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Research article

Stochastic analysis for measles transmission with Lévy noise: a case study

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Abstract: In this paper, we deal with a Lévy noise-driven epidemic model reflecting the dynamics of measles infection subject to the effect of vaccination. After model formulation, the feasibility of the system was studied by using the underlying existence and uniqueness theory. Moreover, we discussed the behavior of solution around the infection-free and disease-present steady states. To check the persistence and extinction of the infection, we calculated the threshold parameter $\mathbf{R_s}$ and it was determined that the disease vanishes whenever $\mathbf{R_s} < 1$. From January to October 2019, the reported measles cases in Pakistan wear used and the model was fitted against this data by using the well-known fitting techniques. The values of the parameter were estimated and future behavior of the infection was predicted by simulating the model. The model was further simulated and theoretical findings of the study were validated.

Keywords: white noise; Lévy jump; Lyapunov function; persistence; parameter estimation; positive recurrence
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1. Introduction

Measles is one of the major global public health concerns and most importantly in underdeveloped countries. Measles is a childhood disease brought on by a virus from the family of paramyxovirus that is typically spread through close interaction as well as by air [1, 2]. The virus enters the body through the nose and throat and then moves around the body. Rubella is a human pathogen that has never been observed in animals. Measles, also known as rubeola, is easily transmitted and is potentially severe, if not fatal, in young children. Before the proper implementation of the measles vaccine, about 2.6 million people died due to this disease. Regardless of the wide spread availability of reliable and efficient vaccines, approximately 140,000 lives were lost from measles in 2018, the

majority of whom were children under the age of five [3]. Further, as reported by the World Health Organisation (WHO), approximately 110, 000 lives were lost due to rubella in 2017, primarily children under the age of six [4]. Rubella health problems can include diarrhea and vomiting, pneumonia, pregnancy complications, and ear infection [5]. Sneezing and coughing, interaction with aerosol or nasal secretions, or close contact are all ways to spread rubella. The virus is highly contagious for the first two hours on surface and in the air. The initial symptoms of the virus are: blurry vision, runny nose, cough, highly sore throat and white spots inside the mouth. Generally, the symptoms remain active for 10–12 days. Later, a rash appears, expanding outward out from the nose. The shedding of the virus has the greatest infection phase, lasting four days before and after the appearance of the rashes. Commonly, the virus takes 10 to 14 days to spread in the whole body and during this stage, the body shows no symptoms of the virus [6]. In real situations, some individuals having been vaccinated are also sensitive whenever the vaccination fails or their immunization from the vaccine wears off. From 2000 to 2018, the global deaths due to rubella decreased by 73% due to available vaccines [7, 8]. Rubella remains widespread continually throughout various nations, particularly in Africa and Asia. Statistics suggest that mass vaccination particularly and vaccination in general are the most effective and efficient strategy for decreasing the severity and death rate of an epidemic. For example, the measles vaccines save the lives of more than 3 millions individuals each year in the Nigeria. The measels virus is still endemic in Nigeria and re-emergence of the infection occurs at regular intervals. The disease is present all the time and in seasons in Nigeria. The vaccine boosts an individual's natural immunity and thus, lowers the probability of getting the infection [9]. The most common and commercially available vaccine for measles is measles, mumps, and rubella (MMR) vaccine. This vaccine is considered to be highly safe and effective for both adults and children. A single MMR dose is about 92% effective in controlling of measles, while two dose of the vaccines is 95% effective. The MMR vaccine is also helpful in preventing other diseases like mumps [10].

In the WHO Asia Pacific countries, Pakistan is one of the countries facing a high number of measles infections [11]. With a regular interval of 8 to 10 years, the disease shows emergence and re-emergence. It was found that 2845 cases of measles were diagnosed in Pakistan in the year 2016. The statistics further showing that these figures approached 6791 and 33,007 respectively in 2017 and 2018. In the region of Eastern Mediterranean which contain 22 countries, these Pakistani reported cases contribute to for approximately 44%, 20% and 51% of the entire reported cases in the region. In 2017, approximately 130 children died due to measles and this number raised to above 300 in 2018 [12].

Literature suggests that the use of mathematical modeling tools is strongly recommended for examining the transmission mechanism and management of an epidemic [8, 12–14]. Mathematical models can achieve a balance between accuracy in depicting the history of an infection and the strength of their connection to data by incorporating biological realism. Mathematical models of measles, at both outbreak and population levels, have exposed a diverse array of disease dynamics. Environmental noise is a crucial factor in physical systems and biological processes, and it is likewise an important role in the transmission dynamics of measles [15]. The development and spread of an epidemic are inherently unpredictable due to the variability of person-to-person interactions and other population features. As a result, the epidemic's state is influenced by the unpredictable and diverse nature of the environment.

In stochastic studies, Lévy noise is significant as it enters the space of different threshold parameters through drifting velocities. The inclusion of Lévy noise in stochastic models yields better results or

more mutual information for various infectious diseases. This improvement was derived by applying the Lipschitz condition to the epidemic model. Compared to Gaussian noise, Lévy noise is superior in epidemiological modeling because it introduces ambiguity into the model [16–19]. The jumping diffusion problem associated with Lévy noise is preferable to the generalized Lévy model in terms of the efficiency evolution of neurons walls. Therefore, we can conclude that Lévy noise is more advantageous for time-varying stability.

The work in this research paper is structured as: We introduce a stochastic system that governs the mechanism of transmission dynamics of the epidemic measles in Section 2. In Section 3, we calculate the stochastic reproductive quantity of the epidemic problem and identify its equilibrium points. Section 4 establishes the existence as well as the uniqueness analysis of the model solution with positive global properties. Sections 5 and 6 then provide the conditions of persistence and extinction for the stochastic model that is under consideration. To further support our proposed model, we optimize it with real data reported in Pakistan from January to October of 2019, which is presented in Section 7. Theoretical results are quantitatively validated and graphically illustrated in Section 8. In Section 9, we summarize the study's results and put forward a road map for the future work.

2. Model description

In a recent study by Kuddus et al. [20], a compartmental model for measles transmission dynamics was developed. The model considers various compartments that are mutually exclusive, including susceptible population denoted by ($\mathbb{S}$) which are not yet infected but are at risk. Those having taken only the first dose of the vaccine are symbolized by ($\mathbb{V}_1$), while those having taken the second dose as well are denoted by ($\mathbb{V}_2$). The individuals which are infected but have no symptoms are exposed individuals and accordingly denoted by ($\mathbb{E}$). Moreover, the individuals that are infectious and spread the disease to other are described by ($\mathbb{I}$), and ($\mathbb{R}$) is assumed to represent the recovered individuals, who become healthy after getting infected. People in the recovery phase of the disease are no longer susceptible to it and cannot transmit it to others, and may include individuals who are undergoing treatment, in isolation, no longer in contact with others, or deceased.

The entire population symbolized by $\mathbb{N}(t)$ is presumed to be well-mixed and constant, that is,

$$\mathbb{N}(t) = \mathbb{R}(t) + \mathbb{E}(t) + \mathbb{S}(t) + \mathbb{V}_1(t) + \mathbb{V}_2(t) + \mathbb{I}(t).$$

In order to maintain a constant size of population, we consider total deaths to be replaced by newborns in susceptible individuals group. This contains natural deaths, which occur in every group at a fixed rate denoted by μ, as well as measles related death given by δ. When individuals in the susceptible group got the first vaccine dose, they will move to vaccinated group of individuals at the rate τ. We also assume that individuals which have taken only first dose of vaccine ($\mathbb{V}_1$) may lose the immunity and return to the susceptible group of individuals at the rate ρ, and the remaining portion of the individuals moves to the two-dose vaccinated group of individuals ($\mathbb{V}_2$) at a rate of σ. Individuals in the group of two-dose vaccinated group move to the recovery class at ω rate. Further, let as assume that if β is the disease transmission rate, then the susceptible group $\mathbb{S}$ gets the infection of measles virus at $\lambda = \beta\mathbb{S}\mathbb{I}$. After getting infected, the susceptible individuals then move to the exposed class ($\mathbb{E}$). After getting exposed to the disease, a fraction of the exposed individuals become infected and move to the infected group of individuals at the rate α. The rate at which individuals of the infected group move to the

recovery class because of the treatment as well as due to natural recovery is denoted by the parameter γ. The schematic process of the disease is illustrated in Figure 1.

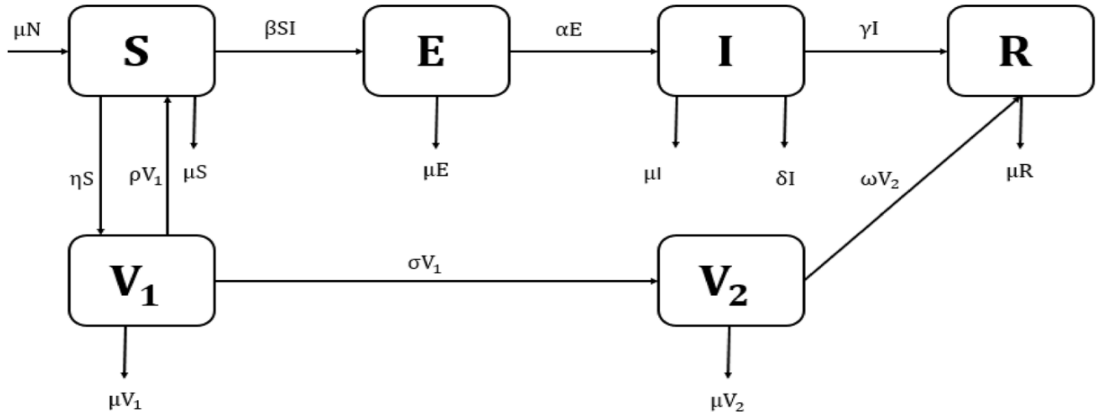


Figure 1. Schematic diagram of measles model (2.1) [21].

Thus, the above assumption as well as the schematic process of the disease leads to the dynamical system which can be described by a system of nonlinear differential equations, which include the previously mentioned components:

$$\begin{aligned}
\frac{d\mathbb{S}(t)}{t} &= \phi - (\mu+\tau)\mathbb{S}(t) - \beta\mathbb{S}(t)\mathbb{I}(t) + \rho\mathbb{V}_1(t),\\
\frac{d\mathbb{V}_1(t)}{t} &= \tau\mathbb{S}(t) - (\rho+\sigma+\mu)\mathbb{V}_1(t),\\
\frac{d\mathbb{V}_2(t)}{t} &= \sigma\mathbb{V}_1(t) - (\omega+\mu)\mathbb{V}_2(t),\\
\frac{d\mathbb{E}(t)}{t} &= \beta\mathbb{I}(t)\mathbb{S}(t) - (\mu+\alpha)\mathbb{E}(t),\\
\frac{d\mathbb{I}(t)}{t} &= \alpha\mathbb{E}(t) - (\delta+\mu+\gamma)\mathbb{I}(t),\\
\frac{d\mathbb{R}(t)}{t} &= \gamma\mathbb{I}(t) - \mu\mathbb{R}(t) + \omega\mathbb{V}_2(t).
\end{aligned} \tag{2.1}$$

The main aim of the study is to modify problem (2.1) by incorporating a perturbed term with Lévy noises, taking into account the effect of non-linear incidence. We use two noises: white noises and Lévy noises, which areused for the continuity and jump sections respectively. This modification results in a stochastic version of the originally idealistic problem (2.1) which can be expressed as follows:

$$\begin{aligned}
d\mathbb{S} &= \left[\phi - \frac{\beta\mathbb{S}(t)\mathbb{I}(t)}{\mathbb{N}(t)} + \omega\mathbb{V}(t) - (\tau+\mu)\mathbb{S}(t)\right]dt + \eta_1\mathbb{S}(t)d\mathbb{B}_1(t) + \int_Y \mathbb{X}_1(u)\mathbb{S}\left(t^-\right)\tilde{\mathbb{N}}(dt,du),\\
d\mathbb{V}_1 &= \left[\tau\mathbb{S}(t) - (\rho+\sigma+\mu)\mathbb{V}_1(t)\right]dt + \eta_2\mathbb{V}_1(t)d\mathbb{B}_2(t) + \int_Y \mathbb{X}_2(u)\mathbb{V}_1\left(t^-\right)\tilde{\mathbb{N}}(dt,du),\\
d\mathbb{V}_2 &= \left[-(\omega+\mu)\mathbb{V}_2(t) + \sigma\mathbb{V}_1(t)\right]dt + \int_Y \mathbb{X}_3(u)\mathbb{V}_2(t)\left(t^-\right)\tilde{\mathbb{N}}(dt,du) + \mathbb{V}_2\eta_3 d\mathbb{B}_2(t),\\
d\mathbb{E} &= \left[\frac{\beta\mathbb{S}(t)\mathbb{I}(t)}{\mathbb{N}(t)} - (\alpha+\mu)\mathbb{E}(t)\right]dt + \eta_4\mathbb{E}(t)d\mathbb{B}_4(t) + \int_Y \mathbb{X}_4(u)\mathbb{E}\left(t^-\right)\tilde{\mathbb{N}}(dt,du),
\end{aligned}$$

$$d\mathbb{I} = \Big[\alpha\mathbb{E}(t) - (\gamma + \delta + \mu)\mathbb{I}(t)\Big]dt + \eta_5\mathbb{I}(t)d\mathbb{B}_5(t) + \int_Y \mathbb{X}_5(u)\mathbb{I}\left(t^-\right)\tilde{\mathbb{N}}(dt, du),$$
$$d\mathbb{R} = \Big[\gamma\mathbb{I}(t) + \omega\mathbb{V}_2(t) - \mu\mathbb{R}(t)\Big]dt + \eta_6\mathbb{R}(t)d\mathbb{B}_6(t) + \int_Y \mathbb{X}_6(u)\mathbb{R}\left(t^-\right)\tilde{\mathbb{N}}(dt, du). \tag{2.2}$$

Here $\mathbb{B}_1(t), \mathbb{B}_2(2), \mathbb{B}_3(t), \mathbb{B}_4(t), \mathbb{B}_5(t)$ and $\mathbb{B}_6(t)$ mathematically represent Brownian motions and $\eta_1, \eta_2, \eta_3, \eta_4, \eta_5$ and η_6 stand for the intensity of the noises. The Brownian motions are subject to the conditions $\mathbb{B}_i(0) = 0$ for $i = 1, 2, 3 \ldots, 6$. The function $M\left(t^-\right)$ denotes the left limit of $M(t)$. The compensation stochastic measure is denoted by $\tilde{\mathbb{N}}$ can be expressed as $\tilde{\mathbb{N}} = \mathbb{N}(du, dt) - \nu(du)dt$ and $\mathbb{N}(du, dt)$ is a counting Poisson's measure with the stationary compensatory $\nu(dy)dt.\nu$ defind on a measurable sub-set Q of $[0, +\infty)$ with $\nu(\mathrm{Q}) < \infty$ and $\mathbb{X}_i > -1, i = 1, 2, 3, 4, 5, 6$.

Keeping in view model (2.2), our focus is on addressing the following inquiries:

Q_1 : Does the inclusion of Lévy noises impact the dynamics of the measles disease?

Q_2 : How does a contaminated vaccination contribute to the spread of the measles disease?

Q_3 : What criteria can be used to determine the existence of extinction in the model?

Q_4 : What criteria can be used to determine the existence of persistence in the model?

3. Stability analysis

Generally, a dynamical system that governs the dynamics of an epidemic exhibits two types of equilibrium solutions: Disease-free, which is derived if the basic reproduction quantity is less than unity ($\mathbf{R_0^D} < 1$), and the endemic solution is calculated if the reproductive quantity is greater than unity ($\mathbf{R_0^D} > 1$). These will be discussed below in sequence.

3.1. The disease-free state $\left(\mathbf{X}^0\right)$

We derive the disease-free equilibrium (DFE) of the epidemic problem as reported by system (2.1) where the disease epidemic is eradicated by setting $\mathbb{E} = 0 = \mathbb{I}$. Therefore, the disease-free state takes the form:

$$\begin{aligned}\mathrm{X}^0 &= \left(\mathbb{S}^0, \mathbb{V}_1^0, \mathbb{V}_2^0, 0, 0\right)\\ &= \left(\frac{(\rho+\mu+\sigma)\phi}{(\rho+\mu+\sigma)(\mu+\eta)-\eta\rho}, \frac{\eta\phi}{(\rho+\mu+\sigma)(\mu+\eta)-\eta\rho}, \frac{\sigma\eta\phi}{(\mu+\omega)((\sigma+\rho+\mu)(\eta+\mu)-\rho\eta)}, 0, 0\right).\end{aligned}$$

This equilibrium state denotes a scenario where there are no infections within the population, and the whole populace size $\mathbb{N}$ remains constant at $t = 0$.

3.2. Basic reproductive number $\mathbf{R_0^D}$

The method of next-generation matrix can be applied to determine this quantity. This matrix is obtained by multiplying matrices T and $-\Sigma^{-1}$, where T denotes the rate of infection transmission in groups $\mathbb{E}$ and $\mathbb{I}$, and Σ is the rate of all other transfers between epidemiological groups of the model. These matrices are expressed as:

$$\mathrm{T} = \begin{pmatrix} 0 & \beta S^0 \\ 0 & 0 \end{pmatrix} \text{ and } \Sigma = \begin{pmatrix} -(\alpha+\mu) & 0 \\ \alpha & -(\gamma+\delta+\mu) \end{pmatrix}.$$

The next-generation matrix is

$$\mathrm{K} = \mathrm{T} \times \left(-\Sigma^{-1}\right) = \begin{pmatrix} 0 & \beta S^0 \\ 0 & 0 \end{pmatrix} \times \begin{pmatrix} \frac{1}{(\alpha+\mu)} & 0 \\ \frac{\alpha}{(\mu+\alpha)(\delta+\gamma+\mu)} & \frac{1}{(\delta+\gamma+\mu)} \end{pmatrix} = \begin{pmatrix} \frac{\beta \mathbb{S}^0 \alpha}{(\mu+\alpha)(\delta+\gamma+\mu)} & \frac{\beta \mathbb{S}^0}{(\delta+\gamma+\mu)} \\ 0 & 0 \end{pmatrix}.$$

We compute the largest eigenvalue of the next-generation matrix (K), which can be obtained using the matrices T and $-\Sigma^{-1}$ described above. Thus

$$\mathbf{R_0^D} = \frac{\beta S^0 \alpha}{(\alpha+\mu)(\delta+\gamma+\mu)} = \frac{(\rho+\mu+\sigma)\alpha\beta\phi}{(\mu+\alpha)(\delta+\gamma+\mu)((\rho+\mu+\sigma)(\mu+\eta)-\eta\rho)},$$

is the basic reproductive quantity for the model that is under consideration.

3.3. Endemic equilibrium point (X^*)

By assuming the non-trivial infected groups, we can find the endemic state (EE) of the epidemiological model (2.1). Therefore, we assume that $\mathrm{X}^* = \left(\mathbb{S}^*, \mathbb{V}_1^*, \mathbb{V}_2^*, \mathbb{E}^*, \mathbb{I}^*\right)$ is the endemic state then

$$\begin{cases} \mathbb{S}^* = \frac{(\mu+\alpha)(\delta+\gamma+\mu)}{\alpha\beta}, \\ \mathbb{V}_1^* = \frac{(\alpha+\mu)(\gamma+\delta+\mu)\eta}{\alpha\beta(\rho+\delta+\mu)}, \\ \mathbb{V}_2^* = \frac{\sigma\eta(\alpha+\mu)(\gamma+\delta+\mu)}{\alpha\beta(\omega+\mu)(\rho+\delta+\mu)}, \\ \mathbb{E}^* = \frac{(\alpha+\mu)(\gamma+\delta+\mu)^2((\eta+\mu)(\rho+\sigma+\mu)-\rho\eta)(\mathrm{R}_0-1)}{((\mu+\alpha)(\gamma+\mu+\delta))\alpha(\rho+\sigma+\mu)}, \\ \mathbb{I}^* = \frac{(\alpha+\mu)(\mathrm{R}_0-1)(\gamma+\mu+\delta)((\mu+\eta)(\rho+\sigma+\mu)-\rho\eta)}{((\alpha+\mu)(\delta+\gamma+\mu))(\sigma+\rho+\mu)}. \end{cases}$$

It is easy to show that for $\mathbf{R_0^D}$, the endemic state X^* is stable.

4. Existence and uniqueness of positive solution

In this part of the study, we intend to explore the existence of a solution to the model, particularly proving the global existence and positivity of the solution by following the techniques of [16]. We assume that the conditions ($\mathbf{C_1}$) and ($\mathbf{C_2}$) are sufficient for the proof of the existence and uniqueness of a global non-negative solution for the system (2.2).

($\mathbf{C_1}$) To each $\mathbb{Q} > 0 \; \exists \; \mathbb{L}_{\mathbb{Q}} > 0$,

$$\int_Y |A_i(x_1, u) - A_i(x_2, u)|^2 \, v(du) \leq \mathbb{L}_{\mathbb{Q}} |x_1 - x_2|^2, \, i = 1, 2, 3, 4, 5, 6$$

for $|y_1| \vee |y_2| \leq \mathbb{M}$ here

$$\begin{aligned} A_1(x,u) &= \mathbb{X}_1(u)x \text{ at } \; x = \mathbb{S}(t^-), \\ A_2(x,u) &= \mathbb{X}_2(u)x \text{ at } \; x = \mathbb{V}_1(t^-), \\ A_3(x,u) &= \mathbb{X}_3(u)x \text{ at } \; x = \mathbb{V}_2(t^-), \\ A_4(x,u) &= \mathbb{X}_4(u)x \text{ at } \; x = \mathbb{E}(t^-), \\ A_5(x,u) &= \mathbb{X}_5(u)x \text{ at } \; x = \mathbb{I}(t^-), \\ A_6(x,u) &= \mathbb{X}_6(u)x \text{ at } \; x = \mathbb{R}(t^-). \end{aligned}$$

($\mathbf{C_2}$) $C \geq \left|\log\left(\mathbb{X}_i(x)\right)\right|$ for $-1 < \mathbb{X}_i(x), \quad i = 1, \cdots, 6$, where the constant C appearing in the expression is positive.

Theorem 1. *Subject to an initial condition* $(\mathbb{S}(0), \mathbb{V}_1(0), \mathbb{V}_2(0), \mathbb{E}(0), \mathbb{I}(0), \mathbb{R}(0)) \in \mathbb{R}^6_+$*, system (2.2) exhibits a nonlocal and unique solution given by* $(\mathbb{S}(t), \mathbb{V}_1(t), \mathbb{V}_2(t), \mathbb{E}(t), \mathbb{I}(t), \mathbb{R}(t))$ *and such solution remains in* $\mathbb{R}^6_+$ *for all t greater than zero a.s.*

Proof. Referring $(\mathbb{C}_\nvDash)$, one can easily verify that the terms of the diffusion are Lipschitz locally. Thus, the underlying problem has a unique solution $(\mathbb{S}, \mathbb{V}_1, \mathbb{V}_2, \mathbb{E}, \mathbb{I}, \mathbb{R})$ of the local nature over the interval $[0, \tau_e)$ and for a given initial values of the population $(\mathbb{S}(0), \mathbb{V}_1(0), \mathbb{V}_2(0), \mathbb{E}(0), \mathbb{I}(0), \mathbb{R}(0))$ from the space $\mathbb{R}^6_+$, here the notion τ_e physically describes the expulsion time. The next step is prove that this solution is global actually and, for this, one needs to prove that a.s. $\tau_e = \infty$. Initially, we must show that the solution does not become unbounded within finite time. Let us consider a large enough positive real number k_0 such that the solution to the problem is in the interval $[\frac{1}{k_0}, k_0]$. The stopping time is defined by

$$\tau_k = \inf\left\{t \in [0, \tau_e) / (\mathbb{S}(t), \mathbb{V}_1(t), \mathbb{V}_2(t), \mathbb{E}(t), \mathbb{I}(t), \mathbb{R}(t)) \notin \left(\frac{1}{k}, k\right)\right\}, \tag{4.1}$$

for all $k \leq k_0$.

Let us assume that the $\inf \emptyset = \infty$ and $\tau^+ \leq \tau_e$. Thus, if we prove that τ^+ approaches ∞ a.s, that means $\tau_e \to \infty$. On the contrary, let τ^+ be less than ∞, then there exist surely a positive T such that $0 < \mathbb{P}(\tau^+ < T)$.

Define the following Lyapunov function

$$\begin{aligned}\mathbb{F}(\mathbb{S}, \mathbb{V}_1, \mathbb{V}_2, \mathbb{E}, \mathbb{I}, \mathbb{R}) = \mathbb{S} + \mathbb{V}_1 + \mathbb{V}_2 + \mathbb{E} + \mathbb{I} + \mathbb{R} - 6 \\ - (\log\mathbb{S} + \log\mathbb{V}_1 + \log\mathbb{V}_2 + \log\mathbb{E} + \log\mathbb{I} + \log R).\end{aligned} \tag{4.2}$$

By applying the well-known *Itô* formula to the function F on over the interval $t \in [0, \tau^+]$, we have

$$\begin{aligned}d\mathbb{F} = L\mathbb{F}(\mathbb{S}, \mathbb{V}_1, \mathbb{V}_2, \mathbb{E}, \mathbb{I}, \mathbb{R})dt + \eta_1(\mathbb{S} - 1)d\mathbb{B}_1(t) + \eta_2(\mathbb{V}_1 - 1)d\mathbb{B}_2(t) + \eta_3(\mathbb{V}_2 - 1)d\mathbb{B}_3(t) \\ + \eta_4(\mathbb{E} - 1)d\mathbb{B}_4(t) + \eta_5(\mathbb{I} - 1)d\mathbb{B}_5(t) + \eta_6(\mathbb{R} - 1)d\mathbb{B}_6(t) \\ + \int_\Upsilon \left[\mathbb{X}_1(u)\mathbb{S} - log\left(\mathbb{X}_1(u) + 1\right)\right]\tilde{\mathbb{N}}(dt, du) + \int_\Upsilon \left[\mathbb{X}_2(u)\mathbb{V}_1 - log\left(1 + \mathbb{X}_2(u)\right)\right]\tilde{\mathbb{N}}(dt, du) \\ + \int_\Upsilon \left[\mathbb{X}_4(u)\mathbb{E} - \log\left(1 + \mathbb{X}_4(u)\right)\right]\tilde{\mathbb{N}}(dt, du) + \int_\Upsilon \left[\mathbb{X}_5(u)\mathbb{I} - \log\left(1 + \mathbb{X}_5(u)\right)\right]\tilde{N}(dt, du) \\ + \int_\Upsilon \left[\mathbb{X}_3(u)\mathbb{V}_2 - \log\left(1 + \mathbb{X}_3(y)\right)\right]\tilde{\mathbb{N}}(dt, du) + \int_\Upsilon \left[\mathbb{X}_6(u)\mathbb{R} - \log\left(1 + \mathbb{X}_6(u)\right)\right]\tilde{\mathbb{N}}(dt, du).\end{aligned} \tag{4.3}$$

In Eq (4.3), $L\mathbb{F} : \mathbb{R}^6_+ \to \mathbb{R}_+$ is represented with the help of condition $(\mathbb{C}_\nvDash)$, thus

$$\begin{aligned}L\mathbb{F} \leq \phi + \rho + \sigma + \tau + \omega + \alpha + \gamma + \delta + 6\mu + \frac{\eta_1^2 + \eta_2^2 + \eta_3^2 + \eta_4^2 + \eta_5^2 + \eta_6^2}{2} \\ + \int_Y \left[\mathbb{X}_1(u) - \log\left(1 + \mathbb{X}_1(u)\right)\right]\nu(du) + \int_Y \left[\mathbb{X}_2(u) - \log\left(1 + \mathbb{X}_2(u)\right)\right]\nu(du) \\ + \int_Y \left[\mathbb{X}_3(u) - \log\left(1 + \mathbb{X}_3(u)\right)\right]\nu(du) + \int_Y \left[\mathbb{X}_4(u) - \log\left(1 + \mathbb{X}_4(u)\right)\right]\nu(du) \\ + \int_Y \left[\mathbb{X}_5(u) - \log\left(1 + \mathbb{X}_5(u)\right)\right]\nu(du) + \int_Y \left[\mathbb{X}_6(u) - \log\left(1 + \mathbb{X}_6(u)\right)\right]\nu(du).\end{aligned} \tag{4.4}$$

The remainIng steps are very much similar to the steps provided in the proof of Theorem 2.1 in Fatin et al. [16] and therefore, we must skip these steps and hence the result.

5. Extinction for system (2.2)

In order to mitigate the impact of a disease on a society over time, certain conditions must be met, which can be determined by studying the dynamic behavior of the epidemic. In this part of the study, we will explore the necessary criteria under which the infection could be eradicated, using a stochastic modeling approach. First, we provide the threshold parameters for the Ordinary differential equation (ODE) system (2.2), which are given below:

$$\mathbf{R_0^D} = \frac{\alpha\beta\phi(\rho+\sigma+\mu)}{(\mu+\alpha)(\delta+\gamma+\mu)((\eta+\mu)(\rho+\mu+\sigma)-\eta\rho)}. \tag{5.1}$$

The threshold for the stochastic approach can also be determined by analyzing the dynamic behavior of the stochastic system with jumps given by Eq (2.2). The stochastic version of the model allows us to calculate the threshold parameter, denoted by $\mathbf{R_s}$, which is given by:

$$\mathbf{R_s} = \frac{\alpha}{\left[(\mu+\alpha)(\mu+\delta+\gamma)+\frac{\eta_4^2}{2}+\frac{\eta_5^2}{2}+\int_y\{(\mathbb{X}_4(y)+\mathbb{X}_5(y))-\ln(1+(\mathbb{X}_4(t)+\mathbb{X}_5(y)))\}\,\nu(du)\right]}. \tag{5.2}$$

Before moving further, let us describe the following notion $\langle\mathbb{X}(t)\rangle = \frac{1}{t}\int_0^t \mathbb{X}(s)ds$.

Lemma 1. *(Strong law) [21, 22] If $\mathbb{Q} = \{\mathbb{Q}\}_{0\le t}$ be a real valued continuous local martingale vanishing at $t \to 0$, then*

$$\begin{aligned}
&\lim_{t\to\infty}\langle\mathbb{Q},\mathbb{Q}\rangle_t = \infty,\ a.s.,\ \Rightarrow\ \lim_{t\to\infty}\frac{\mathbb{Q}_t}{\langle\mathbb{Q},\mathbb{Q}\rangle_t} = 0,\ a.s.\\
&\limsup_{t\to\infty}\frac{\langle\mathbb{Q},\mathbb{Q}\rangle_t}{t} < 0,\ a.s.,\ \Rightarrow\ \lim_{t\to\infty}\frac{\mathbb{Q}_t}{t} = 0,\ a.s.
\end{aligned} \tag{5.3}$$

Theorem 2. *Consider $(\mathbb{S}(t),\mathbb{V}_1(t),\mathbb{V}_2(t),\mathbb{E}(t),\mathbb{I}(t),\mathbb{R}(t))$ to be the required solution to the problem (2.2) having the intial conditions $(\mathbb{S}(0),\mathbb{V}_1(0),\mathbb{V}_2(0),\mathbb{E}(0),\mathbb{I}(0),\mathbb{R}(0)) \in \mathbb{R}^6$. Further, if $q > \frac{(\eta_1^2\vee\eta_2^2\vee\eta_3^2\vee\eta_4^2\vee\eta_5^2\vee\eta_6^2)}{2}$ and $\mathbf{R_s} < 1$, then*

$$\lim_{t\to\infty}\frac{\log\langle\mathbb{E}(t)\rangle}{t} < 0, \quad \textit{and} \quad \lim_{t\to\infty}\frac{\log\langle\mathbb{I}(t)\rangle}{t} < 0, a.s.$$

This means that the infected groups $\mathbb{E}(t)$ and $\mathbb{I}(t)$ will exponentially approach to 0 over time a.s. and ultimately, the infection will leave the population with unit probability. Moreover,

$$\begin{aligned}
\lim_{t\to\infty}\langle\mathbb{S}(t)\rangle &= \frac{\phi(\rho+\sigma+\mu)}{(\mu+\eta)(+\mu+\sigma\rho)-\eta\rho},\\
\lim_{t\to\infty}\langle\mathbb{V}_1(t)\rangle &= \frac{\phi\eta}{(\mu+\eta)(+\mu+\sigma\rho)-\eta\rho},\\
\lim_{t\to\infty}\langle\mathbb{V}_2(t)\rangle &= \frac{\eta\sigma\phi}{(+\mu+\omega)((\mu+\eta)(+\mu+\sigma\rho)-\eta\rho)},\\
\lim_{t\to\infty}\langle\mathbb{E}(t)\rangle &= 0,\\
\lim_{t\to\infty}\langle\mathbb{I}(t)\rangle &= 0,\\
\lim_{t\to\infty}\langle\mathbb{R}(t)\rangle &= 0.
\end{aligned} \tag{5.4}$$

Proof. Let $(\mathbb{S}(t), \mathbb{V}_1(t), \mathbb{V}_2(t), \mathbb{E}(t), \mathbb{I}(t), \mathbb{R}(t))$ subject to the initial conditions

$$(\mathbb{S}(0), \mathbb{V}_1(0), \mathbb{V}_2(0), \mathbb{E}(0), \mathbb{I}(0), \mathbb{R}(0)) \in \mathbb{R}^6_+$$

be a solution to model (2.2). We have

$$H_1(t) = \alpha\mathbb{E}(t) + (\alpha + \mu)\mathbb{I}(t). \tag{5.5}$$

Differentiating relation Eq (5.5) with the help of Ito's formula, gives us

$$\begin{aligned}
d(lnH_1(t)) &= \frac{1}{H_1}\left[\frac{\alpha\beta\mathbb{S}\mathbb{I}}{\mathbb{N}} - (+\mu+\alpha)(+\delta+\mu+\gamma)\mathbb{I}\right] - \frac{\alpha^2\mathbb{E}^2\eta_4^2 + (+\mu+\alpha)^2\eta_5^2\mathbb{I}^2}{2(H_1)^2} \\
&+ \frac{\alpha\eta_4}{[\alpha\mathbb{E}(t) + (\alpha+\mu)\mathbb{I}]}\mathbb{E}d\mathbb{B}_4(t) + \frac{(\alpha+\mu)\eta_5}{[\mathbb{E} + (\alpha+\mu)\mathbb{I}]}\mathbb{I}d\mathbb{B}_5(t) \\
&+ \int_y \left\{\ln\left(1 + \frac{\beta\mathbb{X}_4(t)\mathbb{E} + (\alpha+\mu)\mathbb{X}_5(u)\mathbb{I}}{H_1}\right) - \frac{\beta\mathbb{X}_4(y)\mathbb{E} + (\alpha+\mu)\mathbb{X}_5(u)\mathbb{I}}{H_1}\right\}\nu(du) \\
&+ \int_y \ln\left(1 + \frac{\beta\mathbb{X}_5(u)\mathbb{E}(t^-) + (\mu+\beta)\mathbb{X}_5\mathbb{I}(t^-)}{H_1(t^-)}\right)\tilde{\mathbb{N}}(dt, du) \\
&\leq \frac{1}{H_1}\left[\alpha\beta\mathbb{I} - (\mu+\alpha)(\gamma+\delta+\mu)\mathbb{I}\right] - \frac{\alpha^2\mathbb{E}^2\eta_4^2}{2(H_1)^2} - \frac{(\mu+\alpha)^2\eta_5^2\mathbb{I}^2}{2(H_1)^2} \\
&- \int_y \left\{\frac{\beta\mathbb{X}_4(u)\mathbb{E} + (\mu+\alpha)\mathbb{X}_5(u)\mathbb{I}}{H_1} - \ln\left(1 + \frac{\alpha\mathbb{X}_4(t)\mathbb{E} + (\mu+\alpha)\mathbb{X}_5(u)\mathbb{I}}{H_1}\right)\right\}\nu(du) \\
&+ \frac{\alpha\eta_4}{[\alpha\mathbb{E}(t) + (\mu+\alpha)\mathbb{I}]}\mathbb{E}d\mathbb{B}4(t) + \frac{(\mu+\alpha)\eta_5}{[\mathbb{E} + (\mu+\alpha)\mathbb{I}]}\mathbb{I}d\mathbb{B}_5(t) \\
&+ \int_y \ln\left(1 + \frac{\alpha\mathbb{X}_4(u)\mathbb{E}(t^-) + (\mu+\alpha)\mathbb{X}_5\mathbb{I}(t^-)}{H_1(t^-)}\right)\tilde{\mathbb{N}}(dt, du), \quad [\because \mathbb{S} \leq \mathbb{N}] \\
&\leq \frac{1}{(+\alpha+\mu)}\left[\beta - (+\mu+\alpha)(+\mu+\delta+\gamma)\right] - \frac{\eta_4^2}{2} - \frac{\eta_5^2}{2} \\
&- \int_y \{(\mathbb{X}_4(u) + \mathbb{X}_5(5)) - \ln(1 + (\mathbb{X}_4(t) + \mathbb{X}_5(u)))\}\nu(du) \\
&+ \frac{\alpha\eta_4}{[\alpha\mathbb{E}(t) + (\mu+\alpha)\mathbb{I}]}\mathbb{E}d\mathbb{B}_4(t) + \frac{(\mu+\alpha)\eta_5}{[\mathbb{E} + (\mu+\beta)\mathbb{I}]}\mathbb{I}d\mathbb{B}_4(t) \\
&+ \int_y \ln\left(1 + \frac{\alpha\mathbb{X}_4(u)\mathbb{E}(t^-) + (\mu+\alpha)\mathbb{X}_5\mathbb{I}(t^-)}{H_1(t^-)}\right)\tilde{\mathbb{N}}(dt, du), \quad [\because \mathbb{I} \leq \mathbb{I} + \frac{\alpha\mathbb{E}}{(\mu+\alpha)}].
\end{aligned} \tag{5.6}$$

By performing integration on both sides of the last inequality over the interval from 0 to t, we obtain:

$$\begin{aligned}
lnH_1(t) &\leq \frac{1}{(\mu+\alpha)}\left\{\beta - \left[(+\alpha+\mu)(\gamma+\delta+\mu) + \frac{\eta_4^2}{2} + \frac{\eta_5^2}{2}\right.\right. \\
&\left.\left. + \int_y \{(\mathbb{X}_4(u) + \mathbb{X}_5(u)) - \ln(1 + (\mathbb{X}_4(t) + \mathbb{X}_5(u)))\}\nu(u)\right]\right\} \\
&+ \int_0^t \frac{\eta_4\mathbb{E}\beta d\mathbb{B}_4(t)}{[(\mu+\alpha)\mathbb{I} + \alpha\mathbb{E}(t)]} + \int_0^t \frac{(\mu+\alpha)\eta_5\mathbb{I}dB_5(t)}{[\mathbb{E} + (\mu+\alpha)\mathbb{I}]}
\end{aligned}$$

$$
\begin{aligned}
&+\int_0^t\int_y \ln\left(1+\frac{\beta\mathbb{X}_4(u)\mathbb{E}(t^-)+(\mu+\alpha)\mathbb{X}_5\mathbb{I}(t^-)}{H_1(t^-)}\right)\tilde{\mathbb{N}}(dt,du)\\
&\leq \frac{1}{(\mu+\alpha)}\Bigg\{\beta-\Bigg[(\mu+\alpha)(\gamma+\delta+\mu)+\frac{\eta_4^2}{2}+\frac{\eta_5^2}{2}\\
&+\int_y\{(\mathbb{X}_4(u)+\mathbb{X}_5(u))-\ln(1+(\mathbb{X}_4(t)+\mathbb{X}_5(u)))\}\,\nu(du)\Bigg]\Bigg\}\\
&+\int_0^t\frac{\eta_4\mathbb{E}\beta d\mathbb{B}_3(t)}{[(\mu+\alpha)\mathbb{I}+\alpha\mathbb{E}(t)]}+\int_0^t\frac{(\mu+\alpha)\eta_5\mathbb{I}d\mathbb{B}_5(t)}{[\mathbb{E}+(\mu+\alpha)\mathbb{I}]}\\
&+\int_0^t\int_y \ln\left(1+\frac{\alpha\mathbb{X}_4(y)\mathbb{E}(t^-)+(+\alpha+\mu)\mathbb{X}_5\mathbb{I}(t^-)}{H_1(t^-)}\right)\tilde{\mathbb{N}}(dt,du)\\
&\leq \frac{\left[(\mu+\alpha)(\mu+\delta+\gamma)+\frac{\eta_4^2+\eta_5^2}{2}+\int_y\{(\mathbb{X}_4(u)+\mathbb{X}_5(u))-\ln(1+(\mathbb{X}_4(t)+\mathbb{X}_5(y)))\}\,\nu(du)\right]}{(\mu+\beta)}\Big[\mathbf{R_s}-1\Big]\\
&+\int_0^t\frac{\eta_4\mathbb{E}\beta d\mathbb{B}_3(t)}{[(\mu+\alpha)\mathbb{I}+\alpha\mathbb{E}(t)]}+\int_0^t\frac{(\mu+\alpha)\eta_5\mathbb{I}d\mathbb{B}_4(t)}{[\mathbb{E}+(\mu+\alpha)\mathbb{I}]}\\
&+\int_0^t\int_y \ln\left(1+\frac{\beta\mathbb{X}_4(u)\mathbb{E}(t^-)+(\mu+\alpha)\mathbb{X}_5\mathbb{I}(t^-)}{H_1(t^-)}\right)\tilde{\mathbb{N}}(dt,du). \qquad (5.7)
\end{aligned}
$$

Taking limit superior as $t\to\infty$ after multiplying Eq (5.7) by $\frac{1}{t}$ and using Lemma 1, we get

$$
\limsup_{t\to\infty}(lnH_1(t))\leq\frac{\left[(\mu+\alpha)(\mu+\delta+\gamma)+\frac{\eta_4^2}{2}+\frac{\eta_5^2}{2}+\int_y\{(\mathbb{X}_4(u)+\mathbb{X}_5(u))-\ln(1+(\mathbb{X}_4(t)+\mathbb{X}_5(y)))\}\nu(du)\right]}{(\mu+\alpha)}\Big[\mathbf{R_s}-1\Big]. \qquad (5.8)
$$

If $\mathbf{R_s}<1$, then $\lim_{t\to\infty}H_1=\lim_{t\to\infty}[\alpha\mathbb{E}(t)+(\mu+\alpha)\mathbb{I}(t)]=0$, a.s. Again $\alpha>0,(\mu+\alpha)>0$, we assert that $\lim_{t\to\infty}[\alpha\mathbb{E}(t)+(\mu+\alpha)\mathbb{I}(t)]=0\Longrightarrow\lim_{t\to\infty}\mathbb{E}=\lim_{t\to\infty}\mathbb{I}=0$. Hence the result.

6. Persistence in mean

This section deals with the long-term effects of the disease. Firstly, we define the persistence in the average as given in [16].

Definition 1. [16, 19] The phenomenon of persistence could be observed in system (2.2) if the following holds

$$
\liminf_{t\to\infty}\frac{1}{t}\int_0^t\mathbb{Q}(r)dr>0,\ \text{ a.s.} \qquad (6.1)
$$

The following assertion is also a basic requirement for the persistence of an epidemic as could be seen in [16, 17].

Lemma 2. *Let $h\in C([0,\infty)\times\Omega,(0,\infty))$ and $K\in C([0,\infty)\times\Omega,\mathbb{R})$ such that $\lim_{t\to\infty}\frac{K(t)}{t}=0$ a.s. If for all $t\geq 0$*

$$
\log h(t)\geq\lambda_0 t-\lambda\int_0^t h(s)ds+K(t),\ a.s.
$$

Then $\liminf_{t\to\infty}\langle k(t)\rangle\geq\frac{\lambda_0}{\lambda}$, a.s, where λ_0 is nonnegative and λ is positive real numbers.

Here we will outline the hypothesis necessary for the mean persistence of model (2.2). The below result summarizes the objective of this section:

Theorem 3. *If* $\mathbf{R_0^s} > 1$, *then for* $(\mathbb{S}(0), \mathbb{V}_1(0), \mathbb{V}_2(0), \mathbb{E}0), \mathbb{I}(0), \mathbb{R}(0)) \in \mathbb{R}^6_+$, *the infectious group* $\mathbb{I}(t)$ *will satisfy the following inclusion*

$$\liminf_{t\to\infty} \langle \mathbb{I}(t) \rangle \geq \frac{3\phi\left(\sqrt{\mathbf{R_0^s}} - 1\right)}{C_1\beta}, \quad a.s., \tag{6.2}$$

where $A_1 = \frac{\phi}{(\tau+\mu+\frac{\eta_1^2}{2}+\int_Y \mathbb{X}_1(u)+\log(1+\mathbb{X}_1(u))\nu(du))}$, *meaning, the infection will persist in the community.*

Let

$$\mathbf{R_0^s} = \frac{\alpha\beta}{abc}. \tag{6.3}$$

Where

$$\begin{aligned} a &= \left(\tau + \mu + \frac{\eta_1^2}{2} + \int_Y \mathbb{X}_1(u) + \log\left(1 + \mathbb{X}_1(u)\right) \nu(du)\right), \\ b &= \left(\mu + \beta + \frac{\eta_3^2}{2} + \int_Y \mathbb{X}_3(u) + \log\left(1 + \mathbb{X}_3(u)\right) \nu(du)\right), \\ c &= \left(\gamma + \delta + \mu + \frac{\eta_4^2}{2} + \int_Y \mathbb{X}_4(u) + \log\left(1 + \mathbb{X}_4(u)\right) \nu(du)\right). \end{aligned} \tag{6.4}$$

Proof. Let define a suitable stochastic Lyapunov function

$$H_1 = -A_1 ln\mathbb{S} - A_2 ln\mathbb{E} - A_3 ln\mathbb{I}, \tag{6.5}$$

here A_1, A_2 and A_3 are constant and need to be defined later on.

By the using *Itô* formula on Eq (6.5), we get

$$\begin{aligned} dH_1 = LH_1 &- A_1\eta_1 d\mathbb{B}_1(t) - A_2\eta_4 d\mathbb{B}_4(t) - A_3\eta_5 d\mathbb{B}_5(t) \\ &- \int_Y \left[\log\left(1 + \mathbb{X}_1(u)\right)\right] \tilde{\mathbb{N}}(dt, du) - \int_Y \left[\log\left(1 + \mathbb{X}_4(u)\right)\right] \tilde{\mathbb{N}}(dt, du) - \int_Y \left[\log\left(1 + \mathbb{X}_5(u)\right)\right] \tilde{\mathbb{N}}(dt, du), \end{aligned} \tag{6.6}$$

where

$$\begin{aligned} H_1 = &- \frac{A_1\phi}{\mathbb{S}} + \frac{A_1\beta\mathbb{I}}{\mathbb{N}} - \frac{A_1\rho\mathbb{V}_1}{\mathbb{S}} + A_1(\tau + \mu) - A_2\frac{\beta\mathbb{S}\mathbb{I}}{\mathbb{E}} + A_2(\alpha + \mu) - A_3\frac{\alpha\mathbb{E}}{\mathbb{I}} + A_3(\gamma + \delta + \mu) \\ &+ \frac{A_1\eta_1^2}{2} + \frac{A_2\eta_4^2}{2} + \frac{A_3\eta_5^2}{2} + \int_Y A_1\mathbb{X}_1(u) + A_1\log\left(1 + \mathbb{X}_1(u)\right)\nu(du) \\ &+ \int_Y A_2\mathbb{X}_4(u) + A_2\log\left(1 + \mathbb{X}_4(u)\right)\nu(du) + \int_Y A_3\mathbb{X}_5(u) + A_2\log\left(1 + \mathbb{X}_5(u)\right)\nu(du) \\ &\leq -\frac{A_1\phi}{\mathbb{S}} - A_2\frac{\beta\mathbb{S}\mathbb{I}}{\mathbb{E}} - A_3\frac{\alpha\mathbb{E}}{\mathbb{I}} + \frac{A_1\beta\mathbb{I}}{\mathbb{N}} \\ &+ A_1(\tau + \mu + \frac{\eta_1^2}{2} + \int_Y \mathbb{X}_1(u) + \log\left(1 + \mathbb{X}_1(u)\right)\nu(du)) \end{aligned}$$

$$+ A_2(\mu + \beta + \frac{\eta_3^2}{2} + \int_Y \mathbb{X}_3(u) + \log(1 + \mathbb{X}_3(u))\,\nu(du))$$
$$+ A_3(\gamma + \delta + \mu + \frac{\eta_4^2}{2} + \int_Y \mathbb{X}_4(u) + \log(1 + \mathbb{X}_4(u))\,\nu(du)). \tag{6.7}$$

Let

$$\begin{aligned} A_1(\tau + \mu + \frac{\eta_1^2}{2} + \int_Y \mathbb{X}_1(u) + \log(1 + \mathbb{X}_1(u))\,\nu(du)) &= \phi, \\ A_2(\mu + \beta + \frac{\eta_3^2}{2} + \int_Y \mathbb{X}_3(u) + \log(1 + \mathbb{X}_3(u))\,\nu(du)) &= \phi, \\ A_2(\gamma + \delta + \mu + \frac{\eta_4^2}{2} + \int_Y \mathbb{X}_4(u) + \log(1 + \mathbb{X}_4(u))\,\nu(du)) &= \phi. \end{aligned} \tag{6.8}$$

Let

$$\begin{aligned} a &= \Big(\tau + \mu + \frac{\eta_1^2}{2} + \int_Y \mathbb{X}_1(u) + \log(1 + \mathbb{X}_1(u))\,\nu(du)\Big), \\ b &= \Big(\mu + \beta + \frac{\eta_3^2}{2} + \int_Y \mathbb{X}_3(u) + \log(1 + \mathbb{X}_3(u))\,\nu(du)\Big), \\ c &= \Big(\gamma + \delta + \mu + \frac{\eta_4^2}{2} + \int_Y \mathbb{X}_4(u) + \log(1 + \mathbb{X}_4(u))\,\nu(du)\Big). \end{aligned}$$

Then

$$\begin{aligned} LH_1 \leq & -3\sqrt{\frac{A_1\phi}{\mathbb{S}} \times \frac{A_3\beta\mathbb{S}\mathbb{I}}{\mathbb{E}} \times \frac{A_3\alpha\mathbb{E}}{\mathbb{I}}} + A_1\beta\mathbb{I} \\ & + A_1(\tau + \mu + \frac{\eta_1^2}{2} + \int_Y \mathbb{X}_1(u) + \log(1 + \mathbb{X}_1(u))\,\nu(du)) \\ & + A_2(\mu + \beta + \frac{\eta_3^2}{2} + \int_Y \mathbb{X}_3(u) + \log(1 + \mathbb{X}_3(u))\,\nu(du)) \\ & + A_3(\gamma + \delta + \mu + \frac{\eta_4^2}{2} + \int_Y \mathbb{X}_4(u) + \log(1 + \mathbb{X}_4(u))\,\nu(du)) \\ & = -3\sqrt{\frac{\phi^3\alpha\beta}{abc}} + 3\phi + A_1\beta\mathbb{I} = -3\phi\Big(\sqrt{\mathbf{R_0^s}} - 1\Big) + A_1\beta\mathbb{I}. \end{aligned} \tag{6.9}$$

By plugging Eq (6.9) into Eq (6.5) and then considering the integration of the underlying stochastic measles model (2.2), we have

$$\begin{aligned} & \frac{H_1(\mathbb{S}(t), \mathbb{E}(t), \mathbb{I}(t)) - G_1(\mathbb{S}(0), \mathbb{E}(0), \mathbb{I}(0))}{t} \\ \leq & -3\phi\Big(\sqrt{\mathbf{R_0^s}} - 1\Big) + A_1\beta\mathbb{I} - A_1\eta_1 d\mathbb{B}_1(t) - A_2\eta_4 d\mathbb{B}_5(t) - A_3\eta_5 d\mathbb{B}_5(t) \\ & - \int_Y [\log(1 + \mathbb{X}_1(u))]\,\tilde{\mathbb{N}}(dt, du) - \int_Y [\log(1 + \mathbb{X}_4(u))]\,\tilde{\mathbb{N}}(dt, du) - \int_Y [\log(1 + \mathbb{X}_5(u))]\,\tilde{\mathbb{N}}(dt, du), \end{aligned} \tag{6.10}$$

where

$$\begin{aligned}\Psi(t) = &- A_1\eta_1 d\mathbb{B}_1(t) - A_2\eta_4 d\mathbb{B}_4(t) - A_3\eta_5 d\mathbb{B}_5(t) - \int_Y \left[\log\left(1 + \mathbb{X}_1(u)\right)\right]\tilde{\mathbb{N}}(dt, du)\\ &- \int_Y \left[\log\left(1 + \mathbb{X}_4(u)\right)\right]\tilde{\mathbb{N}}(dt, du) - \int_Y \left[\log\left(1 + \mathbb{X}_5(u)\right)\right]\tilde{\mathbb{N}}(dt, du).\end{aligned} \tag{6.11}$$

By virtue of Lemma 1, we get

$$\lim_{t\to\infty} \Psi(t) = 0. \tag{6.12}$$

From Eq (6.11), we have

$$\begin{aligned}C_1\alpha\langle\mathbb{I}(t)\rangle &\geq 3\phi\left(\sqrt{\mathbf{R}_0^s} - 1\right) - \Psi(t) + \frac{G_1(\mathbb{S}(t), \mathbb{E}(t), \mathbb{I}(t)) - G_1(\mathbb{S}(0), \mathbb{E}(0), \mathbb{I}(0))}{t},\\ \langle\mathbb{I}(t)\rangle &\geq \frac{3\phi\left(\sqrt{\mathbf{R}_0^s} - 1\right)}{A_1\beta} - \frac{\Psi(t)}{A_1\beta} - \frac{1}{A_1\beta}\left(\frac{G_1(\mathbb{S}(t), \mathbb{E}(t), \mathbb{I}(t)) - G_1(\mathbb{S}(0), \mathbb{E}(0), \mathbb{I}(0))}{t}\right).\end{aligned} \tag{6.13}$$

By Lemma 1 and Eq (6.12), the limit superior of Eq (4.4), we have

$$0 \leq \liminf_{t\to\infty} \langle\mathbb{I}(t)\rangle \geq \frac{3\phi\left(\sqrt{\mathbf{R}_0^s} - 1\right)}{A_1\beta}, \quad \text{a.s,} \tag{6.14}$$

alternatively, this means $0 \leq \liminf_{t\to\infty} \langle\mathbb{I}(t)\rangle$. It completes the proof of Theorem 3.

7. Parameter estimation

One of the well-known and significant strategies used in whole-system analysis is the use of practical observations to obtain conclusions for a few missing epidemiological unknowns in biological systems under inquiry, is called parameter estimation. The parameter estimation was carried for the system (2.2):

- The approximate solution for the system of ODEs and stochastic differential equations was obtained using the Runge-Kutta Method (RK4), with selected initial conditions and parameters.
- The results obtained from the model are checked with real-world and a Levenberg-Marquardt optimization algorithm [23, 24] is employed for getting new values of the parameter that lead to an even best fit of the system outcomes to the observed data.
- Once the optimizer determines new parameter values, the system of ODEs is simulated with these updated values. The resulting output of the model are once again compared with the data at hand.
- The process of updating parameters and solving the system of ODEs numerically using the Runge-Kutta method, specifically the Dormand-Prince pairs [25, 26], is repeated until the criteria for parameter convergence are met.
- During the parameter estimation process, a random process is used to select around one thousand values for every parameter that need to be fitted.
- The final parameter estimation was achieved after conducting simulations above 10^3.

The proposed model involves nine parameters that are initially unknown. While the values of ϕ (recruitment rate) and the mortality rate μ can be determined from the local population, the remaining

parameters require estimation from field data. Table 1 presents the estimated values for the parameters used in the model. During the year 2019, particularly from January to October, the reported measles data of Pakistan is used for the fitting [12]. In Figure 2, the simulation findings for the Measles cases obtained by fitted the proposed system (2.2) by using the data of Table 2. We can see in Figure 2 gives a good declines fit.

Table 1. The proposed measles pandemic model utilizes the above biological parameters.

Parameter	Description	Source
ϕ	260,479	Estimated
β	$1.253133e-03$	Estimated
ω	0.97	Estimated
τ	$1.60056e-07$	Fitted
μ	9.3408	Fitted
α	$9.2373e-01$	Fitted
δ	$5.8306e-01$	Fitted
ρ	0	Estimated
γ	$5.8306e-01$	Fitted

Table 2. Data of reported measles patients in Pakistan during the year 2019 from January to October [12].

Jan.	Feb.	Mar.	Apr.	May	June	July	Aug.	Sep.	Oct.
237	252	397	399	276	168	70	28	23	19

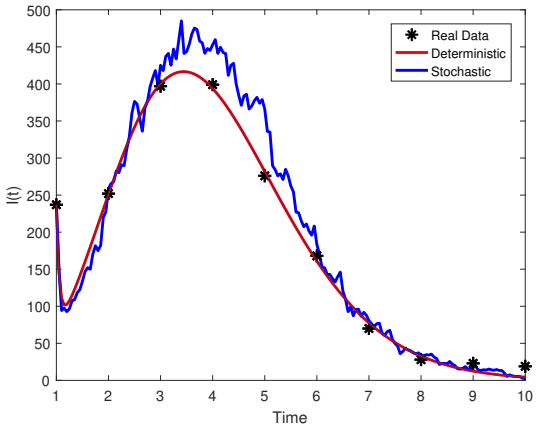


Figure 2. Fitting the proposed measles model to actual statistical data by utilizing the parameters presented in Table 1.

8. Numerical simulations

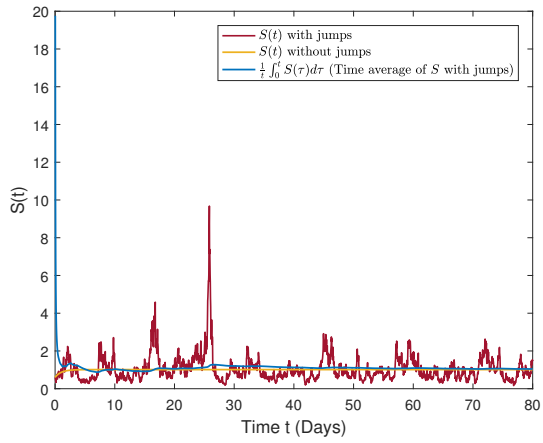
In the current section, we present numerical simulations that are based on the theoretical methodology outlined in the previous section. To demonstrate our results, we use numerical calculations to generate trajectories for both the stochastic disease system (2.2) and its associated ODEs form. We consider the time interval $[0, 100]$ units, and the initial values are given by: $(\mathbb{S}_0, \mathbb{V}_{10}, \mathbb{V}_{20}, \mathbb{E}_0, \mathbb{I}_0, \mathbb{R}_0) = (0.6, 0.5, 0.3, 0.5, 0.3, 0.2)$, and $\Delta = 0.3$.

8.1. The realizations of the extinction

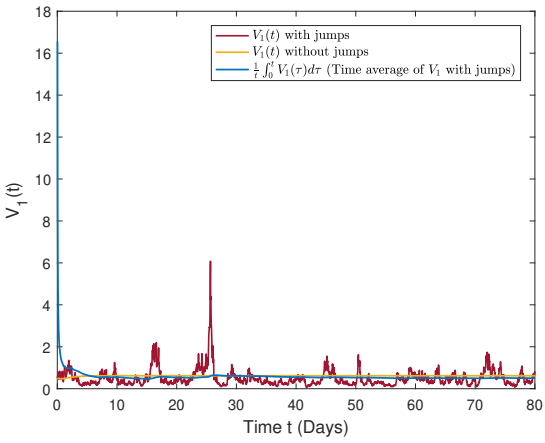
We use graphical simulations to examine the approximate solution and confirm the biological well-posedness of the model (2.2). Figure 3 illustrates that when $\mathbf{R_0^D} < 1$, the solution of model (2.2) with Lévy jumps reaching the DFE of the corresponding ODE model, indicating the elimination of the infection. Theorem 2 provides those criteria which is needed for the eradication of the disease which is dynamically described by model (2.2). The visual presentation confirms the validity of Theorem 2 in light of the hypothesis $\mathbf{R_s} < 1$. The values of parameters are chosen as: $\phi = 0.9$, $\tau = 0.3$, $\beta = 0.3$, $\mu = 0.03$, $\alpha = 0.05$, $\delta = 0.04$, $\gamma = 0.03$, $\rho = 0.02$, $\sigma = 0.6$, $\omega = 0.01$, and the noise intensities assumed $\eta_1 = 0.45$, $\eta_2 = 0.65$, $\eta_3 = 0.45$, $\eta_4 = 0.35$, $\eta_5 = 0.23$, $\eta_6 = 0.55$ and $\mathbb{X}_i(u) = \frac{-k_i Z(u)}{1+Z(u)^2}$, $u = 0.7$, with $k_1 = 0.60$, $k_2 = 0.55$, $k_3 = 0.45$, $k_4 = 0.23$, $k_5 = 0.25$, $k_6 = 0.34$. By applying Theorem 2, it can be inferred that the disease will eventually vanish. This extinction of the epidemic is evidently observable in the stochastic system's behavior, as illustrated in Figure 3a–3f. It is observed that increasing $\mathbb{S}(t)$ and goes to the equilibrium point can see in Figure 3a and for vaccination class we can see in Figure 3b.

8.2. The realizations of persistence in the mean

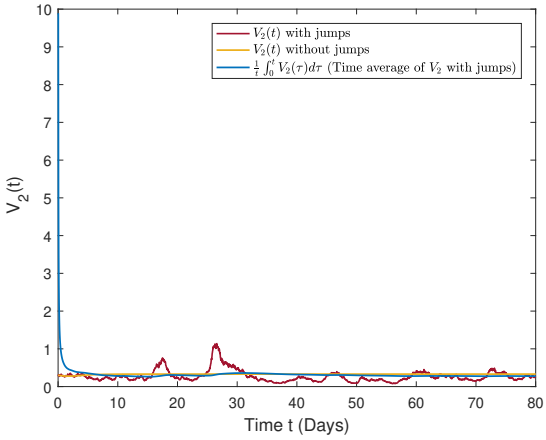
Numerical assessments of the system (2.2) are presented in Figures 4 and 5, respectively. The solution profiles for infected groups shown in Figure 5. It is observed that increasing the noise and vaccination rates greatly caused reduction in infected classes. Further, the results of Theorem 3 indicate that the disease will persist in the system (2.2) due to $\mathbf{R_0^D} > 1$, which implies that $\mathbf{R_0^s} > 1$ with small intensities of noise. This assertion is supported by the numerical simulations shown in Figures 3f–4d, which verify the mean behavior of the epidemic model (2.2) in accordance with Theorem 3. The values of parameters for this simulations are: $\phi = 0.5$, $\tau = 0.44$, $\beta = 0.5$, $\mu = 0.6$, $\alpha = 0.05$, $\delta = 0.4$, $\gamma = 0.2$, $\rho = 0.1$, $\sigma = 0.31$,$\omega = 0.2$, and the noise intensities assumed $\eta_1 = 0.55$, $\eta_2 = 0.25$, $\eta_3 = 0.20$, $\eta_4 = 0.45$, $\eta_5 = 0.55$, $\eta_6 = 0.24$ and $\mathbb{X}_i(u) = \frac{-k_i Z(u)}{1+Z(u)^2}$, $u = 0.7$, where $k_1 = 0.50$, $k_2 = 0.35$, $k_3 = 0.30$, $k_4 = 0.35$, $k_5 = 0.10$, $k_6 = 0.20$. The stochastic threshold $\mathbf{R_0^s} > 1$ and clearly $\mathbf{R_0^D} > 1$ and can be calculated easily. The mean simulation results in Figure 4 confirm that the disease will persist, which support findings of Theorem 3. Figure 4a–4f also show that the epidemic will continue in the mean in the population.



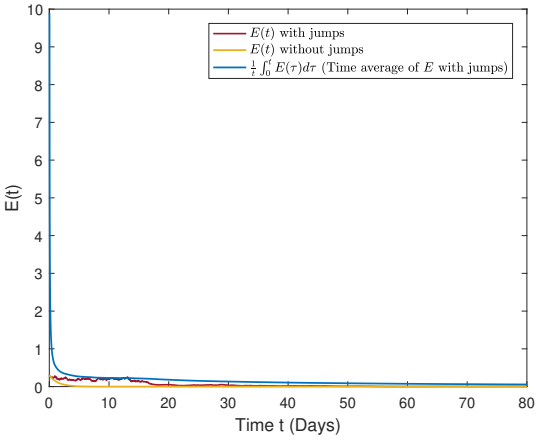
(a) $\mathbb{S}(t)-$Population



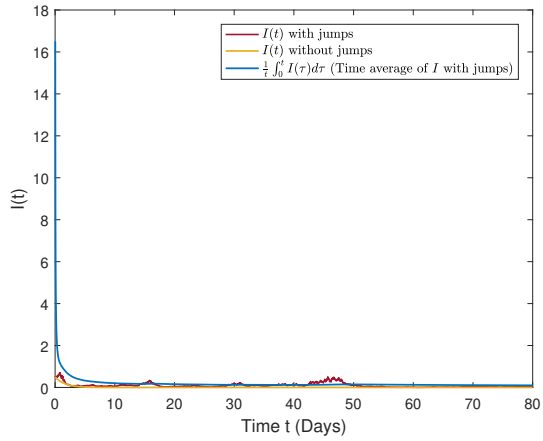
(b) $\mathbb{V}_1(t)-$Population



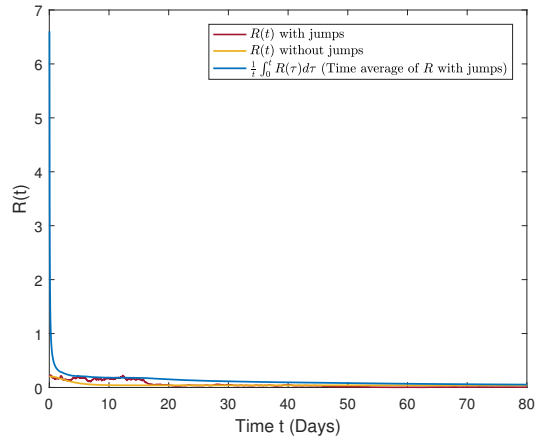
(c) $\mathbb{V}_2(t)-$Population



(d) $\mathbb{E}(t)-$Population

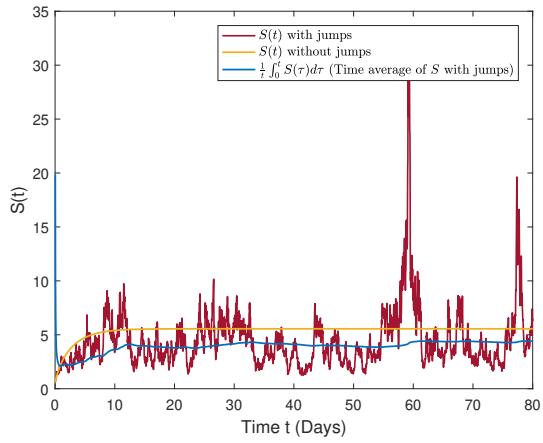


(e) $\mathbb{I}(t)-$Population

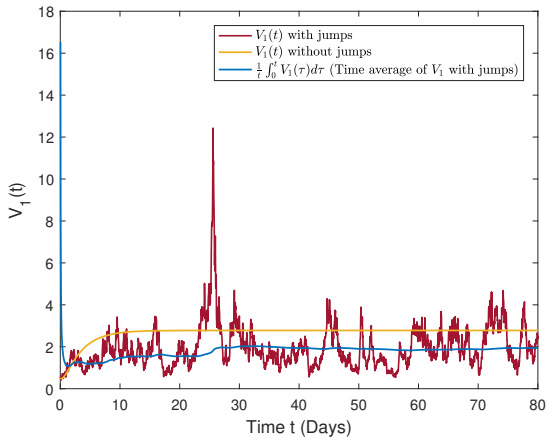


(f) $\mathbb{R}(t)-$Population

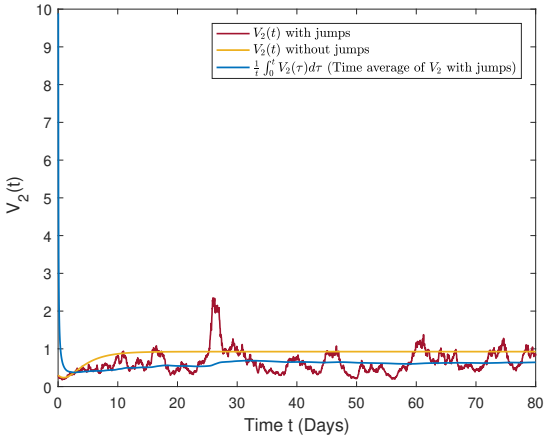
Figure 3. Extinction of the stochastic system (2.2) and the deterministic system (2.1).



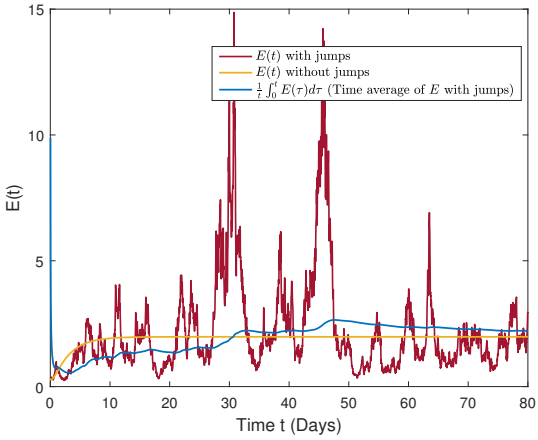
(a) $\mathbb{S}(t)-$Population



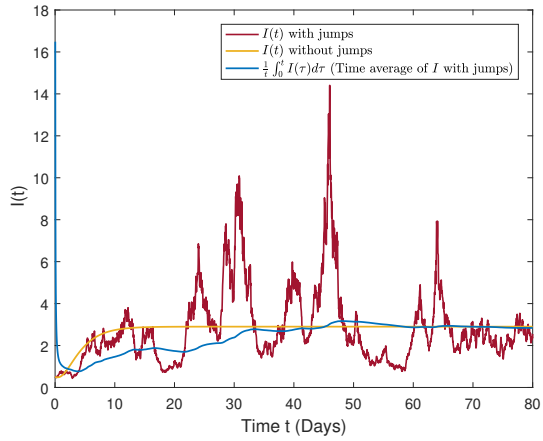
(b) $\mathbb{V}_1(t)-$Population



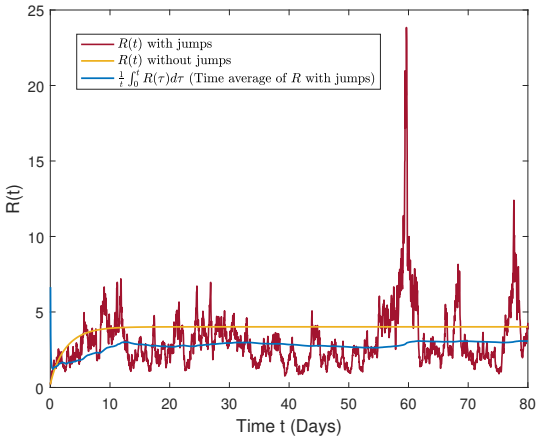
(c) $V_2(t)-$Population



(d) $\mathbb{E}(t)-$Population



(e) $\mathbb{I}(t)-$Population

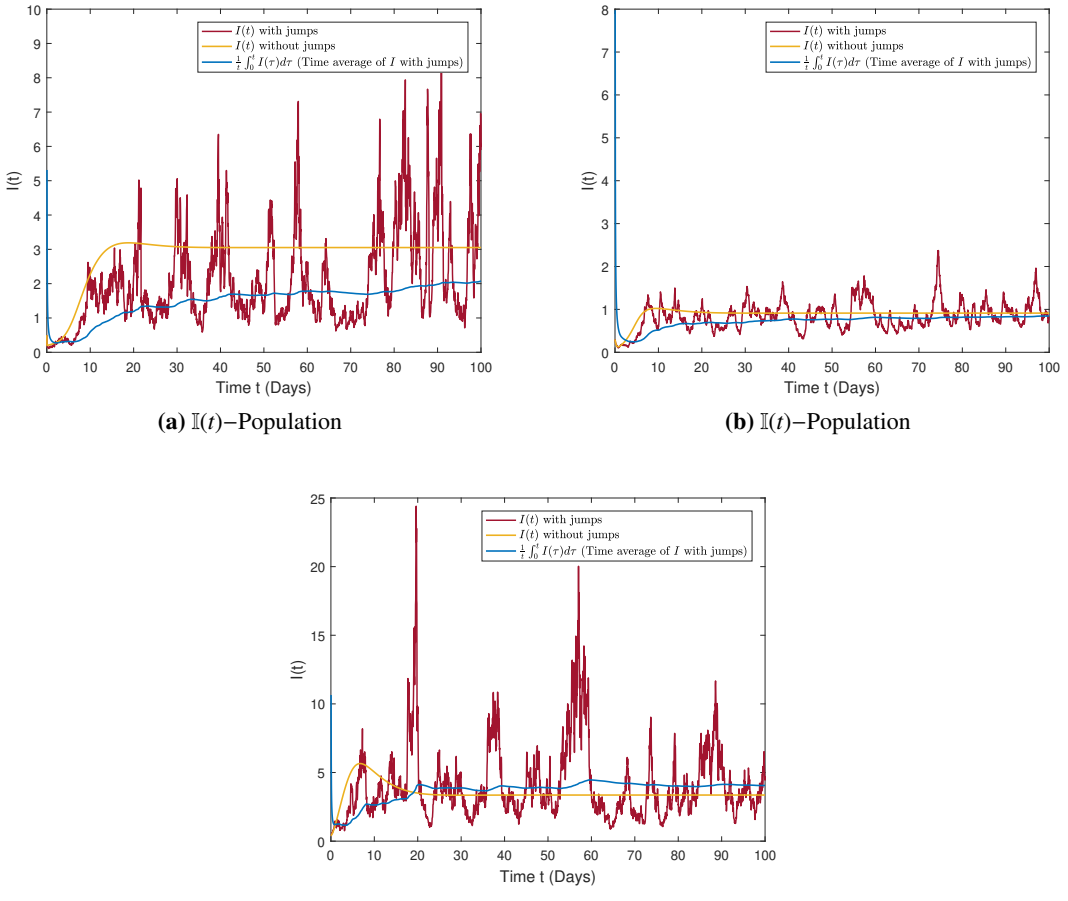


(f) $\mathbb{R}(t)-$Population

Figure 4. Simulations for the persistence of the stochastic system (2.2) and the deterministic system (2.1).

8.3. *The impact of Lévy noise on $\mathbb{I}$-class*

The impact on the class $\mathbb{I}$ of the intensity of the noises correspond to system (2.2) is shown in Figure 5a–5c. These figures suggest that by increasing the values of η_i for $i = 1, \cdots, 6$ leads towards the extinction of the disease. This means, the size of the infected class approaches zero as we increase the values of the intensity of the noises. Further, this indicates that for small values of the intensities, the infected group oscillates around the endemic steady state $\mathbb{I}^\star$ which confirm the result of Theorem 3. Nonetheless, when the white noise terms are large enough, the corresponding solution $\mathbb{I}$ does not oscillate in the vicinity of the EE. This demonstrates that continuous efforts to increase stochastic disruptions through mass recovery of susceptible individuals, as well as effective treatment and care of the affected persons, could significantly lower the spread and circulation of the Measles virus in the population.



(a) $\mathbb{I}(t)-$Population

(b) $\mathbb{I}(t)-$Population

(c) $\mathbb{I}(t)-$Population

Figure 5. Simulations of $\mathbb{I}(t)$ based on the stochastic and deterministic systems, showing the effect of the intensities on the class $\mathbb{I}$, when $\phi = 0.80$, $\tau = 0.50$, $\beta = 0.40$, $\mu = 0.60$, $\alpha = 0.05$, $\delta = 0.4$, $\gamma = 0.2$, $\rho = 0.2$, $\sigma = 0.25$, $\omega = 0.3$, and the noise intensities assumed $\eta_1 = 0.45$, $\eta_2 = 0.20$, $\eta_3 = 0.45$, $\eta_4 = 0.45$, $\eta_5 = 0.35$, $\eta_6 = 0.20$ and $\mathbb{X}_i(u) = \frac{-k_i Z(u)}{1+Z(u)^2}$, $u = 0.7$, where $k_1 = 0.60$, $k_2 = 0.34$, $k_3 = 0.30$, $k_4 = 0.35$, $k_5 = 0.10$, $k_6 = 0.20$, and the $(\mathbb{S}(0), \mathbb{V}_1(0), \mathbb{V}_2(0), \mathbb{E}(0), \mathbb{I}(0), \mathbb{R}(0)) = (0.5, 0.4, 0.5, 0.3, 0.2, 0.1)$.

9. Conclusions

In conclusion, this study developed a Levy noise-driven epidemic model for measles infection that incorporates the effect of vaccination. The feasibility of the model was founded through the use of existence and uniqueness theory, and its behavior around steady states was analyzed using appropriate Lyapunov function. The threshold parameter $\mathbf{R}_0^s$ was calculated to check the persistence and extinction of the disease and it was found that the disease will become extinct when $\mathbf{R}_0^s < 1$. The model was also fitted against reported measles cases in Pakistan from January to October 2019 using established fitting techniques. The estimated parameter values were used to predict the future behavior of the infection through numerical simulations, and the analytical results of the study were verified through further simulation. Overall, this study provides valuable insights into the dynamical behavior of measles infection subject to a vaccination program and noise, which can aid in the development of effective disease control strategies. We plan to explore the effects of regime switching and temporary immunity in the system (2.2) in our upcoming research.

Use of AI tools declaration

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

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Conflict of interest

The authors declare that there are no conflicts of interest.

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