Journal of Mathematical Techniques in Modeling; Vol.1, Issue.1, 2024
https://doi.org/10.56868/jmtm.v1i1.31

Research Paper https://www.ufpub.com/index.php/jmtm/index

Stochastic Model on the Transmission of Worms in Wireless Sensor Network

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ABSTRACT

Due to their severe operating limitations, wireless sensor networks (WSNs) confront a major "Network Security" problem. The root of the problem is worm penetration into the wireless network. Worms may spread quickly and uncontrollably throughout the network from a single compromised node, infecting other nodes with the virus. In the present manuscript, a stochastic Susceptible-Infectious-vaccinated-Susceptible (SIVR) model for Wireless sensor networks is proposed. Firstly, we prove that the global positive solution exists and is unique. We then infer adequate circumstances for the malware to endure and to go extinct. Our results demonstrate that the introduction of sporadic environmental disturbances can prevent the malware from spreading. Stated differently, the deterministic model overestimates the ability of the malware to spread because it ignores unpredictable disturbances. To demonstrate the analytical results, numerical simulations are carried out. Comparing the proposed (SIVR) model to other models, it offers a better method of controlling the spread of worms

Keywords: Wireless Sensor Networks; Epidemic Model; Noise; Persistence

1 Introduction

The advancement of information technology has led to a noticeable rise in hostile activities that target wireless networks. These actions put people at risk in addition to posing a security risk to countries. People in the present day would benefit greatly from a wireless communication network that ensures efficiency, security, and dependability. An intelligent, reasonably priced, and small gadget is a sensor node in a wireless network. Mission-critical installations for recurring data gathering are just two uses for wireless sensor networks (WSNs). They have vital uses in many different domains, including tracking military targets, monitoring agricultural objects, managing disasters, exploring dangerous environments, monitoring pollution and the environment, detecting floods, tracking vehicles, monitoring traffic, detecting gas, monitoring water quality, seismic sensing, and applications in the healthcare industry (Akyildiz, Su, Sankarasubramaniam, [1–3]). Sensor nodes are, however, reasonably priced and clever gadgets. Moreover, based on sources [4, 5], they function within resource limits that include battery life, memory, and processing capacity restrictions. Because of their limited resources

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Received: January 26, 2024; **Received in the revised form:** March 7, 2024; **Accpted:** March 10, 2024
Available online: March 21, 2024

and decentralized architecture, it is therefore very difficult to establish wireless connections and provide security across these networks. As mentioned in reference [6], security risks are more common in wireless networks than in traditional networks because of their greater sensitivity. Although several security measures have been put in place to protect these networks from attacks, hackers are still able to take advantage of enduring software bugs and vulnerabilities. In the case of WSNs, the aforementioned difficulties are much more pressing. The reference [7] highlights that sensor nodes have a restricted communication range and send the data they have acquired in a multi-hop fashion. Reference [8] indicates that these limitations make sensor nodes less able to defend themselves against malware assaults, such as worms, viruses, and dangerous signals. Ensuring the network's viability requires effective control over the spread of worms. In light of this, research on mathematical modeling and understanding the propagation pattern of the harmful signals are essential, as suggested by [9–12] and references cited therein.

A vital and simple tool for analyzing and forecasting the dynamic behavior of different epidemics is mathematical modeling [13–16]. In reality, the force at which infection occurs is directly linked and is significantly influenced by various stochastic disturbance parameters such as precipitation, temperature, and absolute humidity. Recognizing this effect enables us to add randomness to deterministic biological models and so expose the effects of environmental variability, such as random noise in differential systems or oscillations in parameters [17–19]. In addition, stochastic models offer more realism and freedom than their comparable deterministic counterparts. Stochastic population dynamics affected by white noise (or Brownian motion) has been the subject of much research by several writers (see [20, 21] for references).

By clearly demonstrating and investigating the proposed SIVR model, this study makes a substantial contribution and gives researchers a useful tool for effectively communicating and handling the dynamics of worms within WSNs. Through the integration of perturbations of the white noise type, we reveal the influence of both ambient noise and parameter changes. The SIVR model is used in this article to clearly and succinctly explain the methods for limiting virus spread across network users. The suggestion clarifies the interaction of metamorphism which a node may have in different infectious stages. It conceptualizes the SIVR model and applies the idea of stochastic epidemic theory to study malware spread within the networks. Thorough simulation results provide unequivocal confirmation of the correctness of the suggested model and the analytical results carried out during the analysis.

The subsequent sections are arranged in the following order inside the manuscript. In Section 2, we provide the stochastic epidemic model pertaining to worm propagation within a wireless sensor network. The solution of the type of global positive of the underlying model and its dynamical properties are described in Section 3. We show that the worm epidemic experiences exponential extinction under certain conditions in Section 4. Establishing necessary criteria for the existence of persistence is the focus of Section 5. Part 6 contains numerical simulations that support the theory behind the obtained conclusions, which are confirmed both qualitatively and quantitatively. In Section 7, the study comes to a finish with some closing thoughts and suggestions for more investigation.

2 Proposed model

At any time t, a wireless sensor network with N nodes which are evenly dispersed across a certain region is assumed to formulate the model. It is further assumed that the worms propagate within the network like an epidemic spread within a human population. The average density of the nodes is ρ, uniformly distributed over a region of L^2. Around πr^2, each node may sense a region with a distance of r. At least one node must be located inside the covered zone in order for communication to occur. Every node has the ability to communicate information to the sink directly or via nearby nodes. At first, we take it for granted that every node in the network is vulnerable to virus attack, which means that worm assaults can target them. Four state variables are taken into consideration in this study: susceptible nodes $\mathsf{S}(t)$, which are vulnerable to malware attacks but have not yet contracted the

infection; infectious nodes $\mathsf{I}(t)$, which have contracted the infection and are able to infect other nodes; recovered nodes $\mathsf{R}(t)$, which have been outfitted with a detection tool to help them find and remove malware infections; and vaccinated nodes $\mathsf{V}(t)$. Every node has a sensing range of r and a potential spreading region of πr^2. The density of susceptible nodes per unit area is denoted by the expression $\rho(t) = \frac{S(t)}{L\times L}$. The expression $S'(t) = \frac{S(t)\pi r^2}{L^2}$ represents the size of the nodes inside a node's sensing region. To make things easier, let $\beta = \frac{\pi r^2}{L^2}\beta$ be the parameter. Given the following differential equation system, it may be used to explain the worm's behavior inside the network:

$$\begin{aligned}
\frac{d\mathsf{S}(t)}{dt} &= (1-p)\mu - \frac{\beta \mathsf{S}(t)\mathsf{I}(t)}{\mathsf{N}(t)} - \mu \mathsf{S}(t),\\
\frac{d\mathsf{I}(t)}{dt} &= \frac{\beta \mathsf{S}(t)\mathsf{I}(t)}{\mathsf{N}(t)} - (\gamma+\gamma_1+\mu)\mathsf{I}(t),\\
\frac{d\mathsf{V}(t)}{dt} &= p\mu - \mu \mathsf{V}(t),\\
\frac{d\mathsf{R}(t)}{dt} &= \gamma_1 \mathsf{I}(t) - \mu \mathsf{R}(t).
\end{aligned} \tag{2.1}$$

A detailed interpretation of the model' parameters is presented in Table 1.

Symbol	Description of symbol
p	Denote the vaccination rate
β	The rate of contacts that causes infection
μ	Represent the natural death
γ	Death Rate by the infection
γ_1	Recovery rate

Table 1: Interpretation of the parameters used in the model.

We need to set $\frac{d\mathsf{S}}{dt} = 0, \frac{d\mathsf{I}}{dt} = 0, \frac{d\mathsf{V}}{dt} = 0$ as well as $\frac{d\mathsf{R}}{dt} = 0$ in order to calculate the underlying equilibria of the model. Following the required computations, the worm-free equilibrium (WFE) value is as follows:

$$¶_0 = \left(\mathsf{S}^0, \mathsf{I}^0, \mathsf{V}^0, \mathsf{R}^0\right) = (1-p, 0, p, 0)\,.$$

The threshold number for the associated system of ODEs, denoted as $\mathbb{R}_0$, is obtained as follows:

$$\mathbb{R}_0 = \frac{\mu\beta(1-p)}{\mathsf{N}(\gamma+\gamma_1+\mu)}.$$

where $N = 1$ is assumed. The endemic equilibrium (EE), also known as the state in which the worm exists inside the network, is described as $¶^* = (\mathsf{S}^*, \mathsf{E}^*, \mathsf{I}^*, \mathsf{R}^*)$ and is defined by

$$\begin{aligned}
\mathsf{S}^* &= \frac{\mathsf{N}(\gamma+\gamma_1+\mu)}{\beta},\\
\mathsf{I}^* &= \frac{\mu(\beta(1-p)-(\gamma+\gamma_1+\mu))}{\beta(\gamma+\gamma_1+\mu)},\\
\mathsf{V}^* &= p,\\
\mathsf{R}^* &= \frac{\gamma_1}{\mu} I^*.
\end{aligned}$$

When addressing real-world situations, epidemics—whether caused by infectious illnesses or something else entirely—are prone to intricate and unpredictable fluctuations. Given the intrinsic unpredictability of epidemic events, using stochastic models to represent them would be a better strategy.

The introduction of the white noise model (2.1) is the main goal of the current effort. Next, the stochastically perturbed format of the deterministic system (2.1) may be represented as follows:

$$\begin{aligned} d\mathsf{S}(t) &= \left[(1-p)\mu - \frac{\beta \mathsf{S}(t)\mathsf{I}(t)}{\mathsf{N}(t)} - \mu \mathsf{S}(t)\right]dt + \eta_1 \mathsf{S}(t) d\mathsf{B}_1(t), \\ d\mathsf{I}(t) &= \left[\frac{\beta \mathsf{S}(t)\mathsf{I}(t)}{\mathsf{N}(t)} - (\gamma + \gamma_1 + \mu)\mathsf{I}(t)\right]dt + \eta_2 \mathsf{I} d\mathsf{B}_2(t), \\ d\mathsf{V}(t) &= \left[p\mu - \mu \mathsf{V}(t)\right]dt + \eta_3 \mathsf{V}(t) d\mathsf{B}_3(t), \\ d\mathsf{R}(t) &= \left[\gamma_1 \mathsf{I}(t) - \mu \mathsf{R}(t)\right]dt + \eta_4 \mathsf{R}(t) d\mathsf{B}_4(t). \end{aligned} \tag{2.2}$$

Here the fluctuating dynamics are represented by $\mathsf{B}_i(t)$ for $i = 1, \cdots, 4$, and the noise intensities are represented by η_1, η_2, η_3, and η_4. Undoubtedly, it includes the results of $\mathsf{B}_i(0) = 0$ for the values of $i = 1, 2, \cdots, 4$.

3 Qualitative Analysis of positive solution

Finding a nonlocal solution inside the permissible space is essential for examining the dynamic behavior of system (2.2). This may be achieved by confirming that the system's related parameters (2.2) fulfill the growth and Lipschitz criteria. Fulfilling these conditions ensures that the underlying model has a non-negative solution. Indeed, further analysis of Lyapunov function approaches is necessary for the exploration of positive and non-local solutions [12, 19, 23–28]. These methods offer insightful information on the system's convergence and stability characteristics, facilitating a more thorough comprehension of its behavior. To investigate the features described above, let us first express a crucial claim and then provide its proof.

Theorem 3.1. *Subject to a positive initial set of data for the state variables, a global solution* $(\mathsf{S}, \mathsf{I}, \mathsf{V}, \mathsf{R})(t) \in \mathbb{R}^4_+$ *for system (2.2) exist for all* $0 \leq t$ *a.s.*

Proof. The coefficients of system (2.2) are evidently continuous and Lipschitz locally, considering any an initial values $(\mathsf{S}_0, \mathsf{I}_0, \mathsf{V}_0, \mathsf{R}_0) \in \mathbb{R}^4_+$. Consequently, a unique local solution $(\mathsf{S}(t), \mathsf{I}(t), \mathsf{V}(t), \mathsf{R}(t))$ exists for $t \in [0, \tau_e)$, here τ_e represents the explosion time. In order to establish that infact this solution is global, it is necessary to demonstrate that $\tau_e = \infty$ almost surely. Choose a sufficiently large integer $k_0 \geq 0$ such that the initial values $(\mathsf{S}(0), \mathsf{I}(0), \mathsf{V}(0), \mathsf{R}(0))$ lie within the interval $\left[\frac{1}{k_0}, k_0\right]$. For every integer $k \geq k_0$, we define the following stopping time:

$$\tau_k = \inf\left\{t \in [0, \tau_e) : \min((\mathsf{S}(t), \mathsf{I}(t), \mathsf{V}(t), \mathsf{R}(t)) \leq \frac{1}{\mathrm{k}} \text{ or } \max\{((\mathsf{S}(t), \mathsf{I}(t), \mathsf{V}(t), \mathsf{R}(t))\} \geq \mathrm{k}\right\}.$$

Using $\inf \emptyset = \infty$ as a definition, we observe that $\tau_e \geq \tau^+$, suggesting that $\tau^+ = \infty$ is very certainly the case. This fact certainly shows that $\tau_e = +\infty$. Given that τ^+ is not infinite, $0 < \mathbb{P}(\tau^+ < T)$ must exist for some nonnegative number T and

$$P\{\tau_\infty \leq T\} > \epsilon.$$

As a result, there exists a real number $k_0 \leq k_1$ for which

$$P\{T \geq \tau_k\} > \epsilon \ \forall \ \mathrm{k} \geq \mathrm{k}_1. \tag{3.1}$$

To proceed further, consider a function (a C^2-function) $V : \mathbb{R}^4_+ \to \overline{\mathbb{R}}_+$ where

$$\{x : x \text{ is non-negative real number}\} = \overline{\mathbb{R}}_+,$$

by

$$V(S,I,V,R) = (-1+S-\ln S)+(-1+I-\ln I)+(-1+V-\ln V)+(-1+R-\ln R) + \int_0^t \zeta I(s)ds.$$

By employing the *Itô* formula, we derive

$$dV = LVdt + (S-1)\eta_1 dB_1(t) + (I-1)\eta_2 dB_2(t) + (V-1)\eta_3 dB_3(t) + (R-1)\eta_4 dB_4(t),$$

where

$$\begin{aligned}
LV &= (1-\frac{1}{S})((1-p)\mu - \frac{\beta SI}{N} - \mu S) + \frac{\eta_1^2}{2} + (1-\frac{1}{I})(\frac{\beta SI}{N} - (\gamma+\gamma_1+\mu)I) + \frac{\eta_2^2}{2} \\
&+ (1-\frac{1}{V})(p\mu - \mu V) + \frac{\eta_3^2}{2} + (1-\frac{1}{R})(\gamma_1 I - \mu R) + \frac{\eta_4^2}{2}, \\
&= (1-p)\mu - \frac{\beta SI}{N} - \mu S - \frac{(1-p)\mu}{S} + \frac{\beta I}{N} + \mu + \frac{\beta SI}{N} - (\gamma+\gamma_1+\mu)I - \frac{\beta S}{N} + (\gamma+\gamma_1+\mu) \\
&+ p\mu - \mu V - \frac{p\mu}{V} + \mu + \gamma_1 I - \mu R - \frac{\gamma_1 I}{R} + \mu + \frac{\eta_1^2+\eta_2^2+\eta_3^2+\eta_4^2}{2}, \\
&= 5\mu + \frac{\beta I}{N} + \gamma + \gamma_1 + \frac{\eta_1^2+\eta_2^2+\eta_3^2+\eta_4^2}{2}, \\
&\leq 5\mu + \beta + \gamma + \gamma_1 + \frac{\eta_1^2+\eta_2^2+\eta_3^2+\eta_4^2}{2}.
\end{aligned}$$

We have,

$$LV \leq 5\mu + \beta + \gamma + \gamma_1 + \frac{\eta_1^2+\eta_2^2+\eta_3^2+\eta_4^2}{2} := M. \tag{3.2}$$

The remaining work for the proof can be same as in Theorem 2.1 of [24], therefore we skip it. ∎

4 Extinction analysis of the worm-free equilibrium

The investigation of the system's extinction (2.2) and the establishment of a threshold to ascertain if the disease will eventually disappear or continue are the main topics of this portion of the work. It infers the circumstances that caused the illness to go extinct. Just to be clear, let's define

$$\langle X(t)\rangle = \frac{1}{t}\int_0^t X(s)\,ds.$$

Lemma 4.1. *Let* $(S(t), I(t), V(t), R(t))$ *be the solution of the system (2.2) with initial value* $(S(0), I(0), V(0), R(0)) \in \mathbb{R}^4_+$, *then* $\limsup_{t\to\infty}(S(t)+I(t)+V(t)+R(t)) < \infty$.

Moreover,

$$\lim_{t\to\infty}\frac{S(t)}{t} = 0, \quad \lim_{t\to\infty}\frac{I(t)}{t} = 0. \lim_{t\to\infty}\frac{V(t)}{t} = 0, \quad \lim_{\to\infty}\frac{R(t)}{t} = 0, \quad a.s.$$

$$\lim_{t\to\infty}\frac{\ln S(t)}{t} = 0, \quad \lim_{t\to\infty}\frac{\ln I(t)}{t} = 0. \lim_{t\to\infty}\frac{\ln V(t)}{t} = 0, \quad \lim_{\to\infty}\frac{\ln R(t)}{t} = 0, \quad a.s.$$

and

$$\lim_{t\to\infty}\frac{\int_0^t S(s)dB_1(s)}{t} = 0, \quad \lim_{t\to\infty}\frac{\int_0^t I(s)dB_2(s)}{t} = 0,$$

$$\lim_{t\to\infty}\frac{\int_0^t V(s)dB_3(s)}{t} = 0, \quad \lim_{t\to\infty}\frac{\int_0^t R(s)dB_4(s)}{t} = 0, \quad a.s.$$

Proof. From the model (2.2), we can have

$$d(\mathsf{S}+\mathsf{I}+\mathsf{V}+\mathsf{R}) = \mu - \mu(\mathsf{S}+\mathsf{I}+\mathsf{V}+\mathsf{R}) - \gamma\mathsf{I} + \eta_1\mathsf{S}d\mathsf{B}_1 + \eta_2\mathsf{I}d\mathsf{B}_2 + \eta_3\mathsf{V}d\mathsf{B}_3 + \eta_4\mathsf{R}d\mathsf{B}_4$$

Solving this equation, we can get ∎

Theorem 4.2. *If the threshold number $\mathbb{R}_0^s < 1$, then the disease $\mathsf{I}(t)$ in system (2.2) will almost surely tend to zero following an exponential function.*

Proof. Equations resulting from the integration of the model (2.2) are as follows:

$$\frac{\mathsf{S}(t)-\mathsf{S}(0)}{t} = (1-p)\mu - \frac{\beta\langle\mathsf{SI}\rangle}{\langle N\rangle} - \mu\langle\mathsf{S}\rangle + \frac{\eta_1}{t}\int_0^t \mathsf{S}(t)\,dB_1(t)$$

$$\frac{\mathsf{I}(t)-\mathsf{I}(0)}{t} = \frac{\beta\langle\mathsf{SI}\rangle}{\langle N\rangle} - (\gamma+\gamma_1+\mu)\langle\mathsf{I}\rangle + \frac{\eta_2}{t}\int_0^t \mathsf{I}(t)\,dB_2(t)$$

$$\frac{\mathsf{V}(t)-\mathsf{V}(0)}{t} = p\mu - \mu\langle\mathsf{V}\rangle + \frac{\eta_3}{t}\int_0^t \mathsf{V}(t)\,d\mathsf{B}_3(t)$$

$$\frac{\mathsf{R}(t)-\mathsf{R}(0)}{t} = \gamma_1\langle\mathsf{I}\rangle - \mu\langle\mathsf{R}\rangle + \frac{\eta_4}{t}\int_0^t \mathsf{R}(t)\,d\mathsf{B}_4(t)$$

Differentiating the $2nd$ equation of model (2.2) using $Itô$ formula, one can get

$$\begin{aligned}
d ln\mathsf{I}(t) &= \left[\frac{\beta\mathsf{SI}}{N} - (\gamma+\gamma_1+\mu)\mathsf{I}\right]\frac{1}{\mathsf{I}}dt - \frac{\eta_2^2}{2}dt + \eta_2 d\mathsf{B}_2,\\
&= \left[\frac{\beta\mathsf{S}}{N} - (\gamma+\gamma_1+\mu)\right]dt - \frac{\eta_2^2}{2}dt + \eta_2 d\mathsf{B}_2,\\
&\leq \left[\beta - (\gamma+\gamma_1+\mu+\frac{\eta_2^2}{2})\right]dt + \eta_2 d\mathsf{B}_2,\\
&\leq (\gamma+\gamma_1+\mu+\frac{\eta_2^2}{2})\left[\frac{\beta}{(\gamma+\gamma_1+\mu+\frac{\eta_2^2}{2})} - 1\right]dt + \eta_2 d\mathsf{B}_2,\\
&\leq (\gamma+\gamma_1+\mu+\frac{\eta_2^2}{2})(\mathbb{R}_0^s - 1)dt + \eta_2 d\mathsf{B}_2,
\end{aligned} \tag{4.1}$$

where,

$$\mathbb{R}_0^s = \frac{\beta}{(\gamma+\gamma_1+\mu+\frac{\eta_2^2}{2})}.$$

Therefore, we have

$$d ln\mathsf{I}(t) \leq (\gamma+\gamma_1+\mu+\frac{\eta_2^2}{2})(\mathbb{R}_0^s - 1)dt + \eta_2 d\mathsf{B}_2. \tag{4.2}$$

Taking integration of the inequality (4.2) within the range 0 and t, employing Lemma 4.1, we have

$$\lim_{t\to\infty}\sup\frac{\ln\mathsf{I}(t)}{t} \leq (\gamma+\gamma_1+\mu+\frac{\eta_2^2}{2})(\mathbb{R}_0^s - 1),\ \ a.s, \tag{4.3}$$

which shows that

$$\lim_{t\to\infty}\mathsf{I}(t) = 0. \tag{4.4}$$

In the view of above given result, our model A equations satisfy

$$\frac{\mathsf{R}(t)-\mathsf{R}(0)}{t} = \gamma_1\langle\mathsf{I}\rangle - \mu\langle\mathsf{R}\rangle + \frac{\eta_4}{t}\int_0^t \mathsf{R}(t)\,dB_4(t) \tag{4.5}$$

Applying limits with respect to $t \to \infty$, we can have

$$\lim_{t\to\infty} \frac{\mathsf{R}(t) - \mathsf{R}(0)}{t} = \gamma_1 \lim_{t\to\infty} \langle \mathsf{I} \rangle - \mu \lim_{t\to\infty} \langle \mathsf{R} \rangle + \frac{\eta_4}{t} \lim_{t\to\infty} \int_0^t \mathsf{R}(t)\, dB_4(t) \tag{4.6}$$

according to Lemma 4.1 and above result, we can get

$$\lim_{t\to\infty} \mathsf{R}(t) = 0. \tag{4.7}$$

and the rest of the equations of model A are given as

$$\lim_{t\to\infty} \mathsf{V}(t) = p. \tag{4.8}$$

and

$$\lim_{t\to\infty} \mathsf{S}(t) = 1 - p. \tag{4.9}$$

This demonstrates how the system behaves asymptotically, and hence supporting the primary goal of the theorem. ∎

5 Persistence

In this part of the manuscript, we lay out a few necessary criteria for the worm to persist in the networks. We start by introducing this definition:

Definition 5.1. *The proposed model (2.2) is said to be persistence in the mean, if*

$$\lim_{t\to\infty} \inf \frac{1}{t} \int_0^t I(r)dr > 0, \ \ a.s.$$

Theorem 5.2. *If* $\mathbb{R}^s = \frac{(1-p)\mu\beta}{(\mu+\frac{\eta_1^2}{2})(\gamma+\gamma_1+\mu+\frac{\eta_1^2}{2})} > 1$, *then for a positive initial data, that is,* $(S, I, V, R)(0) \in R_4^+$, *the malware infected nodes* $I(t)$ *of (2.2) has the property*

$$\lim_{t\to\infty} \inf \left\langle I(t) \right\rangle \geq \frac{2\Lambda(\sqrt{\mathbb{R}^s} - 1)}{c_1\beta}, \ \ a.s. \tag{5.1}$$

where $c_1 = \frac{\Lambda}{(\mu+\frac{\eta_1^2}{2})}$ *and* $c_2 = \frac{\Lambda}{(\gamma+\gamma_1+\mu\frac{\eta_2^2}{2})}$. *In other words, the worms will persist in the networks for* $\mathbb{R}^s > 1$.

Proof. Set

$$V_1 = -c_1 ln\mathsf{S} - c_2 ln\mathsf{I}.$$

In this case, c_1 and c_2 values will be found later. Using the Itô formula, we arrive at

$$dV_1 = LV_1 dt - c_1\eta_1 d\mathsf{B}_1(t) - c_2\eta_2 d\mathsf{B}_2(t), \tag{5.2}$$

where

$$\begin{aligned} LV_1 &= c_1 L(-lnS) + c_2 L(lnI) \\ &= -c_1[\frac{(1-p)\mu}{\mathsf{S}} - \frac{\beta\mathsf{I}(t)}{\mathsf{N}(t)} - \mu - \frac{\eta_1^2}{2}] - c_2[\frac{\beta\mathsf{S}(t)}{\mathsf{N}(t)} - (\gamma+\gamma_1+\mu) - \frac{\eta_2^2}{2}] \\ &= -c_1\frac{(1-p)\mu}{S} + c_1\frac{\beta\mathsf{I}(t)}{\mathsf{N}(t)} + c_1\mu + c_1\frac{\eta_1^2}{2} - c_2\frac{\beta\mathsf{S}(t)}{\mathsf{N}(t)} + c_2(\gamma+\gamma_1+\mu) + c_2\frac{\eta_2^2}{2} \\ &\leq -2\sqrt{c_1c_2(1-p)\mu\beta} + c_1(\mu+\frac{\eta_1^2}{2}) + c_2(\gamma+\gamma_1+\mu\frac{\eta_2^2}{2}) + \frac{c_2\beta I(t)}{\mathsf{N}(t)}, \end{aligned} \tag{5.3}$$

let

$$c_1 = \frac{\Lambda}{(\mu + \frac{\eta_1^2}{2})}$$

$$c_2 = \frac{\Lambda}{(\gamma + \gamma_1 + \mu + \frac{\eta_2^2}{2})}$$

$$\begin{aligned} LV_1 &\leq -2\sqrt{\frac{\Lambda^2(1-p)\mu\beta}{(\mu+\frac{\eta_1^2}{2})(\gamma+\gamma_1+\mu\frac{\eta_2^2}{2})}} + 2\Lambda + \frac{c_1\beta I(t)}{\mathsf{N}(t)} \\ &\leq -2\Lambda\Big(\sqrt{\frac{(1-p)\mu\beta}{(\mu+\frac{\eta_1^2}{2})(\gamma+\gamma_1\mu+\frac{\eta_2^2}{2})}} - 1\Big) + \frac{c_1\beta I(t)}{\mathsf{N}(t)} \\ &\leq -2\Lambda\big(\sqrt{R_0^S} - 1\big) + \frac{c_1\beta I(t)}{\mathsf{N}(t)}. \end{aligned} \tag{5.4}$$

Put Eq. (5.4) in Eq. (5.2) and then integrate both sides of Eq. (5.2) we will get

$$\frac{V_1(\mathsf{S}(t),\mathsf{I}(t) - V_1(\mathsf{S}(0),\mathsf{I}(0))}{t} \leq -2\Lambda(\sqrt{\mathbb{R}^s} - 1) + \frac{c_1\beta\langle I(t)\rangle}{\langle \mathsf{N}(t)\rangle} - \frac{c_1\eta_1\mathsf{B}_1(t)}{t} - \frac{c_2\eta_2\mathsf{B}_2(t)}{t}.$$

$$\frac{V_1(\mathsf{S}(t),\mathsf{I}(t) - V_1(\mathsf{S}(0),\mathsf{I}(0))}{t} \leq -2\Lambda(\sqrt{\mathbb{R}^s} - 1) + \frac{c_1\beta\langle I(t)\rangle}{\langle N(t)\rangle} + \psi(t). \tag{5.5}$$

where $\psi(t) = -\frac{c_1\eta_1\mathsf{B}_1(t)}{t} - \frac{c_2\eta_2\mathsf{B}_2(t)}{t}$. From strong law of large number it follows that

$$\lim_{t\to\infty} \psi(t) = 0.a.s. \tag{5.6}$$

From Eq. (5.5) we have

$$\langle I(t)\rangle \geq \frac{2\Lambda(\sqrt{\mathbb{R}^s} - 1)}{c_1\beta} - \frac{\psi(t)}{c_1\beta} + \frac{1}{c_1\beta}\Big(\frac{(V_1(S(t),I(t)) - V_1(0),I(0))}{t}\Big). \tag{5.7}$$

By taking the limit it will yield to

$$\lim_{t\to\infty} inf\langle I(t)\rangle \geq \frac{2\Lambda(\sqrt{\mathbb{R}^s} - 1)}{c_1\beta}, a.s.$$

Here we finish the proof of Theorem 5.2. ∎

6 Simulations

To demonstrate the previously proven analytical results, we shall simulate the model for numerical solution in this portion of the manuscript. To discretize system (2.2), we shall use the conventional higher-order Milstein's approach [25,29] and the scheme is presented as follows:

$$\begin{aligned} \mathsf{S}_{i+1} &= \mathsf{S}_i + \left[(1-p)\mu - \frac{\beta S_i I_i}{N} - \mu S_i\right] \triangle t + \eta_1 \mathsf{S}_i\sqrt{\triangle t}\xi_{1,i} + \frac{\alpha_1^2}{2}\mathsf{S}_i(\xi_{1,i}^2 - 1) \triangle t, \\ \mathsf{I}_{i+1} &= \mathsf{I}_i + \left[\frac{\beta S_i I_i}{N} - (\gamma+\gamma_1+\mu)I_i\right] \triangle t + \alpha_2 \mathsf{I}_i\sqrt{\triangle t}\xi_{1,i} + \frac{\alpha_2^2}{2}\mathsf{I}_i(\xi_{1,i}^2 - 1) \triangle t, \\ \mathsf{V}_{i+1} &= \mathsf{I}_i + \left[p\mu - \mu V_i\right] \triangle t + \alpha_3 V_i\sqrt{\triangle t}\xi_{1,i} + \frac{\alpha_3^2}{2}V_i(\xi_{1,i}^2 - 1) \triangle t, \\ \mathsf{R}_{i+1} &= \mathsf{R}_i + \left[\gamma_1 I_i - \mu R_i\right] \triangle t + \alpha_4 \mathsf{R}_i\sqrt{\triangle t}\xi_{1,i} + \frac{\alpha_4^2}{2}\mathsf{R}_i(\xi_{1,i}^2 - 1) \triangle t. \end{aligned} \tag{6.1}$$

In the above, ξ_i such that $i = 1, \cdots, 4$ are the independent Gaussian random variables that follow the Gaussian distribution $\mathsf{N}(0,1)$ and the term Δt indicates a positive uniform time-step. Tested sets include $[0, 5000]$ and $\Delta = 0.5$.

6.1 Numerical simulations of the extinction

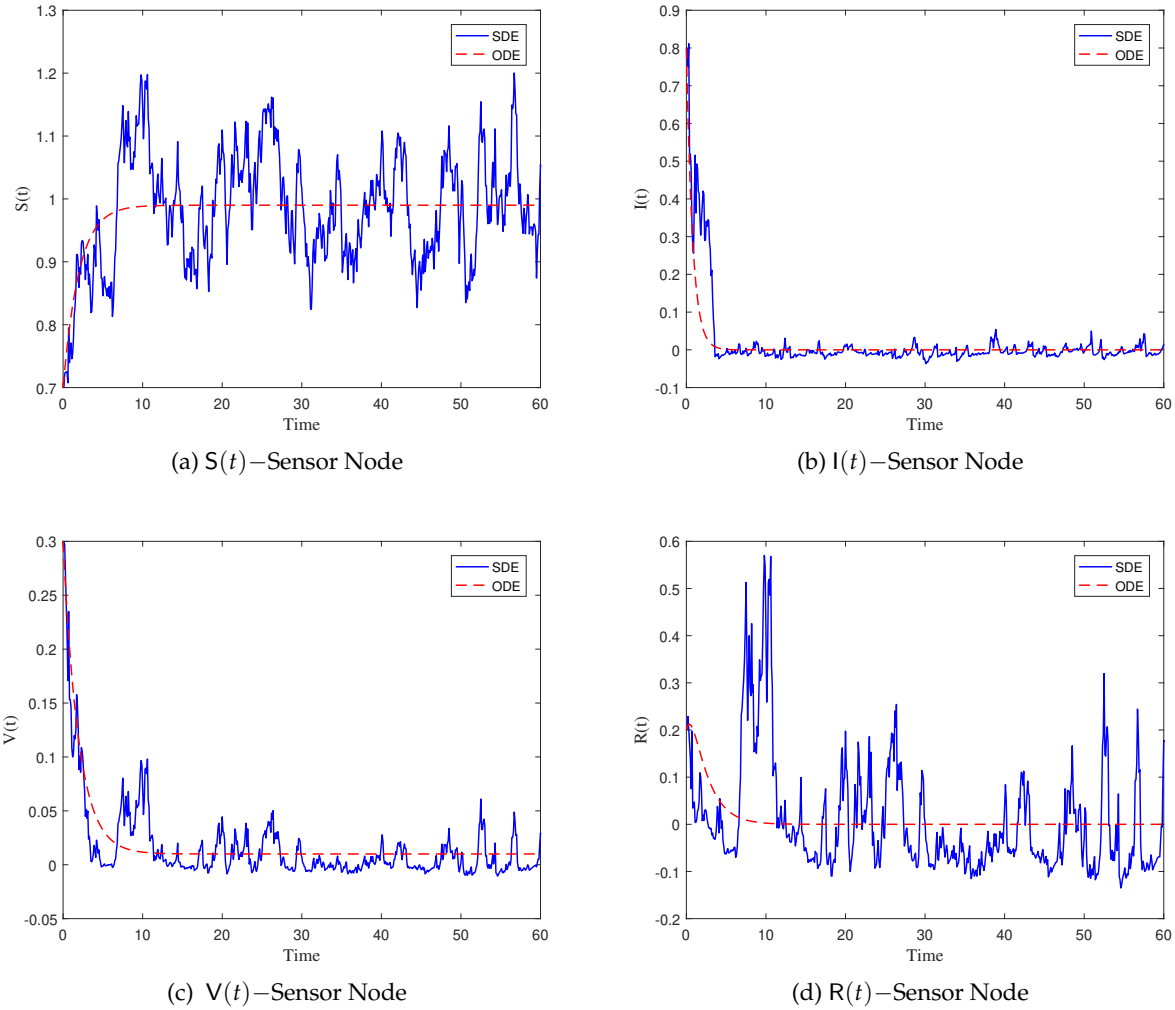
It is clear from the analytical sections studied so far that the term $\mathbb{R}_0^s$ acts as a threshold value. In the case $\mathbb{R}_0^s < 1$, the infected nodes' portion goes toward zero. This numerical validation support the findings of Theorem 4.2, which states that the worm consistently and progressively vanishes from the network when $\mathbb{R}_0^s < 1$. Figures 1a-1d, which come from the first experiment, can be used to infer this finding. Table 2 shows the associated parameters and starting values of the system (2.2). In the long term, the nodes infected by the worms are approaching zero whenever $\mathbb{R}_0^s < 1$. Rational worms in the network will ultimately approach zero, as theorem 4.2 suggests. Using either deterministic or stochastic versions of the model, Figures 1a-1d guarantee the removal of worms from the networks, exhibiting the same result. Table 2 shows the parameter settings and the initial data used to simulate system (2.2). The reproduction number $\mathbb{R}_0$ is the threshold value for the epidemic-free equilibrium in (2.1), as seen in Fig. 1. The trajectories of model (2.2), which displays variations, converges towards the worm-free equilibrium point of the deterministic counterpart, signifying the epidemic's disappearance. MATLAB (R2017a) was used to simulate the model in order to investigate the effects of different settings.

Parameter	Value	Source
p	0.04	Estimated
μ	0.01	Estimated
β	0.07	Estimated
γ	0.02	Estimated
γ_1	0.03	Estimated
$\mathsf{S}(0)$	0.70	Estimated
$\mathsf{I}(0)$	0.80	Estimated
$\mathsf{V}(0)$	0.30	Estimated
$\mathsf{R}(0)$	0.20	Estimated
η_1	0.10	Estimated
η_2	0.30	Estimated
η_3	0.45	Estimated
η_4	0.40	Estimated

Table 2: Values of the parameter used in simulating models (2.1), and (2.2).

6.2 Numerical simulations for persistence

In this part of the paper, we intend to validate the persistence of the worms within the network under certain conditions as suggested by Theorem 5.2. We calculated $\mathbb{R}^s > 1$ by using data from 3, which indicates that the size of the infected nodes is non-negative and eventually converges to the corresponding equilibrium. This implies that, in line with Theorem 5.2, the malware will continue to exist in the networks. Taking into account both temporal and geographical modifications of the parameters, these simulations explore the dynamics of malware inside the network. Furthermore, Theorem 5.2 attests to the suggested model's durability. As thus, the malware will continue to exist in the network without decreasing, creating a steady state of endemic equilibrium. Moreover, it has been shown that improved network connection is a direct result of increased sensor node connectivity.



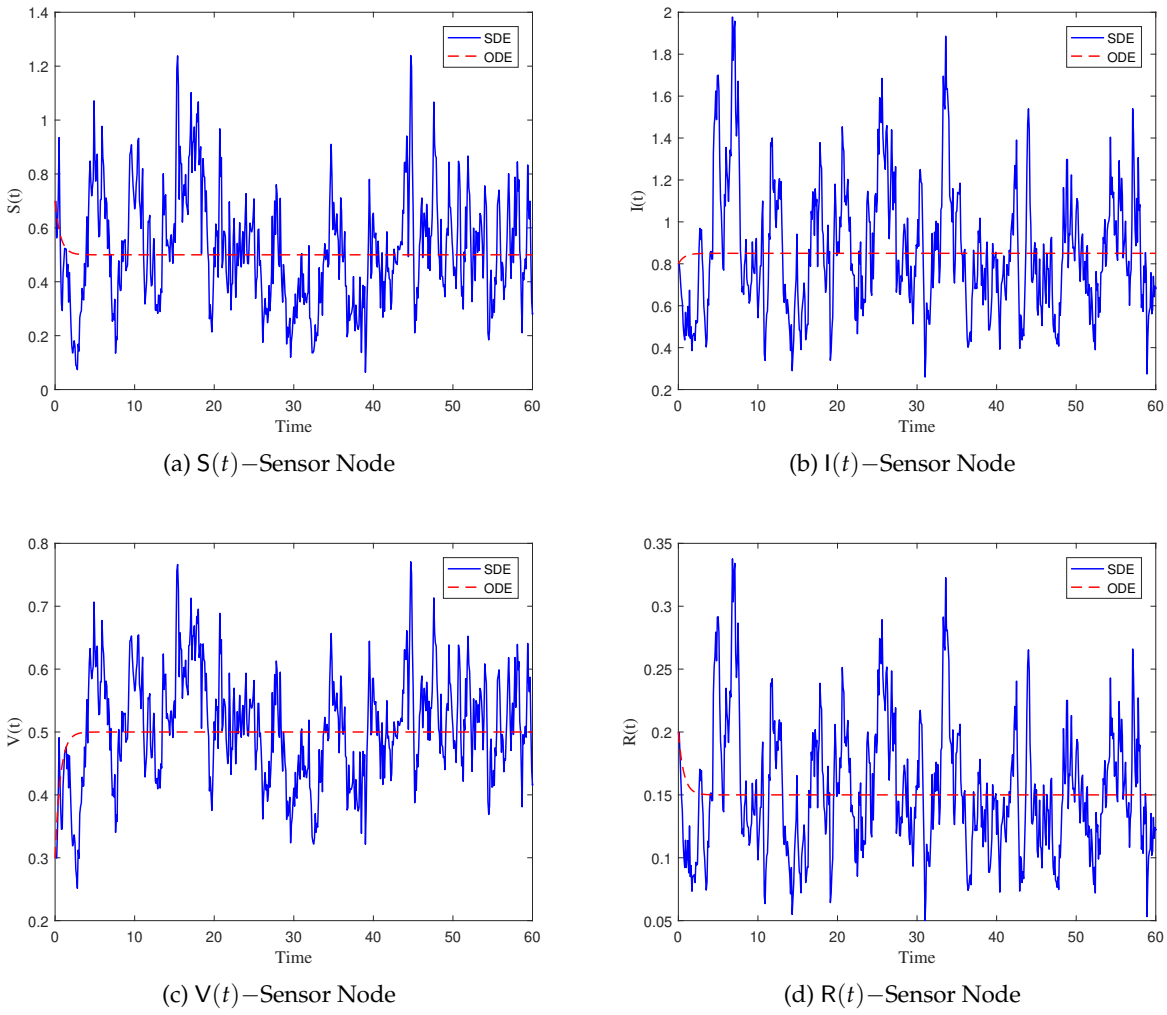
(a) $\mathsf{S}(t)-$Sensor Node (b) $\mathsf{I}(t)-$Sensor Node

(c) $\mathsf{V}(t)-$Sensor Node (d) $\mathsf{R}(t)-$Sensor Node

Figure 1: The corresponding simulations of the system (2.2) and the deterministic system (2.1).

Parameter	Value	Source
p	1.40	Estimated
μ	0.20	Estimated
β	0.50	Estimated
γ	0.20	Estimated
γ_1	0.40	Estimated
$\mathsf{S}(0)$	0.70	Estimated
$\mathsf{I}(0)$	0.80	Estimated
$\mathsf{V}(0)$	0.30	Estimated
$\mathsf{R}(0)$	0.20	Estimated
η_1	0.50	Estimated
η_2	0.70	Estimated
η_3	0.45	Estimated
η_4	0.25	Estimated

Table 3: Values of the parameters for simulating model (2.1), and (2.2) ensuring the condition $\mathbb{R}^s > 1$.



(a) $\mathsf{S}(t)-$Sensor Node

(b) $\mathsf{I}(t)-$Sensor Node

(c) $\mathsf{V}(t)-$Sensor Node

(d) $\mathsf{R}(t)-$Sensor Node

Figure 2: The corresponding simulations of the system (2.2) and the deterministic system (2.1).

7 Conclusion

We introduced a stochastic SIVR model in this study to tackle the problem of lowering the worm spread in wireless sensor networks. The model incorporates random components that come from fluctuations in the environment, represented as Gaussian white noise. We defined an adequate set of conditions to determine if worm spread in WSNs would endure or cease to exist in terms of mean behavior. These settings offer insightful information on the system's long-term dynamics, which advances our knowledge of the dynamics of worm propagation and control techniques in WSNs. First, using the Lyapunov function method, we showed that our model permits a global, positive, and feasible solution. We then calculated our stochastic system's basic reproductive threshold, which coincided with the basic threshold value $\mathbb{R}_0$ of the deterministic system, excluding noise oscillation. We concluded that the worms will eventually die out if $\mathbb{R}_0^s$ is less than or equal to 1. On the other hand, the average nodes will continue to be infected if $\mathbb{R}_0^s > 1$. Lastly, using simulations, we contrasted our analytical findings with approximate solutions.

The thorough findings of this investigation provide compelling evidence that the suggested paradigm improves data efficiency and prolongs the lifetime of wireless sensor networks. These results have practical implications for software companies, providing knowledge that can be applied to create antivirus programs that are more successful in preventing malware assaults in wireless sensor networks.

To further strengthen the entire security architecture and lessen future threats, the investigation will help end users recover affected nodes and install antivirus software on sensor nodes with caution.

Further study paths may also include the inclusion of mobile and diverse nodes and the investigation of other variables like quarantined and vaccinated classes. These factors might improve the model's applicability and provide more information on the dynamics of worm spread and mitigation techniques in WSNs.

Author contributions

Sayed Murad Ali Shah: Conceptualization, Methodology, Software, Investigation, Writing – Original Draft Preparation.
Hassan Tahir: Conceptualization, Methodology, Software, Investigation, Writing – Original Draft Preparation.
Asaf Khan: Writing – Review & Editing, Supervision, Project Administration, Funding Acquisition.
Wajahat Ali Khan: Writing – Review & Editing, Supervision, Project Administration, Funding Acquisition.
Alishba Arshad: HSoftware, Writing – Review & Editing.

Acknowledgment

The authors are grateful to the anonymous referees for their valuable comments and suggestions, which led to the improvement of this paper.

Conflict of interests

This work does not have any potential conflicts of interest.

Data Availability Statement

The associated data is available upon request from the corresponding author.

Grant/Funding information

There are no funders to report for this submission.

Declaration Statement of Generative AI

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

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