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Research article

Impact of supervise neural network on a stochastic epidemic model with Levy noise

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Abstract: This paper primarily focused on analyzing a stochastic $\mathcal{SVIR}$ epidemic model that incorporates Levy noises. The population may be divided into four distinct compartments: vulnerable class ($\mathcal{S}$), vaccinated individuals ($\mathcal{V}$), infected individuals ($\mathcal{I}$), and recovered individuals ($\mathcal{R}$). To achieve this, we chose existing and unique techniques as the most feasible solution. In the nexus, the stochastic model was theoretically analyzed using a suitable Lyapunov function. This analysis broadly covered the existence and uniqueness of the non-negative solution, as well as the dynamic properties related to both the disease-free equilibrium and the endemic equilibrium. In order to eradicate diseases, a stochastic threshold value denoted as "$\mathbf{R}_0$" was used to determine if they may be eradicated. If $\mathbf{R}_0 < 1$, it means that the illnesses have the potential to become extinct. Moreover, we provided numerical performance results of the proposed model using the artificial neural networks technique combined with the Bayesian regularization method. We firmly believe that this study will establish a solid theoretical foundation for comprehending the spread of an epidemic, the implementation of effective control strategies, and addressing real-world issues across various academic disciplines.

Keywords: white noise; Levy noise; epidemic model; artificial neural networks; numerical solutions
Mathematics Subject Classification: 92B20, 93E03

1. Introduction

An epidemic is a significant disease caused by various pathogens that pose a threat to human health. It can be spread from person to animal, animal to animal, or person to person. Human survival and development have always been challenged by infectious diseases. Humans have been engaged in a long battle against such diseases.

For instance, in 1988, Sungai Nipah, a village in Malaysia, experienced an outbreak of the Nipah

virus (Niv) [1]. The continued emergence of new major infectious diseases in the new century (such as SARS in 2003, A/H1H1 flu in 2009 and MERS in 2012), and the epidemic of the novel coronavirus (COVID-19) have had a tremendous influence on the normal social life and people's health. As a result, the study of how infectious illnesses are transmitted has always been a key area of research in academia.

For a significant duration of time, "chamber" models have been the primary mathematical systems used in the theory of epidemics. These systems are still widely used and constantly being developed. Many scholars [2–8] have studied the classic "chamber" systems, such as ($\mathcal{SIS}$), ($\mathcal{SIR}$) or ($\mathcal{SIRS}$). Vaccination is the most effective way to prevent infectious illnesses, according to evidence. Moreover, prevention is regarded as one of the primary strategies to control these illnesses. Consequently, the ($\mathcal{SVIR}$) model of infectious illnesses with vaccination is extremely important. Recently, some researchers have studied the dynamic behavior of the original classical model and proposed the $\mathcal{SVIR}$ model. A chamber "$\mathcal{V}$" was added to the ($\mathcal{SIS}$) model by Velasco-Hernandez and Kribs-Zaleta [9]. This chamber shows that rather than going straight to the vulnerable people, the population recovering from the illness returns to the immune compartment. On the basis of this, Lin and Takeuchi [10] constructed a ($\mathcal{SVIR}$) system with a continuous vaccination strategy. Later, Ashrafu and Zou [11] developed a vaccination distribution model with vaccination priority. The model of vaccination strategy is not perfect. From a biological perspective, they neglected the possibility that recipients of vaccinations might contract the disease again if they are not completely immune. In real life, vaccines do not always provide complete protection against infections. However, they greatly reduce the likelihood of getting infected [12].

Therefore, in the system of infectious illnesses, the case of incomplete immunity must be taken into account.

The article [13] describes a simple ($\mathcal{SVIR}$) epidemic system that takes the age of vaccination into account. It states that the system allows for vaccinated individuals to become vulnerable again once the vaccine protective properties diminish over time. Based on this, the $\mathcal{SVIR}$ infectious disease model was examined, and techniques for combining the LaSalle invariance principle, Itô's formula, the Lyapunov method, and a numerical simulation were presented in order to analyse the ($\mathcal{SVIR}$) model's global dynamics [14]. Moreover, the corresponding continuous model was given to the Mickens nonstandard finite difference format by Geng and Xu [15]. Based on this model [16,17], many researchers have also examined age and time lag. Liao and Yang [18] examined the best control for the basics deterministic ($\mathcal{SVIR}$) model. Wang and Xu [19] constructed a nonlinear incidence rate into the ($\mathcal{SIS}$) epidemiological system of ilness age and then examined the corresponding characteristic equation to establish the local stability of every steady state of the system.

In the real world, population models are constantly subjected to random and complex variations. A more appropriate approach to modeling epidemics would be to employ stochastic models. Additionally, research has demonstrated that stochastic models are more grounded in reality than deterministic ones. Numerous researchers [20–23] have shown interest in stochastic epidemic systems perturbation in the last few years. Cao et al. [24] developed a stochastic epidemic system with quarantine and standard incidence rate. They also defined the parameters for the disease's extinction based on white noise and basic reproductive value of the stochastic model. To illustrate the persistence, they showed the system's stationary distribution. White noise's effect on the basic ($\mathcal{SIRS}$) model and the global stability of a deterministic ($\mathcal{SIR}$) epidemic model with a ratio-dependent incidence rate

were both studied by Cai et al. [25]. Sufficient conditions have been proven for both the extinction and the existence of only one endemic stationary distribution, depending on the threshold value. In all the cited works above, while abrupt environmental disruptions like tsunamis, volcanoes, or hurricanes. can affect population systems, the suggested models were disturbed by white noise. This kind of phenomenon was not clear within the traditional stochastic model. Thus, incorporating the Levy jump process into the underlying population pattern is one way to solve the problem (see, [26–29]). The dynamics of a ($\mathcal{SIR}$) epidemic system with media coverage were studied by Lui et al. [30], who also explored the conditions related to the persistence and extinction of interactive populations. In [31], Jian Wu investigated the stability of a stochastic model consisting of one prey and two predators with Levy noise and delay. Motivated by the above discussions, we reformulated the ($\mathcal{SVIR}$) epidemic model with white and Levy noises [10].

$$\begin{aligned}
d\mathcal{S}(t) &= \left(\Lambda - \frac{\beta\mathcal{S}(t)\mathcal{I}(t)}{\mathcal{N}(t)} - (d+\rho)\mathcal{S}(t)\right)dt + \alpha_1\mathcal{S}(t)d\mathcal{W}_1(t) + \int_{\mathcal{X}} p_1(\mathcal{K})\mathcal{S}(t^-)\tilde{\mathcal{D}}(dt, d\mathcal{K}),\\
d\mathcal{V}(t) &= \left(\rho\mathcal{S}(t) - \frac{\beta_1\mathcal{V}(t)\mathcal{I}(t)}{\mathcal{N}(t)} - (\delta_1 + d)\mathcal{V}(t)\right)dt + \alpha_2\mathcal{V}(t)d\mathcal{W}_2(t) + \int_{\mathcal{X}} p_2(\mathcal{K})\mathcal{V}(t^-)\tilde{\mathcal{D}}(dt, d\mathcal{K}),\\
d\mathcal{I}(t) &= \left(\frac{\beta\mathcal{I}(t)\mathcal{S}(t)}{\mathcal{N}(t)} + \frac{\beta_1\mathcal{V}\mathcal{I}(t)}{\mathcal{N}(t)} - (\delta + d)\mathcal{I}(t)\right)dt + \alpha_3\mathcal{I}(t)d\mathcal{W}_3(t) + \int_{\mathcal{X}} p_3(\mathcal{K})\mathcal{I}(t^-)\tilde{\mathcal{D}}(dt, d\mathcal{K}),\\
d\mathcal{R}(t) &= (\delta\mathcal{I}(t) + \delta_1\mathcal{V}(t) - d\mathcal{R}(t))\,dt + \alpha_4\mathcal{R}(t)d\mathcal{W}_4(t) + \int_{\mathcal{X}} p_4(\mathcal{K})\mathcal{R}(t^-)\tilde{\mathcal{D}}(dt, d\mathcal{K}).
\end{aligned} \tag{1.1}$$

The population's recruiting rate and natural mortality ratio are denoted by Λ. Let δ denote the infection-related recovery rate and β the disease transmission rate when susceptible individuals come into contact with individuals who are ill. People who have recovered are said to have acquired immunity, often referred to as natural immunity, against the infection.

Let $\mathcal{V}$ represent a newly defined group, distinct from others, that quantifies the density of vaccines initiating the vaccination process, with the objective of incorporating the vaccination program. The individuals within the group $\mathcal{V}$ are distinct from those in groups $\mathcal{S}$ and $\mathcal{R}$. Let ρ represent the frequency of individuals; those who are vulnerable are introduced to the vaccinations process. Subsequent to the procedure, individuals will acquire immunity induced by the vaccine. Let δ_1 denote the average rate (and thus the average duration) of those who become immune and become part of the population that has recovered. In this case, we do not distinguish between natural immunity and immunity resulting from vaccinations, because vaccine-induced immunity can also endure for a long period. We assume that prior to developing immunity, coming into contact with infected individuals carries a risk of infection with the vaccinations at a rate of β_1. As individuals undergo vaccination, they may acquire temporary immunity or become better at identifying characteristics associated with disease transmission, thus potentially reducing the quantity of effective contacts with infected individuals. It is possible to consider that the parameter β may exceed the parameter β_1.

$\mathcal{W}_1(t)$, $\mathcal{W}_2(t)$, $\mathcal{W}_3(t)$ and $\mathcal{W}_4(t)$ are Brownian motions defined on the complete probability space $(\Omega, \mathcal{F}, \mathbb{P})$ with filtration $\{\mathcal{F}\}_{t\geq 0}$, satisfying the identical assumptions (i.e., it is growing and right continuous while $\mathcal{F}$ holds all $\mathbb{P}$-empty sets); $\alpha_i (i = 1, ..., 4)$ represents the intensity of white noise. The left limit for $\mathcal{S}(t), \mathcal{V}(t), \mathcal{I}(t)$ and $\mathcal{R}(t)$ are $\mathcal{S}(t^-), \mathcal{V}(t^-), \mathcal{I}(t^-)$ and $\mathcal{R}(t^-)$, respectively. $\tilde{\mathcal{D}}(dt, d\mathcal{K}) = \mathcal{D}(dt, d\mathcal{K}) - \upsilon(\mathcal{K})dt, \mathcal{K}$ is a Poisson counting measure with characteristics measure υ

on the measurable subset $\mathcal{X}$ of $[0,\infty)$, with $\upsilon(\mathcal{X}) < \infty$, and $p_i : Z \times \Omega \to \mathbb{R}, (i = 1, ..., 4)$, show the impact of random perturbation, which is considered to be bounded and continuous with regard to υ and is $\mathcal{B}(X) \times \mathcal{F}_t$-measurable.

The remaining sections of this article are organized as follows: In Section 2, we examine an existence analysis of the feasible or positive root to the system 1.1. To reduce the epidemic, we use a stochastic threshold in Section 3. In Section 4, we present ample evidence to establish the persistence of mean for an epidemic. Section 5 presents the numerical performance of the proposed model using Bayesian regularization neural networks (BRNNs).

2. Well-posedness for the system

The current section of this article uses the methods described in [32] to demonstrate the existence and uniqueness of the non-negative globalized solution of the stochastic disease problem 1.1. Two standardized suppositions, (A_1) and (A_2), are used to support this assertion.

(A_1). For every $\mathbb{Q} > 0$, such that $L_{\mathbb{Q}} > 0$

$$\int_X |K_i(z_1, (\mathcal{K})) - K_i(z_2, (\mathcal{K}))|^2 V dx \leq L_{\mathbb{Q}} |z_1 - z_2|^2, i = 1, ..., 4,$$

with $|(\mathcal{K})_1| \vee |(\mathcal{K})_2| \leq \mathbb{Q}$, where

$$\begin{aligned} K_1(z, (\mathcal{K})) &= p_1((\mathcal{K}))\, z \; for \; z = \mathcal{S}(t^-), \\ K_2(z, (\mathcal{K})) &= p_2((\mathcal{K}))\, z \; for \; z = \mathcal{V}(t^-), \\ K_3(z, (\mathcal{K})) &= p_3((\mathcal{K}))\, z \; for \; z = \mathcal{I}(t^-), \\ K_4(z, (\mathcal{K})) &= p_4((\mathcal{K}))\, z \; for \; z = \mathcal{R}(t^-). \end{aligned}$$

(A_2). $|\ln(1 + p_i((\mathcal{K}))| \leq m$, for $p_i((\mathcal{K})) > -1, \quad i = 1, ..., 4$, with non-negative constant $'m'$.

Theorem 2.1. *For every $t \geq 0$, model (1.1) will fulfill only one characteristic of the global output $(\mathcal{S}, \mathcal{V}, \mathcal{I}, \mathcal{R}) \in \mathbb{R}^4_+$ for the starting approximation $(\mathcal{S}(0), \mathcal{V}(0), \mathcal{I}(0), \mathcal{R}(0)) \in \mathbb{R}^4_+$.*

Proof. Drifting and mixing are localized Lipschitzian, because by assumption (A1), then t and ζ_e for any initial approximation $(\mathcal{S}(0), \mathcal{V}(0), \mathcal{I}(0), \mathcal{R}(0)) \in \mathbb{R}^4_+$, we suppose $(\mathcal{S}, \mathcal{V}, \mathcal{I}, \mathcal{R})$ on $t \in [0, \zeta_e)$ is only one localized solution. To justify the globalized solution, we have to show that $\zeta_e = \infty$ a.s. First, we show that $(\mathcal{S}, \mathcal{V}, \mathcal{I}, \mathcal{R})$ can never tend to ∞ in a defined time interval. Suppose $f_0 > 0$ large enough for $(\mathcal{S}(0), \mathcal{V}(0), \mathcal{I}(0), \mathcal{R}(0))$ belonging to the interval $[\frac{1}{f_0}, f_0]$. For every constant, let us establish the stopping time $f \leq f_0$.

$$\zeta_f = \inf \left\{ t \in [0, \zeta_e) \backslash (\mathcal{S}, \mathcal{V}, \mathcal{I}, \mathcal{R}) \notin (\frac{1}{f}, f) \right\}. \tag{2.1}$$

It is obvious that $\zeta^+ \leq \zeta_e$ when we set $\inf \phi = \infty$; this implies $\zeta^+ = \infty$. a.s., which demonstrates that $\zeta_e = +\infty$ a.s. Next, by assuming $\zeta^+ < \infty$, then $0 < \Upsilon$ exists, implying that

$$\mathbb{P}\{\Upsilon \geq \zeta_k\} > 0.$$

By taking into account the function, one can use it in the following way $H : \mathbb{R}^4_+ \to \mathbb{R}_+$ from the C^2 space as described.

$$V(\mathcal{S}, \mathcal{V}, \mathcal{I}, \mathcal{R}) = (\mathcal{S} - \ln \mathcal{S}) + (\mathcal{V} - \ln \mathcal{V}) + (\mathcal{I} - \ln \mathcal{I}) + (\mathcal{R} - \ln \mathcal{R}) - 4. \tag{2.2}$$

Applying the Itos on Eq (2.2), for every $t \in [0, \zeta^+]$,

$$\begin{aligned} dV(\mathcal{S},\mathcal{V},\mathcal{I},\mathcal{R}) &= LV(\mathcal{S},\mathcal{V},\mathcal{I},\mathcal{R}) + \alpha_1(-1+\mathcal{S})d\mathcal{W}_1(t) \\ &+ \alpha_2(-1+\mathcal{V})d\mathcal{W}_2(t) + \alpha_3(-1+\mathcal{I})d\mathcal{W}_3(t) + \alpha_4(-1\mathcal{R})d\mathcal{W}_4(t) \\ &+ \int_{\mathcal{X}} [p_1((\mathcal{K}))\mathcal{S} - \ln(1+p_1(\mathcal{K}))]\widetilde{\mathcal{D}}(dt, d(\mathcal{K})) \\ &+ \int_{\mathcal{X}} [p_2((\mathcal{K}))V - \ln(1+p_2(\mathcal{K}))]\widetilde{\mathcal{D}}(dt, d(\mathcal{K})) \\ &+ \int_{\mathcal{X}} [p_3((\mathcal{K}))I - \ln(1+p_3(\mathcal{K}))]\widetilde{\mathcal{D}}(dt, d(\mathcal{K})) \\ &+ \int_{\mathcal{X}} [p_4((\mathcal{K}))R - \ln(1+p_4(\mathcal{K}))]\widetilde{\mathcal{D}}(dt, d(\mathcal{K})). \end{aligned} \tag{2.3}$$

According to A_2, we get

$$\begin{aligned} LV \leq \Lambda + \rho + 4d + \delta + \delta_1 + \beta \ &+ \frac{{\alpha_1}^2 + {\alpha_2}^2 + {\alpha_3}^2 + {\alpha_4}^2}{2} \\ &+ \int_{\mathcal{X}} [p_1(\mathcal{K}) - \ln(1+p_1(\mathcal{K})]n(d\mathcal{K}) \\ &+ \int_{\mathcal{X}} [p_2(\mathcal{K}) - \ln(1+p_2(\mathcal{K})]n(d\mathcal{K}) \\ &+ \int_{\mathcal{X}} [p_3(\mathcal{K}) - \ln(1+p_3(\mathcal{K})]n(d\mathcal{K}) \\ &+ \int_{\mathcal{X}} [p_4(\mathcal{K}) - \ln(1+p_4(\mathcal{K})]n(d\mathcal{K}) := \mathbf{K}. \end{aligned} \tag{2.4}$$

When considering expectations and taking integration on both sides of Eq (2.3) from 0 to $\zeta_m \wedge \Upsilon$,

$$\begin{aligned} 0 < \mathbb{E}\Big[V(\mathcal{S}(\zeta_f \wedge \Upsilon), \mathcal{V}(\zeta_f \wedge \Upsilon), \mathcal{I}(\zeta_f \wedge \Upsilon), \mathcal{R}(\zeta_f \wedge \Upsilon))\Big] \\ \leq V(\mathcal{S}(0), \mathcal{V}(0), \mathcal{I}(0), \mathcal{R}(0)) + \mathbb{E}\Big[\int_0^{\zeta_f \wedge \Upsilon} \mathbf{K}\Big] \\ \leq V(\mathcal{S}(0), \mathcal{V}(0), \mathcal{I}(0), \mathcal{R}(0)) + \Upsilon\mathbf{K}. \end{aligned} \tag{2.5}$$

We get $\mathbb{P}(\zeta_f) \geq \epsilon$, for $f_1 \leq f$, by taking $\zeta_f = \{\zeta_f \leq \Upsilon\}$. Moreover, there exists $\mathcal{S}(\zeta_f, \omega)$, $\mathcal{V}_{(\zeta_f,\zeta)}, \mathcal{I}_{(\zeta_f,\zeta)}, \mathcal{R}_{(\zeta_f,\zeta)})$ equals to $\frac{1}{f}$ or f for each ζ in ζ_f.

As $V(\mathcal{S}_{(\zeta_f)}, \mathcal{V}_{(\zeta_f)}, \mathcal{I}_{(\zeta_f)}, \mathcal{R}_{(\zeta_f)}) \geq \frac{1}{f} - 1 + \ln f$ or $f - 1 - \ln f$. So

$$\mathbb{E}\Big[\Big(f - 1 - \ln f\Big) \wedge \Big(\frac{1}{f} - 1 + \ln f\Big)\Big] \leq V\Big(\mathcal{S}_{(\zeta_f)}, \mathcal{V}_{(\zeta_f)}, \mathcal{I}_{(\zeta_f)}, \mathcal{R}_{(\zeta_f)}\Big). \tag{2.6}$$

According to $\mathbb{P}(\zeta^+ < T)$ and Eq (2.5), we have

$$\begin{aligned} &\leq \epsilon\Big[\Big(\frac{1}{f} - 1 + \ln f\Big) \wedge \Big(f - 1 - \ln f\Big)\Big] \leq \mathbb{E}\Big[1_{\Omega(\omega)} V\Big(\mathcal{S}_{(\zeta_f)}, \mathcal{V}_{(\zeta_f)}, \mathcal{I}_{(\zeta_f)}, \mathcal{R}_{(\zeta_f)}\Big)\Big] \\ &\leq V(\mathcal{S}(0), \mathcal{V}(0), \mathcal{I}(0), \mathcal{R}(0)) + \mathbf{K}\Upsilon. \end{aligned} \tag{2.7}$$

$1_{\Omega(\omega)}$ denotes the Ω. Then, f tends to ∞, to reach the contradiction $\infty > V(\mathcal{S}(0), \mathcal{V}(0), \mathcal{I}(0), \mathcal{R}(0)) + \mathbf{MT} = \infty$. Therefore, the system has unique well posedness. □

3. Extinction for the system

To reduce the impact of a disease in the society over time, certain necessary conditions are derived from studying the dynamic behavior of the epidemic. In this section, we will explore the necessary conditions for the eradication of the disease through stochastic techniques. For the stochastic system 1.1, the threshold value R_0, can be shown as

$$\mathbf{R}_0 = \frac{\Lambda\beta}{d(\delta + d + \frac{\alpha_3^2}{2} + \int_{\mathcal{X}}[p_3(\mathcal{K}) - \ln(1 + p_3(\mathcal{K})]n(d\mathcal{K})}. \tag{3.1}$$

Here, the following notations are assigned:

$$\langle z\rangle = \frac{1}{t}\int_0^t z(r)dr.$$

Lemma 1. *If model (1.1), $(\mathcal{S}(t), \mathcal{V}(t), \mathcal{I}(t), \mathcal{R}(t))$ are the root with initial approximation $(\mathcal{S}(0), \mathcal{V}(0), \mathcal{I}(0), \mathcal{R}(0)) \in \mathbb{R}^4_+$, then a.s.,*

$$\lim_{t\to\infty}\frac{\mathcal{N}(t)}{t} = 0. \tag{3.2}$$

Moreover, if $d > \frac{1}{2}(\alpha_1^2 \vee \alpha_2^2 \vee \alpha_3^2 \vee \alpha_4^2)$, then

$$\begin{aligned}
&\lim_{t\to\infty}\frac{1}{t}\int_0^t \mathcal{S}(s)d\mathcal{W}_1(s) = 0,\\
&\lim_{t\to\infty}\frac{1}{t}\int_0^t \mathcal{V}(s)d\mathcal{W}_2(s) = 0,\\
&\lim_{t\to\infty}\frac{1}{t}\int_0^t \mathcal{I}(s)d\mathcal{W}_3(s) = 0,\\
&\lim_{t\to\infty}\frac{1}{t}\int_0^t \mathcal{R}(s)d\mathcal{W}_4(s) = 0 \quad a.s.
\end{aligned} \tag{3.3}$$

Theorem 3.1. *Assume $(\mathcal{S}, \mathcal{V}, \mathcal{I}, \mathcal{R})$* is the root of model (1.1) with starting approximation $(\mathcal{S}(0), \mathcal{V}(0), \mathcal{I}(0), \mathcal{R}(0)) \in \mathbb{R}^4$. Further, If $d > \frac{\alpha_1^2\vee\alpha_2^2\vee\alpha_3^2\vee\alpha_4^2}{2}$, and $\boldsymbol{R}_0$, is less than 1, then

$$\lim_{t\to\infty}\frac{\ln\langle\mathcal{I}(t)\rangle}{t} < 0, \ a.s.$$

Here, the exponential mapping of $\mathcal{I}(t)$ goes to 0 a.s., demonstrating that the illness vanishes with chance one. Moreover,

$$\begin{aligned}
&\lim_{t\to\infty}\ln\langle\mathcal{S}(t)\rangle = \frac{\Lambda}{(d+\rho)},\\
&\lim_{t\to\infty}\ln\langle\mathcal{V}(t)\rangle = \frac{\rho\Lambda}{(\delta_1 + d)(\rho + d)},\\
&\lim_{t\to\infty}\ln\langle\mathcal{I}(t)\rangle = 0,\\
&\lim_{t\to\infty}\ln\langle\mathcal{R}(t)\rangle = \frac{\delta_1\rho\Lambda}{(\delta_1 + d)(d+\rho)}.
\end{aligned} \tag{3.4}$$

Proof. Using the suggested system 1.1 in direct integration, we get the following calculations:

$$\begin{aligned}
\frac{\mathcal{S}(t)}{t} - \frac{\mathcal{S}(0)}{t} &= \Lambda - \frac{\beta \langle \mathcal{I}(t)\rangle \langle \mathcal{S}(t)\rangle}{\langle \mathcal{N}(t)\rangle} - (d+\rho)\langle \mathcal{S}(t)\rangle + \frac{\alpha_1}{t}\int_0^t \mathcal{S}(s)d\mathcal{W}_1(s) \\
&\quad + \frac{1}{t}\int_0^t \Big[\int_{\mathcal{X}} p_1(\rho)\mathcal{S}(t^-)\widetilde{\mathcal{D}}(dt, d\mathcal{K})\Big]ds, \\
\frac{\mathcal{V}(t) - \mathcal{V}(0)}{t} &= \rho\langle \mathcal{S}(t)\rangle - \frac{\beta_1 \langle \mathcal{V}(t)\rangle \langle \mathcal{I}(t)\rangle}{\langle \mathcal{N}(t)\rangle} - (d+\delta_1)\langle \mathcal{V}(t)\rangle + \frac{\alpha_2}{t}\int_0^t \mathcal{V}(s)d\mathcal{W}_2(s) \\
&\quad + \frac{1}{t}\int_0^t \Big[\int_{\mathcal{X}} p_2(\mathcal{K})\mathcal{V}(t^-)\widetilde{\mathcal{D}}(dt, d\mathcal{K})\Big]ds, \\
\frac{\mathcal{I}(t)}{t} - \frac{\mathcal{I}(0)}{t} &= \frac{\beta \langle \mathcal{S}(t)\rangle \langle \mathcal{I}(t)\rangle}{\langle \mathcal{N}(t)\rangle} - \frac{\beta_1 \langle \mathcal{V}(t)\rangle \langle \mathcal{I}(t)\rangle}{\langle \mathcal{N}(t)\rangle} - (d+\delta)\langle \mathcal{I}(t)\rangle + \frac{\alpha_3}{\tau}\int_0^t \mathcal{I}(s)d\mathcal{W}_3(s) \\
&\quad + \frac{1}{t}\int_0^t \Big[\int_{\mathcal{X}} p_3(\mathcal{K})\mathcal{I}(t^-)\widetilde{\mathcal{D}}(dt, d\mathcal{K})\Big]ds, \\
\frac{\mathcal{R}(t)}{t} - \frac{\mathcal{R}(0)}{t} &= \delta_1\langle \mathcal{V}(t)\rangle + \delta\langle \mathcal{I}(t)\rangle\rangle\mathcal{R}(t)\rangle + \frac{\alpha_4}{t}\int_0^t \mathcal{R}(s)d\mathcal{W}_4(s) \\
&\quad + \frac{1}{t}\int_0^t \Big[\int_{\mathcal{X}} p_4(\mathcal{K})\mathcal{R}(t^-)\widetilde{\mathcal{D}}(dt, d\mathcal{K})\Big]ds.
\end{aligned} \tag{3.5}$$

Equation (3.5) makes it easy to calculate the value of $\langle \mathcal{S}\rangle$.

$$\begin{aligned}
d\langle \mathcal{S}\rangle &= \Lambda + \frac{\alpha_1}{t}\int_0^t \mathcal{S}(s)d\mathcal{W}_1(s) + \frac{1}{t}\int_0^t \Big[\int_{\mathcal{X}} p_1(\mathcal{K})\mathcal{S}(t^-)\widetilde{\mathcal{D}}(dt, d\mathcal{K})\Big]ds \\
&\quad + \frac{\alpha_2}{t}\int_0^t \mathcal{V}(s)d\mathcal{W}_2(s) + \frac{1}{t}\int_0^t \Big[\int_{\mathcal{X}} p_2(\mathcal{K})\mathcal{S}(t^-)\widetilde{\mathcal{D}}(dt, d\mathcal{K})\Big]ds \\
&\quad + \frac{\alpha_3}{t}\int_0^t \mathcal{I}(s)d\mathcal{W}_3(s) + \frac{1}{t}\int_0^t \Big[\int_{\mathcal{X}} p_3(\mathcal{K})\mathcal{S}(t^-)\widetilde{\mathcal{D}}(dt, d\mathcal{K})\Big]ds \\
&\quad -(d+\delta)\langle \mathcal{I}\rangle - d\langle \mathcal{S}\rangle - (\delta_1 + d)\langle \mathcal{V}\rangle \\
&\quad -\Big[\frac{\mathcal{N} - \mathcal{R}(0)}{t} + \frac{\mathcal{N}(0) - \mathcal{R}(0)}{t}\Big].
\end{aligned} \tag{3.6}$$

Computation becomes

$$\langle \mathcal{S}(t)\rangle = \frac{\Lambda}{d} - \frac{(\delta_1 + d)}{d}\langle \mathcal{V}\rangle - \frac{(\delta + d)}{d}\langle I\rangle + \Omega(t), \tag{3.7}$$

where

$$\begin{aligned}
\Omega(t) &= \frac{1}{d}\Big\{\frac{\alpha_1}{t}\int_0^t \mathcal{S}(s)d\mathcal{W}_1(s) + \frac{1}{t}\int_0^t \Big[\int_{\mathcal{X}} p_1(\mathcal{K})\mathcal{S}(t^-)\widetilde{\mathcal{D}}(dt, d\mathcal{K})\Big]ds \\
&\quad + \frac{\alpha_2}{t}\int_0^t \mathcal{V}(s)d\mathcal{W}_2(s) + \frac{1}{t}\int_0^t \Big[\int_{\mathcal{X}} p_2(\mathcal{K})\mathcal{S}(t^-)\widetilde{\mathcal{D}}(dt, d\mathcal{K})\Big]ds \\
&\quad + \frac{\alpha_3}{t}\int_0^t \mathcal{I}(s)d\mathcal{W}_3(s) + \frac{1}{t}\int_0^t \Big[\int_{\mathcal{X}} p_3(\mathcal{K})\mathcal{S}(t^-)\widetilde{\mathcal{D}}(dt, d\mathcal{K})\Big]ds \\
&\quad - \Big[\frac{\mathcal{N}(t) - \mathcal{R}(t)}{t} + \frac{\mathcal{N}(0) - \mathcal{R}(0)}{t}\Big]\Big\}.
\end{aligned} \tag{3.8}$$

To solve the third Equation of the model (1.1), we will utilize Ito's formula:$\mathbb{V} = \ln \mathcal{I}(t)$

$$d\ln \mathcal{I} = L\mathbb{V}dt + \alpha_3 d\mathcal{W}_3(t) + \int_{\mathcal{X}} [\ln(1 + p_3(\mathcal{K}))]\widetilde{\mathcal{D}}(dt, d\mathcal{K}), \tag{3.9}$$

where

$$L\mathbb{V} = \left[\frac{\beta\mathcal{S}}{\mathcal{N}} + \frac{\beta_1\mathcal{V}}{\mathcal{N}} - (\delta + d) - \frac{\alpha_3^2}{2}\right]dt - \int_{\mathcal{X}} [p_3(\mathcal{K}) - \ln(1 + p_3)]n(d\mathcal{K}). \tag{3.10}$$

By integrating Eq (3.9) and dividing with t over the interval $[0, t]$, we acquire

$$\begin{aligned}
\frac{\ln \mathcal{I}(t)}{t} - \frac{\ln \mathcal{I}(0)}{t} &= \frac{\beta\langle\mathcal{S}\rangle}{\langle\mathcal{N}\rangle} + \frac{\beta_1\langle\mathcal{V}\rangle}{\langle\mathcal{N}\rangle} - (d + \delta) + \frac{\alpha_3^2}{2} + \frac{\alpha_3}{t}\int_0^t d\mathcal{W}_3(s) \\
&\quad + \frac{1}{t}\int_{\mathcal{X}} [\ln(1 + p_3(\mathcal{K}))]\widetilde{\mathcal{D}}(dt, d\mathcal{K}) - \int_{\mathcal{X}} [p_3(\mathcal{K}) - \ln(1 + p_3(\mathcal{K}))]n(d\mathcal{K}) \\
&\leq \beta\,\langle\mathcal{S}\rangle + \beta_1 - (\delta + d) + \frac{\alpha_3^2}{2} + \frac{1}{t}\int_{\mathcal{X}} [\ln(1 + p_3(\mathcal{K}))]\widetilde{\mathcal{D}}(dt, d\mathcal{K}) \\
&\quad + \frac{\alpha_3}{t}\int_0^t d\mathcal{W}_3(s) - \int_{\mathcal{X}} [p_3(\mathcal{K}) - \ln(1 + p_3(\mathcal{K}))]n(d\mathcal{K}).
\end{aligned} \tag{3.11}$$

Using Eq (3.7), then

$$\begin{aligned}
\frac{\ln \mathcal{I}(t) - \ln \mathcal{I}(0)}{t} &\leq \beta\left[\frac{\Lambda}{d} - \frac{d + \delta_1}{d}\langle\mathcal{V}\rangle - \frac{(\delta + d)}{d}\langle\mathcal{I}\rangle + \Omega(t)\right] + \beta_1 - (\delta + d) - \frac{\alpha_3^2}{2} \\
&\quad + \frac{\alpha_3}{t}\int_0^t d\mathcal{W}_3(s) - \int_{\mathcal{X}} [p_3(\mathcal{K}) - \ln(1 + p_3(\mathcal{K}))]n(d\mathcal{K}) \\
&\quad \frac{1}{t}\int_{\mathcal{X}} [\ln(1 + p_3(\mathcal{K}))]\widetilde{\mathcal{D}}(dt, \mathcal{K}) \\
&\leq \frac{\beta\Lambda}{d} + \beta_1 - \left(\delta + d + \frac{\alpha_3^2}{2} + \int_{\mathcal{X}} [p_3(\mathcal{K}) - \ln(1 + p_3(\mathcal{K}))]n(d\mathcal{K})\right) \\
&\quad + \frac{\alpha_3}{t}\int_0^t d\mathcal{W}_3(s) + \frac{1}{t}\int_{\mathcal{X}} [\ln(1 + p_3(\mathcal{K}))]\widetilde{\mathcal{D}}(dt, d\mathcal{K}) + \beta\Omega(t).
\end{aligned} \tag{3.12}$$

Assume $\mathbb{M} = \frac{\alpha_3}{t}\int_0^t d\mathcal{W}_3(s) + \frac{1}{t}\int_{\mathcal{X}} [\ln(1 + p_3(\mathcal{K}))]\widetilde{\mathcal{D}}(dt, d\mathcal{K})$, which is commonly referred to as the continuous locally martingale if $\mathbb{M}(0) = 0$, and in the same way $\Omega(0) = 0$,

By using (Lemma 1) and t tending to ∞, we have

$$\limsup_{t\to\infty} \frac{\Omega(t)}{t} = 0, \textit{and} \limsup_{t\to\infty} \frac{\mathbb{M}(t)}{t} = 0. \tag{3.13}$$

If $\mathbb{R}_0$ is less than 1, Eq (3.11) will be transformed

$$\limsup_{t\to\infty} \frac{\ln \mathcal{I}(t)}{t} \leq \left(d + \delta + \frac{\alpha_3^2}{2} + \int_{\mathcal{X}} [p_3(\mathcal{K}) - \ln(1 + p_3)]n(d\mathcal{K})\right)(R_0 - 1) < 0,\ a.s. \tag{3.14}$$

Equation (3.14) above indicates that

$$\lim_{t\to\infty} \mathcal{I}(t) = 0\ \ a.s. \tag{3.15}$$

Furthermore, in order to solve the second Equation of model (1.1), we have to use Eq (3.15). Taking integration from 0 to t, and then dividing the final computation by t, we acquire

$$\langle R\rangle = \frac{\rho\Lambda\delta_1}{(d+\delta_1)(\rho+d)}. \tag{3.16}$$

Which proves the conclusion. □

4. Persistence in mean

The long-term effects of the illness are covered in this section. First, we will introduce the average persistency, which is presented in Khan et al. [20].

Definition 1. Din and Li [33] and Zhao and Jiang [34] made the following assumptions, which are related to the maintenance or persistence of system (1.1).

$$\liminf_{t\to\infty} \frac{1}{t}\int_0^t \mathcal{I}(s)ds > 0 \; a.s. \tag{4.1}$$

To verify the persistence of the epidemic, one may additionally need to meet the following basic outcomes, which are specified in El Fatini and Sekkak [32].

Lemma 2. *[(Strong law) [20, 34]]. If a mapping that is continuous, $\mathbb{M} = \mathbb{M}_{0\le t}$, exists as a local martingale such that it vanishes as t tends to* 0*, then*

$$\begin{aligned} &\lim_{t\to\infty}\langle \mathbb{S},\mathbb{S}\rangle_t = \infty, \; a.s., \\ &\Rightarrow \lim_{t\to\infty}\frac{\mathbb{S}t}{\langle \mathbb{S},\mathbb{S}\rangle t} = 0, \; a.s. \\ \textit{also} \\ &\limsup_{t\to\infty}\frac{\langle \mathbb{S},\mathbb{S}\rangle_t}{t} < 0, \; a.s., \\ &\Rightarrow \lim_{t\to\infty}\frac{\mathbb{S}_t}{t} = 0, \; a.s. \end{aligned} \tag{4.2}$$

Lemma 3. *Let $b \in c([0,\infty)\times\Omega,(0,\infty))$ and $\mathbb{B} \in c([0,\infty)\times\Omega,\mathbb{R})\exists \lim_{t\to\infty}\frac{\mathbb{B}(t)}{t} = 0$ a.s. if*

$$\ln b(t) \ge \eta_0 t - \eta\int_0^t b(r)dr + \mathbb{B}(t) \; a.s.$$

Then

$$\liminf_{t\to\infty}\langle b(t)\rangle \ge \eta_0\backslash\eta \; a.s.$$

Where $\{\eta,\eta_0 \in \mathbb{R} \ni \eta > 0 \;\&\; \eta_0 \ge 0\}$ *[32, 33].*

The following theorem states that the correct task of the given section is to show the assumptions for the persistence in the mean of model (1.1).

Theorem 4.1. If $\mathbf{R}0^s > 1$, then for initial approximation $(\mathcal{S}(0), \mathcal{V}(0), \mathcal{I}(0), \mathcal{R}(0)) \in \mathbb{R}+^4$, the epidemic $\mathcal{I}(t)$ may have an axiom

$$\liminf_{t\to\infty} \langle \mathcal{I} \rangle \geq \frac{2\Lambda\sqrt{\boldsymbol{R}_0^s} - 1}{g_1\beta} \; a.s. \tag{4.3}$$

In the same way, $\liminf_{t\to\infty} \langle \mathcal{I} \rangle \geq 0$, where $g_1 = \frac{\Lambda}{\left(\rho+d+\frac{\alpha_1^2}{2}+\int_{\mathcal{X}}[p_1(\mathcal{K})-\ln(1+p_1(\mathcal{K}))]n(d\mathcal{K})\right)}$. Then, we can conclude that the disease will exist within the society.

Let us define

$$\boldsymbol{R}0^s = \frac{\Lambda\beta}{d\left(\delta_1 + d + \frac{\alpha_3^2}{2} + \int \mathcal{X}[p_3(\mathcal{K}) - \ln(1 + p_3(\mathcal{K}))]n(d\mathcal{K})\right)}. \tag{4.4}$$

Proof.

$$Let\ \mathbb{G}_1 = -g_1 \ln \mathcal{S} - g_2 \ln \mathcal{I}. \tag{4.5}$$

The constants g_1 and g_2 are currently presented and will be discussed later.
By applying Itos formula on Eq (4.5), we get

$$\begin{aligned} d\mathbb{G}_1 = {} & L\mathbb{G}_1 - g_1\alpha_1 d\mathcal{W}_1(t) - g_2\alpha_3 d\mathcal{W}_3(t) \\ & - g_1 \int_{\mathcal{X}} [p_1(\mathcal{K}) - \ln(1 + p_1(\mathcal{K})]\widetilde{\mathcal{D}}(dt, d\mathcal{K}) \\ & - g_2 \int_{\mathcal{X}} [p_3(\mathcal{K}) - \ln(1 + p_3(\mathcal{K}))]\widetilde{\mathcal{D}}(dt, d\mathcal{K}), \end{aligned} \tag{4.6}$$

where

$$\begin{aligned} L\mathbb{G}_1 &= g_1 L(-\ln \mathcal{S}) + g_2 L(-\ln \mathcal{I}), \\ L\mathbb{G}_1 &= -g_1\frac{\Lambda}{\mathcal{S}} + g_1\frac{\beta\mathcal{I}(t)}{\mathcal{N}(t)} - g_2\frac{\beta\mathcal{S}(t)}{\mathcal{N}(t)} - g_2\frac{\beta_1\mathcal{V}(t)}{\mathcal{N}(t)} \\ &\quad + g_1(\rho + d + \frac{\alpha_1^2}{2}) + g_2(\delta + d + \frac{\alpha_3^2}{2}) \\ &\quad + g_1 \int_{\mathcal{X}} [p_1(\mathcal{K}) - \ln(1 + p_1(\mathcal{K}))]n(d\mathcal{K}) \\ &\quad + g_2 \int_{\mathcal{X}} [p_3(\mathcal{K}) - \ln(1 + p_3(\mathcal{K}))]n(d\mathcal{K}), \\ &\leq \left(-g_1\frac{\Lambda}{\mathcal{S}(t)} - g_2\frac{\beta\mathcal{S}(t)}{\mathcal{N}(t)}\right) + g_1\beta\mathcal{I}(t) \\ &\quad + g_1\left(\rho + d + \frac{\alpha_1^2}{2} + \int_{\mathcal{X}} [p_1(\mathcal{K}) - \ln(1 + p_1(\mathcal{K}))]n(d\mathcal{K})\right) \\ &\quad + g_2\left(\delta + d + \frac{\alpha_3^2}{2} + \int_{\mathcal{X}} [p_3(\mathcal{K}) - \ln(1 + p_3(\mathcal{K}))]n(d\mathcal{K})\right) \end{aligned}$$

$$
\begin{aligned}
&\leq -2\Big(g_1\frac{\Lambda}{\mathcal{S}(t)}\times\ g_2\frac{\beta\mathcal{S}(t)}{\mathcal{N}(t)}\Big)^{\frac{1}{2}}+g_1\beta\mathcal{I}(t)\\
&\quad+g_1\Big(\rho+d+\frac{\alpha_1^2}{2}+\int_{\mathcal{X}}[p_1(\mathcal{K})-\ln(1+p_1(\mathcal{K}))]n(d\mathcal{K})\Big)\\
&\quad+g_2\Big(\delta+d+\frac{\alpha_3^2}{2}+\int_{\mathcal{X}}[p_3(\mathcal{K})-\ln(1+p_3(\mathcal{K}))]n(d\mathcal{K})\Big).
\end{aligned}
\tag{4.7}
$$

Assume

$$
\begin{aligned}
g_1&=\frac{\Lambda}{\rho+d+\frac{\alpha_1^2}{2}+\int_{\mathcal{X}}[p_1(\mathcal{K})-\ln(1+p_1(rho))]n(d\mathcal{K})},\\
g_2&=\frac{\Lambda}{\delta+d+\frac{\alpha_3^2}{2}+\int_{\mathcal{X}}[p_3(\mathcal{K})-\ln(1+p_3(\mathcal{K}))]n(d\mathcal{K})}.
\end{aligned}
\tag{4.8}
$$

Let

$$
\begin{aligned}
a&=\rho+d+\frac{\alpha_1^2}{2}+\int_{\mathcal{X}}[p_1(\mathcal{K})-\ln(1+p_1(\mathcal{K}))]n(d\mathcal{K}),\\
b&=\delta+d+\frac{\alpha_3^2}{2}+\int_{\mathcal{X}}[p_3(\mathcal{K})-\ln(1+p_3(\mathcal{K}))]n(d\mathcal{K}).
\end{aligned}
$$

$$
\begin{aligned}
L\mathbb{G}_1&\leq -2\sqrt{\frac{\beta\Lambda^3}{ab}}+2\Lambda+g_1\beta\mathcal{I},\\
L\mathbb{G}_1&\leq -2\Lambda\Big[\sqrt{\frac{\beta\Lambda}{ab}}-1\Big]+g_1\beta\mathcal{I},\\
L\mathbb{G}_1&\leq -2\Lambda\Big[\sqrt{\mathbf{R}_0^s}-1\Big]+g_1\beta\mathcal{I}.
\end{aligned}
\tag{4.9}
$$

Integrating both sides of the stochastic system (1.1) after inserting the value of Eq (4.9) into Eq (4.5):

$$
\begin{aligned}
\frac{\mathbb{G}_1(\mathcal{S}(t),\mathcal{I}(t))}{t}-\frac{\mathbb{G}_1(\mathcal{S}(0),\mathcal{I}(0))}{t}&\leq -2\Lambda\Big[\sqrt{\mathbf{R}_0^s}-1\Big]+g_1\beta\langle\mathcal{I}\rangle\\
&\quad-\frac{g_1\alpha_1\mathcal{W}_1(t)}{t}-\frac{g_2\alpha_1\mathcal{W}_3(t)}{t}\\
&\quad-\frac{g_1\int_{\mathcal{X}}[p_1(\mathcal{K})-\ln(1+p_1(\mathcal{K}))]\widetilde{\mathcal{D}}(dt,d\mathcal{K})}{t}\\
&\quad-\frac{g_2\int_{\mathcal{X}}[p_3(\mathcal{K})-\ln(1+p_3(\mathcal{K}))]\widetilde{\mathcal{D}}(dt,d\mathcal{K})}{t}\\
&\leq -2\Lambda\Big[\sqrt{\mathbf{R}_0^s}-1\Big]+g_1\beta\langle\mathcal{I}\rangle+\mathbb{Z}(t),
\end{aligned}
\tag{4.10}
$$

where

$$\mathbb{Z}(t) = -\frac{g_1\alpha_1\mathcal{W}_1(t)}{t} - \frac{g_2\alpha_3\mathcal{W}_3(t)}{t} - \frac{g_1\int_{\mathcal{X}}[p_1(\mathcal{K}) - \ln(1+p_1(\mathcal{K}))]\widetilde{\mathcal{D}}(dt,d\mathcal{K})}{t} - \frac{g_2\int_{\mathcal{X}}[p_3(\mathcal{K}) - \ln(1+p_3(\mathcal{K}))]\widetilde{\mathcal{D}}(dt,d\mathcal{K})}{t}.$$

Strong law, as expressed in lemma 2, gives us

$$\lim_{t\to\infty}\Phi(t) = 0$$

By Eq (4.10), we get

$$\langle\mathcal{I}(t)\rangle \geq \frac{2\Lambda\left[\sqrt{\mathbf{R}_0^s} - 1\right]}{g_1\beta} - \frac{\Phi(t)}{g_1\beta} + \frac{1}{g_1\beta}\Big(\frac{\mathbb{G}_1(\mathcal{S}(t) - \mathcal{S}(0)}{t}) - \frac{\mathbb{G}_1(\mathcal{I}(0) - \mathcal{I}(0)}{t})\Big). \tag{4.11}$$

By lemma 3, and taking the superior limit of Eq (2.2), we acquire

$$\liminf_{t\to\infty}\langle\mathcal{I}(t)\rangle \geq \frac{2\Lambda\left[\sqrt{\mathbf{R}_0^s} - 1\right]}{g_1\beta}. \tag{4.12}$$

The proof of theorem 4.1 is completed. □

5. Results and analysis

The numerical solution for three different cases of the ($\mathcal{SVIR}$) epidemic model by applying Bayesian regularization neural networks (BRNNs) is shown in this section. The numerical representations for solving three cases based on the three different initial conditions are listed in Table 1, while the other parameter values are $\Lambda = 0.2, \beta = 0.01, d = 0.31, \rho = 0.24, \beta_1 = 0.421, \delta = 0.1$, and $\delta_1 = 0.41$.

Table 1. Initial values for model 1.1.

	CASE 1	CASE 2	CASE 3
$\mathcal{S}_0$	80	100	120
$\mathcal{V}_0$	70	80	110
$\mathcal{I}_0$	40	60	80
$\mathcal{R}_0$	20	30	40

The numerical solutions by utilizing the BRNNs to solve the $\mathcal{SVIR}$ epidemic system were achieved using the MATLAB nftool command in combination with 10 neurons, 70% of the data used for training, and 15% used for validation and testing.

The MSE convergence for training, validation, epochs, testing, and complexity investigations provided in Table 2 is accomplished to solve the $\mathcal{SVIR}$ epidemic model.

Table 2. The differential model of $\mathcal{SVEIR}$ is evaluated using the $\mathcal{L}-\mathcal{MBNN}s$ method.

Case	MSE		Gradient	Performance	Epoch	Mu
	Training	Testing				
1	6.63×10^{-10}	7.36×10^{-10}	2.33×10^{-06}	6.63×10^{-10}	1000	5000
2	1.52×10^{-08}	1.89×10^{-08}	4.02×10^{-06}	1.53×10^{-08}	1000	5000
3	3.40×10^{-08}	8.0064×10^{-08}	2.14×10^{-05}	2.27×10^{-08}	1000	500

The graphic illustrations of the BRNNs to solve the $\mathcal{SVIR}$ epidemic model are shown in Figures 1–5. The appropriate numerical schemes of each scenario of the $\mathcal{SVIR}$ epidemic model are obtained in Figure 1(a, c, e) utilizing the performances. The proposed technique has the finest validation performance in Figure 1(a, c, e) since the error is minimized after a few training epochs, but may grow on the validation data set if the network starts to over fit the training data. The performance from the epoch with the lowest validation error is chosen as the best. The performance results for the $\mathcal{SVIR}$ epidemic model at epoch 1000 show the best results. These results are given as 6.63×10^{-10}, 1.5331×10^{-08}, and 2.2714×10^{-08}. Figure1(b, d, f) authenticates the gradient measures using the BRNNs to solve the dynamical $\mathcal{SVIR}$ epidemic model. The gradient performances are given as 2.33×10^{-06}, 4.02×10^{-06}, and 2.14×10^{-05}. These illustrations indicate the exactness, convergence, and accuracy of the proposed BRNNs to simulate the $\mathcal{SVIR}$ epidemic model. The fitting curve values for each variation of the $\mathcal{SVIR}$ epidemic model are shown in Figure 1(b, d, f), which shows a comparison between the solution of BRNNs and reference solutions.

Figure 2(a, c, e) displays the valuations of the results-based authentication for the dynamical $SVIR$ epidemic model targets, training outputs, error curves, test scores, and fitness. Figure 2(b, d, f) displays the zero error performances through the error histograms (EHs) values of test, train, and authentication for the dynamical $\mathcal{SVIR}$ epidemic system. The EHs are represented as 3.54×10^{-06}, 1.38×10^{-06}, and 3.0×10^{-06} for the dynamical SVIR epidemic model the dynamical form of the $\mathcal{SVIR}$ epidemic system. Figure 3 uses the train, authentication, and test measures for the dynamical $\mathcal{SVIR}$ epidemic model to authenticate the correlation operator values. The correlation is reported as a perfect representation of the model. Figures 4 and 5 provide a detailed comparison between the numerical solutions obtained from the proposed BRNNs technique and those derived from the RK method, along with the corresponding error plots. Specifically, Figure 4 shows, for each variation of the dynamical $\mathcal{SVIR}$ epidemic system, the accuracy of the BRNNs scheme using the comparison procedure. Figure 5 uses the BRNNs to authenticate the values of the absolute error (AE) for each variation of the dynamical $\mathcal{SVIR}$ epidemic model. The AE measures for the dynamical $\mathcal{SVIR}$ epidemic model's special predator category $\mathcal{S}(t)$ are 10^{-4} to 10^{-5}, 10^{-4} to 10^{-7}, and 10^{-3} to 10^{-5}, for cases 1, 2, and 3, respectively. The absolute error measure indicates the magnitude of the difference between the numerical results from the BRNNs technique and the RK method. An overall error of less than 10^{-7} signifies a high degree of accuracy and a strong agreement between the two sets of numerical results, confirming the effectiveness of the proposed BRNNs technique.

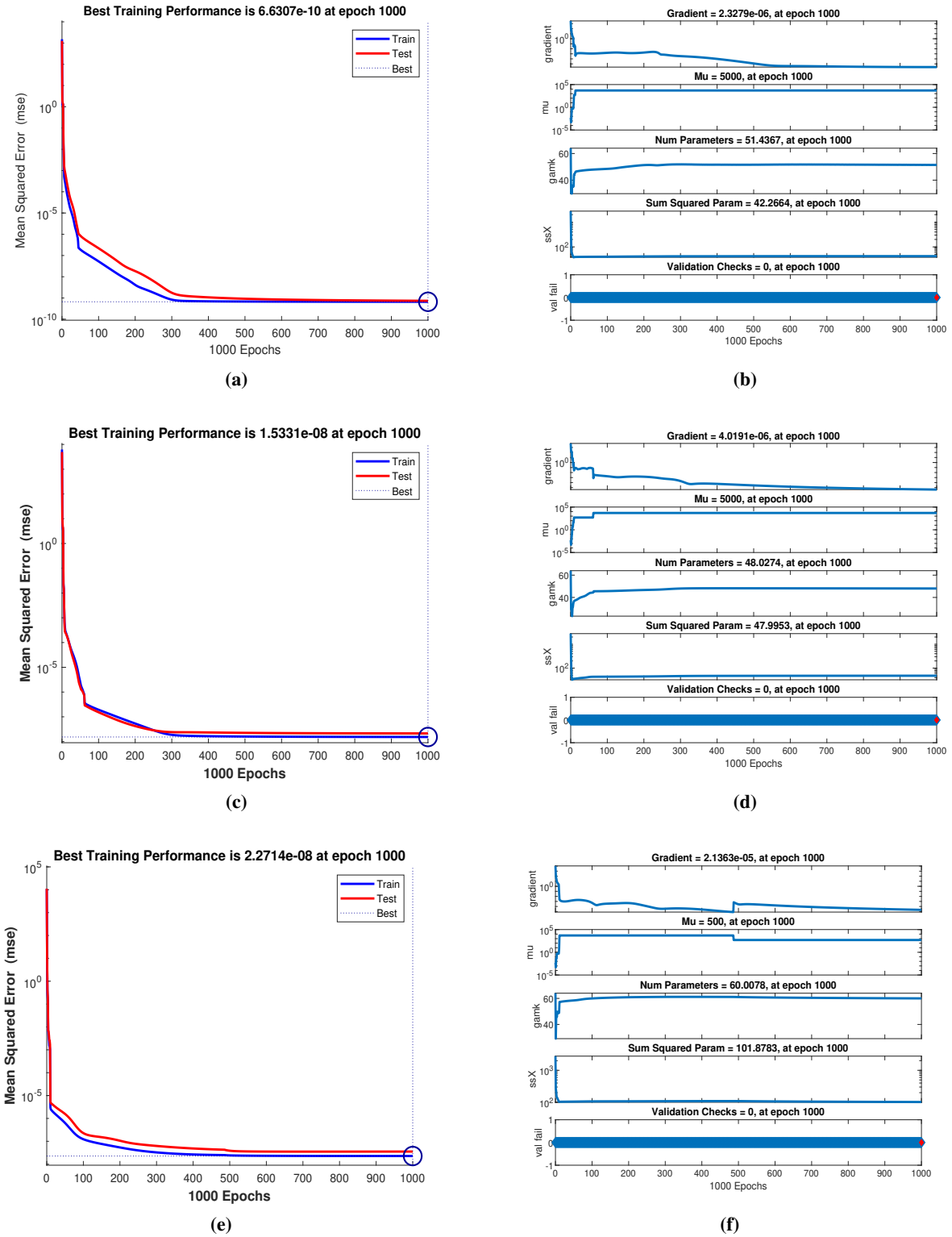

Figure 1. The dynamical $\mathcal{SVIR}$ epidemic model performance as solved by MSE and STs.

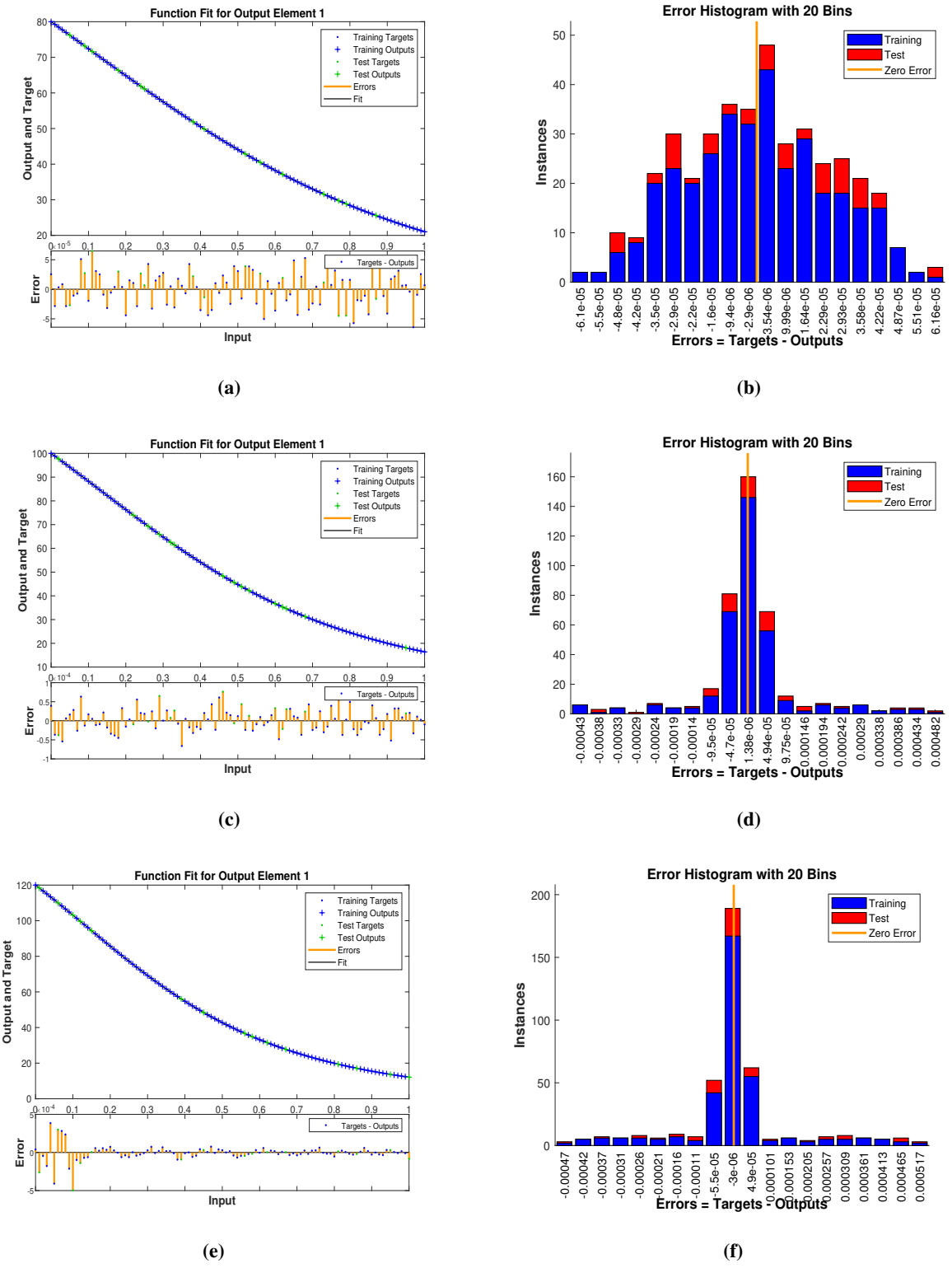

Figure 2. Error histograms measures and results estimations for the dynamical $\mathcal{SVIR}$ epidemic model.

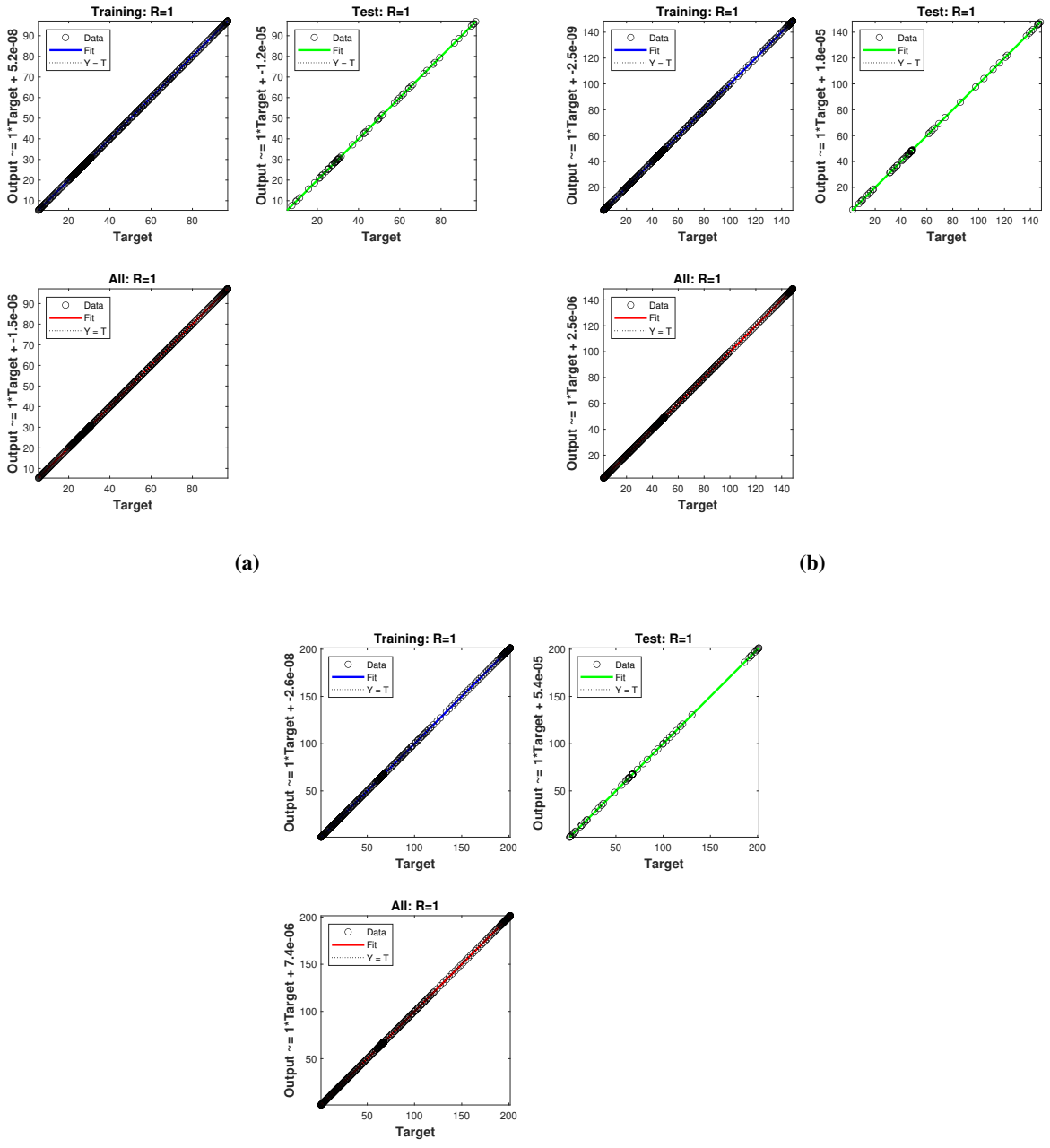

Figure 3. The performance of the regression for the dynamical $\mathcal{SVIR}$ epidemic model.

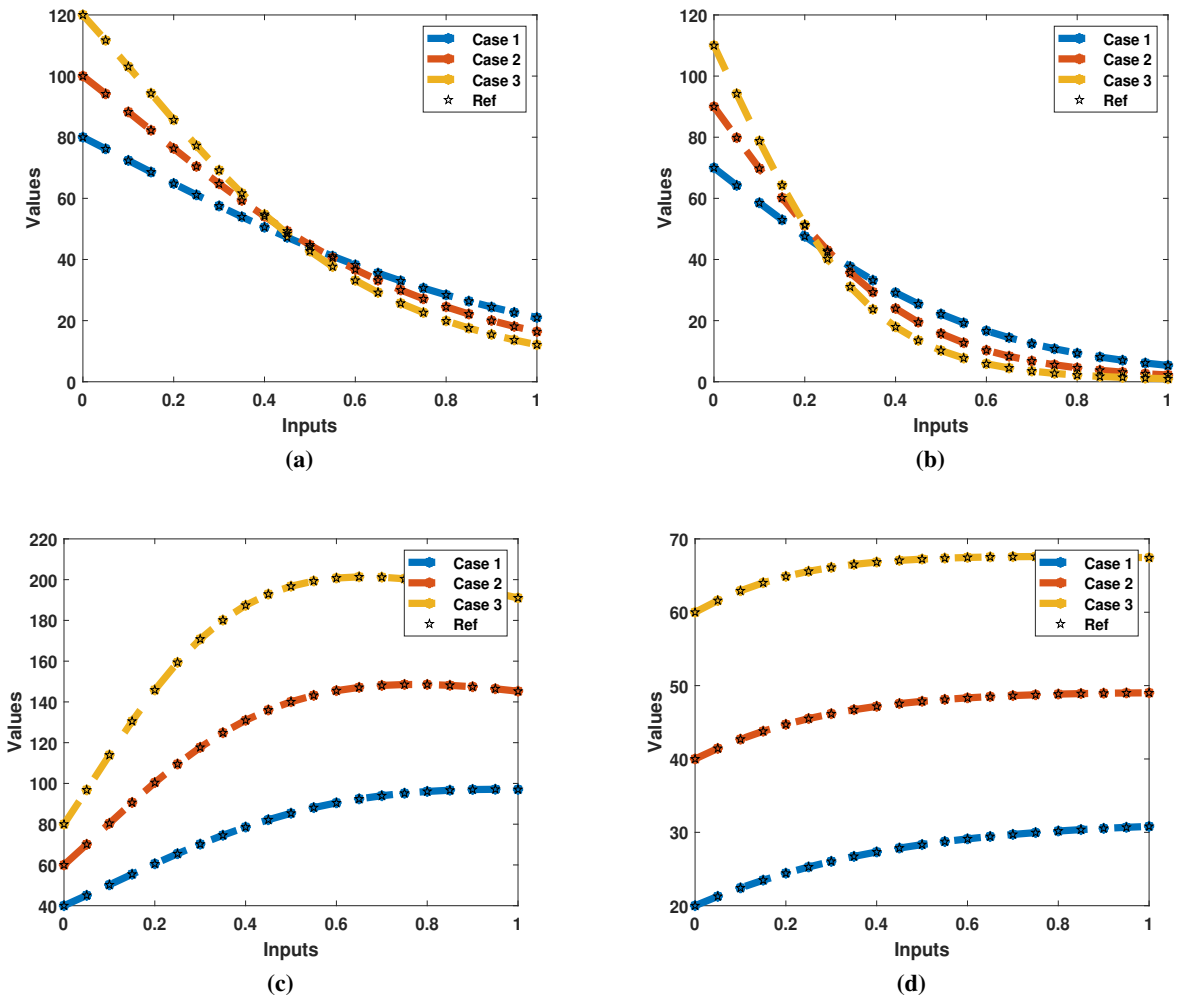

Figure 4. Achieved and reference results of the $\mathcal{SVIR}$ epidemic model.

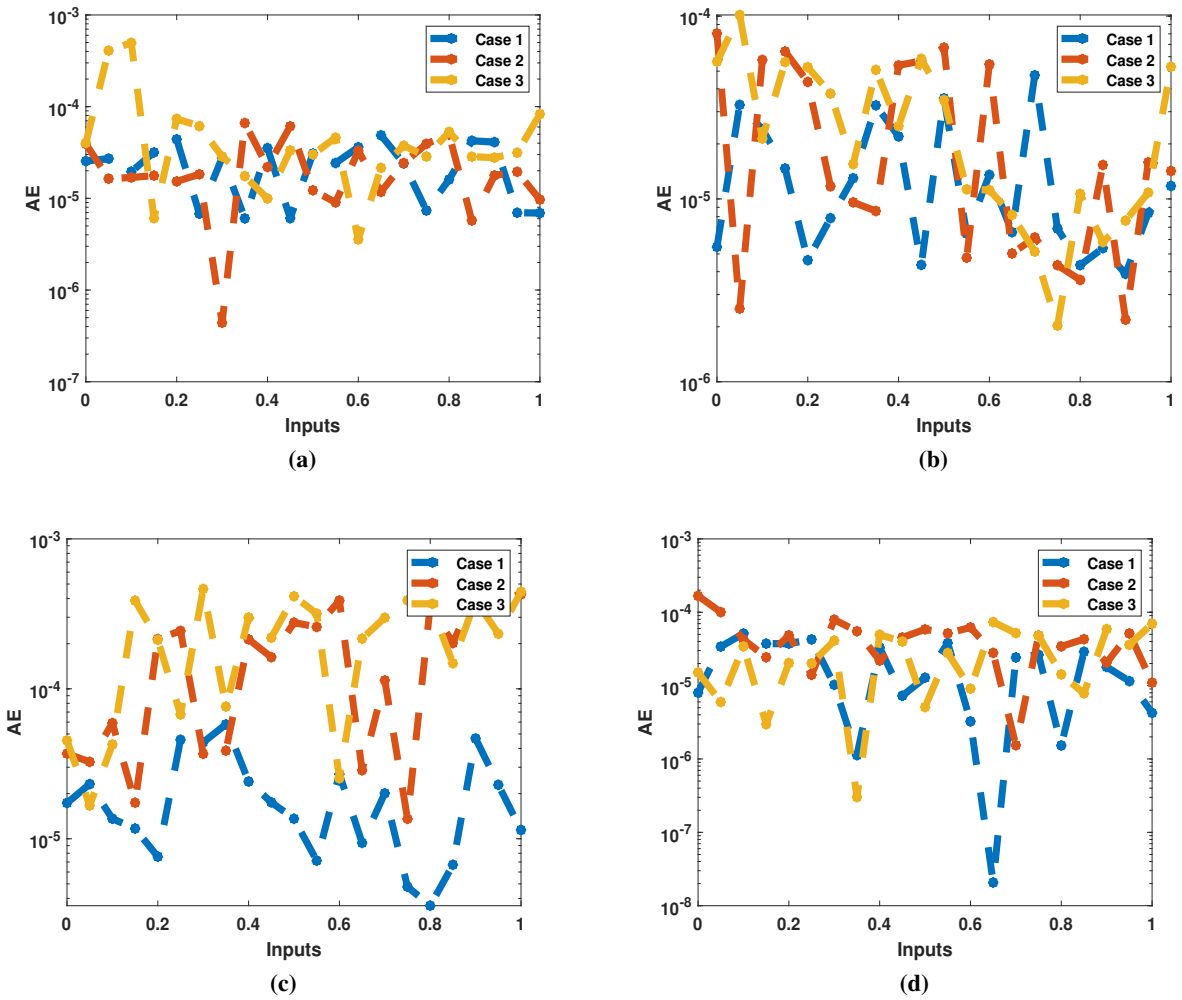

Figure 5. The values of the absolute error for the $\mathcal{SVIR}$ epidemic model.

6. Conclusions

Crucial conclusions can be made based on the research and analysis done in this article, which examined the effects of vaccination, Levy noises, and the dynamics of the infectious stochastic system on the $\mathcal{SVIR}$ epidemic model with a variable immunized system. The proposed $\mathcal{SVIR}$ epidemic model's non-negative and non-global solutions are analyzed as part of the phased process to achieve the research objectives. In order to account for this, the generalization of time and the realistic stochastic technique incorporate the external effect of Levy noises. The solution was obtained by constructing the Lyapunov function. We have conducted numerical investigations aimed at establishing a stochastic computing platform utilizing BRNNs for solving the $\mathcal{SVIR}$ epidemic model.

This research work will be implemented in the future by adding the time delay term to the stochastic version $\mathcal{SVIR}$ epidemic model.

Author contributions

R. Ikram: Formal analysis, Writing–original draft, Validation, Software; A. Khan: Conceptualization, Supervision, Visualization, Methodology; A. Raezah: Writing–review and editing, Investigation.

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 no conflict of interest.

References

1. P. Agarwal, R. Singh, Modelling of transmission dynamics of Nipah virus (Niv): a fractional order approach, *Physica A*, **547** (2020), 124243. https://doi.org/10.1016/j.physa.2020.124243
2. J. Amador, D. Armesto, A. Gómez-Corral, Extreme values in SIR epidemic models with two strains and cross-immunity, *Math. Biosci. Eng.*, **16** (2019), 1992–2022. https://doi.org/10.3934/mbe.2019098
3. K. Okuwa, H. Inaba, T. Kuniya, Mathematical analysis for an age-structured SIRS epidemic model, *Math. Biosci. Eng.*, **16** (2019), 6071–6102. https://doi.org/10.3934/mbe.2019304
4. S. Kim, J. H. Byun, I. H. Jung, Global stability of an SEIR epidemic model where empirical distribution of incubation period is approximated by Coxian distribution, *Adv. Differ. Equ.*, **2019** (2019), 469. https://doi.org/10.1186/s13662-019-2405-9
5. H. Qi, L. Liu, X. Meng, Dynamics of a nonautonomous stochastic SIS epidemic model with double epidemic hypothesis, *Complexity*, **2017** (2017), 4861391. https://doi.org/10.1155/2017/4861391
6. Q. Liu, D. Jiang, Dynamical behavior of a stochastic multigroup SIR epidemic model, *Physica A*, **526** (2019), 120975. https://doi.org/10.1016/j.physa.2019.04.211
7. Q. Liu, D. Jiang, Stationary distribution and extinction of a stochastic SIR model with nonlinear perturbation, *Appl. Math. Lett.*, **73** (2017), 8–15. https://doi.org/10.1016/j.aml.2017.04.021
8. Z. Liu, C. Tian, A weighted networked SIRS epidemic model, *J. Differ. Equations*, **269** (2020), 10995–11019. https://doi.org/10.1016/j.jde.2020.07.038
9. C. M. Kribs-Zaleta, J. X. Velasco-Hernández, A simple vaccination model with multiple endemic states, *Math. Biosci.*, **164** (2000), 183–201. https://doi.org/10.1016/S0025-5564(00)00003-1

10. X. Liu, Y. Takeuchi, S. Iwami, SVIR epidemic models with vaccination strategies, *J. Theor. Biol.*, **253** (2008), 1–11. https://doi.org/10.1016/j.jtbi.2007.10.014

11. S. M. A. Rahman, X. Zou, Modelling the impact of vaccination on infectious diseases dynamics, *J. Biol. Dyn.*, **9** (2015), 307–320. https://doi.org/10.1080/17513758.2014.986545

12. W. Halota, M. Muszyska, M. Pawowska, Hepatitis B virus serologic markers and anti-hepatitis B vaccination in patients with diabetes, *Med. Sci. Monit.*, **8** (2002), 516–519.

13. X. Duan, S. Yuan, X. Li, Global stability of an SVR model with age of vaccination, *Appl. Math. Comput.*, **226** (2014), 528–540. https://doi.org/10.1016/j.amc.2013.10.073

14. P. Raúl, C. Vargas-De-León, P. Miramontes, Global stability results in a SVIR epidemic model with immunity loss rate depending on the vaccine-age, *Abstr. Appl. Anal.*, **2015** (2015), 341854. http://doi.org/10.1155/2015/341854

15. Y. Geng, J. Xu, Stability preserving NSFD scheme for a multi-group SVIR epidemic model, *Math. Method. Appl. Sci.*, **40** (2017), 4917–4927. https://doi.org/10.1002/mma.4357

16. W. Li, Y. Ding, Stability and branching analysis of a class of time-delay SVIR model with saturation incidence, *Journal of Lanzhou University of Arts and Science (Natural Science Edition)*, **32** (2018), 1–6.

17. R. Zhang, S. Liu, Traveling waves for SVIR epidemic model with nonlocal dispersal, *Math. Biosci. Eng.*, **16** (2019), 1654–1682. https://doi.org/10.3934/mbe.2019079

18. S. Liao, W. Yang, A SVIR optimal control model with vaccination, (Chinese), *Journal of Southwest University (Natural Science)*, **37** (2015), 72–78. https://doi.org/10.13718/j.cnki.xdzk.2015.01.011

19. Z. Wang, R. Xu, Global dynamics of an SVIR epidemiological model with infection age and nonlinear incidence, *J. Biol. Syst.*, **25** (2017), 419–440. https://doi.org/10.1142/S0218339017500206

20. T. Khan, A. Khan, G. Zaman, The extinction and persistence of the stochastic hepatitis B epidemic model, *Chaos Soliton. Fract.*, **108** (2018), 123–128. https://doi.org/10.1016/j.chaos.2018.01.036

21. M. Song, W. Zuo, D. Jiang, T. Hayat, Stationary distribution and ergodicity of a stochastic cholera model with multiple pathways of transmission, *J. Frank. Inst.*, **357** (2020), 10773–10798. https://doi.org/10.1016/j.jfranklin.2020.04.061

22. Q. Liu, D. Jiang, T. Hayat, A. Alsaedi, B. Ahmad, Dynamical behavior of a higher order stochastically perturbed SIRI epidemic model with relapse and media coverage, *Chaos Soliton. Fract.*, **139** (2020), 110013. https://doi.org/10.1016/j.chaos.2020.110013

23. A. Lahrouz, A. Settati, A. Akharif, Effects of stochastic perturbation on the SIS epidemic system, *J. Math. Biol.*, **74** (2017), 469–498. https://doi.org/10.1007/s00285-016-1033-1

24. Z. Cao, W. Feng, X. Wen, L. Zu, M. Cheng, Dynamics of a stochastic SIQR epidemic model with standard incidence, *Physica A*, **527** (2019), 121180. https://doi.org/10.1016/j.physa.2019.121180

25. Y. Cai, Y. Kang, W. Wang, A stochastic SIRS epidemic model with nonlinear incidence rate, *Appl. Math. Comput.*, **305** (2017), 221–240. https://doi.org/10.1016/j.amc.2017.02.003

26. X.-B. Zhang, X.-H. Zhang, The threshold of a deterministic and a stochastic SIQS epidemic model with varying total population size, *Appl. Math. Model.*, **91** (2021), 749–767. https://doi.org/10.1016/j.apm.2020.09.050

27. S. Wang, G. Hu, T. Wei, L. Wang, Permanence of hybrid competitive Lotka-Volterra system with Lévy noise, *Physica A*, **540** (2020), 123116. https://doi.org/10.1016/j.physa.2019.123116

28. A. El Koufi, A. Bennar, N. Yousfi, Dynamics behaviors of a hybrid switching epidemic model with levy noise, *Appl. Math. Inform. Sci.*, **15** (2021), 131–142. http://dx.doi.org/10.18576/amis/150204

29. Y. Zhou, S. Yuan, D. Zhao, Threshold behavior of a stochastic SIS model with Levy jumps, *Appl. Math. Comput.*, **275** (2016), 255–267. https://doi.org/10.1016/j.amc.2015.11.077

30. Y. Liu, Y. Zhang, Q. Wang, A stochastic SIR epidemic model with Lévy jump and media coverage, *Adv. Differ. Equ.*, **2020** (2020), 70. https://doi.org/10.1186/s13662-020-2521-6

31. J. Wu, Dynamics of a two-predator one-prey stochastic delay model with Lévy noise, *Physica A*, **539** (2020), 122910. https://doi.org/10.1016/j.physa.2019.122910

32. M. El Fatini, I. Sekkak, Lévy noise impact on a stochastic delayed epidemic model with Crowly-Martin incidence and crowding effect, *Physica A*, **541** (2020), 123315. https://doi.org/10.1016/j.physa.2019.123315

33. A. Din, Y. Li, Stationary distribution extinction and optimal control for the stochastic hepatitis B epidemic model with partial immunity, *Phys. Scr.*, **96** (2021), 074005. https://doi.org/10.1088/1402-4896/abfacc

34. Y. Zhao, D. Jiang, The threshold of a stochastic SIRS epidemic model with saturated incidence, *Appl. Math. Lett.*, **34** (2014), 90–93. https://doi.org/10.1016/j.aml.2013.11.002

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