(I(t) + gC(t) \right) \left(b_1 - \frac{b_2 I(t)}{p + I(t)} \right) - (m + \beta)E(t) \right) dt + dL_2(t), \\
 dC(t) &= \left((1 - \vartheta) \beta E(t) - (m + \omega_C + s_C + h_C) C(t) \right) dt + dL_3(t), \\
 dI(t) &= \left(\beta \vartheta E(t) - (m + \omega_I + s_I + h_I) I(t) \right) dt + dL_4(t), \\
 dQ(t) &= \left(aS(t) - (m + \varepsilon) Q(t) \right) dt + dL_5(t), \\
 dZ(t) &= \left(\omega_I I(t) + \omega_C C(t) - (m + h_Z + s_Z) Z(t) \right) dt + dL_6(t), \\
 dP(t) &= \left(s_Z Z(t) + s_I I(t) + s_C C(t) - mP(t) \right) dt + dL_7(t),
 \end{aligned} \tag{2}$$

where

$$\begin{aligned}
 dL_1(t) &= \beta_1 S(t) dX_1(t) + \int_{\mathcal{Z}} \gamma_1(z) S(t_-) \mathbb{Y}_-(ds, dz), \\
 dL_2(t) &= \beta_2 E(t) dX_2(t) + \int_{\mathcal{Z}} \gamma_2(z) E(t_-) \mathbb{Y}_-(ds, dz), \\
 dL_3(t) &= \beta_3 C(t) dX_3(t) + \int_{\mathcal{Z}} \gamma_3(z) C(t_-) \mathbb{Y}_-(ds, dz), \\
 dL_4(t) &= \beta_4 I(t) dX_4(t) + \int_{\mathcal{Z}} \gamma_4(z) I(t_-) \mathbb{Y}_-(ds, dz), \\
 dL_5(t) &= \beta_5 Q(t) dX_5(t) + \int_{\mathcal{Z}} \gamma_5(z) Q(t_-) \mathbb{Y}_-(ds, dz), \\
 dL_6(t) &= \beta_6 Z(t) dX_6(t) + \int_{\mathcal{Z}} \gamma_6(z) Z(t_-) \mathbb{Y}_-(ds, dz), \\
 dL_7(t) &= \beta_7 P(t) dX_7(t) + \int_{\mathcal{Z}} \gamma_7(z) P(t_-) \mathbb{Y}_-(ds, dz).
 \end{aligned}$$

In this setting, X_ℓ ($\ell = 1, \dots, 7$), are Wiener processes with positive amplitudes β_ℓ ($i = 1, \dots, 7$) respectively. \mathbb{Y}_- is the compensated Poisson measure associated with a Lévy measure Λ_+ defined on $(\mathbb{R}^7 \setminus \{0\}, \mathcal{B}(\mathbb{R}^7 \setminus \{0\}))$. $\gamma_\ell : \mathcal{Z} \subset \mathbb{R}^7 \setminus \{0\} \rightarrow \mathbb{R}$ are measurable functions and $\gamma_\ell(z) > -1$.

The organization of the remaining parts is as follows: In Section 2, we present the probabilistic analysis of a perturbed SECIQZP model (2) by offering the associated hypothetical framework and giving the condition of said properties. In Section 3, we numerically check our obtained results. In Section 4, we summarize the main results of our study.

2 Stochastic analysis of a perturbed SECIQZP model

Hypothetical framework

The first step of this section is to clearly define the hypothetical framework of our analysis. This includes the specification of the well-posedness of our perturbed system, the necessary assumptions and the useful techniques. Regarding the underlying assumption, we assume the following:

(A) The quantities $\int_{\mathcal{Z}} \gamma_k^2(z) \Lambda_+(dz)$ and $\int_{\mathcal{Z}} \left(\gamma_k(z) - \ln(1 + \gamma_k(z)) \right) \Lambda_+(dz)$ are finite for all $k \in$

$\{1, \dots, 7\}$.

Under **(A)**, system (2) is mathematically well-defined and has a unique positive solution [24]. In other words, it is a model that provides a reliable and accurate representation of the biological disease being studied. To derive some large-time estimates of the solution, we need to add a second assumption:

(B) Suppose that for a given $s > 2$, we have

$$m - 0.5(s - 1) \left(\beta_1^2 \vee \beta_2^2 \vee \beta_3^2 \vee \beta_4^2 \vee \beta_5^2 \vee \beta_6^2 \vee \beta_7^2 \right) - s^{-1} \mathbf{I}_s(z) > 0,$$

where

$$\begin{aligned} \mathbf{I}_s(z) = \int_{\mathcal{Z}} & \left(\left(1 + \overbrace{\gamma_1(z) \vee \gamma_2(z) \vee \gamma_3(z) \vee \gamma_4(z) \vee \gamma_5(z) \vee \gamma_6(z) \vee \gamma_7(z)}^{\gamma^*(z)} \right)^s - 1 \right. \\ & \left. - s \left(\underbrace{\gamma_1(z) \wedge \gamma_2(z) \wedge \gamma_3(z) \wedge \gamma_4(z) \wedge \gamma_5(z) \wedge \gamma_6(z) \wedge \gamma_7(z)}_{\gamma_*(z)} \right) \right) \Lambda_+(dz). \end{aligned}$$

Lemma 1. Under **(B)**, we have the following properties:

- (a) $\lim_{t \rightarrow \infty} \frac{y_\ell(t)}{t} = 0 \quad a.s. \quad \forall \ell \in \{1, \dots, 7\}$.
- (b) $\lim_{t \rightarrow \infty} \frac{1}{t} \int_0^t y_\ell(s) d\mathbb{X}_\ell(s) = 0 \quad a.s. \quad \forall \ell \in \{1, \dots, 7\}$.
- (c) $\lim_{t \rightarrow \infty} \frac{1}{t} \int_0^t \int_{\mathcal{Z}} \gamma_\ell(z) y_\ell(s_-) \mathbb{Y}_-(ds, dz) = 0 \quad a.s. \quad \forall \ell \in \{1, \dots, 7\}$.

The proof of the above lemma is similar to a previously proven result in [25], it is better to omit the proof in order to avoid redundancy and to streamline the presentation of the argument.

Asymptotic extinction

Asymptotic extinction refers to the extinction of a species or population due to random, unpredictable events that occur in the environment, rather than gradual, predictable changes. These events may include natural disasters, epidemics, or fluctuations in the availability of resources. It can be difficult to predict or prevent, as it is often influenced by factors beyond human control. However, conservation efforts can help mitigate the effects of stochasticity by protecting habitat and promoting genetic diversity within populations. Theoretically, we can provide sufficient conditions for disease extinction which are provided in the following theorem.

Theorem 1. Assume that **(A)** and **(B)** hold. Then, we have the following inequality:

$$\limsup_{t \rightarrow \infty} \frac{1}{t} \ln \left(E(t) + C(t) + I(t) \right) \leq b_1 \mathbf{S}^\bullet - m - \frac{1}{6} \left(\beta_2^2 \wedge \beta_3^2 \wedge \beta_4^2 \right) - \tilde{\chi} = Cte \quad a.s.,$$

where $\mathbf{S}^\bullet = \frac{\Pi}{m} \times \frac{m + \varepsilon}{a + m + \varepsilon}$ and

$$\tilde{\chi} = \int_{\mathcal{Z}} \left\{ \left(\ln(1 + \gamma^*(z)) - \gamma^*(z) \right) \mathbb{1}_{\{\gamma^*(z) \leq 0\}} + \left(\ln(1 + \gamma_*(z)) - \gamma_*(z) \right) \mathbb{1}_{\{\gamma^*(z) > 0\}} \right\} \Lambda_+(dz).$$

More precisely, the asymptotic extinction of a virus occurs when $Cte < 0$.

Proof By employing the stochastic Itô's formula, we get

$$\begin{aligned} & d \ln (E(t) + C(t) + I(t)) \\ &= \left\{ \frac{1}{E(t) + C(t) + I(t)} \left(S(t) (I(t) + gC(t)) \left(b_1 - b_2 \frac{I(t)}{p + I(t)} \right) \right. \right. \\ &\quad \left. \left. - (\omega_C + s_C + h_C) C(t) - (\omega_I + s_I + h_I) I(t) \right) - m - \frac{\beta_2^2 E^2(t) + \beta_3^2 C^2(t) + \beta_4^2 I^2(t)}{2(E(t) + C(t) + I(t))^2} \right. \\ &\quad \left. + \int_{\mathcal{Z}} \left\{ \ln \left(1 + \frac{\gamma_2(z)E(t) + \gamma_3(z)C(t) + \gamma_4(z)I(t)}{E(t) + C(t) + I(t)} \right) - \frac{\gamma_2(z)E(t) + \gamma_3(z)C(t) + \gamma_4(z)I(t)}{E(t) + C(t) + I(t)} \right\} \Lambda_+(dz) \right\} dt \\ &\quad + \frac{1}{E(t) + C(t) + I(t)} \left(\beta_2 E(t) d\mathbb{X}_2(t) + \beta_3 C(t) d\mathbb{X}_3(t) + \beta_4 I(t) d\mathbb{X}_4(t) \right) \\ &\quad + \int_{\mathcal{Z}} \ln \left(1 + \frac{\gamma_2(z)E(t_-) + \gamma_3(z)C(t_-) + \gamma_4(z)I(t_-)}{E(t_-) + C(t_-) + I(t_-)} \right) \mathbb{Y}_-(dt, dz). \end{aligned}$$

Thanks to the positivity of the solution, we get

$$\begin{aligned} & d \ln (E(t) + C(t) + I(t)) \\ &\leq \left\{ S(t) \left(b_1 - b_2 \frac{I(t)}{p + I(t)} \right) - m - 0.5(\beta_2^2 \wedge \beta_3^2 \wedge \beta_4^2) \times \frac{E^2(t) + C^2(t) + I^2(t)}{(E(t) + C(t) + I(t))^2} \right\} dt \\ &\quad + \int_{\mathcal{Z}} \left\{ \ln \left(1 + \frac{\gamma_2(z)E(t) + \gamma_3(z)C(t) + \gamma_4(z)I(t)}{E(t) + C(t) + I(t)} \right) - \frac{\gamma_2(z)E(t) + \gamma_3(z)C(t) + \gamma_4(z)I(t)}{E(t) + C(t) + I(t)} \right\} \Lambda_+(dz) dt \\ &\quad + \frac{\beta_2 E(t)}{E(t) + C(t) + I(t)} d\mathbb{X}_2(t) + \beta_4 \frac{C(t)}{E(t) + C(t) + I(t)} d\mathbb{X}_3(t) + \beta_5 \frac{I(t)}{E(t) + C(t) + I(t)} d\mathbb{X}_4(t) \\ &\quad + \int_{\mathcal{Z}} \ln \left(1 + \frac{\gamma_2(z)E(t_-) + \gamma_3(z)C(t_-) + \gamma_4(z)I(t_-)}{E(t_-) + C(t_-) + I(t_-)} \right) \mathbb{Y}_-(dt, dz). \end{aligned}$$

By employing Cauchy-Schwartz inequality, we get $E^2(t) + C^2(t) + I^2(t) \geq \frac{1}{3}(E(t) + C(t) + I(t))$.

Furthermore, we can easily check that

$$\int_{\mathcal{Z}} \left\{ \ln \left(1 + \frac{\gamma_2(z)E(t) + \gamma_3(z)C(t) + \gamma_4(z)I(t)}{E(t) + C(t) + I(t)} \right) - \frac{\gamma_2(z)E(t) + \gamma_3(z)C(t) + \gamma_4(z)I(t)}{E(t) + C(t) + I(t)} \right\} \Lambda_+(dz) \leq -\tilde{\chi}.$$

Consequently,

$$\begin{aligned} d \ln (E(t) + C(t) + I(t)) &\leq \left\{ b_1 S(t) - m - \frac{(\beta_2^2 \wedge \beta_3^2 \wedge \beta_4^2)}{6} - \tilde{\chi} \right\} dt + \frac{\beta_2 E(t)}{E(t) + C(t) + I(t)} d\mathbb{X}_2(t) \\ &\quad + \frac{\beta_3 C(t)}{E(t) + C(t) + I(t)} d\mathbb{X}_3(t) + \frac{\beta_4 I(t)}{E(t) + C(t) + I(t)} d\mathbb{X}_4(t) \quad (3) \\ &\quad + \int_{\mathcal{Z}} \ln \left(1 + (\gamma_2(z) \vee \gamma_3(z) \vee \gamma_4(z)) \right) \mathbb{Y}_-(dt, dz). \end{aligned}$$

We integrate (3) from 0 to t , and we divide by t on both sides, we obtain

$$\begin{aligned}
 \frac{\ln(E(t) + C(t) + I(t))}{t} &\leq \frac{\mathfrak{b}_1}{t} \int_0^t S(s) ds - \mathfrak{m} - \frac{1}{6} (\beta_2^2 \wedge \beta_3^2 \wedge \beta_4^2) - \tilde{\chi} \\
 &+ \frac{\beta_2}{t} \int_0^t \frac{E(s)}{E(s) + C(s) + I(s)} d\mathbb{X}_2(s) + \frac{\beta_3}{t} \int_0^t \frac{C(s)}{E(s) + C(s) + I(s)} dB_3(s) \\
 &+ \frac{\beta_4}{t} \int_0^t \frac{I(s)}{E(s) + C(s) + I(s)} d\mathbb{X}_4(s) + \frac{\ln(E(0) + C(0) + I(0))}{t} \\
 &+ \frac{1}{t} \int_0^t \int_{\mathcal{Z}} \ln(1 + (\gamma_2(z) \vee \gamma_3(z) \vee \gamma_4(z))) \mathbb{Y}_-(ds, dz). \tag{4}
 \end{aligned}$$

Now, we need to estimate $\frac{1}{t} \int_0^t S(s) ds$. From (2), we remark that

$$\begin{aligned}
 S(t) &= \Pi t - (\mathfrak{m} + \mathfrak{a}) \int_0^t S(s) ds - \int_0^t S(s) (I(s) + gC(s)) \left(\mathfrak{b}_1 - \frac{\mathfrak{b}_2 I(s)}{p + I(s)} \right) ds + \varepsilon \int_0^t Q(s) ds \\
 &+ \beta_1 \int_0^t S(s) d\mathbb{X}_1(s) + \int_0^t \int_{\mathcal{Z}} \gamma_1(z) S(s_-) \mathbb{Y}_-(ds, dz) + S(0) \\
 &\leq \Pi t - (\mathfrak{m} + \mathfrak{a}) \int_0^t S(s) ds + \varepsilon \int_0^t Q(s) ds + \beta_1 \int_0^t S(s) d\mathbb{X}_1(s) \\
 &+ \int_0^t \int_{\mathcal{Z}} \gamma_1(z) S(s_-) \mathbb{Y}_-(ds, dz) + S(0).
 \end{aligned}$$

Then

$$\begin{aligned}
 \frac{1}{t} \int_0^t S(s) ds &\leq \frac{1}{\mathfrak{m} + \mathfrak{a}} \left(\Pi + \frac{\varepsilon}{t} \int_0^t Q(s) ds + \frac{\beta_1}{t} \int_0^t S(s) d\mathbb{X}_1(s) \right. \\
 &\left. + \frac{1}{t} \int_0^t \int_{\mathcal{Z}} \gamma_1(z) S(s_-) \mathbb{Y}_-(ds, dz) + \frac{S(0)}{t} - \frac{S(t)}{t} \right). \tag{5}
 \end{aligned}$$

Again, we need to estimate $\frac{1}{t} \int_0^t Q(s) ds$. From system (2), we have

$$Q(t) = \mathfrak{a} \int_0^t S(s) ds - (\mathfrak{m} + \varepsilon) \int_0^t Q ds + \beta_2 \int_0^t Q(s) d\mathbb{X}_5(s) + \int_0^t \int_{\mathcal{Z}} \gamma_1(z) Q(s_-) \mathbb{Y}_-(ds, dz) + Q(0),$$

which implies that

$$\frac{1}{t} \int_0^t Q(s) ds = \frac{1}{\mathfrak{m} + \varepsilon} \left(\mathfrak{a} \int_0^t S(s) ds + \frac{\beta_5}{t} \int_0^t Q(s) d\mathbb{X}_5(s) + \frac{1}{t} \int_0^t \int_{\mathcal{Z}} \gamma_5(z) Q(s_-) \mathbb{Y}_-(ds, dz) + \frac{Q(0) - Q(t)}{t} \right). \tag{6}$$

Combining (5) with (6) yields

$$\begin{aligned} \frac{1}{t} \int_0^t S(s) \, ds &\leq \frac{1}{m+a} \left\{ \Pi + \varepsilon \left(\frac{a}{(m+\varepsilon)t} \int_0^t S(s) \, ds + \frac{\beta_5}{(m+\varepsilon)t} \int_0^t Q(s) \, dX_5(s) \right. \right. \\ &\quad \left. \left. + \frac{1}{(m+\varepsilon)t} \int_0^t \int_{\mathcal{Z}} \gamma_5(z) Q(s_-) Y_-(ds, dz) + \frac{Q(0)}{(m+\varepsilon)t} \right) \right. \\ &\quad \left. + \frac{\beta_1}{t} \int_0^t S(s) \, dX_1(s) + \frac{1}{t} \int_0^t \int_{\mathcal{Z}} \gamma_1(z) S(s_-) Y_-(ds, dz) + \frac{S(0)}{t} \right\}. \end{aligned}$$

Thus,

$$\begin{aligned} \frac{1}{t} \int_0^t S(s) \, ds &\leq \frac{\Pi(m+\varepsilon)}{m(a+m+\varepsilon)} + \frac{\varepsilon\beta_2}{m(a+m+\varepsilon)t} \int_0^t Q(s) \, dX_5(s) + \frac{(m+\varepsilon)\beta_1}{m(m+a+\varepsilon)t} \int_0^t S(s) \, dX_1(s) \\ &\quad + \frac{\varepsilon}{m(a+m+\varepsilon)t} \int_0^t \int_{\mathcal{Z}} \gamma_5(z) Q(s_-) Y_-(ds, dz) \\ &\quad + \frac{(m+\varepsilon)}{m(m+a+\varepsilon)t} \int_0^t \int_{\mathcal{Z}} \gamma_1(z) S(s_-) Y_-(ds, dz) \\ &\quad + \frac{\varepsilon Q(0)}{m(m+a+\varepsilon)t} + \frac{(m+\varepsilon)S(0)}{m(m+a+\varepsilon)t}. \end{aligned} \tag{7}$$

From Lemma 1, we obtain

$$\lim_{t \rightarrow \infty} \frac{1}{t} \int_0^t S(s) \, ds \leq \frac{\Pi}{m} \times \frac{m+\varepsilon}{a+m+\varepsilon} = \mathbf{S}^\bullet. \tag{8}$$

An application direct of the strong law of large numbers for local martingales gives

$$\begin{cases} \lim_{t \rightarrow \infty} \frac{1}{t} \int_0^t \int_{\mathcal{Z}} \ln \left(1 + (\gamma_2(z) \vee \gamma_3(z) \vee \gamma_4(z)) \right) Y_-(ds, dz) = 0 \quad \text{a.s.} \\ \lim_{t \rightarrow \infty} \frac{\beta_2}{t} \int_0^t \frac{E(s)}{E(s) + C(s) + I(s)} \, dX_2(s) = 0 \quad \text{a.s.}, \\ \lim_{t \rightarrow \infty} \frac{\beta_3}{t} \int_0^t \frac{A(s)}{E(s) + C(s) + I(s)} \, dX_3(s) = 0 \quad \text{a.s.}, \\ \lim_{t \rightarrow \infty} \frac{\beta_4}{t} \int_0^t \frac{I(s)}{E(s) + C(s) + I(s)} \, dX_4(s) = 0 \quad \text{a.s.} \end{cases} \tag{9}$$

Finally and from results (4), (8) and (9), we conclude that

$$\limsup_{t \rightarrow \infty} \frac{1}{t} \ln \left(E(t) + C(t) + I(t) \right) \leq \mathbf{b}_1 \mathbf{S}^\bullet - m - \frac{1}{6} \left(\beta_2^2 \wedge \beta_3^2 \wedge \beta_4^2 \right) - \tilde{\chi} \quad \text{a.s.}$$

If $\underline{Cte} < 0$, then asymptotic extinction will occur fine. ■

Remark 1. *The positivity of the solution allows us to affirm that $\lim_{t \rightarrow \infty} E(t) = 0$, $\lim_{t \rightarrow \infty} C(t) = 0$ and $\lim_{t \rightarrow \infty} I(t) = 0$ a.s. Here the total extinction of the virus is mentioned.*

Corollary 1. According to the hypothesis and the context of Theorem 1, we get

$$\lim_{t \rightarrow \infty} \frac{1}{t} \int_0^t S(s) ds = \mathbf{S}^\bullet \text{ a.s.}, \quad \lim_{t \rightarrow \infty} \frac{1}{t} \int_0^t Q(s) ds = \mathbf{Q}^\bullet \text{ a.s.}$$

Proof From system (2), we obtain

$$\begin{aligned} d(S(t) + E(t)) &= \left\{ \Pi - (\mathbf{m} + \mathbf{a}) S(t) + \varepsilon Q(t) - (\mathbf{m} + \beta) E(t) \right\} dt \\ &\quad + \beta_1 S(t) d\mathbf{X}_1(t) + \int_{\mathcal{Z}} \gamma_1(z) S(t_-) \mathbf{Y}_-(dt, dz) \\ &\quad + \beta_2 E(t) d\mathbf{X}_2(t) + \int_{\mathcal{Z}} \gamma_2(z) E(t_-) \mathbf{Y}_-(dt, dz). \end{aligned} \quad (10)$$

We integrate (10) from 0 to t , and we divide by t on both sides, we get

$$\begin{aligned} \frac{1}{t} (S(t) + E(t)) &= \Pi - \frac{(\mathbf{m} + \mathbf{a})}{t} \int_0^t S(s) ds + \frac{\varepsilon}{t} \int_0^t Q(s) ds - \frac{(\mathbf{m} + \beta)}{t} \int_0^t E(s) ds \\ &\quad + \frac{S(0) + E(0)}{t} + \beta_1 \int_0^t S(s) d\mathbf{X}_1(s) + \int_0^t \int_{\mathcal{Z}} \gamma_1(z) S(s_-) \mathbf{Y}_-(ds, dz) \\ &\quad + \beta_2 \int_0^t E(s) d\mathbf{X}_2(s) + \int_0^t \int_{\mathcal{Z}} \gamma_2(z) E(s_-) \mathbf{Y}_-(ds, dz). \end{aligned}$$

From the expression (6), we have

$$\begin{aligned} \frac{1}{t} (S(t) + E(t)) &= \Pi - \frac{(\mathbf{m} + \mathbf{a})}{t} \int_0^t S(s) ds + \frac{\varepsilon}{(\varepsilon + \mathbf{m})t} \left(\mathbf{a} \int_0^t S(s) ds - \frac{Q(t) - Q(0)}{t} \right. \\ &\quad \left. + \frac{\beta_5}{t} \int_0^t Q(s) d\mathbf{X}_5(s) + \int_0^t \int_{\mathcal{Z}} \gamma_5(z) Q(s_-) \mathbf{Y}_-(ds, dz) \right) - \frac{(\mathbf{m} + \beta)}{t} \int_0^t E(s) ds \\ &\quad + \frac{S(0) + E(0)}{t} + \beta_1 \int_0^t S(s) d\mathbf{X}_1(s) + \int_0^t \int_{\mathcal{Z}} \gamma_1(z) S(s_-) \mathbf{Y}_-(ds, dz) \\ &\quad + \beta_2 \int_0^t E(s) d\mathbf{X}_2(s) + \int_0^t \int_{\mathcal{Z}} \gamma_2(z) E(s_-) \mathbf{Y}_-(ds, dz). \end{aligned}$$

Then

$$\begin{aligned} \frac{1}{t} \left(-\frac{\varepsilon \mathbf{a}}{\varepsilon + \mathbf{m}} + \mathbf{m} + \mathbf{a} \right) \int_0^t S(s) ds &= \Pi + \frac{\varepsilon}{\mathbf{m} + \varepsilon} \frac{Q(0) - Q(t)}{t} - \frac{S(t) + E(t)}{t} - \frac{(\mathbf{m} + \beta)}{t} \int_0^t E(s) ds \\ &\quad + \frac{\varepsilon \beta_5}{(\mathbf{m} + \varepsilon)t} \int_0^t Q(s) d\mathbf{X}_5(s) + \frac{\varepsilon}{(\mathbf{m} + \varepsilon)t} \int_0^t \int_{\mathcal{Z}} \gamma_5(z) Q(s_-) \mathbf{Y}_-(ds, dz) \\ &\quad + \frac{S(0) + E(0)}{t} + \frac{\beta_1}{t} \int_0^t S(s) d\mathbf{X}_1(s) + \frac{1}{t} \int_0^t \int_{\mathcal{Z}} \gamma_1(z) S(s_-) \mathbf{Y}_-(ds, dz) \\ &\quad + \frac{\beta_2}{t} \int_0^t E(s) d\mathbf{X}_2(s) + \frac{1}{t} \int_0^t \int_{\mathcal{Z}} \gamma_2(z) E(s_-) \mathbf{Y}_-(ds, dz). \end{aligned}$$

By using Lemma 1, we directly obtain that

$$\lim_{t \rightarrow \infty} \frac{1}{t} \left(-\frac{\varepsilon \alpha}{\varepsilon + m} + m + \alpha \right) \int_0^t S(s) \, ds = \Pi - \lim_{t \rightarrow \infty} \frac{(m + \beta)}{t} \int_0^t E(s) \, ds \quad \text{a.s.},$$

From Remark 1, we indicated that $\lim_{t \rightarrow \infty} E(t) = 0$ a.s., which implies that $\lim_{t \rightarrow \infty} \frac{1}{t} \int_0^t E(s) \, ds = 0$ a.s. So

$$\lim_{t \rightarrow \infty} \frac{1}{t} \int_0^t S(s) \, ds = \Pi \left(-\frac{\varepsilon \alpha}{\varepsilon + m} + m + \alpha \right)^{-1} = \frac{\Pi}{m} \times \frac{m + \varepsilon}{\alpha + m + \varepsilon} = \mathbf{S}^\bullet,$$

and

$$\lim_{t \rightarrow \infty} \frac{1}{t} \int_0^t Q(s) \, ds = \frac{q}{(\Pi + m)t} \int_0^t S(s) \, ds - \frac{Q(t)}{(\Pi + m)t} + \frac{Q(0)}{(\Pi + m)t} + \frac{\beta_2}{\Pi + m} \times \frac{1}{t} \int_0^t Q(s) \, dB_2(s).$$

Therefore,

$$\lim_{t \rightarrow \infty} \frac{1}{t} \int_0^t Q(s) \, ds = \frac{\alpha \mathbf{S}^\bullet}{\varepsilon + m} = \mathbf{Q}^\bullet.$$

■

Remark 2. From the above results, we can directly infer that

$$\lim_{t \rightarrow \infty} \frac{1}{t} \int_0^t Z(s) \, ds = 0 \text{ a.s.}, \quad \text{and} \quad \lim_{t \rightarrow \infty} \frac{1}{t} \int_0^t P(s) \, ds = 0 \text{ a.s.}$$

Asymptotic persistence

Asymptotic persistence of a virus refers to the scenario where the infection becomes endemic in a population, meaning that it becomes present at a relatively constant level within that population over time. This can occur when the virus has a low but steady transmission rate, allowing it to continue spreading even when there are no major outbreaks. In this subsection, we give an optimal condition for the continuation of the virus which is presented in the following theorem.

Theorem 2. Assume that (A) and (B) hold. If $f_1(\hat{u}) > f_2 + f_3$, then we have the following inequality

$$\liminf_{t \rightarrow \infty} \frac{1}{t} \int_0^t (I(s) + C(s)) \, ds \geq \frac{1}{b_1} (f_1(\hat{u}) - f_2 - f_3) = \underline{Cte} \quad \text{a.s.},$$

where

- $f_1(u) = 3 \left(\sqrt[3]{g(1-d)u} + \sqrt[3]{d \times (1-u)} \right) \sqrt[3]{\Pi(b_1 - b_2)} \beta, \quad \forall u \in (0, 1),$
- $f_2 = 7m + \beta + (\omega_C + s_C + h_C) + (\omega_I + s_I + h_I) + (h_Z + s_Z) + |\Pi - \alpha|,$
- $f_3 = \frac{1}{2} \sum_{i=1}^7 \beta_i^2 + \sum_{i=1}^7 \int_{\mathcal{Z}} \left(\gamma_i(z) - \ln(1 + \gamma_i(z)) \right) \Lambda_+(dz),$
- $\hat{u} = \sqrt{g(1-d)} \left(\sqrt{g(1-d)} + \sqrt{d} \right)^{-1}, \quad (0 < \hat{u} < 1).$

More precisely, the asymptotic persistence of a virus occurs when $\underline{Cte} > 0$.

Proof We define the function $\widehat{\mathcal{F}}(y) = \sum_{\ell=1}^7 \ln(y_\ell)$. Direct use of Itô's formula gives

$$\begin{aligned} d\widehat{\mathcal{F}}(y(t)) = & \left\{ \left(\frac{\Pi}{S(t)} - (m + a) - (I(t) + gC(t)) \left(b_1 - \frac{b_2 I(t)}{\vartheta + I(t)} \right) + \frac{\varepsilon Q(t)}{S(t)} \right) \right. \\ & + \left(\frac{S(t)}{E(t)} (I(t) + gC(t)) \left(b_1 - \frac{b_2 I(t)}{\vartheta + I(t)} \right) - (m + \beta) \right) \\ & + \left((1 - \vartheta) \beta \frac{E(t)}{C(t)} - (m + \omega_C + s_C + h_C) \right) \\ & + \left(\beta \vartheta \frac{E(t)}{I(t)} - (m + \omega_I + s_I + h_I) \right) + \left(a \frac{S(t)}{Q(t)} - (m + \varepsilon) \right) \\ & + \left(\omega_I \frac{I(t)}{Z(t)} + \omega_C \frac{C(t)}{Z(t)} - (m + h_Z + s_Z) \right) + \left(s_Z \frac{Z(t)}{P(t)} + s_I \frac{I(t)}{P(t)} + s_C \frac{C(t)}{P(t)} - m \right) \\ & - 0.5 \sum_{i=1}^7 \beta_i^2 - \sum_{i=1}^7 \int_{\mathcal{Z}} (\gamma_i(z) - \ln(1 + \gamma_i(z))) \Lambda_+(dz) \Big\} dt \\ & + \sum_{i=1}^7 \beta_i dX_i(t) + \sum_{i=1}^7 \int_{\mathcal{Z}} \ln(1 + \gamma_i(z)) \Upsilon_-(dt, dz). \end{aligned}$$

Then

$$\begin{aligned} d\widehat{\mathcal{F}}(y(t)) \geq & \left\{ \frac{\Pi}{S(t)} - b_1(I(t) + gC(t)) + (\varepsilon \wedge a) \left(\frac{Q(t)}{S(t)} + \frac{S(t)}{Q(t)} \right) + \frac{S(t)}{E(t)} (b_1 - b_2) (I(t) + gC(t)) \right. \\ & + (1 - \vartheta) \beta \frac{E(t)}{C(t)} + \beta \vartheta \frac{E(t)}{I(t)} - \sum_{i=1}^7 \int_{\mathcal{Z}} (\gamma_i(z) - \ln(1 + \gamma_i(z))) \Lambda_+(dz) \\ & - \left[7m + \Pi + a + \beta + (\omega_C + s_C + h_C) + (\omega_I + s_I + h_I) + (h_Z + s_Z) + 0.5 \sum_{i=1}^7 \beta_i^2 \right] \Big\} dt \\ & + \sum_{i=1}^7 \beta_i dX_i(t) + \sum_{i=1}^7 \int_{\mathcal{Z}} \ln(1 + \gamma_i(z)) \Upsilon_-(dt, dz). \end{aligned}$$

By remarking that $2(\Pi \wedge a) = \Pi + a - |\Pi - a|$; and $(S^2(t) + Q^2(t)) \geq 2S(t)Q(t)$, we get

$$\begin{aligned} d\widehat{\mathcal{F}}(X(t)) \geq & \left(\left[\frac{(1 - \widehat{u})\Pi}{S(t)} + (b_1 - b_2) \frac{S(t)I(t)}{E(t)} + \beta \vartheta \frac{E(t)}{I(t)} \right] \right. \\ & + \left[\frac{\widehat{u}\Pi}{S(t)} + g(b_1 - b_2) \frac{S(t)C(t)}{E(t)} + (1 - \vartheta) \beta \frac{E(t)}{C(t)} \right] \\ & \left. - b_1(I + gC(t)) - f_2 - f_3 \right) dt + \sum_{i=1}^7 \beta_i dX_i(t) + \sum_{i=1}^7 \int_{\mathcal{Z}} \ln(1 + \gamma_i(z)) \Upsilon_-(dt, dz). \end{aligned}$$

By employing the arithmetic-geometric inequality, we directly obtain

$$\begin{aligned}
 d\widehat{\mathcal{F}}(X(t)) &\geq \left(3\sqrt[3]{(1-\widehat{u})\Pi(\mathbf{b}_1-\mathbf{b}_2)\beta\mathfrak{d}} + 3\sqrt[3]{\widehat{u}\Pi(\mathbf{b}_1-\mathbf{b}_2)g\beta(1-\mathfrak{d})} - \mathbf{b}_1(I+gC(t)) - f_2 - f_3 \right) dt \\
 &\quad + \sum_{i=1}^7 \beta_i d\mathbb{X}_i(t) + \sum_{i=1}^7 \int_{\mathcal{Z}} \ln(1+\gamma_i(z)) \mathbb{Y}_-(dt, dz) \\
 &\geq \left((f_1(\widehat{u}) - f_2 - f_3) - \mathbf{b}_1(I+gC(t)) \right) dt \\
 &\quad + \sum_{i=1}^7 \beta_i d\mathbb{X}_i(t) + \sum_{i=1}^7 \int_{\mathcal{Z}} \ln(1+\gamma_i(z)) \mathbb{Y}_-(dt, dz).
 \end{aligned} \tag{11}$$

An integration from 0 to t on both sides of (11) leads to

$$\begin{aligned}
 \frac{1}{t} \left(\widehat{\mathcal{F}}(y(t)) - \widehat{\mathcal{F}}(y(0)) \right) &\geq (f_1(\widehat{u}) - f_2 - f_3) - \frac{\mathbf{b}_1}{t} \int_0^t (I(s) + gC(s)) ds \\
 &\quad + \frac{1}{t} \sum_{i=1}^7 \beta_i \mathbb{X}_i(t) + \frac{1}{t} \sum_{i=1}^7 \int_0^t \int_{\mathcal{Z}} \ln(1+\gamma_i(z)) \mathbb{Y}_-(dt, dz).
 \end{aligned}$$

Hence,

$$\begin{aligned}
 \frac{1}{t} \int_0^t (I(s) + C(s)) ds &\geq \int_0^t (I(s) + gC(s)) ds \geq \frac{1}{\mathbf{b}_1} \left(\frac{\widehat{\mathcal{F}}(X(0)) - \widehat{\mathcal{F}}(X(t))}{t} + (f_1(\widehat{u}) - f_2 - f_3) \right) \\
 &\quad + \frac{1}{t} \sum_{i=1}^7 \int_0^t \int_{\mathcal{Z}} \ln(1+\gamma_i(z)) \mathbb{Y}_-(dt, dz).
 \end{aligned}$$

Thanks to the strong law of large numbers for local martingales and Lemma 1, we finally get

$$\liminf_{t \rightarrow \infty} \frac{1}{t} \int_0^t (I(s) + C(s)) ds \geq \frac{1}{\mathbf{b}_1} (f_1(\widehat{u}) - f_2 - f_3) = \underline{Cte} \quad \text{a.s.}$$

If $Cte > 0$, then asymptotic persistence will occur almost surely. That is to say that all classes of the population persist in the mean. ■

Remark 3. *In the context of a disease, persistence in the mean refers to the tendency of the disease incidence or prevalence to revert back to its long-term average over time. This means that if the incidence or prevalence of a disease is higher (or lower) than its long-term average in one period, it is likely to be closer to the average in the next period.*

3 Numerical verification

A numerical verification of theoretical results involves using computational methods to simulate a mathematical model or theory and comparing the simulation results to the analytical predictions. This process helps to validate the theoretical results and to gain a better understanding of the underlying phenomena. For this reason, we present the following two examples in order to validate the outcomes of Theorems 1 and 2.

Example 1: Asymptotic extinction

We consider the following initial data:

$$(S(0), E(0), C(0), I(0), Q(0), Z(0), P(t)) = (3, 1.6, 1.2, 1.3, 1.8, 0, 5, 0.2).$$

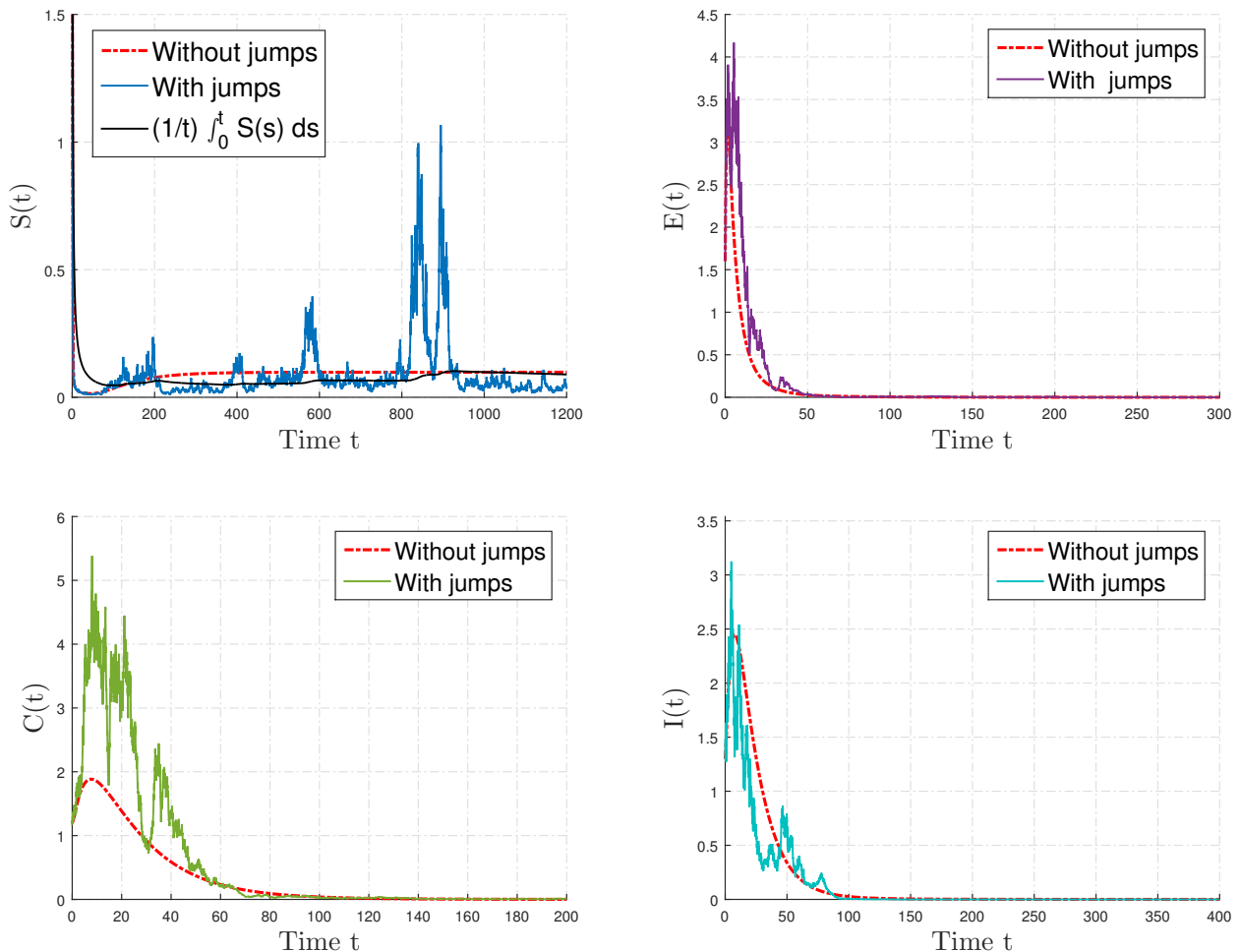
For deterministic parameters, we select $\Pi = 0.01$, $m = 0.014$, $h_C = 0.0005$, $h_I = 0.0008$, $h_Z = 0.004$, $\alpha = 0.1003$, $\epsilon = 0.071$, $b_1 = 1.2$, $b_2 = 0.1$, $p = 0.71$, $g = 0.0594$, $\beta = 0.6201$, $\nu = 0.2$, $\omega_I = 0.033$, $\omega_C = 0.024$, $s_Z = 0.02$, $s_I = 0.0183$ and $s_C = 0.0139$. For stochastic parameters, we select $\beta_\ell = 0.25$ and $\gamma_\ell = 0.12$. Then

$$m - 0.5(s - 1) (\beta_1^2 \vee \beta_2^2 \vee \beta_3^2 \vee \beta_4^2 \vee \beta_5^2 \vee \beta_6^2 \vee \beta_7^2) - s^{-1} \mathbf{I}_s(z) = 0.00145 > 0,$$

and

$$\limsup_{t \rightarrow \infty} \frac{1}{t} \ln (E(t) + C(t) + I(t)) \leq b_1 \mathbf{S}^\bullet - m - \frac{1}{6} (\beta_2^2 \wedge \beta_3^2 \wedge \beta_4^2) - \tilde{\chi} = -0.0075 < 0.$$

Consequently, the conditions of Theorem 1 are verified and the infection will asymptotically extinct (see Figure 2).



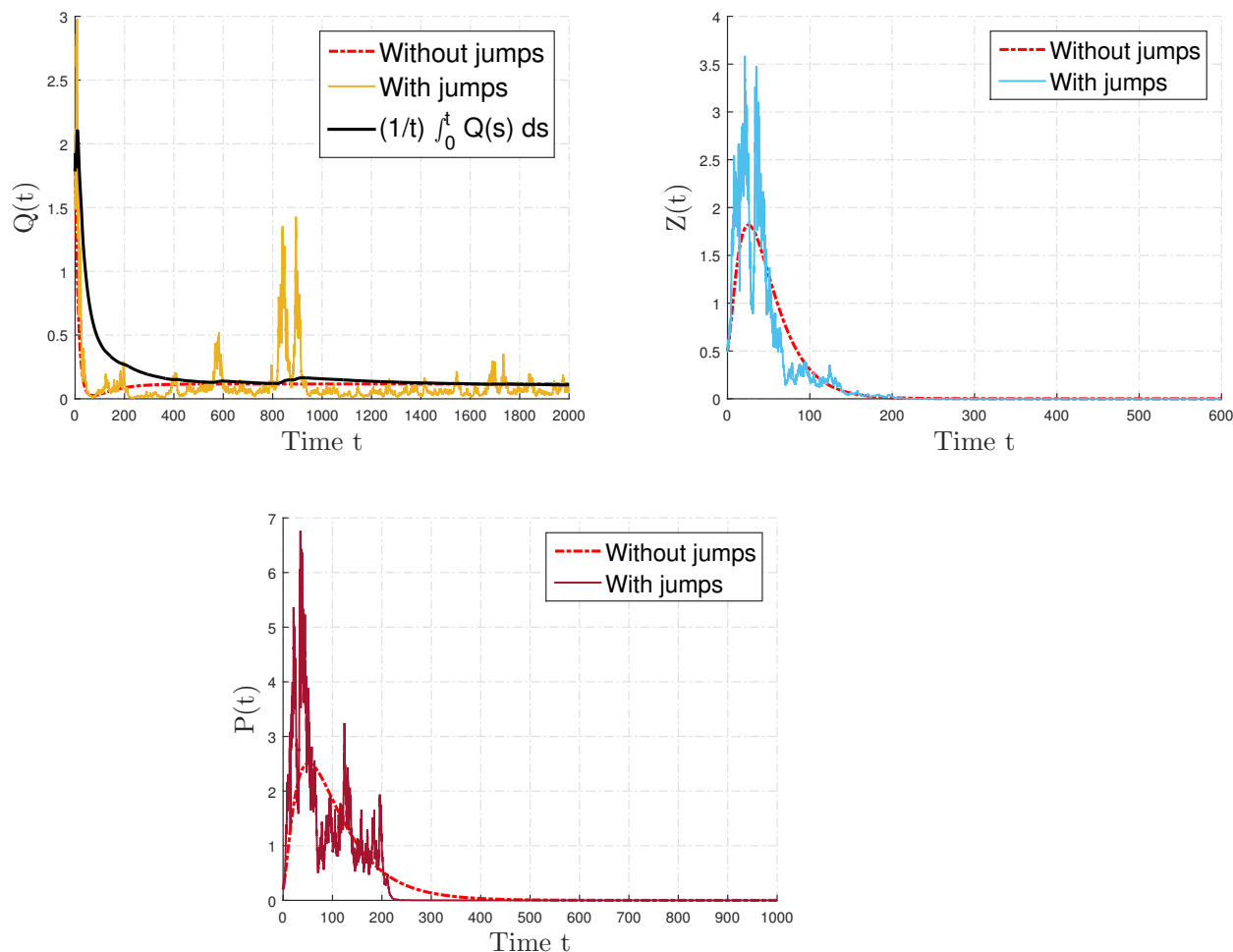


Figure 2. Random paths of perturbed model (2) in the case of asymptotic extinction

Example 2: Asymptotic persistence

We consider the following initial data:

$$\left(S(0), E(0), C(0), I(0), Q(0), Z(0), P(t) \right) = \left(3, 1.6, 1.2, 1.3, 1.8, 0, 5, 0.2 \right).$$

For deterministic parameters, we select $\Pi = 0.04$, $m = 0.014$, $h_C = 0.0005$, $h_I = 0.0008$, $h_Z = 0.004$, $\alpha = 0.1003$, $\epsilon = 0.071$, $b_1 = 1.2$, $b_2 = 0.1$, $p = 0.71$, $g = 0.0594$, $\beta = 0.6201$, $\nu = 0.2$, $\omega_I = 0.033$, $\omega_C = 0.024$, $s_Z = 0.02$, $s_I = 0.0183$ and $s_C = 0.0139$. For stochastic parameters, we select $\beta_\ell = 0.15$ and $\gamma_\ell = 0.08$. Then $f_1(\hat{u}) = 0.145 > f_2 + f_3 = 0.0378$, and

$$\liminf_{t \rightarrow \infty} \frac{1}{t} \int_0^t \left(I(s) + C(s) \right) ds \geq \frac{1}{b_1} (f_1(\hat{u}) - f_2 - f_3) = 0.0893 > 0.$$

From Figure 4, we confirm the result of Theorem 1. Therefore, all model classes persist almost surely, which are shown in Figures 3 and 4.

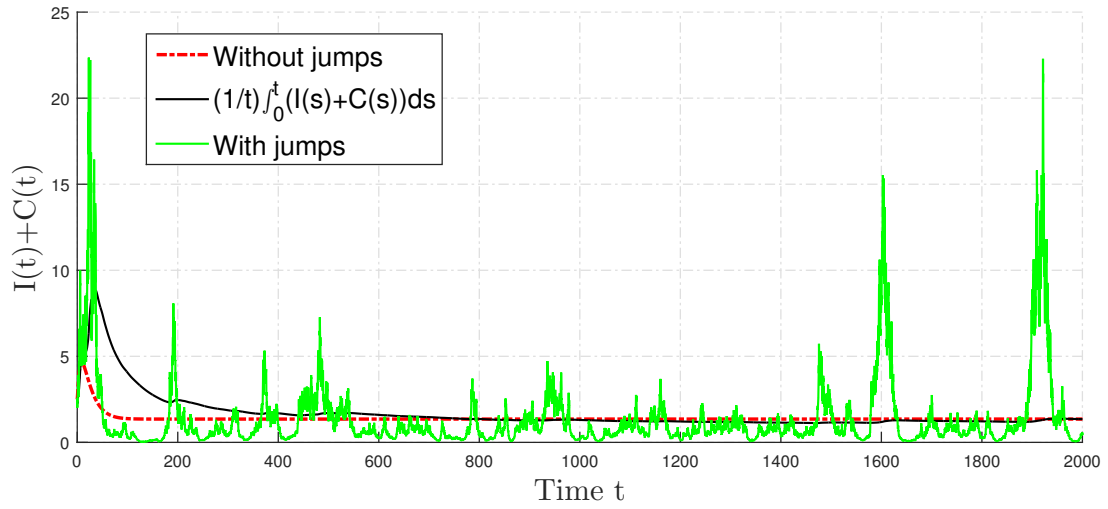
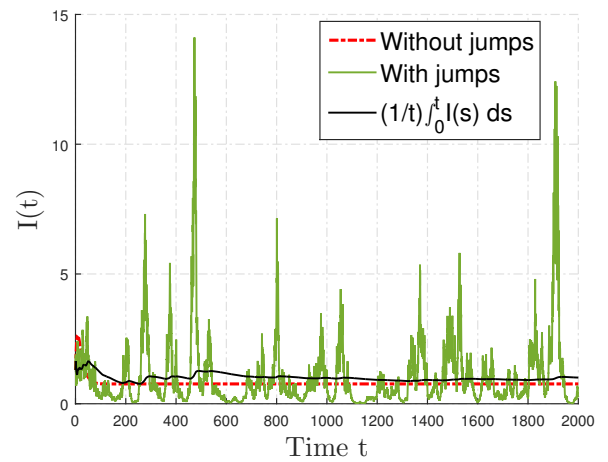
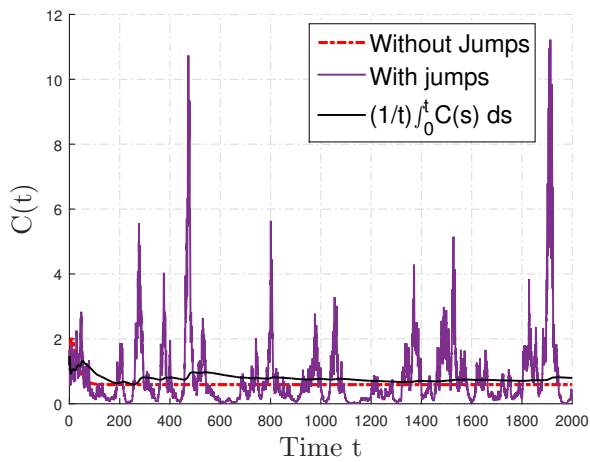
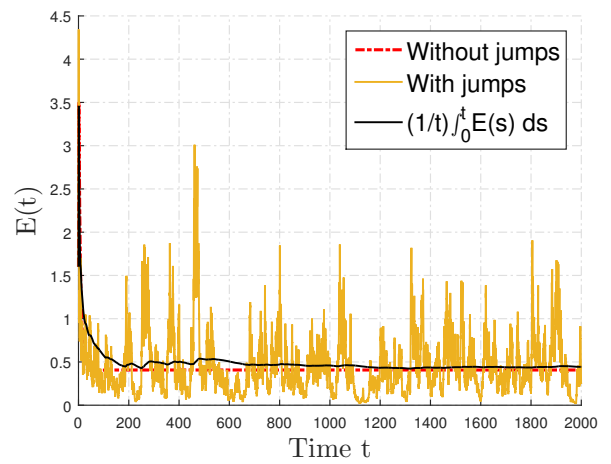
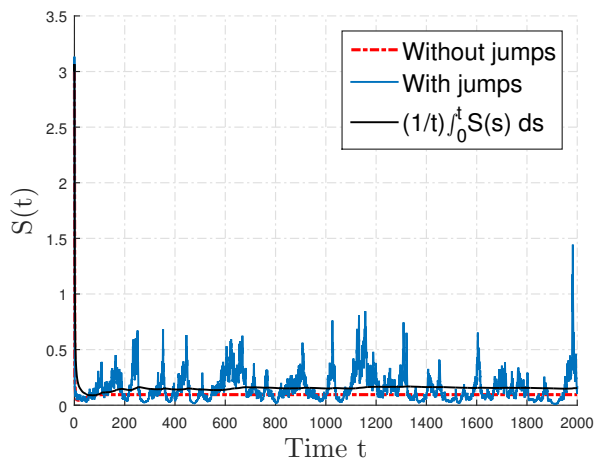


Figure 3. Random paths of the solution $I(t) + C(t)$ in the case of asymptotic persistence



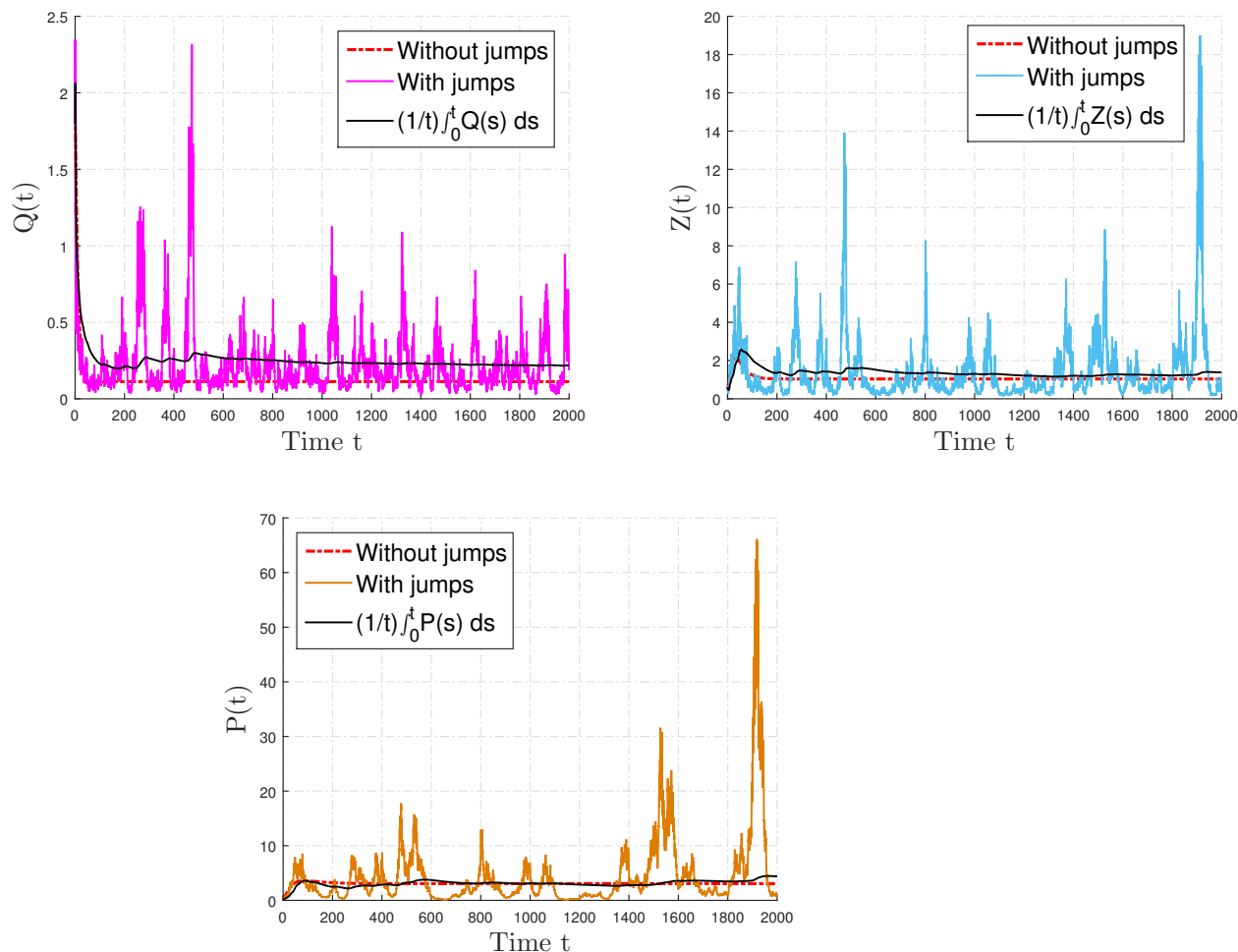


Figure 4. Random paths of perturbed model (2) in the case of asymptotic persistence

4 Concluding remarks

Intervention measures are strategies implemented in epidemiology to prevent or control the spread of infectious diseases. These measures can be classified into primary, secondary, and tertiary prevention. In this article, we have proposed a general epidemic model that takes into consideration various measurement interventions such as media, isolation, and therapy. Our model is extended to a more general and real context by considering the effect of discontinuities. Epidemiological leaps refer to sudden increases in the number of cases of a particular disease within a population or geographic area. These jumps can occur for a variety of reasons, including changes in the environment, behaviors, or characteristics of the pathogen. It is important to understand these factors to develop effective strategies to control and manage the spread of the disease. For this reason, we have provided the conditions for the extinction and persistence of the infection. Finally, we performed some numerical experiments to validate our study.

In general, we point out that this study generalizes many previous works to the case of standard Lévy jumps. Furthermore, this study offers a few new insights for understanding the transmission of the disease with complex real-world assumptions. In other words, the techniques and models investigated in this work open up several research opportunities for future studies.

Declarations

Ethical approval

Not applicable.

Consent for publication

Not applicable.

Conflicts of interest

The author declares that he has no conflict of interest.

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Author's contributions

The author confirmed the results and contributed to the final manuscript.

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References

- [1] Brauer, F. and Castillo-Chavez, C. *Mathematical Models in Population Biology and Epidemiology* (Vol. 40). Springer: New York, (2012).
- [2] Capasso, V. *Mathematical Structures of Epidemic Systems* (Vol. 97). Springer, (2008).
- [3] Safarishahrbiari, A., Lawrence, T., Lomotey, R., Liu, J., Waldner, C. and Osgood, N. Particle filtering in a SEIRV simulation model of H1N1 influenza. In *2015 Winter Simulation Conference (WSC)*, pp. 1240-1251, Huntington Beach, CA, USA, (2015, December). [[CrossRef](#)]
- [4] Brauer, F. Compartmental models in epidemiology. In *Mathematical Epidemiology* (pp. 19-79). Berlin, Heidelberg: Springer, (2008). [[CrossRef](#)]
- [5] Ming, W.K., Huang, J. and Zhang, C.J. Breaking down of healthcare system: Mathematical modelling for controlling the novel coronavirus (2019-nCoV) outbreak in Wuhan, China. *BioRxiv*, (2020). [[CrossRef](#)]
- [6] Nesteruk, I. Statistics-based predictions of coronavirus epidemic spreading in mainland China, Igor Sikorsky Kyiv Polytechnic Institute, MedRxiv, (2020).
- [7] Li, R., Pei, S., Chen, B., Song, Y., Zhang, T., Yang, W. and Shaman, J. Substantial undocumented infection facilitates the rapid dissemination of novel coronavirus (SARS-CoV-2). *Science*, 368(6490), 489-493, (2020). [[CrossRef](#)]
- [8] Russell, T.W., Hellewell, J., Abbott, S., Golding, N., Gibbs, H., Jarvis, C.I., et al. Using a delay-adjusted case fatality ratio to estimate under-reporting. *Centre for Mathematical Modeling of Infectious Diseases Repository*, 22, (2020).
- [9] Pal, D., Ghosh, D., Santra, P.K. and Mahapatra, G.S. Mathematical analysis of a COVID-19 epidemic model by using data-driven epidemiological parameters of diseases spread in India. *Biophysics*, 67(2), 231-244, (2022). [[CrossRef](#)]
- [10] Hu, Z., Cui, Q., Han, J., Wang, X., Wei, E.I. and Teng, Z. Evaluation and prediction of the COVID-19 variations at different input population and quarantine strategies, a case study

- in Guangdong province, China. *International Journal of Infectious Diseases*, 95, 231-240, (2020). [[CrossRef](#)]
- [11] Darnell, M.E., Subbarao, K., Feinstone, S.M. and Taylor, D.R. Inactivation of the coronavirus that induces severe acute respiratory syndrome, SARS-CoV. *Journal of Virological Methods*, 121(1), 85-91, (2004). [[CrossRef](#)]
- [12] Wu, J., Tang, B., Bragazzi, N.L., Nah, K. and McCarthy, Z. Quantifying the role of social distancing, personal protection and case detection in mitigating COVID-19 outbreak in Ontario, Canada. *Journal of Mathematics in Industry*, 10, 1-12, (2020). [[CrossRef](#)]
- [13] Mohsen, A.A., Al-Husseiny, H.F., Zhou, X. and Hattaf, K. Global stability of COVID-19 model involving the quarantine strategy and media coverage effects. *AIMS Public Health*, 7(3), 587, (2020). [[CrossRef](#)]
- [14] Ivorra, B., Ferrández, M.R., Vela-Pérez, M. and Ramos, A.M. Mathematical modeling of the spread of the coronavirus disease 2019 (COVID-19) taking into account the undetected infections. *The Case of China. Communications in Nonlinear Science and Numerical Simulation*, 88, 105303, (2020). [[CrossRef](#)]
- [15] Karimi-Zarchi, M., Neamatzadeh, H., Dastgheib, S.A., Abbasi, H., Mirjalili, S.R., Behforouz, A., Ferdosian F. and Bahrami, R. Vertical transmission of coronavirus disease 19 (COVID-19) from infected pregnant mothers to neonates: a review. *Fetal and Pediatric Pathology*, 39(3), 246-250, (2020). [[CrossRef](#)]
- [16] Lu, D., Sang, L., Du, S., Li, T., Chang, Y. and Yang, X.A. Asymptomatic COVID-19 infection in late pregnancy indicated no vertical transmission. *Journal of Medical Virology*, 92(9), 1660-1664, (2020). [[CrossRef](#)]
- [17] Schwartz, D.A. and Dhaliwal, A. Infections in pregnancy with COVID-19 and other respiratory RNA virus diseases are rarely, if ever, transmitted to the fetus: experiences with coronaviruses, parainfluenza, metapneumovirus respiratory syncytial virus, and influenza. *Archives of Pathology & Laboratory Medicine*, 144(8), 920-928, (2020). [[CrossRef](#)]
- [18] Naim, M., Sabbar, Y. and Zeb, A. Stability characterization of a fractional-order viral system with the non-cytolytic immune assumption. *Mathematical Modelling and Numerical Simulation with Applications*, 2(3), 164-176, (2022). [[CrossRef](#)]
- [19] Kiouach, D. and Sabbar, Y. The threshold of a stochastic SIQR epidemic model with Levy jumps. In *Trends in Biomathematics: Mathematical Modeling for Health, Harvesting, and Population Dynamics* (pp. 87-105). Cham: Springer, (2019). [[CrossRef](#)]
- [20] Zhang, X.B., Huo, H.F., Xiang, H., Shi, Q. and Li, D. The threshold of a stochastic SIQS epidemic model. *Physica A: Statistical Mechanics and Its Applications*, 482, 362-374, (2017). [[CrossRef](#)]
- [21] Liu, Q. and Jiang, D. The dynamics of a stochastic vaccinated tuberculosis model with treatment. *Physica A: Statistical Mechanics and its Applications*, 527, 121274, (2019). [[CrossRef](#)]
- [22] Cai, Y., Kang, Y. and Wang, W. A stochastic sirs epidemic model with nonlinear incidence rate. *Applied Mathematics and Computation*, 305, 221-240, (2017). [[CrossRef](#)]
- [23] Kiouach, D. and Sabbar, Y. Threshold analysis of the stochastic Hepatitis B epidemic model with successful vaccination and Levy jumps. In *IEEE 2019 4th World Conference on Complex Systems (WCCS)*, pp. 1-6, Ouarzazate, Morocco, (2019, April). [[CrossRef](#)]
- [24] Sabbar, Y., Din, A. and Kiouach, D. Influence of fractal–fractional differentiation and independent quadratic Lévy jumps on the dynamics of a general epidemic model with vaccination

strategy. *Chaos, Solitons & Fractals*, 171, 113434, (2023). [[CrossRef](#)]

[25] Zhao, Y. and Jiang, D. The threshold of a stochastic SIS epidemic model with vaccination. *Applied Mathematics and Computation*, 243, 718-727, (2014). [[CrossRef](#)]

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