Correlated Stochastic Epidemic Model for the Dynamics of SARS-CoV-2 with Vaccination

Tahir Khan (✉ tkhan87@uom.edu.pk)

Department of Computing, Muscat College, Muscat Oman

Roman Ullah

Department of Computing, Muscat College, Muscat Oman

Gul Zaman

Department of Mathematics, University of Malakand, Chakdara, Dir (Lower), Khyber Pakhtunkhawa, Pakistan

Youssef Khatib

Department of Mathematical Sciences, UAE University, Al-Ain, P.O. Box 15551, United Arab Emirates

Research Article

Keywords: Novel SARS-CoV-2 correlated model, various sources of randomness, vaccination, existence, uniqueness, extinction, persistence, numerical simulation

Posted Date: November 10th, 2021

DOI: <https://doi.org/10.21203/rs.3.rs-957724/v1>

License: © ⓘ This work is licensed under a Creative Commons Attribution 4.0 International License.

[Read Full License](#)

Correlated stochastic epidemic model for the dynamics of SARS-CoV-2 with vaccination

Tahir Khan^{a,b,†}, Roman Ullah^a, Gul Zaman^b and Youssef El Khatib^c

a. Department of Computing, Muscat College, Muscat Oman

b. Department of Mathematics, University of Malakand, Chakdara, Dir (Lower), Khyber Pakhtunkhawa, Pakistan

c. Department of Mathematical Sciences, UAE University, Al-Ain, P.O. Box 15551, United Arab Emirates

Corresponding Author email address: tkhan87@uom.edu.pk

Abstract

We formulate a mathematical model has been proposed to describe the stochastic influence of SARS-CoV-2 virus with various sources of randomness and vaccination. We assume the various sources of randomness in each population groups by different Brownian motion. We develop the correlated stochastic model by taking into account the various sources of randomness by different Brownian motions and distributed the total human population in three groups of susceptible, infected and recovered with reservoir class. Because reservoir play a significant role in the transmission of SARS-CoV-2 virus spreading. Moreover, the vaccination of susceptible are also accorded. Once we formulate the correlated stochastic model, the existence and uniqueness of positive solution will be discussed to show the problem feasibility. The SARS-CoV-2 extinction as well as persistency will be also discussed and we will obtain the sufficient conditions for it. At the last all the theoretical results will be supported via numerical/graphical findings.

Key words: Novel SARS-CoV-2 correlated model, various sources of randomness, vaccination, existence, uniqueness, extinction, persistence, numerical simulation

1 Introduction

In Wuhan, China a respiratory disease outbreak have been started in December, 2019. Later on it was identified as a novel corona-virus (COVID-19), which is known as SARS-CoV-2 virus. The initial spreading source of the novel disease was an animal. But pandemic rises from the human interaction. Total of 116,216,580 number of infected individuals have been reported yet while 2,581,649 deaths occurred till 5th March, 2021 around the world. Vaccination is an important weapon against controlling a disease. In case of SARS-CoV-2 virus disease vaccination is very important and there are many vaccines that could shown its effectiveness. World Health Organization (WHO) investigates that reliable vaccinations programme will change the situation. But precautionary measures could be necessary for the time being as it is still doubtful that vaccine of SARS-CoV-2 provide how many degree of safeness.

Modeling the real world problem is an emerging area in the field of science and technology. Mathematical models play a very significant role to explore the dynamics of a disease and to predict for future. Also effective control programmes have been forecasted to suggest useful guidelines for the health officials. On the basis of these guidelines it could be easily implemented by taking serious steps to control the disease. Researchers studied epidemiological models to discuss the dynamical behavior of a disease with suggesting control mechanism [1, 2, 3, 4, 5, 6]. The Covid-19 also called SARS-CoV-2 virus and its vaccination is a challenging task, which attracts the attention of many researches, (see [7, 8, 9, 10, 11]). The reported literature reveals that the mathematical models which have been analyzed are simple and used a deterministic approaches. However the SARS-CoV-2 virus transmission is influenced by different factors (social behavior, age, mobility, and virus mutation etc.,) which can affect the dynamics [12, 13, 14, 15, 16, 17, 18]. So from the characteristic of the disease it could be very interesting if the stochastic approach will be used. A stochastic mathematical model has been studied for the novel corona virus by *Khan et al.*, [19] very recently, where the random fluctuation are assumed in transmission rate only, while as reported above that the due to many factors the SARS-CoV-2 virus is influenced

We formulate a stochastic mathematical models for capturing the realistic nature of the disease. For this we will extend the work of *Khan et al.*, by incorporating various random sources in which every individuals class has a various Brownian motion according to the disease characteristics. The vaccination of susceptible individuals are also assumed to investigate the

efficiency of vaccination and its role minimization of the infection. First the models will be formulated and then analyzing to discuss the detail dynamics. We will discuss the existence as well as the uniqueness of the proposed problem to show the well-posedness and feasibility of the problem. We then show that under what conditions the SARS-CoV-2 virus disease extinct as well as persist. Finally, all analytical findings will be supported by using some graphical representation in the form of a large scale numerical simulation by using Euler Maruyama scheme. It will be performed via coding the proposed problem with the help of MATLAB and we will show the analytical finding graphically.

2 Formulation of the model with fundamental analysis

Let us assume a filtered probability space $(\Omega, \mathcal{F}_T, (\mathcal{F}_t)_{t \in [0, T]}, P)$ on which lives $W := (W(t))_{t \in [0, T]}$ with $W(t) := (W_i(t) : \text{such that } i = 1, \dots, 4)$, where W is a Brownian motion of 4th dimension. Moreover, the natural filtration $(\mathcal{F}_t)_{t \in [0, T]}$ is assumed generated by the Brownian motion W . For $k = 1, 2, 3, 4$, we consider the correlated 1-dimensional Brownian motions $(B_k(t))_{t \in [0, T]}$ given by

$$B_k(t) := \sum_{i=1}^4 \rho_{ki}(t) W_i(t) \quad \text{where } \rho_{ki} \text{ are constant in } [-1, 1].$$

We classify the total human population in three human population groups and one class of reservoir. The three population groups are susceptible, SARS-CoV-2 virus infected and recovered, which are symbolized by $s(t)$, $i(t)$ and $r(t)$ respectively, while the reservoir class is denoted by $w(t)$. All the populations groups and reservoir are distributed by different Brownian motion. Thus we suggest a correlated stochastic epidemic model by the following system:

$$\begin{aligned} ds(t) &= \{\Pi - (\beta_1 i(t) + \beta_2 w(t) + \mu + v) s(t)\} dt + \eta_1 s(t) dB_1(t), \\ di(t) &= \{(\beta_1 i(t) + \beta_2 w(t)) s(t) - (\sigma + d_1 + \mu) i(t)\} dt + \eta_2 i(t) dB_2(t), \\ dr(t) &= \{\sigma i(t) + v s(t) - \mu r(t)\} dt + \eta_3 r(t) dB_3(t), \\ dw(t) &= \{\alpha i(t) - \eta w(t)\} dt + \eta_4 w(t) dB_4(t). \end{aligned} \tag{1}$$

In Eq.(1) the various parameters are characterized as: the rate of new born is symbolized with Π , and $\beta_i, i = 1, 2$, are routes of disease transmission from

the infected human as well as from reservoir. Moreover, v is the vaccination of susceptible population and μ is the natural death rate while death from the disease is described with d_1 . We also symbolize the recovery rate by σ and a rate contribute to the virus to the seafood market by α . The removing SARS-CoV virus rate is denoted by η .

If $\rho = 1$ then $B_1 = B_2 = B_3 = B_4$ and the model is reduced to the stochastic model studied in *Khan et al.*, [19]. Also it could be clearly noted that the above system (1) will reduce to the deterministic form, whenever $\eta_1 = \eta_2 = \eta_3 = \eta_4 = 0$. In addition the disease-free and the endemic equilibriums of the associated deterministic form of the model are respectively symbolized with $E_0 = (s_0, 0, 0, r_0)$ and $E^* = (s^*, i^*, r^*, w^*)$ with $s_0 = \Pi/\mu$, $r_0 = v\Pi/dq_1$, where $p_1 = \mu + v$. To move towards the endemic equilibrium, we will calculate the *basic reproductive number* first, which is defined to be the average number of secondary infectious produced an infective whenever reached to a totally non-infected population. We assume $X = (i, w)^T$ and $p_2 = \sigma + \mu + d_1$, then the deterministic version of the model (1) yields

$$\frac{dX}{dt}|_{E_0} = -V + F, \text{ and } F = \begin{bmatrix} \beta_1 s_0 & \beta_2 s_0 \\ 0 & 0 \end{bmatrix}, V = \begin{bmatrix} p_2 & 0 \\ -\alpha & \eta \end{bmatrix}. \quad (2)$$

The *basic reproductive number* is then the spectral radius of $\rho(FV^{-1})$ and consequently looks like

$$R_0 = \frac{\Pi\beta_1}{p_1 p_2} + \frac{\Pi\alpha\beta_2}{\eta p_1 p_2}. \quad (3)$$

We use this quantity, to find the components of the endemic equilibrium which may take the form

$$s^* = \frac{\eta q_2}{\eta\beta_1 + \beta_2\alpha}, i^* = \frac{\eta q_1 (R_0 - 1)}{\beta_1\eta + \beta_2\alpha}, r^* = \frac{vs^* + \sigma i^*}{\mu}, w^* = \frac{\alpha}{\eta} i^*. \quad (4)$$

2.1 Sensitivity analysis

In every disease the role of threshold parameter (*basic reproductive number*) is very important and the disease spreads whenever the value of this quantity is more than one and disease dies out if its value is less than unity. We will discuss the sensitivity of threshold parameter to find the relation between *basic reproductive number* and model parameter. We also calculate the sensitivity indexes that which parameters is how much sensitive to the disease control and transmission. Generally the sensitivity index of a parameter say ϕ is denoted by Υ_ϕ and define as $\frac{\phi}{R_0} \frac{\partial R_0}{\partial \phi}$. By following this formula we calculate the sensitivity indices of model parameters as: $\Upsilon_{\beta_1} = 0.9937106918$,

$\Upsilon_{\beta_2} = 0.006289308180$, $\Upsilon_{\alpha} = 0.006289308180$, $\Upsilon_{\nu} = -0.8333333334$ and $\Upsilon_{\sigma} = -0.6862610536$, where the parameters value are taken to be $\Pi = 2$, $\beta_1 = 0.079$, $\beta_2 = 0.001$, $\mu = 0.16$, $\nu = 0.8$, $d_1 = 0.00001$, $\sigma = 0.35$, $\alpha = 0.1$ and $\eta = 0.2$. The biological interpretation of these analysis investigate that the epidemic parameters β_1 , β_2 and α have a positive influence on the threshold quantity while there is a negative influence with the parameters ν and σ . This shows that decreasing in the value of β_1 , β_2 and α , and increasing in the value of ν and σ will decrease the value of the *basic reproductive number*, which is significant in disease elimination. It could be also noted that β_1 and ν got the highest sensitivity index and so are the most sensitive parameters to the disease transmission and control. We observed that increasing the value of β_1 say by 10 percent would significantly increase the value of R_0 by 9.9 percent as depicted in Fig.1, while increasing the value of ν say by 10 percent decreasing the value of R_0 by 8.3 percent as shown in Fig.4. Similarly, β_2 and α collectively effect R_0 by 0.1257861636 whenever these parameters are increased or decreased by 10 percent as depicted by Fig.2 and Fig.3. The relation between σ and R_0 is also an inverse as increased σ by 10 percent would decrease the threshold quantity by 6.86 percent is given in Fig.4.

Figure 1: The picture visualize the variation of the *reproductive number* against β_1 and β_2 .

Figure 2: The graph visualize the variation of the *basic reproductive number* against β_1 and α .

Figure 3: The graph visualize the variation of the *basic reproductive number* against β_2 and α .

Figure 4: The graph visualize the variation of the *basic reproductive number* against σ and ν .

2.2 Existence and uniqueness analysis

In this portion of the manuscript the solutions existence and uniqueness with positivity of Eq.(1) will be discussed.

Here, we describe a Multidimensional Itô formula for getting our results by following the book of stochastic calculus [20].

Lemma 2.1. *Let $a = (\alpha_1, \dots, \alpha_n)$ and $b = (\beta_1, \dots, \beta_n)$ represent the adapted processes with square-integrable n -dimensional. We consider $X = (X_1, \dots, X_n)$, where X_k is driven by the stochastic differential equation and $k \in \{1, \dots, n\}$, thus*

$$dX_k(t) = a_k(t)dt + b_k(t)dB(t), \quad X_k(0) \in \mathbb{R}.$$

Let F is a given twice continuously differentiable function $f : \mathbb{R}^n \rightarrow \mathbb{R}$, then we have

$$dF(X(t)) = \sum_{k=1}^n \frac{\partial F}{\partial x_k}(X(t))dX_k(t) + \sum_{k,l=1}^n \frac{1}{2} \frac{\partial^2 F}{\partial x^k \partial x^l}(X(t))d\langle X_k(t), X_l(t) \rangle,$$

where $d\langle X_k(t), X_l(t) \rangle = b_k(t)b_l(t)dt$, $dt = d\langle B(t), B(t) \rangle$, and $d\langle B(t), t \rangle = d\langle t, B(t) \rangle = 0$.

We use the Lyapunov theory as well as the virtue of Itô formula to prove the solution of Eq.(1) exists globally and is positive. Define

$$\mathbb{D} = \{(s, i, r, w) \in R_+^4 : s \text{ and } r > 0, i, w \geq 0, s + i + r + w \leq 1\}. \quad (5)$$

The result that discusses the existence analysis of the problem is given by the following theorem.

Theorem 2.2. *Let $(s(0), i(0), r(0), w(0))$ be the initial classes and assumed to be in R_+^4 , then the solution $(s(t), i(t), r(t), w(t))$ of the model (1) is unique as well as remains in R_+^4 almost surely (a.s) i.e.,*

$$p\{(s, i, r, w) \in \mathbb{D}, \forall t \geq 0\} = 1.$$

Proof. We use the procedure as adopted in [21] and so in the light of this the local Lipschitz continuity property holds for system (1), therefore the solution symbolized by (s, i, r, w) of the proposed problem in $[0, \tau_e)$ subject to initial conditions in R_+^4 is unique and local for the explosion time τ_e . Moreover, we investigate that $\tau_e = \infty$ a.s as to show the solution globalization. It is assumed that $\kappa_0 \geq 0$ is sufficiently large and $\frac{1}{\kappa_0} < N(0) < \kappa_0$, where $N(0) = (s(0), a(0), c(0), r(0))$. We define the stopping time for every $\kappa \geq \kappa_0$ as:

$$\tau_k = \inf \left\{ t \in [0, \tau_e) : \min(s(t), i(t), r(t), w(t)) \leq \frac{1}{k} \text{ or } \max(s(t), i(t), r(t), w(t)) \right\}. \quad (6)$$

Further, let ϕ is empty set and $\inf \phi = \infty$. Since τ_k depend on k and whenever k increasing τ_k also increasing as k increasing without bound i.e., tend to ∞ . Making use of $\lim = \tau_\infty$ if $t \rightarrow \infty$ with taking $\tau_\infty = \infty$ a.s gives that $(s(t), i(t), r(t), w(t)) \in R_+^4, \forall t \geq 0$ a.s. We now only need to show that $\tau_e = \infty$. For this, we use the assumption that for any two constants, $T > 0$ and $\varepsilon \in (0, 1)$, we have

$$P\{\tau_\infty \leq T\} > \varepsilon. \quad (7)$$

So $k_1 \geq k_0$ is an integer that

$$P\{\tau_k \leq T\} \geq \varepsilon, \text{ for every } k \geq k_1. \quad (8)$$

Let H is twice continuously differentiable function i.e., $H \in C^2$ and $H : R_+^4 \rightarrow R_+$ by

$$H(s, i, r, w) = s - 1 - \log(s) + i - 1 - \log(i) + r - 1 - \log(r) + w - 1 - \log(w). \quad (9)$$

Clearly $H \geq 0$, so for $0 \leq T$ and $k_0 \leq k$, and by the application of Itô formula leads to the assertion

$$dH = LHdt + (s-1)\eta_1 dB_1 + (i-1)\eta_2 dB_2 + (r-1)\eta_3 dB_3 + (w-1)\eta_4 dB_4. \quad (10)$$

In Eq.(10), LH is defined as

$$\begin{aligned} LH = & (1 - 1/s)(\Pi - \beta_1 si - \beta_2 sw - (\mu + v)s) + \frac{1}{2}\eta_1^2 + (1 - 1/i)(\beta_1 si \\ & + \beta_2 sw - (\mu + d_1 + \sigma)i) + \frac{1}{2}\eta_2^2 + (1 - 1/r)(vs + \sigma i - \mu r) + \frac{1}{2}\eta_3^2 + (1 - 1/w) \\ & (\alpha i - \eta w) + \frac{1}{2}\eta_4^2. \end{aligned} \quad (11)$$

Simplifying and re-writing the above equation we may lead to the following inequality

$$LH \leq \Pi + (\beta_1 + \alpha)i + \beta_2 w + vs + 3\mu + v + d_1 + \sigma + \eta. \quad (12)$$

It could be noted from the fact that $s + i + r + w \leq 1$, so the last inequality gives

$$LH \leq \Pi + \beta_1 + \beta_2 + \alpha + 2v + 3\mu + d_1 + \sigma + \eta := K. \quad (13)$$

Plugging Eq.(13) in Eq.(10) we may arrive

$$dH \leq Kdt + (s-1)\eta_1 dB_1 + (i-1)\eta_2 dB_2 + (r-1)\eta_3 dB_3 + (w-1)\eta_4 dB_4.$$

The integration of both sides reveals that

$$\begin{aligned} \int_0^{\tau_k \wedge T} dH \leq & \int_0^{\tau_k \wedge T} Kdt + \int_0^{\tau_k \wedge T} (s-1)\eta_1 dB_1 + \int_0^{\tau_k \wedge T} (i-1)\eta_2 dB_2 \\ & + \int_0^{\tau_k \wedge T} (r-1)\eta_3 dB_3 + \int_0^{\tau_k \wedge T} (w-1)\eta_4 dB_4. \end{aligned} \quad (14)$$

The expectation of both sides provides

$$\begin{aligned} E \left[H(s(\tau_k \wedge T), i(\tau_k \wedge T), r(\tau_k \wedge T), w(\tau_k \wedge T)) \right] & \leq H(s(0), i(0), r(0), w(0)) \\ & + E \left[\int_0^{\tau_k \wedge T} Kdt \right], \end{aligned}$$

which implies that

$$E \left[H(s(\tau_k \wedge T), i(\tau_k \wedge T), r(\tau_k \wedge T), w(\tau_k \wedge T)) \right] \leq H(s(0), i(0), r(0), w(0)) + TK. \quad (15)$$

Setting a notion of $\Omega_k = T \geq \tau_k$ for all $k \geq k_1$. The use of Eq.(7) gives that $P(\Omega_k) \geq \epsilon$. Noted that there is at least one $s(\omega, \tau_k)$ or $i(\omega, \tau_k)$ or $r(\omega, \tau_k)$ or $w(\omega, \tau_k)$ equal $1/k$ or k for all $\omega \in \Omega_k$. Since $\frac{1}{k} + \log k - 1$ or $-\log k + k - 1$. Hence

$$(s(\tau_k, \omega), i(\tau_k, \omega), r(\tau_k, \omega), w(\tau_k, \omega)) \geq \left(\frac{1}{k} - 1 + \log k \right) \wedge (-\log k - 1 + k). \quad (16)$$

So Eq.(7) and (15) gives

$$\begin{aligned} H(N(0)) + TK &\geq E \left[1_{\Omega_k(\omega)} H(s(\tau_k \wedge T), i(\tau_k \wedge T), r(\tau_k \wedge T), w(\tau_k \wedge T)) \right], \\ &= E \left[1_{\Omega_k(\omega)} H(s(\tau_k, T), i(\tau_k, T), r(\tau_k, T), w(\tau_k, T)) \right] \geq E \left[1_{\Omega_k(\omega)} \left(\log k - 1 + \frac{1}{k} \right) \right. \\ &\quad \left. \wedge (-\log k - 1 + k) \right] = \left(\log k - 1 + \frac{1}{k} \right) \wedge (-\log k - 1 + k) E[1_{\Omega_k(\omega)}], \end{aligned}$$

implies that

$$H(N(0)) + TK \geq \epsilon \left(\log k + \frac{1}{k} - 1 \right) \wedge (-\log k + k - 1),$$

where $1_{\Omega_k(\omega)}$ is a function known indicator function for $\Omega_k(\omega)$. Let $k \rightarrow \infty$ we ultimately obtain $\infty > H(N(0)) + KT = \infty$, which contradict, therefore $\infty = \tau_\infty$ a.s. $\square$

Remark 1. *The uniqueness as well as the existence reveals that for any initial compartments $(N(0)) \in R_+^4$, the unique solution with global axiom $(s, i, r, w) \in R_+^4$ almost surely (a.s) exists for the proposed problem under consideration as reported by Eq. (1),*

Theorem 2.3. *Let (s, i, r, w) be the solutions of the stochastic problem as stated by Eq.(1) whenever exist are positive for every $(N(0)) \in R_+^4$ a.s and $t > 0$.*

Proof. To discuss the solutions of Eqn.(1) which becomes

$$X_k(t) = \zeta_k(t) \left[X_k(0) + \int_0^t [\alpha_k(u) - \sum_{j=1}^m \theta_{kj}(u) \gamma_{kj}(u) \rho_{kj}^2] \zeta_k^{-1}(u) du + \sum_{j=1}^m \int_0^t \gamma_{kj}(t) \rho_{kj} \zeta_k^{-1}(u) dW_j(u) \right]. \quad (17)$$

where

$$\zeta_k(t) = \exp \left[\int_0^t \left(a_k(u) - \frac{1}{2} \sum_{j=1}^m b_{kj}^2(u) \right) du + \sum_{j=1}^m \int_0^t b_{kj}(u) dW_j(u) \right]. \quad (18)$$

Here $k = 4$, $m = 4$, $\rho_{kj} = \rho_{kj}$, $\gamma_{kj} = 0$ (for $k, j = 1, 2, 3, 4$) and

$$\begin{aligned} X_1 &= s, \alpha_1 = \Pi, a_1 = -\beta_1 i(t) - \beta_2 w(t) - (\mu + v), b_{1j} = \eta_1 \rho_{1j}, \\ X_2 &= i, \alpha_2 = \beta_2 w(t) s(t), a_2 = \beta_1 s(t) - (\sigma + \mu + d_1), b_{2j} = \eta_2 \rho_{2j}, \\ X_3 &= r, \alpha_3 = v s(t) + \sigma i(t), a_3 = -\mu, b_{3j} = \eta_3 \rho_{3j}, \\ X_4 &= w, \alpha_4 = \alpha i(t), a_4 = -\eta, b_{4j} = \eta_4 \rho_{4j}. \end{aligned}$$

The equations (17) and (18) show clearly that the solution of our model (1) exists and it is unique and positive. This ends the proof. $\square$

2.3 Extinction and persistence

In this section, the extinction and persistence analysis of the stochastic model (1) are discussed. We derive the various conditions in the form of some expression to show the permanence and extinction. These expressions containing the model parameters and intensities of noises. Before the formal analysis we define that

$$\langle g(t) \rangle = \frac{1}{t} \int_0^t g(x) dx. \quad (19)$$

Now it could be described that the persistence of novel corona virus SARS-CoV-2 is subjected to $\lim (\inf \langle i(t) \rangle)$ and $\lim (\inf \langle w(t) \rangle)$ whenever are positive as t increases without bound i.e., to ∞ . Moreover, the stochastic *reproductive number* of corona dynamical system represented by Eq.(1) is symbolized by R_0^S and define as $R_0^S = R_1^S + R_2^S$, where

$$R_1^S = \frac{\Pi \beta_1}{p_1 \left(p_2 + \frac{\eta_2^2}{2} \right)}, \quad R_2^S = \frac{\Pi \beta_2}{p_1 \left(p_2 + \frac{\eta_2^2}{2} \right)}. \quad (20)$$

Similarly, if

$$\liminf_{t \rightarrow \infty} \int_0^t i(x) dx > 0, \text{ a.s.}, \quad (21)$$

and

$$\liminf_{t \rightarrow \infty} \int_0^t w(x) dx > 0, \text{ a.s.}, \quad (22)$$

holds, the epidemic problem represented by Eq.(1) states that the disease will persistent. Thus for the extinction analysis of the proposed problem we state the following subsequent result.

Theorem 2.4. *The SARS-CoV-2 virus will die out exponentially whenever the stochastic reproductive number parameter (R_0^S) is less than unity i.e.,*

$$\limsup_{t \rightarrow \infty} \frac{\log i(t)}{t} \leq \left(p_1 + \frac{1}{2} \xi_2^2 \right) (R_0^S - 1) < 0 \text{ a.s.}$$

Also

$$\lim_{t \rightarrow \infty} s(t) = \frac{\Pi}{p_1}, \quad \lim_{t \rightarrow \infty} r(t) = \frac{v\Pi}{dp_1}, \quad \lim_{t \rightarrow \infty} w(t) = \lim_{t \rightarrow \infty} i(t) = 0, \text{ a.s.} \quad (23)$$

Proof. To prove the result, we integrate system (1) on both sides which leads to

$$\begin{aligned} \int_0^t ds(x) &= \Pi t - \int_0^t (\beta_1 i(x) + \beta_2 w(x) + p_1) s(x) dx + \int_0^t \eta_1 s(x) dB_1(x), \\ \int_0^t di(x) &= \int_0^t (\beta_1 s(x) + \beta_2 w(x) - \sigma - \mu - \mu_1) i(x) dx + \int_0^t \eta_2 i(x) dB_2(x), \\ \int_0^t dr(x) &= \int_0^t (vs(x) + \sigma i(x) - \mu r(x)) dx + \int_0^t \eta_3 r(x) dB_3(x), \\ \int_0^t dw(x) &= \int_0^t (\alpha i(x) - \eta w(x)) dx + \int_0^t \eta_4 w(x) dB_4(x), \end{aligned} \quad (24)$$

implies that

$$\begin{aligned}
\frac{s(t) - s(0)}{t} &= \Pi - \beta_1 \langle i(t)s(t) \rangle - \beta_2 \langle w(t)s(t) \rangle - p_1 \langle s(t) \rangle + \frac{\eta_1}{t} \int_0^t s(x) dB_1(x), \\
\frac{i(t) - i(0)}{t} &= \beta_1 \langle i(t)s(t) \rangle + \beta_2 \langle w(t)s(t) \rangle - p_2 \langle i(t) \rangle + \frac{\eta_2}{t} \int_0^t i(x) dB_2(x), \\
\frac{r(t) - r(0)}{t} &= \sigma \langle i(t) \rangle + v \langle s(t) \rangle - \mu \langle r(t) \rangle + \frac{\eta_3}{t} \int_0^t r(x) dB_3(x), \\
\frac{w(t) - w(0)}{t} &= -\eta \langle w(t) \rangle + \alpha \langle i(t) \rangle + \frac{\eta_4}{t} \int_0^t w(x) dB_4(x).
\end{aligned} \tag{25}$$

The addition of first two equations of the above system i.e., $\frac{s(t)-s(0)}{t} + \frac{i(t)-i(0)}{t}$ may be written as

$$\begin{aligned}
\frac{s(t) - s(0)}{t} + \frac{i(t) - i(0)}{t} &= \Pi - p_1 \langle s(t) \rangle - p_2 \langle i(t) \rangle + \frac{\eta_1}{t} \int_0^t s dB_1(x) \\
&+ \frac{\eta_2}{t} \int_0^t i(x) dB_2(x).
\end{aligned} \tag{26}$$

For the shake of simplicity, the notion $\Phi(t)$ will be used in Eq.(26) with some basic algebra we arrive at

$$\langle s(t) \rangle = \frac{\Pi}{p_1} - \frac{p_2}{p_1} \langle i(t) \rangle + \Phi(t), \tag{27}$$

where

$$\Phi(t) = -\frac{1}{p_1} \left[\frac{i(t) - i(0)}{t} + \frac{s(t) - s(0)}{t} \right] + \frac{\eta_1}{t} \int_0^t s(x) dB_1(x) + \frac{\eta_2}{t} \int_0^t i(x) dB_2(x).$$

It could be noted from the last result that the limiting value of $\Phi(t)$ is zero whenever t approaches ∞ i.e.,

$$\lim_{t \rightarrow \infty} \Phi(t) = 0 \quad a.s. \tag{28}$$

The virtue of Itô formula to the reported epidemic problem (1) gives

$$d \log i(t) = \beta_1 s(t) + \beta_2 \frac{s(t)w(t)}{i(t)} - p_2 - \frac{\eta_2^2}{2} + \eta_2 dB_2(t). \tag{29}$$

The integration of $d \log i(t)$ yields

$$\frac{1}{t} [\log i(t)]_0^t = \beta_1 \langle s(t) \rangle + \beta_2 \left\langle \frac{s(t)w(t)}{i(t)} \right\rangle - p_2 - \frac{\eta_2^2}{2} + \frac{\eta_2 B_2(t)}{t}. \tag{30}$$

It is very much clear from Eq.(5) that $s + i + r + w \leq 1$, thus we noted that $\left\langle \frac{s(t)w(t)}{i(t)} \right\rangle \leq \langle s(t)w(t) \rangle \leq \langle s(t) \rangle$ therefore the above assertion leads to the inequality is given by

$$\frac{1}{t}[\log i(t)]_0^t \leq (\beta_1 + \beta_2)\langle s(t) \rangle - p_2 - \frac{\eta_2^2}{2} + \frac{\eta_2 B_2(t)}{t}. \quad (31)$$

Using the value of $\langle s(t) \rangle$ with some algebraic manipulation and following the well known *strong law of large number* [22] i.e., $\limsup \frac{\xi_2 B_2}{t} = 0$ a.s as $t \rightarrow \infty$ we obtain

$$\limsup_{t \rightarrow \infty} \frac{\log i(t)}{t} \leq \left(p_2 + \frac{\eta_2^2}{2} \right) (R_0^S - 1) < 0 \text{ a.s.}, \quad (32)$$

implies that whenever the condition $R_0^S < 1$ holds, then $\lim i(t) = 0$ and so $\lim \langle i(t) \rangle = 0$ a.s., as $t \rightarrow \infty$. Moreover, the last equation of system (25) implies that

$$\langle w(t) \rangle = \frac{1}{\eta} \left\{ \alpha \langle i(t) \rangle + \frac{\eta_4}{t} \int_0^t w(x) dB_4(x) - \frac{w(t) - w(0)}{t} \right\}. \quad (33)$$

Since the limiting value of $i(t)$ is zero so it implies that $w(t) = 0$ whenever $t \rightarrow \infty$, so the first equation of system (25) looks like

$$\langle s(t) \rangle = \frac{1}{p_1} \left\{ \Pi + \frac{\eta_1}{t} \int_0^t s(x) dB_1(x) - \frac{s(t) - s(0)}{t} \right\}, \quad (34)$$

gives that if $t \rightarrow \infty$, $\lim s(t) = \Pi/p_1$. We conclude that the novel disease extinct continuously depend on the value of R_0^S , and ultimately whenever $R_0^S < 1$, it will extinct. $\square$

Theorem 2.5. *If $R_0^S > 1$ and (s_0, i_0, r_0, w_0) are any initial population sizes in $\mathbb{D}$, then whenever t approaches ∞ , so system (1) holds the conditions given below*

$$i_2 \leq \liminf \langle i(t) \rangle \leq \sup \langle i(t) \rangle \leq i_1 \text{ and } w_2 \leq \liminf \langle w(t) \rangle \leq \sup \langle w(t) \rangle \leq w_1, \quad (35)$$

where

$$\begin{aligned} i_1 &= \frac{p_1}{p_2(\beta_1 + \beta_2)} \left[\left\{ p_2 + \frac{\eta_2^2}{2} \right\} (R_0^S - 1) \right], \quad i_2 = \frac{p_1}{\beta_1 p_2} \left[\left\{ p_2 + \frac{\eta_2^2}{2} \right\} (R_1^S - 1) \right], \\ w_1 &= \frac{\alpha p_1}{\eta p_2(\beta_1 + \beta_2)} \left[\left\{ p_2 + \frac{\eta_2^2}{2} \right\} (R_0^S - 1) \right], \quad w_2 = \frac{\alpha p_1}{\eta \beta_1 p_2} \left[\left\{ p_2 + \frac{\eta_2^2}{2} \right\} (R_0^S - 1) \right]. \end{aligned} \quad (36)$$

Proof. We noted from Eq.(31) that

$$\langle i(t) \rangle \leq \frac{p_1}{p_2(\beta_1 + \beta_2)} \left[\left\{ p_2 + \frac{\eta_2^2}{2} \right\} (R_0^S - 1) \right] + (\beta_1 + \beta_2) \Phi(t) + \frac{\eta_2 B_2(t)}{t} - \frac{1}{t} [\log i(t)]_0^t.$$

The application of lim as t approaches ∞ with sup property to the above equation gives

$$\lim_{t \rightarrow \infty} \sup \langle i(t) \rangle \leq \frac{p_1}{p_2(\beta_1 + \beta_2)} \left[\left\{ p_2 + \frac{\eta_2^2}{2} \right\} (R_0^S - 1) \right] = i_1. \quad (37)$$

We can also write the following assertion from Eq.(31) that

$$\frac{1}{t} [\log i(t)]_0^t \geq \beta_1 \langle s(t) \rangle - p_2 - \frac{\eta_2^2}{2} + \frac{\eta_2 B_2(t)}{t}, \quad (38)$$

implies

$$\lim_{t \rightarrow \infty} \inf \langle i(t) \rangle \geq \frac{p_1}{\beta_1 p_2} \left[\left\{ p_2 + \frac{\eta_2^2}{2} \right\} (R_1^S - 1) \right] = i_2. \quad (39)$$

Now the last equation of system (25) can be re-written as

$$\langle w(t) \rangle = \frac{1}{\eta} \left[\alpha \langle i(t) \rangle + \frac{\eta_4}{t} \int_0^t w(x) dB_4(x) - \frac{w(t) - w(0)}{t} \right]. \quad (40)$$

Taking lim as $t \rightarrow \infty$ and sup of both sides we get

$$\lim_{t \rightarrow \infty} \sup \langle w(t) \rangle \leq \frac{\alpha p_1}{\eta p_2(\beta_1 + \beta_2)} \left[\left\{ p_2 + \frac{\eta_2^2}{2} \right\} (R_0^S - 1) \right] = w_1. \quad (41)$$

On the other hand lim as $t \rightarrow \infty$ with the application of inf property Eq.(40) takes the following form

$$\lim_{t \rightarrow \infty} \inf \langle w(t) \rangle \geq \frac{\alpha p_1}{\eta \beta_1 p_2} \left[\left\{ p_2 + \frac{\eta_2^2}{2} \right\} (R_1^S - 1) \right] = w_2. \quad (42)$$

Thus from Eq.(37)–Eq.(42) it could be noted that $i_2 \leq \liminf \langle i(t) \rangle \leq \limsup \langle i(t) \rangle \leq i_2$ and $w_2 \leq \liminf \langle w(t) \rangle \leq \limsup \langle w(t) \rangle \leq w_2$ whenever t tend to ∞ . $\square$

3 Numerical simulation

In this section we present the numerical simulation to verify the analytical work. Let us give a short over view to simulate the stochastic differential equations. Let

$$dX(t) = \alpha(t, X(t))dt + b(t, X(t))dB(t), \quad X(0) = X_0. \quad (43)$$

Producing a sample $X(t)$ around t with the utilization of the solution of the above equation, we will find $X(t)$ over continuous period of time. Making use of the notation $\tilde{X}_k$, B_k and $\tilde{X}(k\Delta t)$ for simplicity instead of $B(k\Delta t)$. We discretize the Eq.(43) gives

$$\tilde{X}_{\Delta t}, \tilde{X}_{2\Delta t}, \dots, \tilde{X}_{N\Delta t}. \quad (44)$$

In the above equation N symbolizes the time steps and $\Delta t = T/N$. It could be noted that the application of Itô-Taylor expansion leads to the stochastic Euler Maruyama (SEM) method to simulate the problem under consideration. To retrieve the discretized trajectory of $X(t)$ from the Eq.(43), we may use the algorithm of Euler Maruyama:

- a. Simulate ΔB_k as a normal distributed random variable $N(0, \Delta t)$.
- b. Putting $\tilde{X}_0 := X_0$ and applying $\tilde{X}_{k+1}$ by following the formula is given below

$$\tilde{X}_{k+1} = b(k\Delta t, \tilde{X})\Delta B_k + \alpha(k\Delta t, \tilde{X}_k)\Delta t + \tilde{X}_k, \quad (45)$$

for $\Delta B_k = B_{k+1} - B_k$ and $k = 0, \dots, N-1$.

The stochastic Euler Maruyama technique will be applied for the numerical simulation of system reported by Eq.(1) which take the form

$$\begin{aligned} s_{k+1} - s_k &= [\Pi - \beta_1 s_k i_k - \beta_2 s_k w_k - p_1 s_k] \Delta t + \eta_1 s_k \Delta B_{1k}, \\ i_{k+1} - i_k &= [\beta_1 s_k i_k + \beta_2 s_k i_k - p_2 i_k] \Delta t + \eta_2 i_k \Delta B_{2k}, \\ r_{k+1} - r_k &= [\sigma i_k + \nu s_k - \mu r_k] \Delta t + \eta_3 r_k \Delta B_{3k}, \\ w_{k+1} - w_k &= [\alpha i_k - \eta w_k] \Delta t + \eta_4 r_k \Delta B_{4k}, \end{aligned} \quad (46)$$

which implies that

$$\begin{aligned} s_{k+1} &= s_k + [\Pi - \beta_1 s_k i_k - \beta_2 s_k w_k - p_1 s_k] \Delta t + \eta_1 s_k \Delta B_{1k}, \\ i_{k+1} &= i_k + [\beta_1 s_k i_k + \beta_2 s_k i_k - p_2 i_k] \Delta t + \eta_2 i_k \Delta B_{2k}, \\ r_{k+1} &= r_k + [\sigma i_k + \nu s_k - \mu r_k] \Delta t + \eta_3 r_k \Delta B_{3k}, \\ w_{k+1} &= w_k + [\alpha i_k - \eta w_k] \Delta t + \eta_4 r_k \Delta B_{4k}. \end{aligned} \quad (47)$$

Using Matlab software and coding the above algorithm to solve the proposed system. To run our model for large scale numerical findings we use feasible parameters value with time units of 0 to 400 days. Once we execute the algorithm the following graphs are generated as given by Fig.5–Fig.12. This may verify our analytical findings. Moreover, Fig.5 to Fig.8 demonstrate the temporal dynamics of the susceptible, SARS-CoV-2 virus infected, recovered and the reservoir respectively, which theoretically investigate that there will be always susceptible and recovered population while the SARS-CoV-2 virus infected population and reservoir will vanishes. This may verify the results of our extinction analysis. Since the disease extinct continuously depend on the *basic reproductive parameter* and whenever $R_0^S < 1$ the disease could be easily eliminated. So from biological point of view it is very much important to keep this quantity low as much as possible to eliminate disease. On the other hand Fig.9 to Fig.12 visualize the persistence analysis of the proposed problem. We noted that in this the trajectories of susceptible $s(t)$, SARS-CoV-2 virus infected ($i(t)$), recovered ($r(t)$) and reservoir ($w(t)$) reveals that the disease will persist and all these compartment reach to its endemic stage whenever the value of $R_0^S > 1$. So a special attention is required to make a control mechanism. Since the sensitivity analysis reveals that the disease transmission co-efficient has the highest sensitivity index and a great influence on the threshold parameter therefore minimization of this parameter would significantly decrease the value of the threshold parameter. It could be also noted from the sensitivity index of the vaccination parameter that vaccination has also a strong influence and so increasing the vaccination would strongly decrease the value of *basic reproductive number*. Finally we also noted a relation between the noise intensity with disease extinction and persistence i.e., there is a direct relation between intensity of white noise and extinction while inverse relation between intensity of white noise and persistence.

4 Conclusion

We developed a correlated stochastic epidemic model and to discuss temporal dynamics of SARS-CoV-2 virus keeping in view the various source of randomness and vaccination of susceptible individuals. We studied the basic axioms and proved that the model is well posed both mathematically and biologically. We also discussed the SARS-CoV-2 extinction analysis and persistence, and obtained the conditions for it. The detailed sensitivity analysis has been performed and showed that the disease transmission

Figure 5: The graph visualizes the temporal dynamics of the epidemic problem described by model (1) in the large scale for the class of susceptible individuals ($s(t)$) in case of extinction. The parameters value used are taken from S_1 while $(0.5, 0.3, 0.2, 0.1)$ are assumed to be the initial size of population

Figure 6: The graph visualizes the time dynamics of the model (1) in case of extinction for the class of asymptomatic population ($a(t)$) against parametric values taken from S_1 and $(0.5, 0.3, 0.2, 0.1)$ are the initial sizes for compartmental population.

Figure 7: The graph visualize the temporal dynamics of the model under consideration (1) in the long run for the symptomatic class ($c(t)$) against the parametric value taken from S_1 and initial classes $(0.5, 0.3, 0.2, 0.1)$.

Figure 8: The graph visualizes the time dynamics of the reported model (1) in case of extinction for the recovered population ($r(t)$) subject to the parametric values of S_1 and $(0.5, 0.3, 0.2, 0.1)$ initial populations.

Figure 9: The graph visualizes the dynamics of the epidemic problem described by model (1) in case of persistence for the susceptible class ($s(t)$) against the parameters values taken from S_2 and $(0.5, 0.3, 0.2, 0.1)$ are the initial sizes of population.

Figure 10: The graph visualizes the persistence of the epidemic problem framed by model (1) for the asymptomatic class ($a(t)$) against parameters value taken from S_2 and various sizes of initial population (0.5, 0.3, 0.2, 0.1).

Figure 11: The graph visualizes the time dynamics of model (1) in large scale for symptomatic population ($c(t)$) against the parametric value of S_2 and (0.5, 0.3, 0.2, 0.1) initial population.

Figure 12: The graph visualizes the large scale numerical simulation of the recovered class ($r(t)$) of the model reported by Eq.(1) against parameters value S_2 and $(0.5, 0.3, 0.2, 0.1)$.

co-efficient and vaccination parameters are the highest sensitive parameters to the disease transmission and control. It could be justified that the intensity of noise and vaccination has a major role on the temporal dynamics of the SARS-CoV-2. Also we noted that vaccination and the white noise intensity are directly related with SARS-CoV-2, such that rising the value of these two parameters would significantly increases the disease extinction. However there is an inverse relation of disease persistence with vaccination and white noise intensity are observed. The disease persistence reduction is subjected to the speedily vaccination and therefore there is a need of a fast vaccination immunization. We present some numerical findings at the end of the manuscript and supported the analytical results.

References

- [1] G. Zaman, Y. H. Kang, and I. H. Jung, “Stability analysis and optimal vaccination of an sir epidemic model,” *BioSystems*, vol. 93, no. 3, pp. 240–249, 2008.
- [2] Y. Wang and J. Cao, “Global dynamics of a network epidemic model for waterborne diseases spread,” *Applied Mathematics and Computation*,

vol. 237, pp. 474–488, 2014.

- [3] H. Abboubakar, J. C. Kamgang, and D. Tieudjo, “Backward bifurcation and control in transmission dynamics of arboviral diseases,” *Mathematical biosciences*, vol. 278, pp. 100–129, 2016.
- [4] T. Khan, G. Zaman, and M. I. Chohan, “The transmission dynamic and optimal control of acute and chronic hepatitis b,” *Journal of biological dynamics*, vol. 11, no. 1, pp. 172–189, 2017.
- [5] J. K. K. Asamoah, F. Nyabadza, B. Seidu, M. Chand, and H. Dutta, “Mathematical modelling of bacterial meningitis transmission dynamics with control measures,” *Computational and mathematical methods in medicine*, vol. 2018, 2018.
- [6] M. A. Dokuyucu and H. Dutta, “A fractional order model for ebola virus with the new caputo fractional derivative without singular kernel,” *Chaos, Solitons & Fractals*, vol. 134, p. 109717, 2020.
- [7] W. C. C. for Infectious Disease Modelling, M. C. for Global Infectious Disease Analysis, A. L. J. I. for Disease, E. Analytics, and I. C. London, “Impact of non-pharmaceutical interventions (npis) to reduce covid-19 mortality and healthcare demand,” 2020.
- [8] A. J. Kucharski, T. W. Russell, C. Diamond, Y. Liu, J. Edmunds, S. Funk, R. M. Eggo, F. Sun, M. Jit, J. D. Munday, *et al.*, “Early dynamics of transmission and control of covid-19: a mathematical modelling study,” *The lancet infectious diseases*, vol. 20, no. 5, pp. 553–558, 2020.
- [9] T. Kuniya, “Prediction of the epidemic peak of coronavirus disease in japan, 2020,” *Journal of clinical medicine*, vol. 9, no. 3, p. 789, 2020.
- [10] R. O. Stutt, R. Retkute, M. Bradley, C. A. Gilligan, and J. Colvin, “A modelling framework to assess the likely effectiveness of facemasks in combination with lock down in managing the covid-19 pandemic,” *Proceedings of the Royal Society A*, vol. 476, no. 2238, p. 20200376, 2020.
- [11] Z. Tang, X. Li, and H. Li, “Prediction of new coronavirus infection based on a modified seir model,” *medRxiv*, 2020.

- [12] H. M. Dobrovolny, “Modeling the role of asymptomatics in infection spread with application to sars-cov-2,” *Plos one*, vol. 15, no. 8, p. e0236976, 2020.
- [13] H. M. Dobrovolny, “Quantifying the effect of remdesivir in rhesus macaques infected with sars-cov-2,” *Virology*, vol. 550, pp. 61–69, 2020.
- [14] S. Mandal, T. Bhatnagar, N. Arinaminpathy, A. Agarwal, A. Chowdhury, M. Murhekar, R. R. Gangakhedkar, and S. Sarkar, “Prudent public health intervention strategies to control the coronavirus disease 2019 transmission in india: A mathematical model-based approach,” *The Indian journal of medical research*, vol. 151, no. 2-3, p. 190, 2020.
- [15] R. F. Reis, B. de Melo Quintela, J. de Oliveira Campos, J. M. Gomes, B. M. Rocha, M. Lobosco, and R. W. Dos Santos, “Characterization of the covid-19 pandemic and the impact of uncertainties, mitigation strategies, and underreporting of cases in south korea, italy, and brazil,” *Chaos, Solitons & Fractals*, vol. 136, p. 109888, 2020.
- [16] M. Aguiar, J. B. Van-Dierdonck, J. Mar, N. Cusimano, D. Knopoff, V. Anam, and N. Stollenwerk, “Critical fluctuations in epidemic models explain covid-19 post-lockdown dynamics,” *Scientific Reports*, vol. 11, no. 1, pp. 1–12, 2021.
- [17] R. Ma, X. Zheng, P. Wang, H. Liu, and C. Zhang, “The prediction and analysis of covid-19 epidemic trend by combining lstm and markov method,” *Scientific Reports*, vol. 11, no. 1, pp. 1–14, 2021.
- [18] J. Tao, Y. Ma, C. Luo, J. Huang, T. Zhang, and F. Yin, “Summary of the covid-19 epidemic and estimating the effects of emergency responses in china,” *Scientific reports*, vol. 11, no. 1, pp. 1–9, 2021.
- [19] T. Khan, G. Zaman, and Y. E. Khatib, “Modeling the dynamics of novel coronavirus (covid-19) via stochastic epidemic model,” *Results in Physics*, p. 104004, 2021.
- [20] H. Kuo, “Introduction to stochastic integration springer,” *Berlin Heidelberg*, 2006.
- [21] Q. Lei and Z. Yang, “Dynamical behaviors of a stochastic siri epidemic model,” *Applicable Analysis*, vol. 96, no. 16, pp. 2758–2770, 2017.

- [22] T. Birkel, “A note on the strong law of large numbers for positively dependent random variables,” *Statistics & Probability Letters*, vol. 7, no. 1, pp. 17–20, 1988.