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Dynamics of a stochastic coronavirus (COVID-19) epidemic model with Markovian switching

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Abstract

In this paper, we analyze a stochastic coronavirus (COVID-19) epidemic model which is perturbed by both white noise and telegraph noise incorporating general incidence rate. Firstly, we investigate the existence and uniqueness of a global positive solution. Then, we establish the stochastic threshold for the extinction and the persistence of the disease. The data from Indian states, are used to confirm the results established along this paper.

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1. Introduction

Today, the world is facing the ongoing COVID-19 pandemic, caused by the SARS-CoV2 coronavirus. The novel coronavirus has been a serious threat to public health [4]. In late December 2019, the disease COVID-19 was first discovered in Wuhan (Hubei province) and caused the first pandemic of this century. The virus appears to be transferred mostly through narrow respiratory droplets by coughing, sneezing, or peoples interaction in close proximity (usually less than one meter) with each other for a certain time frame. However, it might be possible that other unobserved environmental exposures may have facilitated the rate the disease spreads through human-to-human transmission. In [9], it is reported that COVID-19 infected individuals generally develop symptoms, including mild respiratory symptoms and fever, on an average of 5–6 days after infection (mean 5–6 days, range 1–14 days). At the present, there is no effective treatment for COVID-19 in the world. Therefore the only way to stop the spread of this disease is to quarantine or isolate the initially infected population as showed by guide line of World Health Organization. By the end of June 2020, the COVID-19 virus has infected more than 10,927,025 people and died at least 521,512 in all over the world [23]. However, since the randomness of population mobility and uncertainty of control measures, methods of predicting COVID-19 and then preventing and controlling the disease for public health departments still remain unclear.

Recently, the novel coronavirus COVID-19 has attracted much attention from many researchers and various comprehensions have been made to deepen understanding and grasping the valuable inferences through mathematical modeling [10–12]. Therefore, it is of great significance to establish and study the model of infectious diseases. Mathematical modelling is an important decision tool that can be useful to analyze the spread and understand the level of manageability and the effect of prevention and control mechanisms applied to the pandemic. A numerous number of models are being used to project the current COVID-19 pandemic. Wang et al. [13] developed an SEIR model to estimate the epidemic trends in Wuhan, assuming the prevention and control measures were either sufficient or insufficient to control the epidemic. Hellewell et al. [14] developed a transmission model and found that highly effective contact tracing and case isolation are enough to control a new outbreak of COVID-19 within three months in most scenarios. In another recent work, Chakraborty and Ghosh [24] have considered a hybrid ARIMA-WBF model to forecast various COVID-19 affected countries throughout the globe. Several other models established a stochastic transition model to evaluate the transmission of COVID-19 and also emphasized the necessity of interventions such as social-distancing, isolation and quarantine [15,17].

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2. Model and preliminaries

2.1. Ordinary differential equation model

In [20], Mandal et al. consider a mathematical model of COVID-19 where human populations are subdivided into five time-dependant classes, namely, Susceptible $S(t)$, Exposed $E(t)$, Quarantined $Q(t)$, Hospitalized infected $I(t)$ and Recovered or Removed $R(t)$. They have assumed that the virus COVID-19 is spreading when a vulnerable person comes into contact with an exposed person. The model is a system of five first order ordinary differential equations shown as below:

\[
\begin{align*}
\dot{S}(t) &= A - \beta (1 - \rho_1)(1 - \rho_2)S(t)E(t) + b_1 Q(t) - \mu S(t), \\
\dot{E}(t) &= \beta (1 - \rho_1)(1 - \rho_2)S(t)E(t) - (b_2 + \alpha + v + \mu)E(t), \\
\dot{Q}(t) &= b_2 E(t) - (b_1 + c + \mu)Q(t), \\
\dot{I}(t) &= \alpha E(t) + \mu I(t) - (\eta + \mu + \delta)I(t), \\
\dot{R}(t) &= \eta I(t) + vE(t) - \mu R(t),
\end{align*}
\]

where all parameters are positive numbers. $A$ is the constant recruitment rate to the susceptible population; $\beta$ stands for the disease transmission rate; $\rho_1 (0 < \rho_1 < 1)$ is portion of susceptible human would maintain proper precaution measure and $\rho_2 (0 < \rho_2 < 1)$ represents portion of the exposed class would take proper precaution measure for disease transmission (i.e., use of face mask, social distancing and implementing hygiene). Therefore $(1 - \rho_1)S$ denotes portion of susceptible individuals due to the contact of $(1 - \rho_2)E$ portion of exposed individuals; $\alpha$ and $b_2$ are the portions of the exposed class going to the infected class and quarantine class, respectively. However, $b_1$ and $c$ represent the portions of the quarantine class moving to susceptible class and infected class, respectively. $\eta$ and $v$ stand for the recovery rate of hospitalized infected population $I$ and exposed class $E$; $\mu$ denotes the natural death rate and $\delta$ is the COVID-19 induced death rate.

According to the theory in Mandal et al. [20], the system (1) always has a disease-free equilibrium $E^0 = \left( \frac{A}{\mu}, 0, 0, 0, 0 \right)$.

If the basic reproduction number $R_0 = \frac{A\beta(1-\rho_1)(1 - \rho_2)}{\mu(b_2 + \alpha + v + \mu)} > 1$, there exists a unique endemic equilibrium $E^* = (S^*_d, E^*_d, Q^*_d, I^*_d, R^*_d)$, where

\[
\begin{align*}
S^*_d &= \frac{b_2 + \alpha + v + \mu}{\beta(1 - \rho_1)(1 - \rho_2)}, \\
E^*_d &= \frac{A\beta(1 - \rho_1)(1 - \rho_2) - \mu(b_2 + \alpha + v + \mu)}{\beta(1 - \rho_1)(1 - \rho_2)(b_2(c + \mu) + (\alpha + v + \mu)(b_1 + c + \mu))}, \\
Q^*_d &= \frac{b_2(1 - \rho_1)(1 - \rho_2) - \mu(b_2 + \alpha + v + \mu)(b_1 + c + \mu)}{\beta(1 - \rho_1)(1 - \rho_2)(b_2(c + \mu) + (\alpha + v + \mu)(b_1 + c + \mu))}, \\
I^*_d &= \frac{\eta I^*_d + vE^*_d}{\mu}, \\
R^*_d &= \frac{\eta I^*_d + vE^*_d}{\mu}.
\end{align*}
\]

Mandal et al. [20] established the following theoretical results about the stability of the equilibriums:

(i) If $R_0 < 1$, then the disease-free equilibrium $E^0$ of system (1) is locally asymptotically stable.

(ii) If $R_0 = 1$, the system (1) passes through a transcritical bifurcation around its disease-free equilibrium.

(iii) If $R_0 > 1$, then the endemic equilibrium $E^*$ of system (1) is locally asymptotically stable.

2.2. Stochastic differential equation model

In fact, the COVID-19 epidemic model is unavoidably subjected to the environmental noise, which made the parameters involved in the system often fluctuate randomly around some average values as the surrounding environment fluctuation. See [1–3,5–7,16,25] and references therein for epidemic models with environmental noise. Therefore, it is necessary to include random fluctuations in the process of COVID-19 modelling. In this paper, we propose a stochastic COVID-19 model adopting a generalized incidence function [21,22] as follows:

\[
\begin{align*}
\dot{S}(t) &= \frac{A - \beta (1 - \rho_1)(1 - \rho_2)S(t)f(E(t)) + b_1 Q(t) - \mu S(t)}{\mu S(t + \sigma^2)dt} - \sigma S(t) f(E(t))dB(t), \\
\dot{E}(t) &= \frac{\beta (1 - \rho_1)(1 - \rho_2)S(t)f(E(t)) - (b_2 + \alpha + v + \mu)E(t)}{dt} + \sigma S(t) f(E(t))dB(t), \\
\dot{Q}(t) &= \frac{b_2 E(t) - (b_1 + c + \mu)Q(t)}{dt}, \\
\dot{I}(t) &= \frac{\alpha E(t) + \mu I(t) - (\eta + \mu + \delta)I(t)}{dt}, \\
\dot{R}(t) &= \frac{\eta I(t) + vE(t) - \mu R(t)}{dt},
\end{align*}
\]

where $B(t)$ is a real-valued Brownian motion and $\sigma^2 > 0$ represents the intensity of the white noise $B(t)$. The function $f(\cdot)$ is generally assumed to be a non-negative twice continuously differentiable function such that $f(0) = 0$, $f'(0) > 0$ and the function $x \mapsto \frac{f(x)}{x}$ is monotonically decreasing on $[0, \infty)$ (this implies that $f(x) < f'(0)$ for any $x > 0$).

Note that the COVID-19 epidemic models may be perturbed by telegraph noise which can causes the system to switch from one environmental regime to another [18]. Mostly the switching between environmental regimes is often memoryless and the waiting time for the next switching follows the exponential distribution [19]. Hence the regime switching can be modelled by a continuous time Markov
where
\begin{align}
B_k(t) = \begin{cases} \beta(t)(1 - \rho(t) - (1 - \rho(t))S(t)f(E(t)) + b_{t}(t)Q - \mu(t)S(t)dt - \sigma(t)S(t)f(E(t))dB(t) \\
\sigma(t)S(t)f(E(t))dB(t) \\
\delta(t)\frac{d(r(t))}{\Delta(t)} - \eta(t)dt + \mu(t)S(t)dt + \mu(t)S(t)f(E(t))dB(t) \\
d(r(t)) = \begin{cases} \eta(t)dt + \mu(t)S(t)dt \\
\mu(t)S(t)dt \\
\end{cases}
\end{cases}
\end{align}

where Markov chain \( r(t) \) is \( \mathcal{F}_t \)-adapted but independent of Brownian motion \( B_t \) and defined on the same complete probability space \((\Omega, \mathcal{F}, \mathbb{P})\). The infinitesimal generator \( \Gamma = (\gamma_{ij})_{N \times N} \) of Markovian chain is defined by

\[ \gamma_{ij} = - \sum_{j \neq i} \gamma_{ij}. \]

In this paper, we assume that \( \gamma_{ij} > 0 \) for \( i, j = 1, \ldots, N \) with \( i \neq j \). This assumption assures that the Markov chain \( r(t) \) is irreducible, which implies that it has a unique stationary distribution \( \pi = (\pi_1, \pi_2, \ldots, \pi_N) \), which can be determined by solving the following equation

\[ \pi \Gamma = 0, \]

subject to

\[ \sum_{k=1}^{N} \pi_k = 1, \quad \pi_k > 0, \quad \text{for any} \ k \in \mathbb{S}. \]

For convenience, we define for any fixed vector \( [\psi(k)]_{k \in \mathbb{S}} \), \( \hat{\psi} = \min_{k \in \mathbb{S}} \psi(k) \) and \( \tilde{\psi} = \max_{k \in \mathbb{S}} \psi(k) \). To begin the analysis of the model, we define the subsets

\[ \mathbb{R}_5^5 = \left\{ (x_1, x_2, x_3, x_4, x_5) \in \mathbb{R}^5 : x_i > 0, i = 1, 2, \ldots, 5 \right\}, \]

\[ \tilde{\mathcal{Y}} = \left\{ (x_1, x_2, x_3, x_4, x_5) \in \mathbb{R}_5^5 : \frac{\tilde{A}}{\mu + \delta} \leq \sum_{i=1}^{5} x_i \leq \frac{\tilde{A}}{\mu} \right\}. \]

Throughout this paper, we carry out the case of small noises: \( \sigma(k) \frac{\tilde{A}}{\mu} f'(0) \leq \beta(k)(1 - \rho_1(k))(1 - \rho_2(k)) \) for any \( k \in \mathbb{S} \).

The structure of the rest of the paper is as follows: In Section 3, we show the existence and uniqueness of a global positive solution to the system (3). In the Sections 4 and 5, we study the existence of a stochastic threshold for the extinction and the persistence in mean of the disease. In last section, we present some numerical simulations to demonstrate our main theoretical results.

3. Existence and uniqueness of a positive solution

To study the dynamical behaviour of an epidemic model, we firstly need to consider whether the solution is global and positive. In this section, we will prove there is a unique global positive solution of system (3).

**Theorem 3.1.** For any given initial value \( (S(0), E(0), Q(0), I(0), R(0)) \in \tilde{\mathcal{Y}} \), there exists a unique solution \( (S(t), E(t), Q(t), I(t), R(t)) \in \tilde{\mathcal{Y}} \) of system (3) on \( t \geq 0 \) and the solution will remain in \( \tilde{\mathcal{Y}} \) with probability 1.

**Proof.** Obviously, the coefficient of model (3) are locally Lipschitz continuous, so there is a unique local solution \( (S(t), E(t), Q(t), I(t), R(t)) \in \mathbb{R}_5^5 \) on \([0, \tau_\epsilon]) \) for any initial value \( (S(0), E(0), Q(0), I(0), R(0)) \in \tilde{\mathcal{Y}} \), where \( \tau_\epsilon \) is the explosion time [27]. If \( \tau_\epsilon = \infty \) a.s., then this local solution is global. To this end, let \( n_0 > 0 \) be sufficiently large for every component of \( (S(0), E(0), Q(0), I(0), R(0)) \) lying within the interval \( [0, \frac{1}{n_0}] \). For each integer \( n \geq n_0 \), define the stopping time

\[ \tau_n = \inf \left\{ t \in [0, \tau_\epsilon) : \min[S(t), E(t), Q(t), I(t), R(t)] \leq 1 \right\}, \]

where throughout this paper we set \( \inf \emptyset = \infty \) (\( \emptyset \) denotes the empty set). Clearly, \( \tau_n \) is increasing as \( n \to \infty \). Set \( \tau_\infty = \lim_{n \to \infty} \tau_n \), which implies \( \tau_\infty < \tau_\epsilon \) a.s. If \( \tau_\infty = \infty \) a.s., then \( \tau_\infty = \infty \) a.s. This means that \( (S(t), E(t), Q(t), I(t), R(t)) \in \mathbb{R}_5^5 \) a.s., for all \( t \geq 0 \). If \( \tau_\epsilon < \infty \) a.s., then there is a pair of constant \( T > 0 \) and \( \epsilon \in (0, 1) \) such that \( \mathbb{P}[\tau_\epsilon \leq T] > \epsilon \). Hence there is an integer \( n_1 \geq n_0 \) such that \( \mathbb{P}[\tau_n \leq T] > \epsilon, \quad \forall n \geq n_1 \).

Denote \( \tilde{N}(t) = S(t) + E(t) + Q(t) + I(t) + R(t) \). For any \( n \geq n_1 \) and \( t \in [0, \tau_n] \), we have

\[ (\mu(t) + \delta(t))(\frac{\tilde{A}}{\mu + \delta} - \tilde{N}(t))dt \leq [A(t) - (\mu(t) + \delta(t))\tilde{N}(t)]dt \]

\[ = d\tilde{N}(t) \leq [A(t) - \mu(t)]\tilde{N}(t)dt \]

\[ \leq \mu(t)(\frac{\tilde{A}}{\mu} - \tilde{N}(t))dt. \]
It then follows that
\[
\frac{d}{dt} \left( \frac{\hat{A}}{\hat{\mu}} - \hat{N}(t) \right) + \mu_{r(t)} \left( \frac{\hat{A}}{\hat{\mu}} - \hat{N}(t) \right) \geq 0,
\]
and
\[
\frac{d}{dt} \left( \frac{\hat{A}}{\hat{\mu} + \delta} - \hat{N}(t) \right) + (\mu_{r(t)} + \delta_{r(t)}) \left( \frac{\hat{A}}{\hat{\mu} + \delta} - \hat{N}(t) \right) \leq 0.
\]
Therefore
\[
\frac{\hat{A}}{\hat{\mu}} - \hat{N}(t) \geq \left( \frac{\hat{A}}{\hat{\mu}} - \hat{N}(0) \right) \exp \left\{ - \int_0^t \mu_{r(s)} \, ds \right\},
\]
and
\[
\frac{\hat{A}}{\hat{\mu} + \delta} - \hat{N}(t) \geq \left( \frac{\hat{A}}{\hat{\mu} + \delta} - \hat{N}(0) \right) \exp \left\{ - \int_0^t (\mu_{r(s)} + \delta_{r(s)}) \, ds \right\}.
\]
Since \( \frac{\hat{A}}{\hat{\mu} + \delta} \leq \hat{N}(0) \leq \frac{\hat{A}}{\hat{\mu}} \), it follows that
\[
\frac{\hat{A}}{\hat{\mu} + \delta} \leq \hat{N}(t) \leq \frac{\hat{A}}{\hat{\mu}}, \quad \forall t \in [0, \tau_n).
\]
Now, define a \( C^2 \)-function \( V : \hat{T} \rightarrow \mathbb{R}_+ \) as follows
\[
V(S, E, Q, I, R) = (S - 1 - \log S) + (E - 1 - \log E) + (Q - 1 - \log Q) + (I - 1 - \log I) + (R - 1 - \log R).
\]
The non-negativity of this function can be obtained from
\[
u - 1 - \log u \geq 0 \quad \text{for any } u > 0.
\]
Let \( n \geq n_1 \) and \( T > 0 \) be arbitrary. For any \( 0 \leq t \leq \min\{\tau_n, T\} \), applying the generalized Itô’s formula [27] to \( V \) yields
\[
dV(S, E, Q, I, R) = \mathcal{L}V(S, E, Q, I, R) \, dt + \left( \frac{1}{S} - \frac{1}{E} \right)\sigma_{r(t)} S f(E) \, dB(t),
\]
where \( \mathcal{L} : \mathbb{R}_+^5 \rightarrow \mathbb{R} \) is defined by
\[
\mathcal{L}V = \left( 1 - \frac{1}{S} \right) \left( A(k) - \beta(k)(1 - \rho_1(k))(1 - \rho_2(k))S(t) f(E) + b_1(k)Q - \mu(k)S \right)
\]
\[
+ \left( 1 - \frac{1}{E} \right) \left( \beta(k)(1 - \rho_1(k))(1 - \rho_2(k))S(t) f(E) - (b_2(k) + \alpha(k) + \nu(k) + \mu(k))E \right)
\]
\[
+ \left( 1 - \frac{1}{Q} \right) \left( b_2(k)E - (b_1(k) + c(k) + \mu(k))Q \right) + \left( 1 - \frac{1}{I} \right) \left( \alpha(k)E + c(k)Q - (\eta(k) + \mu(k) + \delta(k))I \right)
\]
\[
+ \left( 1 - \frac{1}{R} \right) \left( \eta(k)I - \nu(k)E - \mu(k)R \right) + \frac{1}{2} \sigma^2(k) f^2(E) \right) + \frac{1}{2} \sigma^2(k) f^2(Q) + \frac{1}{2} \sigma^2(k) f^2(R)
\]
\[
\leq A(k) + b_1(k)Q + \beta(k)(1 - \rho_1(k))(1 - \rho_2(k))f(E) + 5\mu(k) + b_1(k) + b_2(k) + \alpha(k)
\]
\[
+ \nu(k) + c(k) + \eta(k) + \delta(k) + (b_2(k) + \alpha(k) + \nu(k))E + (c(k)Q + \eta(k)I) + \frac{1}{2} \sigma^2(k) f^2(E) + \frac{1}{2} \sigma^2(k) f^2(Q) + \frac{1}{2} \sigma^2(k) f^2(R)
\]
\[
\leq \hat{A} + \hat{b}_1 \hat{A} \hat{\mu} + \hat{\beta}(1 - \hat{\rho}_1)(1 - \hat{\rho}_2) \hat{A} \hat{f}(0) \right) + 5\hat{\mu} + \hat{b}_1 + \hat{b}_2 + \hat{\alpha} + \hat{\nu} + \hat{\epsilon} + \hat{\eta} + \hat{\delta} + (\hat{b}_2 + \hat{\alpha} + \hat{\nu} + \hat{\epsilon} + \hat{\eta}) \frac{\hat{\mu}}{\hat{\mu}} + \left( \hat{\sigma} \left( \hat{f}(0) \right) \right)^2 := K.
\]
where the inequality \( \frac{\hat{f}(E)}{\hat{f}(E)} \leq f'(0) \) is used and \( K \) is a positive constant which is independent of \( S, E, Q, I, R, k \). The remaining part of the proof is similar to [26] and hence is omitted here. This completes the proof. \( \square \)

4. Extinction of the disease

It is most crucial to deal with the conditions for the extinction of diseases when their dynamics is under investigation. This section is devoted to establish sufficient conditions so that the COVID-19 goes out of the population. First, we need to define the following number
\[
\mathcal{R}^0 = \sum_{k=1}^N \left( \hat{b}_2(k) + \hat{\alpha}(k) + \hat{\nu}(k) + \hat{\mu}(k) + \frac{1}{2} \left( \hat{\sigma} \left( \hat{f}(0) \right) \right)^2 \right).
\]
At this stage, we have the following theorem:

**Theorem 4.1.** Let \( S(t), E(t), I(t), Q(t), R(t) \) be the solution of system (3) with any initial value \( (S(0), E(0), I(0), Q(0), R(0)) \in \mathcal{T} \). If the following condition \( \mathcal{R}^0 < 1 \) is satisfied, then the pandemic goes out of the population with probability one. That is to say
\[
\lim_{t \to \infty} E(t) = \lim_{t \to \infty} Q(t) = \lim_{t \to \infty} I(t) = \lim_{t \to \infty} R(t) = 0 \text{ a.s.}
\]
**Proof.** Applying the generalized Itô’s formula to \(\log E\), we have

\[
d\log E = \left[ \beta_t(1 - \rho_t(1) - (1 - \rho_t(1))) S_t \frac{f(E)}{E} - (b_{2t}(1) + \alpha_t + \nu_t + \mu_t) - \frac{1}{2} \left( \sigma_t S_t \frac{f(E)}{E} \right)^2 \right] dt + \sigma_t S_t \frac{f(E)}{E} dB(t).
\]

Moreover, for any \(k \in \mathbb{R}\), the function defined by \(x \mapsto -\sigma_t^2(k)x^2 + \beta(k)(1 - \rho_t(k)) - (b_{2t}(1) + \alpha_t + \nu_t + \mu_t) + \frac{1}{2} \left( \sigma_t S_t \frac{f(E)}{E} \right)^2\) is increasing on \([0, \beta(k)(1 - \rho_t(k))(1 - \rho_t(1))/\sigma_t^2(k)]\). Using the boundedness of the solution and the fact that \(f(E) \leq f(0)\), we obtain

\[
d\log E \leq \left[ \beta_t(1 - \rho_t(1) - (1 - \rho_t(1))) S_t \frac{f(E)}{E} - (b_{2t}(1) + \alpha_t + \nu_t + \mu_t) - \frac{1}{2} \left( \sigma_t S_t \frac{f(E)}{E} \right)^2 \right] dt + \sigma_t S_t \frac{f(E)}{E} dB(t)
\]

Integrating (5) from 0 to \(t\) and then dividing by \(t\) on both sides lead to

\[
\frac{\log E(t) - \log E(0)}{t} \leq \frac{1}{t} \int_0^t \tilde{R}_t \, ds + \frac{1}{t} \int_0^t \sigma_s S(s) \frac{f(E(s))}{E(s)} dB(s).
\]

It follows from the ergodic property of \(rt\) that

\[
\lim_{t \to \infty} \frac{1}{t} \int_0^t \tilde{R}_t \, ds = \sum_{k=1}^N \pi_k \tilde{R}_k^\infty.
\]

(7)

Taking the superior limit on the both sides of (6) and (7) and making use of the large number theorem for local martingales, we get

\[
\limsup_{t \to \infty} \frac{\log E(t)}{t} \leq \sum_{k=1}^N \pi_k \tilde{R}_k^\infty
\]

\[
= \sum_{k=1}^N \pi_k \left[ b_2(k) + \alpha(k) + \nu(k) + \mu(k) + \frac{1}{2} \left( \sigma_t \frac{\tilde{A}_t}{\tilde{\mu}} f'(0) \right)^2 \right] (\tilde{R}_k^\infty - 1) < 0 \quad \text{a.s.},
\]

which implies that

\[
\lim_{t \to \infty} E(t) = 0 \quad \text{a.s.}
\]

(8)

On the other hand, let \(\Lambda = \{\omega \in \Omega : \lim_{t \to \infty} E(t) = 0\}\). Then (8) implies \(\mathbb{P}(\Lambda) = 1\). Hence, for any \(\omega \in \Lambda\) and \(\epsilon > 0\), there exists a constant \(T(\omega, \epsilon) > 0\) such that

\[
E(\omega, t) \leq \epsilon, \quad \text{for all } t > T.
\]

Substituting this into the third equation of system (3), we obtain for all \(\omega \in \Lambda, t > T\),

\[
dQ(\omega, t) \leq \left[ b_2 - (\tilde{b}_1 + \tilde{c} + \tilde{\mu}) \right] Q(\omega, t) dt
\]

\[
\leq \left[ b_2 \epsilon - (\tilde{b}_1 + \tilde{c} + \tilde{\mu}) \right] Q(\omega, t) dt.
\]

Thus by the comparison theorem we get

\[
\limsup_{t \to \infty} Q(\omega, t) \leq \frac{b_2 \epsilon}{\tilde{b}_1 + \tilde{c} + \tilde{\mu}} \quad \text{a.s.,}
\]

for all \(\omega \in \Lambda\). Note that \(Q(\omega, t) > 0\) for all \(\omega \in \Omega\) and \(t > 0\), in view of the arbitrariness of \(\epsilon > 0\), we get

\[
\lim_{t \to \infty} Q(\omega, t) = 0, \quad \omega \in \Lambda.
\]

Recalling that \(\mathbb{P}(\Lambda) = 1\), hence we obtain

\[
\lim_{t \to \infty} Q(\omega, t) = 0 \quad \text{a.s.}
\]

Similarly, when \(\lim_{t \to \infty} E(t) = 0 \text{ a.s.}, and } \lim_{t \to \infty} Q(t) = 0 \text{ a.s.}, then by using the same approach as above, we can conclude that

\[
\lim I(t) = 0 \text{ a.s., and } \lim R(t) = 0 \text{ a.s.}
\]

This completes the proof. \(\square\)

**Remark 1.** Obviously, the quantity \(R_0^0\) is smaller than \(R_0\). Hence, the extinction of the disease in system (3) could be ensured even if the condition \(R_0 < 1\) is not verified.

### 5. Persistence in mean of the disease

To investigate epidemic dynamical system, we are also interested in when the disease persists in host population. In this section we need to assume that the function \(\frac{dQ}{dt}\) is \(C\)-Lipschitz and we have the following result on the prevailing behaviour of the COVID-19 disease.

**Theorem 5.1.** If \(R_0^0 > 1\), then for any given initial value \((S(0), E(0), I(0), Q(0), R(0)) \in \tilde{T}\), the solution of (3) verifies

\[
\liminf_{t \to \infty} \frac{1}{t} \int_0^t E(s) ds \geq M \left( R_0^0 - 1 \right) \quad \text{a.s.},
\]

where

\[
M \geq \frac{1}{\tilde{b}_1 + \tilde{c} + \tilde{\mu}}\]
\[
\begin{align*}
\liminf_{t \to \infty} \frac{1}{t} \int_0^t Q(s) ds & \geq M_2 (R_2^0 - 1) \quad \text{a.s.,} \\
\liminf_{t \to \infty} \frac{1}{t} \int_0^t I(s) ds & \geq M_3 (R_3^0 - 1) \quad \text{a.s.,} \\
\liminf_{t \to \infty} \frac{1}{t} \int_0^t R(s) ds & \geq M_4 (R_4^0 - 1) \quad \text{a.s.,}
\end{align*}
\]
for some positive constants \(M_i, i = 1, \ldots, 4\).

**Proof.** Applying the Itô's formula on the function \(E \to \log E\), we get
\[
d \log E(t) = F\left( S(t) \frac{f(E(t))}{E(t)} \cdot r(t) \right) dt + \sigma S(t) \frac{f(E(t))}{E(t)} dB(t),
\]
where
\[
F(X, r) = \beta_1 (1 - \rho_{1, r})(1 - \rho_{2, r}) X - (b_{2, r} + \alpha_1 + \nu_r + \mu_r) - \frac{\sigma^2}{2} X^2.
\]
Using the fact that \(\frac{\Lambda}{\mu} \geq \frac{\nu}{r} \leq \frac{f'}{E}\), one can easily show that
\[
\begin{align*}
F\left( \frac{f(E)}{E}, r \right) & \geq F\left( \frac{\Lambda}{\mu} f'(0), r \right) - \left( \beta_1 (1 - \rho_{1, r})(1 - \rho_{2, r}) - \frac{\sigma^2}{2} \frac{\Lambda}{\mu} f'(0) \right) \left( \frac{\Lambda}{\mu} f'(0) - S \frac{f(E)}{E} \right) \\
& \geq F\left( \frac{\Lambda}{\mu} f'(0), r \right) - \left( \hat{\beta}_1 (1 - \hat{\rho}_1)(1 - \hat{\rho}_2) - \frac{\sigma^2}{2} \frac{\Lambda}{\mu} f'(0) \right) \left( \frac{\Lambda}{\mu} f'(0) - S \frac{f(E)}{E} \right).
\end{align*}
\]
From the first equation of (3), we can easily claim that
\[
dS \geq \left[ \frac{\hat{\Lambda}}{\hat{\mu} f'(0)} \right] \left[ \frac{\hat{\Lambda}}{\mu} f'(0) - S \frac{f(E)}{E} \right] - \hat{\mu} S \left( 1 - \frac{\hat{\Lambda}}{\hat{\mu} f'(0)} f(E) \right) - \hat{\beta} (1 - \hat{\rho}_1)(1 - \hat{\rho}_2) \frac{\hat{\Lambda}}{\hat{\mu} f'(0)} dt - \sigma S f(E) dB(t).
\]
Now, let \(u \leq v\). Without loss of generality, assume that \(u < v\). The monotonicity and lipschitzianity assumptions of \(f' \cdot f^{-1}\) implies that \(f(u)/u - f(v)/v \leq C(v - u)\). Extending \(u\) to zero leads to \(f'(0) - \frac{f(0)}{E} \leq CE\). Hence,
\[
S \left( 1 - \frac{f(E)}{f'(0) E} \right) \leq \frac{C}{\mu} f'(0).
\]
Consequently,
\[
dS \geq \left[ \frac{\hat{\Lambda}}{\hat{\mu} f'(0)} \right] \left[ \frac{\hat{\Lambda}}{\mu} f'(0) - S \frac{f(E)}{E} \right] - \hat{\mu} S \left( 1 - \frac{\hat{\Lambda}}{\hat{\mu} f'(0)} f(E) \right) - \hat{\beta} (1 - \hat{\rho}_1)(1 - \hat{\rho}_2) \frac{\hat{\Lambda}}{\hat{\mu} f'(0)} dt - \sigma S f(E) dB(t).
\]
which leads to
\[
- \left( \frac{\Lambda}{\mu} f'(0) - S \frac{f(E)}{E} \right) dt \geq - \frac{\Lambda f'(0)}{\hat{\mu}} \left[ dS + \frac{\hat{\mu}}{\hat{\Lambda}} \left( \frac{\mu C}{f'(0)} + \hat{\beta} (1 - \hat{\rho}_1)(1 - \hat{\rho}_2) f'(0) \right) Edt + \sigma S f(E) dB(t) \right].
\]
Substituting this inequality into (11) and making use of (9), we obtain
\[
d \log E \geq F\left( \frac{\Lambda}{\mu} f'(0), r \right) - m_1 E dt + \hat{A}(t),
\]
where
\[
m_1 = \frac{f(0)}{\Lambda} \left( \frac{\Lambda}{\mu} \right)^2 \left( \hat{\beta} (1 - \hat{\rho}_1)(1 - \hat{\rho}_2) - \frac{\sigma^2}{2} \frac{\Lambda}{\mu} \right) \left( \frac{\mu C}{f'(0)} + \hat{\beta} (1 - \hat{\rho}_1)(1 - \hat{\rho}_2) f'(0) \right),
\]
and
\[
\hat{A}(t) = \left( \frac{\Lambda f'(0)}{\hat{\mu}} \left( \frac{\Lambda}{\mu} f'(0) - S \frac{f(E)}{E} \right) \right) [dS + \sigma S f(E) dB(t)] + \sigma S f(E) dB(t).
\]
Then, applying the generalized Itô's formula on the function \(V(E, k) = \log E + \tilde{\omega}(k)\) and using (12), we have
\[
dV \geq F\left( \frac{\Lambda}{\mu} f'(0), k \right) + \sum_{k=1}^N \gamma_2 \tilde{\omega}(l) - m_1 E dt + \hat{A}(t).
\]
Since the generator \(\Gamma\) is irreducible, then for \(P_0 = (P(1), \ldots, P(N))\) with \(P(k) = F\left( \frac{\Lambda}{\mu} f'(0), k \right)\), there is a solution \(\tilde{\omega} = (\tilde{\omega}(1), \ldots, \tilde{\omega}(N))\) to the system \(\Gamma \omega = -P_0 + \sum_{k=1}^N \pi_k P(k) 1\), where \(1\) is the unit vector of \(\mathbb{R}^N\). That is,
\[
F\left( \frac{\Lambda}{\mu} f'(0), k \right) + \sum_{k=1}^N \gamma_2 \tilde{\omega}(l) = \sum_{k=1}^N \pi_k F\left( \frac{\Lambda}{\mu} f'(0), k \right).
\]
Substituting the above equality into the inequality (13), integrating from 0 to \(t\) and dividing by \(t\), we obtain
\[
\frac{V(t) - V(0)}{t} \geq \sum_{k=1}^{N} \pi_k F(k) - m_1 \frac{1}{t} \int_0^t \bar{E}(s) ds + \frac{1}{t} \int_0^t \bar{A}(s).
\]

The large number theorem for local martingales and the boundedness of \(S\) implies that \(\lim_{t \to \infty} \frac{1}{t} \int_0^t \bar{A}(s) = 0\) a.s. In addition, making use of \(\lim_{t \to \infty} \frac{V(t) - V(0)}{t} \leq 0\), and the fact that
\[
\sum_{k=1}^{N} \pi_k F(k) = \sum_{k=1}^{N} \pi_k \left[ b_2(k) + \alpha(k) + v(k) + \mu(k) + \frac{\sigma^2(k)}{2} \left( \frac{\bar{A}}{\mu} f'(0) \right)^2 \right] \leq m_1 M_1 (R_+^0 - 1)
\]
leads to
\[
\liminf_{t \to \infty} \frac{1}{t} \int_0^t \bar{E}(s) ds \geq M_1 (R_+^0 - 1) \text{ a.s.} \quad (14)
\]

From the third equation of (3), we can establish that
\[
\frac{1}{t} \int_0^t \bar{Q}(s) ds \geq \frac{1}{b_1 + \bar{c} + \bar{\mu}} \left( \frac{b_2}{t} \int_0^t \bar{E}(s) ds \right) - \frac{Q(t) - Q(0)}{t}.
\]

Taking the inferior limit and making use of both the boundedness of \(Q\) and the inequality (14), we get
\[
\liminf_{t \to \infty} \frac{1}{t} \int_0^t \bar{Q}(s) ds \geq M_2 (R_+^0 - 1) \text{ a.s.},
\]
where \(M_2 = \bar{b} M_1 / (\bar{b} + \bar{c} + \bar{\mu})\). Following the same way, we can easily claim that

\[
\liminf_{t \to \infty} \frac{1}{t} \int_0^t \bar{I}(s) ds \geq M_3 (R_+^0 - 1) \text{ a.s.},
\]

\[
\liminf_{t \to \infty} \frac{1}{t} \int_0^t \bar{R}(s) ds \geq M_4 (R_+^0 - 1) \text{ a.s.},
\]

where \(M_3 = (\bar{a} M_1 + \bar{c} M_2) / (\bar{\eta} + \bar{\mu} + \bar{\delta})\) and \(M_4 = (\bar{\nu} M_1 + \bar{\eta} M_3) / \bar{\mu}\). This makes finish of the proof. \(\Box\)

6. Numerical simulations

Based on the Milstein method of [8], the main theoretical results regarding system (3) are illustrated numerically using real COVID-19 data from some Indian states. Namely, Maharashtra, Delhi and Tamil Nadu states. These different parts differ from each other in terms of their environmental conditions and regimes. In this section, we assume that the Markov chain \(\tau\) switches among these states \(S = \{1, 2, 3\}\) with the generator
\[
\Gamma = \begin{bmatrix}
-9 & 4 & 5 \\
2 & -5 & 3 \\
2 & 2 & -4
\end{bmatrix},
\]
and the corresponding stationary distribution \(\pi = \left(\frac{14}{77}, \frac{26}{77}, \frac{37}{77}\right)\). The general Holling type II incidence is considered in the infection force modelling. Namely, \(f(E) = \frac{E}{1 + 0.001 E}\) satisfies the required assumptions along this manuscript. The path of the Markov chain \(\tau\) and the corresponding stationary distribution are plotted in Fig. 1. In this example, we let the state chain takes more time on the third environment.

![Graph](b.png)  
**Fig. 1.** The sample path of the Markov chain \(\tau\) (a) and the corresponding stationary probability density function (b).
Table 1
Parameters values used in numerical simulations.

| Parameter | Maharashtra | Delhi | Tamil Nadu |
|-----------|-------------|-------|------------|
| $\mu$     | 0.0058      | 0.0036| 0.0071     |
| $\alpha$  | 1/5.2       | 1/4   | 1/5.4      |
| $\delta$  | 0.0685      | 0.0232| 0.0122     |
| $\eta$    | 1/14        | 1/14  | 1/14       |
| $\rho_1$  | 0.64        | 0.72  | 0.62       |
| $\rho_2$  | 0.78        | 0.82  | 0.75       |
| $b_1$     | 0.07122     | 0.045185| 0.062856   |
| $b_2$     | 1.11013     | 0.78529| 0.157832   |
| $\nu$     | 0.119732    | 0.14029| 0.36186    |

Fig. 2. Simulation results of the means (blue lines) and the associated standard deviations (grey area) over $5 \times 10^3$ samples of $S(t)$, $E(t)$, $Q(t)$, $I(t)$ and $R(t)$ respectively for the system [3]. Red lines, green and black ones stand for deterministic paths of the ODE model (1) with only one régime $r(t) = 1$, $r(t) = 2$ and $r(t) = 3$ respectively. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

The second environment conditions are assumed to be more likely than the first one to show the applicability of the analytical results established along this paper. The parameter values chosen are given in Mandal et al. [20] and reported in the Table 1.
To demonstrate the effect of telegraph noise on the dynamics of COVID-19 disease, in addition to data of Table 1, we set the following settings:

\[
A(1) = 0.012, \quad A(2) = 0.0078, \quad A(3) = 0.015, \\
\beta(1) = 3.5, \quad \beta(2) = 3, \quad \beta(3) = 2.5, \\
\sigma(1) = 0.25, \quad \sigma(2) = 0.19, \quad \sigma(3) = 0.23.
\]

Then, by direct computation, we obtain \( R_0^N = 0.6303 < 1 \). In other words, the conditions of the Theorem 4.1 hold and the Fig. 2 shows the empirical means and the standard deviations of the solution to (3) in a \( 5 \times 10^4 \) samples, as well as the trajectories of the deterministic system (1) without switching for different values of the three considered regimes. So the stochastic process (3) for COVID-19 disease switches over the states 1, 2 and 3 before going to extinction.

On the other hand, when the following parameter values are considered,

\[
A(1) = 0.048, \quad A(2) = 0.051, \quad A(3) = 0.042, \\
\beta(1) = 3.4, \quad \beta(2) = 2.9, \quad \beta(3) = 2.5, \\
\sigma(1) = 0.6, \quad \sigma(2) = 0.55, \quad \sigma(3) = 0.3,
\]

then we get

\[
\sum_{k=1}^{N} \pi_k \beta(k) (1 - \rho_1(k))(1 - \rho_2(k)) \frac{\Delta}{T} f'(0) = 8.5177 \\
\sum_{k=1}^{N} \pi_k \left[ b_2(k) + \alpha(k) + v(k) + \mu(k) + \frac{1}{2} \left( \sigma(k) \Delta f'(0) \right)^2 \right] = 3.2728
\]

which gives that the stochastic threshold \( R_0^N = 2.6026 \) and the condition of the Theorem 5.1 is fulfilled. The persistence of the disease is clarified in the Fig. 3, where the average of the solution to (3) and the associated standard deviation are plotted besides the trajectories of the solution to the deterministic system (1). The processes \( (S(t), E(t), I(t), Q(t), R(t)) \) move up and down stochastically in a neighborhood of the solutions to the model.
corresponding to each state separately and with no random perturbation.

7. Conclusion

This paper investigates a stochastic epidemic model describing COVID-19 dynamics affected by mixture of environmental perturbations modeled by white and telegraph noises. By means of Lyapunov approach, the existence and positivity of a global solution is well proved. In terms of a stochastic threshold $R_0^S$, the extinction and the persistence in mean of the COVID-19 epidemic are investigated. Particularly, under small noises, the condition $R_0^S < 1$ is sufficient to reduce the daily number of confirmed infectives and make the coronavirus disease 2019 extinct. Reciprocally, the persistence of this novel epidemic is inevitable once $R_0^S$ stays away from unity. Based on the data from different states of India, we performed numerical simulations in order to support and illustrate the main results of this paper.

Although many important contributions are made in literature to draw the dynamical properties of the COVID-19, some of them still unidentified and much more efforts are recommended to make it more comprehensible and help humanity to overcome the current pandemic. As a further suggestion, other improvements such as time varying parameters can be considered to make the studied COVID-19 model more realistic. A task which we leave for next works.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

CRediT authorship contribution statement

Brahim Boukanjime: Conceptualization, Writing - original draft, Writing - review & editing. Tomás Caraballo: Validation, Investigation, Conceptualization, Writing - original draft, Writing - review & editing. Mohamed El Fatini: Methodology, Validation, Formal analysis, Writing - original draft, Writing - review & editing. Mohamed El Khalifi: Software, Formal analysis, Writing - original draft, Writing - review & editing.

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References

[1] Boukanjime B, El Fatini M, A stochastic Hepatitis B epidemic model driven by Lévy noise. Phys A 2019;521:796–806.
[2] Boukanjime B, El Fatini M, Laaribi A, Taki R. Analysis of a deterministic and a stochastic epidemic model with two distinct epidemics hypothesis. Phys A 2019;534:122312.
[3] El Fatini M, Pettersson R, Sakkak I, et al. A stochastic analysis for a triple delayed SIRQ epidemic model with vaccination and elimination strategies. J Appl Math Comput 2020. doi:10.1007/s12190-020-01380-1.
[4] Guan WJ, Ni ZY, Hu Y, Liang WH, Ou CQ, He JX, et al. China medical treatment expert group for, clinical characteristics of coronavirus disease 2019 in China. N Engl J Med 2020. doi:10.1056/NEJMoa2002032.
[5] Caraballoa T, El Fatini M, El Khalifi M, Gerlach R, Pettersson R. Analysis of a stochastic distributed delay epidemic model with relapse and gamma distribution kernel. Chaos Solitons Fractals 2020;133. Article ID 109643.
[6] El Fatini M, Sakkak I, Laaribi A, Pettersson R, Wang K. A stochastic threshold of a delayed epidemic model incorporating Lévy processes with harmonic mean and vaccination. Int J Biomath 2020. doi:10.1142/S1793524520500892.
[7] Lahrouz A, Settati A, El Fatini M, Tridane A. The effect of a generalized nonlinear incidence rate on the stochastic SIS epidemic model. Math Method Appl Sci. 2020. doi:10.1002/mma.6765.
[8] Higham DJ. An algorithmic introduction to numerical simulation of stochastic differential equations. SIAM Rev 2001;43:525–46.
[9] Report of the WHO-China joint mission on coronavirus disease. 2019. COVID-19 https://www.who.int/docs/default-source/coronaviruses/who-china-joint-mission-on-covid-19-final-reportpdf.
[10] Chen Y, Cheng J, Jiang Y, Liu K. A time delay dynamic system with external source for the local outbreak of 2019-nCoV. Appl Anal 2020;1:1–12.
[11] Atangana A. Modelling the spread of COVID-19 with new fractal-fractional operators: can the lockdown save mankind before vaccination? Chaos Solitons Fractals 2020;136:109860.
[12] Khan MA, Atangana A. Modeling the dynamics of novel coronavirus (2019-nCoV) with fractional derivative. Alexandria Eng J 2020.
[13] Wang H, Wang Z, Dong Y, et al. Phase-adjusted estimation of the number of coronavirus disease 2019 cases in Wuhan, China. Cell Discov. 2020;6(1):1–8.
[14] Hellewell J, Abbott S, Gimma A, et al. Feasibility of controlling COVID-19 outbreaks by isolation of cases and contacts. Lancet Glob Health. 2020;8(4):e488–96.
[15] Chen TM, Rui J, Wang GP, Zhao ZY, Cui JA, Yin L. A mathematical model for simulating the phase-based transmissibility of a novel coronavirus. Infect Dis Poverty. 2020;9(24). doi:10.1186/s40249-020-00864-3.
[16] El Fatini M, Sakkak I, Taki R, El Guedouz T. A control treatment for a stochastic epidemic model with relapse and Crowly–Martin incidence. J Anal 2020. doi:10.1142/s1447866920500764.
[17] Wang H, Wang Z, Dong Y, Chang R, Xu C, Yu X, et al. Phase-adjusted estimation of the number of coronavirus disease 2019 cases in Wuhan, China. Cell Discov. 2020;6(10). doi:10.1038/s41421-020-0148-0.
[18] Luo Q, Mao X, Jiang J. Stochastic population dynamics under regime switching. Appl Anal 2007;334:6984.
[19] Settati A, Lahrouz A. Stationary distribution of stochastic population systems under regime switching. Appl Math Comput 2014;244:235243.
[20] Mandal M, Jana S, Nandi SK, Khatua A, Adak S, Kar TK. A model based study on the dynamics of COVID-19: prediction and control. Chaos Solitons Fractals 2020;136:109889.
[21] El Fatini M, Boukanjime B. Stochastic analysis of a two delayed epidemic model incorporating Lévy processes with a general non-linear transmission. Stoch Anal Appl 2019;1–16. doi:10.1080/07362994.2019.1680295.
[22] El Fatini M, El Khalifi M, Gerlach R, Laaribi A, Taki R, Stationary distribution and threshold dynamics of a stochastic SIRS model with a general incidence. Phys A 2019;534:120696.
[23] https://www.worldometers.info/coronavirus.
[24] Chakraborty T, Ghosh I. Real-time forecasts and risk assessment of novel coronavirus (COVID-19) cases: a data-driven analysis. Chaos Solitons Fractals 2020. doi:10.1016/j.chaos.2020.109850.
[25] El Fatini M, El Khalifi M, Lahrouz A, Pettersson R, Settati A. The effect of stochasticity with respect to reinfection and nonlinear transition states for some diseases with relapse. Math Meth Appl Sci. 2020;1–12. doi:10.1002/mma.6903.
[26] Mao X, Marion G, Renshaw E. Environmental noise suppresses explosion in population dynamics. Stoch Process Appl 2002;97:95–110.
[27] Mao X, Yuan C. Stochastic differential equations with markovian switching. London: Imperial College Press; 2006.