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

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In this paper, we propose a mathematical model to describe the influence of the SARS-CoV-2 virus with correlated sources of randomness and with vaccination. The total human population is divided into three groups: susceptible, infected, and recovered. Each population group of the model is assumed to be subject to various types of randomness. We develop the correlated stochastic model by considering correlated Brownian motions for the population groups. As the environmental reservoir plays a weighty role in the transmission of the SARS-CoV-2 virus, our model encompasses a fourth stochastic differential equation representing the reservoir. Moreover, the vaccination of susceptible is also considered. Once the correlated stochastic model, the existence and uniqueness of a positive solution are discussed to show the problem’s feasibility. The SARS-CoV-2 extinction, as well as persistency, are also examined, and sufficient conditions resulted from our investigation. The theoretical results are supported through numerical/graphical findings.

In Wuhan, China, a respiratory disease outbreak has been started in December 2019. Later, it was identified as a novel coronavirus (COVID-19), known as the SARS-CoV-2 virus. The initial spreading source of the novel disease was an animal. But the pandemic rises from human interaction. Total of 589 million infected individuals have been reported while around 6.5 million deaths occurred till August 13, 2022, around the world. Vaccination is an important weapon against controlling a disease. In the case of the SARS-CoV-2 virus, disease vaccination is very important and there are many vaccines that could be shown their effectiveness. World Health Organization (WHO) investigates that reliable vaccinations program will change the situation. But precautionary measures could be necessary for the time being as it is still doubtful that the vaccine of SARS-CoV-2 provides how many degrees 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 disease and predicting for future. Also, effective control programs have been forecasted to suggest useful guidelines for 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 dynamic behavior of disease by suggesting control mechanisms¹–⁴. Covid-19 also called the SARS-CoV-2 virus and its vaccination is a challenging task, which attracts the attention of many researchers, (see⁷–¹⁵). The reported literature reveals that the mathematical models which have been analyzed are simple and used deterministic approaches. However, the SARS-CoV-2 virus transmission is influenced by different factors (social behavior, age, mobility, virus mutation, etc.) that can affect the dynamics¹⁶–²². So from the characteristic of the disease, it could be very interesting if the stochastic approach will be used. A stochastic model has been studied for the novel coronavirus by Khan et al.,²³ very recently, where the random fluctuation is assumed in transmission rate only, while as reported above that due to many factors the SARS-CoV-2 virus is influenced. The main contribution of this paper is to suggest an alternative stochastic model for the SARS-CoV-2 virus, where each population group has its own randomness source, but they are all related by correlation factors. In addition, the correlated suggested model includes the vaccination impact. We formulate a stochastic mathematical model 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 individual class has various Brownian motions according to the disease characteristics. The vaccination of susceptible individuals is also assumed to investigate the efficiency of vaccination and its role in the minimization of the infection. First, the models will be formulated and then analyzed to discuss the detailed 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 is extinct as well as persists. It is essential to discuss extinction and persistence when investigating virus spread. The aim of this analysis is to determine when the disease will end (extinct) and under which conditions will stay (persist). Finally, all analytical findings will be supported by using some graphical representation in the form of a large-scale numerical simulation by using the 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.

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 \(W(t) := (W_i(t)) : \) such that \(i = 1, \ldots, 4\), 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

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

We classify the total human population into 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 \(w(t)\). The quantity \(w\) is the environmental reservoir which is an important element in the study of our epidemic model. It represents the concentration of the coronavirus in the environmental reservoir and it includes rates of the infected individuals contributing the coronavirus to the environmental reservoir and the removal rate of the virus from the environment. All the population groups and the reservoir is distributed by different Brownian motions. The schematic diagram for the distribution process of the various population groups is given in Fig. 1. Thus we suggest a correlated stochastic epidemic model by the following system:

\[
\begin{align*}
ds(t) &= \left[ \Pi - (\beta_1 i(t) + \beta_2 w(t) + \mu + \nu) s(t) \right] dt + \eta_1 s(t) dB_1(t), \\
\di(t) &= \left[ (\beta_1 i(t) + \beta_2 w(t)) s(t) - (\sigma + d_1 + \mu) i(t) \right] dt + \eta_2 i(t) dB_2(t), \\
\dr(t) &= \left[ \sigma r(t) + \nu s(t) - \mu r(t) \right] dt + \eta_3 r(t) dB_3(t), \\
dw(t) &= \left[ \alpha i(t) - \eta w(t) \right] dt + \eta_4 w(t) dB_4(t).
\end{align*}
\]  

(1)

The above-proposed model is a generalization of standard epidemic deterministic models. It allows the different quantity of the model to vary stochastically, which mean that the variations are not only time-dependent but also subject to haphazard fluctuations. The random noise detected from real data is considered in the above stochastic model but neglected in deterministic models. In Eq. (1) the various parameters are characterized as: the newborn rate is symbolized with \(\Pi\), and \(\beta_i\), \(i = 1, 2\), are routes of disease transmission from the infected human as well as from the reservoir. Moreover, \(\nu\) is the vaccination of the susceptible population and \(\mu\) is the natural death rate while death from the disease is described with \(d_1\). We also symbolize the recovery rate by \(\sigma\) and a rate contributed to the virus to the environment by \(\alpha\). The removing SARS-CoV virus rate is denoted by \(\eta\). If \(\lambda_{ki} = 1\) for \(k = 1, 2, 3, 4\), and \(\lambda_{ki} = 0\) otherwise, then \(B_1 = B_2 = B_3 = B_4\) and the model is reduced to the stochastic model studied in Khan et al.\(^\text{13}\). Also, it could be clearly noted that the above system (1) will reduce to the deterministic form, whenever \(\eta_1 = \eta_2 = \eta_3 = \eta_4 = 0\). It can be seen also an extension of\(^\text{9}\). In addition
By following this formula we calculate the sensitivity indices of model parameters $\alpha$, $\beta$, $\eta$, and $\sigma$ with respect to $R_0$, $\mu$, $\nu$, and $d_1$, respectively.

The disease-free and endemic equilibriums of the associated deterministic form of the model are respectively symbolized with $E_0 = (S_0, 0, 0, I_0)$ and $E^* = (S^*, I^*, R^*, W^*)$ with $S_0 = V_i/\mu$, $R_0 = \nu (V_i/\eta)$, where $p_1 = \mu + \nu$. 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

$$\frac{dX}{dt} = -V + F,$$

and $F = \begin{bmatrix} \beta_1 S_0 & 0 \\ 0 & 0 \end{bmatrix}$, $V = \begin{bmatrix} p_2 \\ -\alpha \eta \end{bmatrix}$. \hfill (2)

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

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

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

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

**Sensitivity analysis.** In every disease the role of the threshold parameter (basic reproductive number) is very important and the disease spreads whenever the value of this quantity is more than one and the disease dies out if its value is less than unity. We will discuss the sensitivity of threshold parameter to find the relation between sensitive parameters to the disease transmission and control. We observed that increasing the value of $\beta_1$ and $\beta_2$ by 10% decreases the value of $R_0$ by 9.9% as depicted in Fig. 2, while increasing the value of $\nu$ by 10% decreasing the value of $R_0$ by 8.3% as shown in Fig. 5. Similarly, $\beta_2$ and $\sigma$ collectively have the highest sensitivity index and so are the most sensitive parameters to the disease transmission and control. We observed that increasing the value of $\beta_1$ by 10% would significantly increase the value of $R_0$ by 9.9% as depicted in Fig. 2, while increasing the value of $\nu$ by 10% decreasing the value of $R_0$ by 8.3% as shown in Fig. 5. Similarly, $\beta_2$ and $\sigma$ collectively effect $R_0$ by 10% as depicted by Figs. 3 and 4. The relation between $\sigma$ and $R_0$ is also an inverse as increased $\sigma$ by 10% would decrease the threshold quantity by 6.86% is given in Fig. 5.

**Existence and uniqueness analysis.** In this portion of the manuscript the existence of the solution and uniqueness with the positivity of Eq. (1) will be discussed.

It is worth mentioning that the Itô formula is one of the most useful formulas in stochastic calculus. It is utilized, among others, to solve stochastic differential equations. Here, we describe a Multidimensional Itô formula for getting our results by following the book of stochastic calculus.24.
Figure 3. The graph visualizes the variation of the basic reproductive number against $\beta_1$ and $\alpha$.

Figure 4. The graph visualizes the variation of the basic reproductive number against $\beta_2$ and $\alpha$.

Figure 5. The graph visualizes the variation of the basic reproductive number against $\sigma$ and $\nu$. 
Lemma 2.1 Let \( a = (a_1, \ldots, a_n) \) and \( b = (\beta_1, \ldots, \beta_n) \) represent the adapted processes with square-integrable \( n \)-dimensional. We consider \( X = (X_1, \ldots, X_n) \), where \( X_k \) is driven by the stochastic differential equation and \( k \in \{1, \ldots, n\} \), 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 \( f : \mathbb{R}^n \to \mathbb{R} \), then we have
\[
dF(X(t)) = \sum_{k=1}^{n} \frac{\partial F}{\partial x_k}(X(t))dX_k(t) + \sum_{k,l=1}^{n} \frac{1}{2} \frac{\partial^2 F}{\partial x_k \partial x_l}(X(t))d\langle X_k, X_l \rangle(t),
\]
where \( d\langle X_k, X_l \rangle(t) = b_k(t)b_l(t)dt, dt = d\langle B(t), B(t) \rangle, \) and \( d\langle B(t), t \rangle = d\langle t, t \rangle = d\langle t, B(t) \rangle = 0. \)

We use the Lyapunov theory and the virtue of the Itô formula to prove that the solution of Eq. (1) exists globally and is positive. Define
\[
\mathbb{D} = \{(s, i, r, w) \in \mathbb{R}^4_+ : s \text{ and } r > 0, i, w \geq 0, s + i + r + w \leq 1\}. \tag{5}
\]
The result that discusses the existing 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 \( \mathbb{R}^4_+ \), then the solution \( (s(t), i(t), r(t), w(t)) \) of the model (1) is unique as well as remains in \( \mathbb{R}^4_+ \) 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\textsuperscript{25} 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 \( [0, \tau_c] \) subject to initial conditions in \( \mathbb{R}^4_+ \) is unique and local for the explosion time \( \tau_c \). Moreover, we investigate that \( \tau_c = \infty \) a.s as to show the solution globalization. It is assumed that \( k_0 \geq 0 \) is sufficiently large and \( \frac{1}{k_0} < N(0) < k_0 \), where \( N(0) = (s(0), a(0), c(0), r(0)) \). We define the stopping time for every \( k \geq k_0 \) as:
\[
\tau_k = \inf \left\{ t \in [0, \tau_c) : \max(s(t), i(t), r(t), w(t)) \leq \frac{1}{k} \right\}. \tag{6}
\]

Further, let \( \phi \) is empty set and \( \inf \phi = \infty \). Since \( \tau_k \) depend on \( k \) and whenever \( k \) increasing \( \tau_k \) also increasing as \( k \) increases without bound i.e., tend to \( \infty \). Making use of \( \lim_{k \to \infty} \frac{\tau_k}{\tau} = \infty \) with taking \( \tau_c = \infty \) a.s gives that \( (s(t), i(t), r(t), w(t)) \in \mathbb{R}^4_+ \), \forall t \geq 0 \) a.s. We now only need to show that \( \tau_c = \infty \). For this, we use the assumption that for any two constants, \( T > 0 \) and \( \varepsilon \in (0, 1) \), we have
\[
P[\tau_c \leq T] > \varepsilon. \tag{7}
\]
So \( k_1 \geq k_0 \) is an integer that
\[
P[\tau_k \leq T] \geq \varepsilon, \quad \text{for every } k \geq k_1. \tag{8}
\]

Let \( H \) is twice continuously differentiable function i.e., \( H \in C^2 \) and \( H : \mathbb{R}^4_+ \to \mathbb{R}_+ \) by
\[
H(s, i, r, w) = s - 1 - \log(s) + i - (r + w) - 1 - \log(r) + w - 1 - \log(w). \tag{9}
\]

Clearly, \( H \geq 0 \), so for \( 0 \leq T \) and \( k_0 \leq k \), and by the application of the Itô formula leads to the assertion
\[
dH = LHdt + (s - 1)\eta_1dB_1 + (i - 1)\eta_2dB_2 + (r - 1)\eta_3dB_3 + (w - 1)\eta_4dB_4. \tag{10}
\]

In Eq. (10), \( LH \) is defined as
\[
LH = (1 - 1/s)(\Pi - \beta_1 s - \beta_2 s w - (\mu + \nu) s) + \frac{1}{2} \eta_1^2 + (1 - 1/i)\beta_1 si
\]
\[
+ \beta_2 s w - (\mu + d_1 + \sigma)s + \frac{1}{2} \eta_2^2 + (1 - 1/r)(v s + \sigma s - i - \mu r) + \frac{1}{2} \eta_3^2 + (1 - 1/w)
\]
\[
\times (\alpha i - \eta w) + \frac{1}{2} \eta_4^2. \tag{11}
\]

Simplifying and re-writing the above equation may lead to the following inequality
\[
LH \leq \Pi + (\beta_1 + \alpha)i + \beta_2w + vs + 3\mu + v + d_1 + \sigma + \eta. \tag{12}
\]

It could be noted from the fact that \( s + i + r + w \leq 1 \), so the last inequality gives
\[
LH \leq \Pi + \beta_1 + \beta_2 + \alpha + 2v + 3\mu + d_1 + \sigma + \eta \equiv K. \tag{13}
\]

Plugging Eq. (13) in Eq. (10) we may arrive
\[
dH \leq Kdt + (s - 1)\eta_1dB_1 + (i - 1)\eta_2dB_2 + (r - 1)\eta_3dB_3 + (w - 1)\eta_4dB_4.
\]

The integration of both sides reveals that
\[
\int_0^{T_k \wedge T} dH \leq \int_0^{T_k \wedge T} K dt + \int_0^{T_k \wedge T} (s - 1) \eta_1 dB_1 + \int_0^{T_k \wedge T} (i - 1) \eta_2 dB_2 + \int_0^{T_k \wedge T} (r - 1) \eta_3 dB_3 + \int_0^{T_k \wedge T} (w - 1) \eta_4 dB_4.
\] (14)

The expectation of both sides provides
\[
E \left[ H(s(T_k \wedge T), i(T_k \wedge T), r(T_k \wedge T), w(T_k \wedge T)) \right] \leq H(s(0), i(0), r(0), w(0)) + E \left[ \int_0^{T_k \wedge T} K dt \right],
\]

which implies that
\[
E \left[ H(s(T_k \wedge T), i(T_k \wedge T), r(T_k \wedge T), w(T_k \wedge T)) \right] \leq H(s(0), i(0), r(0), w(0)) + TK.
\] (15)

Setting a notion of \( \Omega_k = T \geq T_k \) for all \( k \geq k_i \). The use of Eq. (7) gives that \( P(\Omega_k) \geq \epsilon \). Noted that there is at least one \( s(\omega, T_k) \) or \( i(\omega, T_k) \) or \( r(\omega, T_k) \) or \( w(\omega, T_k) \) equal \( 1/k \) or \( k \) for all \( \omega \in \Omega_k \). Since \( \frac{1}{k} + \log k - 1 < \log k + k - 1 \). Hence
\[
(s(T_k, \omega), i(T_k, \omega), r(T_k, \omega), w(T_k, \omega)) \geq \left( \frac{1}{k} - 1 + \log k \right) \cap \left( -\log k - 1 + k \right).
\] (16)

So Eqs. (7) and (15) gives
\[
H(N(0)) + TK \geq E \left[ 1_{\Omega_k(\omega)} H(s(T_k \wedge T), i(T_k \wedge T), r(T_k \wedge T), w(T_k \wedge T)) \right],
\]

\[
= E \left[ 1_{\Omega_k(\omega)} \left( \log k - 1 + \frac{1}{k} \right) \right] \wedge \left( -\log k - 1 + k \right) = \left( \log k - 1 + \frac{1}{k} \right) \wedge \left( -\log k - 1 + k \right) E[1_{\Omega_k(\omega)}],
\]

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

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

\( \square \)

Remark 1 The uniqueness as well as the existence reveals that for any initial compartments \( (N(0)) \in \mathbb{R}_+^4 \), the unique solution with global axiom \( (s, i, r, w) \in \mathbb{R}_+^4 \) almost surly (a.s) exists for the proposed problem under consideration as reported by Eq. (1). The previous result can be also proved by the next theorem.

Theorem 2.3 Let \( (s, i, r, w) \) be the solutions of the stochastic differential equations of our model as stated by Eq. (1). The solutions \( (s, i, r, w) \)

Proof We follow\textsuperscript{28} to discuss the solutions of Eq. (1) which becomes
\[
X_k(t) = \xi_k(t) \left[ X_k(0) + \int_0^t [a_k(u) - \sum_{j=1}^m \theta_{kj}(u)g_{kj}(u)i^{2}/j_k] \xi_k^{-1}(u)du \right. \\
+ \left. \sum_{j=1}^m \int_0^t \gamma_{kj}(t) \lambda_{kj} \xi_k^{-1}(u)dW_j(u) \right].
\] (17)

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

Here \( k = 4, m = 4, \lambda_{kj} = \lambda_{kj}, g_{kj} = 0 \) for \( k, j = 1, 2, 3, 4 \) and
Now it could be described that the persistence of novel coronavirus SARS-CoV-2 is subjected to analysis we define that and extinction. These expressions containing the model parameters and intensities of noises. Before the formal statement holds, the epidemic problem represented by Eq. (1) states that the disease will persist. Thus for the extinction problem represented by Eq. (1) is symbolized by $R_0^s$ and define as $R_0^s = R_1^s + R_2^s$, where

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

Similarly, if

$$\lim_{t \to \infty} \inf \int_0^t i(x)dx > 0, \ a.s.,$$

and

$$\lim_{t \to \infty} \inf \int_0^t w(x)dx > 0, \ a.s.,$$

holds, the epidemic problem represented by Eq. (1) states that the disease will persist. Thus for the extinction and persistence 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

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

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 expressions to show permanence and extinction. These expressions containing the model parameters and intensities of noises. Before the formal analysis we define that

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

Now it could be described that the persistence of novel coronavirus 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 $\infty$. 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

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

Similarly, if

$$\lim_{t \to \infty} \inf \int_0^t i(x)dx > 0, \ a.s.,$$

and

$$\lim_{t \to \infty} \inf \int_0^t w(x)dx > 0, \ a.s.,$$

holds, the epidemic problem represented by Eq. (1) states that the disease will persist. 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 then unity i.e.,

$$\lim_{t \to \infty} \sup \frac{\log i(t)}{t} \leq \left( p_1 + \frac{1}{2} \xi_2 \right) (R_0^s - 1) < 0 \ a.s.$$

Also

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

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

$$\int_0^t ds(x) = \Pi t - \int_0^t (\beta_1 i(x) + \beta_2 w(x) + p_1) s(x)dx + \int_0^t \eta_1 s(x)dB_1(x),$$

$$\int_0^t di(x) = \int_0^t (\beta_1 s(x) + \beta_2 w(x) - \sigma - \mu - \mu_1) i(x)dx + \int_0^t \eta_2 i(x)dB_2(x),$$

$$\int_0^t dr(x) = \int_0^t (v s(x) + \sigma i(x) - \mu r(x))dx + \int_0^t \eta_3 r(x)dB_3(x),$$

$$\int_0^t dw(x) = \int_0^t (\alpha i(x) - \eta w(x))dx + \int_0^t \eta_4 w(x)dB_4(x),$$

implies that
\[
\frac{s(t) - s(0)}{t} = \Pi - \beta_1 \langle i(t) s(t) \rangle - \beta_2 \langle w(t) s(t) \rangle - p_1 \langle s(t) \rangle + \frac{\eta_1}{t} \int_0^t s(x) dB_1(x),
\]
\[
\frac{i(t) - i(0)}{t} = \beta_1 \langle i(t) s(t) \rangle + \beta_2 \langle w(t) s(t) \rangle - p_2 \langle i(t) \rangle + \frac{\eta_2}{t} \int_0^t i(x) dB_2(x),
\]
\[
\frac{r(t) - r(0)}{t} = \sigma \langle i(t) \rangle + \nu \langle s(t) \rangle - \mu \langle r(t) \rangle + \frac{\eta_3}{t} \int_0^t r(x) dB_3(x),
\]
\[
\frac{w(t) - w(0)}{t} = -\eta \langle w(t) \rangle + \alpha \langle i(t) \rangle + \frac{\eta_4}{t} \int_0^t w(x) dB_4(x).
\]

The addition of the 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
\[
\frac{s(t) - s(0)}{t} + \frac{i(t) - i(0)}{t} = \Pi - p_1 \langle s(t) \rangle - p_2 \langle i(t) \rangle + \frac{\eta_1}{t} \int_0^t s(x) dB_1(x) + \frac{\eta_2}{t} \int_0^t i(x) dB_2(x).
\]

For the sake of simplicity, the notion \( \Phi(t) \) will be used in Eq. (26) with some basic algebra we arrive at
\[
\langle s(t) \rangle = \frac{\Pi}{p_1} - \frac{p_2}{p_1} \langle i(t) \rangle + \Phi(t),
\]
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 \( \infty \) i.e.,
\[
\lim_{t \to \infty} \Phi(t) = 0 \quad \text{a.s.}
\]

The virtue of the Itô formula to the reported epidemic problem (1) gives
\[
d \log i(t) = \beta_1 s(t) + \beta_2 \frac{s(t) w(t)}{i(t)} - p_2 - \frac{\eta_2^2}{2} + \eta_2 dB_2(t).
\]

The integration of \( d \log i(t) \) yields
\[
\frac{1}{t} \log i(t) \bigg|_{t=0}^t = \beta_1 \langle s(t) \rangle + \beta_2 \left( \frac{s(t) w(t)}{i(t)} \right) - p_2 - \frac{\eta_2^2}{2} + \frac{\eta_2 B_2(t)}{t}.
\]

It is very much clear from Eq. (5) that \( s + i + r + w \leq 1 \), thus we noted that \( \langle \frac{s(t) w(t)}{i(t)} \rangle \leq \langle s(t) \rangle \) therefore the above assertion leads to the inequality given by
\[
\frac{1}{t} \log i(t) \bigg|_{t=0}^t \leq (\beta_1 + \beta_2) \langle s(t) \rangle - p_2 - \frac{\eta_2^2}{2} - \frac{\eta_2 B_2(t)}{t}.
\]

Using the value of \( \langle s(t) \rangle \) with some algebraic manipulation and following the well-known strong law of large number\(^2\) i.e., \( \limsup_{t \to \infty} \frac{i(t)}{t} = 0 \) a.s as \( t \to \infty \) we obtain
\[
\lim_{t \to \infty} \sup_{t \geq 0} \frac{\log i(t)}{t} \leq \left( p_2 + \frac{\eta_2^2}{2} \right) \left( \frac{R_0^S}{2} - 1 \right) < 0 \text{ a.s.,}
\]
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 \( t \to \infty \). Moreover, the last equation of system (25) implies that
\[
\langle w(t) \rangle = \frac{1}{\eta} \left\{ \alpha \langle i(t) \rangle + \frac{\eta_4}{t} \int_0^t w(x) dB_4(x) - \frac{w(t) - w(0)}{t} \right\}.
\]

Since the limiting value of \( i(t) \) is zero then \( w(t) = 0 \) whenever \( t \to \infty \), thus the first equation of the system (25) looks like
\[
\langle s(t) \rangle = \frac{1}{p_1} \left\{ \Pi + \frac{\eta_1}{t} \int_0^t s(x) dB_1(x) - \frac{s(t) - s(0)}{t} \right\},
\]
gives that if \( t \to \infty \), \( \lim s(t) = \Pi/p_1 \). We conclude that the novel disease extinct continuously depends on the value of \( R_0^S \), and ultimately whenever \( R_0^S < 1 \), it will extinct. \( \Box \)
We have seen from the previous theorem that the virus will die out exponentially if $R_0^\delta < 1$. The next theorem discusses the case when the stochastic reproductive number parameter $R_0^\delta > 1$ is greater than one.

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

$$i_2 \leq \lim \inf \langle \hat{i}(t) \rangle \leq \sup \langle \hat{i}(t) \rangle \leq i_1 \text{ and } w_2 \leq \lim \inf \langle \hat{w}(t) \rangle \leq \sup \langle \hat{w}(t) \rangle \leq w_1,$$

where

$$i_1 = \frac{p_1}{p_2(\beta_1 + \beta_2)} \left\{ p_2 + \frac{\eta_2^2}{2} \right\} (R_0^\delta - 1), \quad i_2 = \frac{p_1}{\beta_1 p_2} \left\{ p_2 + \frac{\eta_2^2}{2} \right\} (R_0^\delta - 1),$$

$$w_1 = \frac{\alpha p_1}{\eta p_2(\beta_1 + \beta_2)} \left\{ p_2 + \frac{\eta_2^2}{2} \right\} (R_0^\delta - 1), \quad w_2 = \frac{\alpha p_1}{\eta \beta_1 p_2} \left\{ p_2 + \frac{\eta_2^2}{2} \right\} (R_0^\delta - 1).$$

**Proof** We noted from Eq. (31) that

$$\langle \hat{i}(t) \rangle \leq \frac{p_1}{p_2(\beta_1 + \beta_2)} \left\{ p_2 + \frac{\eta_2^2}{2} \right\} (R_0^\delta - 1) + (\beta_1 + \beta_2) \Phi(t) + \frac{\eta_2 B_2(t)}{t} - \frac{1}{t}[\log \langle \hat{i}(t) \rangle]^0.$$ 

The application of $\lim$ as $t$ approaches $\infty$ with $\sup$ property to the above equation gives

$$\lim_{t \to \infty} \sup \langle \hat{i}(t) \rangle \leq \frac{p_1}{p_2(\beta_1 + \beta_2)} \left\{ p_2 + \frac{\eta_2^2}{2} \right\} (R_0^\delta - 1) = i_1.$$ 

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

$$\frac{1}{t}[\log \langle \hat{i}(t) \rangle]^0 \geq \beta_1 \langle \hat{s}(t) \rangle - p_2 - \frac{\eta_2^2}{2} + \frac{\eta_2 B_2(t)}{t},$$

implies

$$\lim_{t \to \infty} \inf \langle \hat{i}(t) \rangle \geq \frac{p_1}{\beta_1 p_2} \left\{ p_2 + \frac{\eta_2^2}{2} \right\} (R_0^\delta - 1) = i_2.$$ 

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

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

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

$$\lim_{t \to \infty} \sup \langle \hat{w}(t) \rangle \leq \frac{\alpha p_1}{\eta p_2(\beta_1 + \beta_2)} \left\{ p_2 + \frac{\eta_2^2}{2} \right\} (R_0^\delta - 1) = w_1.$$ 

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

$$\lim_{t \to \infty} \inf \langle \hat{w}(t) \rangle \geq \frac{\alpha p_1}{\eta \beta_1 p_2} \left\{ p_2 + \frac{\eta_2^2}{2} \right\} (R_0^\delta - 1) = w_2.$$ 

Thus from Eqs. (37)–(42) it could be noted that $i_2 \leq \lim \inf \langle i(t) \rangle \leq \lim \sup \langle i(t) \rangle \leq i_1$ and $w_2 \leq \lim \inf \langle w(t) \rangle \leq \lim \sup \langle w(t) \rangle \leq w_1$ whenever $t$ tend to $\infty$. \hfill $\square$

**Numerical simulation**

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

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

(43)

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

$$\tilde{X}_{\Delta t}, \tilde{X}_{2\Delta t}, \ldots, \tilde{X}_{N\Delta t}.$$ 

(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:
number also a strong influence and so increasing the vaccination would strongly decrease the value of the basic reproductive parameter. It could be also noted from the sensitivity index of the vaccination parameter that vaccination has therefore minimization of this parameter would significantly decrease the value of the threshold the disease transmission co-efficient has the highest sensitivity index and a great influence on the threshold of this parameter which implies that the disease will persist and all these compartments reach to their endemic stage whenever the value of this parameter is large scale for the class of susceptible individuals (s(t)) in case of extinction. The parameters value used are taken from S1 while (0.5, 0.3, 0.2, 0.1) are assumed to be the initial size of population.

Figure 6. The graph visualizes the temporal dynamics of the epidemic problem described by the model (1) on a large scale for the class of susceptible individuals (s(t)) in case of extinction. The parameters value used are taken from S1 while (0.5, 0.3, 0.2, 0.1) are assumed to be the initial size of population.

a. Simulate $\Delta B_k$ 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 given below

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

for $\Delta B_k = B_{k+1} - B_k$ and $k = 0, \ldots, N - 1$. The stochastic Euler Maruyama technique will be applied for the numerical simulation of the system reported by Eq. (1) which takes the form

$$s_{k+1} - s_k = \left[ \Pi - \beta_1 s_k i_k - \beta_2 s_k w_k - p_1 s_k \right] \Delta t + \eta s_k \Delta B_{1k},$$

$$i_{k+1} - i_k = \left[ \beta_1 s_k i_k + \beta_2 s_k i_k - p_2 i_k \right] \Delta t + \eta i_k \Delta B_{2k},$$

$$r_{k+1} - r_k = \left[ \sigma i_k + v s_k - \mu r_k \right] \Delta t + \eta r_k \Delta B_{3k},$$

$$w_{k+1} - w_k = \left[ \alpha i_k - \eta w_k \right] \Delta t + \eta w_k \Delta B_{4k},$$

which implies that

$$s_{k+1} = s_k + \left[ \Pi - \beta_1 s_k i_k - \beta_2 s_k w_k - p_1 s_k \right] \Delta t + \eta s_k \Delta B_{1k},$$

$$i_{k+1} = i_k + \left[ \beta_1 s_k i_k + \beta_2 s_k i_k - p_2 i_k \right] \Delta t + \eta i_k \Delta B_{2k},$$

$$r_{k+1} = r_k + \left[ \sigma i_k + v s_k - \mu r_k \right] \Delta t + \eta r_k \Delta B_{3k},$$

$$w_{k+1} = w_k + \left[ \alpha i_k - \eta w_k \right] \Delta t + \eta w_k \Delta B_{4k}.$$
i.e., there is a direct relation between the intensity of white noise and extinction while inverse relation between the intensity of white noise and persistence.

**Conclusion**

We developed a correlated stochastic epidemic model to discuss the temporal dynamics of the SARS-CoV-2 virus keeping in view the various source of randomness and vaccination of susceptible individuals. We proved the existence and positivity of the solutions which guarantees the well-posedness of the model. In addition, conditions of SARS-CoV-2 extinction analysis and persistence were obtained. A detailed sensitivity analysis has been performed and showed that the disease transmission coefficient and vaccination parameters are the highest sensitive parameters to disease transmission and control. This suggests that the vaccination has a major impact on the dynamics of the SARS-CoV-2. We observed that a rise in this parameter's value would significantly increase disease extinction. Conversely, the disease persistence reduction is subjected to speedy vaccination, and therefore there is a need for a fast vaccination immunization. Numerical findings were conducted and support the analytical results. Results of this study permit supplementary discussion, such as increasing the impact of the noise. We would encourage researchers to investigate adding jumps to our model.
Figure 9. The graph visualizes the time dynamics of the reported model (1) in case of extinction for the reservoir \( w(t) \) subject to the parametric values of \( S_1 \) and \( (0.5, 0.3, 0.2, 0.1) \) initial populations.

Figure 10. The graph visualizes the dynamics of the epidemic problem described by the model (1) in the case of persistence for the susceptible class \( s(t) \) against the values of the parameters taken from \( S_2 \) and \( (0.5, 0.3, 0.2, 0.1) \) are the initial sizes of population.
Figure 11. The graph visualizes the persistence of the epidemic problem framed by model (1) for the infected class \((i(t))\) against parameters value taken from \(S_2\) and various sizes of initial population \((0.5, 0.3, 0.2, 0.1)\).

Figure 12. The graph visualizes the time dynamics of the model (1) on large scale for recovered population \((r(t))\) against the parametric value of \(S_2\) and \((0.5, 0.3, 0.2, 0.1)\) initial population.
Data availability

All data generated or analyzed during this study are included in this published article.

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**Competing interests**
The authors declare no competing interests.

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