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Stochastic SIR Epidemiological Model With Two Levels of Severity

Gerard Kanga[1] & Jacques Tano [1]

[1] Institut de Recherches Mathématiques (IRMA), Felix Houphouët Boigny University, Abidjan, Côte d'Ivoire

Correspondence: Institut de Recherches Mathématiques (IRMA), Felix Houphouët Boigny University, Abidjan, Côte d'Ivoire

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Abstract

In this paper, we develop an epidemic model to analyze the spread of an infectious disease with two levels of severity within a population of varying size. Additionally, we examine the stability of the disease-free equilibrium.

Such a model is suitable for the dynamics of COVID-19 disease spread. If the two types of infected individuals have different recovery rates, then there is no endemic equilibrium; only the disease-free equilibrium will be the subject of our study. We then study both the deterministic model and a stochastic version. The stochastic model is obtained by perturbing the contact rate using white noise. For the deterministic model, we have shown that if the basic reproduction number $\mathcal{R}_0 < 1$, then the equilibrium state is globally asymptotically stable by using Lyapunov function. This implies that the disease will disappear, and the entire population will become susceptible. For the stochastic version, we demonstrate that the system admits a unique positive global solution that exists within a positively invariant domain. Under suitable conditions on the intensity of the white noise perturbations, we prove that the number of infectious individuals converge almost surely exponentially to zero and the disease-free equilibrium of system is stochastically asymptotically stable in the large provided. Finally, we give some numerical simulations to illustrate our theoretical results.

Keywords: SIaIsR epidemic model, Disease-free equilibrum, Global stability

1. Introduction

The mathematical analysis of the dynamics of disease spread within a population has consistently piqued the interest of many researchers. These investigations primarily revolve around mathematical modeling, with the compartmental SIR model remaining the most classical and fundamental in epidemiology due to its robustness and simplicity. Most models are compartmental models which involve dividing the population into disjointed classes, each containing individuals with the same clinical state regarding the disease. In 1927, some researchers (Kermack & McKendrick, 1927) pioneered the study of the compartmental SIR model, followed by other authors (Kaddar et al., 2011; Korobeinikov & Wake, 2002) who delved into models such as SIS, SEIR and SIRS.

With the emergence of the Covid-19 disease in 2019, researchers have become interested in two-compartment models of infected individuals. Indeed, according to researchers (C.Waechter, 2021), there are two states of desease: symptomatic infected individuals who exhibit signs of the disease, and asymptomatic infected individuals. The number of asymptomatic infection cases at a given moment refers to the number of individuals affected by a disease who do not show any symptoms at that specific time. These situations can elude detection because infected individuals do not exhibit any apparent signs of the disease, but they still have the ability to transmit the infection to others.

We can mention the work of some researchers (Liu et al., 2020), who modeled this phenomenon and used data from three countries to make predictions. The autors (Coulibaly & N´zi, 2021) have also worked on the same model, with a jump perturbation.

In these models, the borders remain closed, which means that there is no recruitment. It is true that authorities have implemented various measures to close the borders in order to slow down the spread, but some borders have remained open with entry controls. This allows the entry of healthy and susceptible individuals into the population.

It is important to emphasize that several authors have examined models involving recruitment within the population of susceptible individuals (Chen & Li, 2022; Lahrouz et al., 2011; Settati et al., 2021). However, in the models examined by these authors, there is only one compartment for infected individuals, who are individuals displaying symptoms of infection. This article is devoted to a qualitative study of an SIaIsR model that incorporates a recruitment process within the population.

The rest of the paper is organized as follows: section 2 is dedicated to the mathematical formulation of the model. we provide some preliminary results in Section 3, while our main results are presented in section 4. In the deterministic model,

we establish the stability of the free equilibrium state. In the stochastic model, we justify the existence and positivity of a solution to the system that almost surely resides in a specific domain of $\mathbb{R}^4$.

Subsequently, we show that, under appropriate conditions, the solution of the stochastic system converges almost surely exponentially to the desease-free equilibrium.

Finally, under assumptions regarding the intensity of the white noise, we demonstrate that the free equilibrium state is globally stochastically stable. In section 5, we perform some numerical simulations to compare the dynamic behaviors of deterministic system and stochastic system.

2. Model Formulation

2.1 Deterministic Model

The model that we present is governed by the following system:

$$\begin{cases} \frac{dS(t)}{dt} = \lambda - \mu S(t) - \tau S(t)[I_a(t) + I_s(t)] \\ \frac{dI_a(t)}{dt} = \tau S(t) I_a(t) - (\mu + \gamma_1) I_a(t) \\ \frac{dI_s(t)}{dt} = \tau S(t) I_s(t) - (\mu + \gamma_2) I_s(t) \\ \frac{dR(t)}{dt} = \gamma_1 I_a(t) + \gamma_2 I_s(t) - \mu R(t) \end{cases} \tag{1}$$

where

- $S(t)$ is the number of susceptible individuals at time t, i.e people who are not infected yet but might become infectious individuals in the future.
- $I_a(t)$ is the number of asymptomatic infectious individuals at time t, i.e people who have contracted the disease but have not yet developed it at time t.
- $I_s(t)$ is the number of symptomatic infectious individuals with mild symptoms at time t.
- $R(t)$ is the number of recovered individuals at time t.
- λ represents the recruitment rate, corresponds to the number of individuals joining the population per unit of time.
- μ is the death rate.
- τ is the transmission rate.
- γ_1 and γ_2 are the recovered rate respectively of asymptomatic infectious individuals and symptomatic infectious individuals.

All the parameters are positive.

An equilibrium state (S, I_a, I_s, R) of the system (1) satisfies the following equations:

$$\begin{cases} \lambda - \mu S - \tau S[I_a + I_s] = 0 \\ \tau S I_a - (\mu + \gamma_1) I_a = 0 \\ \tau S I_s - (\mu + \gamma_2) I_s = 0 \\ \gamma_1 I_a + \gamma_2 I_s - \mu R = 0 \end{cases} \tag{2}$$

A disease-free equilibrium (DFE) is an equilibrium state where $I_a = I_s = 0$ in system (2) . Its stability implies the disappearance of the disease. System (1) admits only one disease-free equilibrium $E^0 = (\frac{\lambda}{\mu}, 0, 0, 0)$.

An endemic equilibrium is an equilibrium state where the compartments of infected individuals (I_a and I_s) are non-zero. Its stability implies the persistence of the disease.

If the recovery rates of both groups of infected individuals are identical, then we have a classical SIR model.

If the recovery rates are different, then there is no endemic equilibrium state with the simultaneous presence of both types of infected individuals ($I_a^* \neq 0$ and $I_s^* \neq 0$).

Indeed, the existence of such a state, denoted as

(S^*, I_a^*, I_s^*, R^*), would lead to $S^* = \frac{\mu+\gamma_1}{\tau} = \frac{\mu+\gamma_2}{\tau}$. That is contradictory. However, we can have a single endemic equilibrium where only one of the compartments of infected individuals is non-zero.

The basic reproduction number $\mathcal{R}_0$ representing how many secondary infectious result from the introduction of one infected individual into a population of susceptible. Using the (Van den Driessche & Watmough, 2002) method, we obtain:

$$\mathcal{R}_0 = \max\left\{\frac{\tau\lambda}{\mu(\mu+\gamma_1)}; \frac{\tau\lambda}{\mu(\mu+\gamma_2)}\right\} \tag{3}$$

We consider different recovery rates and study the stability of the disease-free equilibrium.

2.2 Stochastic Model

The system (1) is obviously a deterministic model that abstracts from any randomness in the parameter values. This assumption often deviates from reality.

Indeed, random fluctuations can influence the parameters or variables of the model. Stochastic perturbations can be introduced to account for the inherent uncertainty in epidemiological processes. According to the literature, these perturbations come in two types. Some authors consider perturbations of the numbers of individuals in compartments through independent Brownian motions. The works of (Cai et al., 2017; Zhang & Wang, 2014; Ikram et al., 2022) can be mentioned in this context. On the other hand, others consider a perturbation of the contact rate, denoted as τ. The works of (Lahrouz et al., 2011; N´zi & Kanga, 2016; N´zi & Tano, 2017) can be cited in this regard. The rate of contact between healthy individuals and infected individuals is subject to random phenomena. The reception of the measures taken by the authorities by the populations often leads to a disruption of the contact rate. To account for this aspect, we have formulated a stochastic version of the model by adding white noise to the contact rate.

Moreover, to account for the stochastic nature of the contact rate, we have added white noise $\sigma\frac{dB(t)}{dt}$ to it. Where $(B(t))_{t\geq 0}$ is standard Brownian motion.

We have taken this type of perturbation into account in our model. We get the following system:

$$\begin{cases} dS(t) = \left[\lambda - \mu S(t) - \tau S(t)(I_a(t) + I_s(t))\right]dt \\ \qquad\quad -\sigma S(t)(I_a(t) + I_s(t))dB(t) \\ dI_a(t) = \left[\tau S(t)I_a(t) - (\mu+\gamma_1)I_a(t)\right]dt + \sigma I_a(t)S(t)dB(t) \\ dI_s(t) = \left[\tau S(t)I_s(t) - (\mu+\gamma_2)I_s(t)\right]dt + \sigma I_s(t)S(t)dB(t) \\ dR(t) = \left[\gamma_1 I_a(t) + \gamma_2 I_s(t) - \mu R(t)\right]dt \end{cases} \tag{4}$$

3. Preliminaries

Let

$$\Delta = \left\{x \in (\mathbb{R}_+^*)^4; x_1 + x_2 + x_3 + x_4 < \frac{\lambda}{\mu}\right\}. \tag{5}$$

Its easy to prove that domain Δ is invariant. In effect, the total population in system (1) verifies the equation

$$\frac{dN(t)}{dt} = \lambda - \mu N(t)$$

$$N(t) = \left(N(0) - \frac{\lambda}{\mu}\right)e^{-\mu t} + \frac{\lambda}{\mu} < \frac{\lambda}{\mu}$$

Theorem 2.1 (A.M. Lyapunov:1992)

If there exists a continuously differentiable function" $V : \mathbb{R}^n \to \mathbb{R}$ such that:

- $V(x_e) = 0$,

- $V(x) > 0 \ \forall x \neq x_e$,
- $f^T(x)\frac{\partial V}{\partial x}(x) < 0 \ \forall x \neq x_e$,
- $f^T(x)\frac{\partial V}{\partial x}(x) \to \infty$ lorsque $\|x\| \to \infty$.

Therefore, the equilibrium state x_e is globally asymptotically stable.

Theorem 2.2 The unidimensional Itô's formula(Seidler, 1991) (I. Karatzas and S.E Shere)

Let $(t, x) \mapsto f(t, x)$ be a real function that is twice differentiable in x and once differentiable in t, and let X be an Itô process. Then we have:

$$\begin{aligned} f(t, X_t) &= f(0, X_0) + \int_0^t f_s^{'}(s, X_s)ds + \int_0^t f_x^{'}(s, X_s)dX_s \\ &\quad + \frac{1}{2}\int_0^t f_{xx}^{''}(s, X_s)d\langle X, X\rangle_s. \end{aligned}$$

Theorem 2.3 Comparison Theorem (Ikeda:1976)

Let σ, b_1, and b_2 be three continuous functions defined on $[0 : +\infty[\times\mathbb{R}$ with values in $\mathbb{R}$, such that $b_1(t, x) \leq b_2(t, x)$ for all $t \geq 0$ and for all $x \in \mathbb{R}$. We consider the following stochastic differential equations:

$$dX_t = b_1(t, X_t)dt + \sigma(t, X_t)dB_t \tag{6}$$

$$dX_t = b_2(t, X_t)dt + \sigma(t, X_t)dB_t \tag{7}$$

If $(X_t^1)_{t\geq 0}$ and $(X_t^2)_{t\geq 0}$ are respective solutions of equations (6) and (7) such that $X_0^1 \leq X_0^2$, then $\mathbb{P} - p.s\ X_t^1 \leq X_t^2$ for all $t \geq 0$.

4. Main Results

4.1 Deterministic Model

Theorem 3.1 *If $\mathcal{R}_0 < 1$ then the desease-free equilibrium $(\frac{\lambda}{\mu}, 0, 0, 0)$ is globally asymptotically stable in Δ.*

Proof Let $\tilde{S} = \dfrac{\lambda}{\mu} - S$. Then system (1) becomes as follows

$$\begin{cases} d\tilde{S}(t) = \left[-\mu\tilde{S}(t) + \frac{\tau\lambda}{\mu}(I_a(t) + I_s(t)) - \tau\tilde{S}(t)(I_a(t) + I_s(t))\right]dt \\ dI_a(t) = \left[-\tau\tilde{S}(t)I_a(t) + (\mu + \gamma_1)(\mathcal{R}_0^{(1)} - 1)I_a(t)\right]dt \\ dI_a(t) = \left[-\tau\tilde{S}(t)I_s(t) + (\mu + \gamma_2)(\mathcal{R}_0^{(2)} - 1)I_s(t)\right]dt \\ dR(t) = \left[\gamma_1 I_a(t) + \gamma_2 I_s(t) - \mu R(t)\right]dt \end{cases}$$

where $\mathcal{R}_0^{(i)} = \dfrac{\tau\lambda}{\mu(\mu + \gamma_i)}$, i=1, 2.
Let ϵ and C positive constants such that,

$$0 < \epsilon < \min\left\{\frac{\mu^2}{\tau\lambda}; \frac{2\mu}{\gamma_1 + \gamma_2}\right\} \tag{8}$$

$$0 < C < \min\left\{\frac{\epsilon\mu(\mu + \gamma_1)(1 - R_0^{(1)})}{\tau\lambda + \mu\gamma_1}; \frac{\epsilon\mu(\mu + \gamma_2)(1 - R_0^{(2)})}{\tau\lambda + \mu\gamma_2}\right\} \tag{9}$$

Consider function

$$V_1(\tilde{S}, I_a, I_s, R) = C\tilde{S}^2 + \frac{1}{2}I_a^2 + \frac{1}{2}I_s^2 + CR^2$$

$$\begin{aligned} dV_1(t) &= -2C\mu\tilde{S}^2 - 2C\mu R^2 - (\mu+\gamma_1)(1-\mathcal{R}_0^{(1)})I_a^2 - (\mu+\gamma_2)(1-\mathcal{R}_0^{(2)})I_s^2 \\ &+ \frac{2C\lambda\tau}{\mu}\tilde{S}I_a + \frac{2C\lambda\tau}{\mu}\tilde{S}I_s + 2C\gamma_1 RI_a + 2C\gamma_2 RI_s \\ &- 2\tau C\tilde{S}^2(I_a+I_s) - \tau\tilde{S}I_a^2 - \tau\tilde{S}I_s^2 \end{aligned} \tag{10}$$

The last term in (10) is negative and using Young inequality we have:

$$\tilde{S}I_a \leq \frac{1}{2\epsilon}I_a^2 + \frac{\epsilon}{2}\tilde{S}^2$$

$$\tilde{S}I_s \leq \frac{1}{2\epsilon}I_s^2 + \frac{\epsilon}{2}\tilde{S}^2$$

$$RI_a \leq \frac{1}{2\epsilon}I_a^2 + \frac{\epsilon}{2}R^2$$

$$RI_s \leq \frac{1}{2\epsilon}I_s^2 + \frac{\epsilon}{2}R^2$$

where ϵ is the constant in (8). Those inequalities injincting in (10), we obtain:

$$\begin{aligned} dV_1(t) &\leq -2C\mu\tilde{S}^2 - 2C\mu R^2 - (\mu+\gamma_1)(1-\mathcal{R}_0^{(1)})I_a^2 \\ &- (\mu+\gamma_2)(1-\mathcal{R}_0^{(2)})I_s^2 + \frac{2C\lambda\tau}{\mu}\left[\frac{1}{2\epsilon}I_a^2 + \frac{\epsilon}{2}\tilde{S}^2\right] \\ &+ \frac{2C\lambda\tau}{\mu}\left[\frac{1}{2\epsilon}I_s^2 + \frac{\epsilon}{2}\tilde{S}^2\right] + 2C\gamma_1\left[\frac{1}{2\epsilon}I_a^2 + \frac{\epsilon}{2}R^2\right] \\ &+ 2C\gamma_2\left[\frac{1}{2\epsilon}I_s^2 + \frac{\epsilon}{2}R^2\right] \\ &\leq K_1\tilde{S}^2 + K_2I_a^2 + K_3I_s^2 + K_4R^2 \end{aligned}$$

where

$$K_1 = 2C\left(-\mu + \frac{\tau\lambda}{\mu}\epsilon\right) < 0$$

$$K_2 = -(\mu+\gamma_1)(1-\mathcal{R}_0^{(1)}) + \frac{C}{\epsilon}\left(\frac{\tau\lambda+\mu\gamma_1}{\mu}\right)$$

$$K_3 = -(\mu+\gamma_2)(1-\mathcal{R}_0^{(2)}) + \frac{C}{\epsilon}\left(\frac{\tau\lambda+\mu\gamma_2}{\mu}\right)$$

Using the fact that $\mathcal{R}_0 < 1$ and (9) it esay to verify that $K_2 < 0$ and $K_3 < 0$.

$$K_4 = C\left(-2\mu + (\gamma_1+\gamma_2)\epsilon\right) < 0 \quad see \quad (14)$$

According to the theorem, the free equilibrium state is globally asymptotically stable.

4.2 Stochastic Model

Let $(\Omega, \mathcal{F}, \mathbb{P})$ be a complete probability space with a filtration $\{\mathcal{F}_t\}_{t\geq 0}$ satisfying the usual conditions. We consider the system (4) and the domain Δ (see (5)).

Theorem 3.2 If $(S(0), I_a(0), I_s(0), R(0)) \in \Delta$, then the system admits a unique solution $(S(t), I_a(t), I_s(t), R(t))$ on $t \geq 0$ and this solution remains in Δ with probability one.

Proof Suppose that $(S(0), I_a(0), I_s(0), R(0)) \in \Delta$. The total population verifies the equation

$$\frac{dN(t)}{dt} = \lambda - \mu N(t).$$

Then if $(S(t), I_a(t), I_s(t), R(t)) \in \mathbb{R}_+^4$ for all $0 \leq t \leq T$ a.s.

We get

$$dN(t) \leq (\lambda - \mu N(t))dt \quad a.s.$$

Hence by comparison Theorem we have,

$$dN(t) \leq \frac{\lambda}{\mu} + \left(N(0) - \frac{\lambda}{\mu}\right)e^{-\mu t},$$

for all $t \in [0, T]$ a.s. Then $N(t) \leq \frac{\lambda}{\mu}$, so we have

$$(S(t), I_a(t), I_s(t), R(t)) \in \Delta \tag{11}$$

for all $t \in [0, T]$ a.s.

The coefficients of the system (4) are locally Lipschitz continuous, so for any given initial value $(S(0), I_a(0), I_s(0), R(0))$ there is a unique local solution on $[0, \tau_e]$ where τ_e is the explosion time.

To prove that this solution is global we need to show that $P(\tau_e = \infty) = 1$

Let $\epsilon_0 > 0$ be such that $S(0), I_a(0), I_s(0), R(0) > \epsilon_0$. For $\epsilon \leq \epsilon_0$ considering the stopping times

$$\tau_\epsilon = inf\,\{t \in [0, \tau_e], S(t) \leq \epsilon \; or \; I_a(t) \leq \epsilon \; or \; I_s(t) \leq \epsilon \; or \; R(t) \leq \epsilon\}$$

and

$$\begin{aligned} \tau &= \lim_{\epsilon \to 0} \tau_\epsilon \\ &= inf\,\{t \in [0, \tau_e], S(t) \leq 0 \; or \; I_a(0) \leq 0 \; or \; I_s(0) \leq 0 \; or \; R(t) \leq 0\}. \end{aligned}$$

Consider the function V defined for $X(t) = (S_t, I_t, R_t) \in \mathbb{R}^3_+$ by

$$V_2(S, I_a, I_s, R) = -\ln\left(\frac{\mu S}{\lambda}\right) - \ln\left(\frac{\mu I_a}{\lambda}\right) - \ln\left(\frac{\mu I_s}{\lambda}\right) - \ln\left(\frac{\mu R}{\lambda}\right)$$

By virtue of Itô's Formula, we have for all $T \geq 0$, and for all $t \in [0, T \wedge \tau_\epsilon]$

$$\begin{aligned} dV(X(t)) &= \left[4\mu + \gamma_1 + \gamma_2 + \sigma^2(S(t))^2 + \tau(I_a(t) + I_s(t)) + \frac{1}{2}\sigma^2(I_a + I_s)^2\right]dt \\ &- \left[\frac{\lambda}{S(t)} + 2\tau S(t) + \gamma_1\frac{I_a(t)}{R(t)} + \gamma_2\frac{I_s(t)}{R(t)}\right]dt + \sigma\left[I_a(t) + I_s(t) - 2S(t)\right]dB(t) \end{aligned} \tag{12}$$

By virtue of (11), we assert that $S(t), I_a(t), I_s(t), R(t) \in [0, \frac{\lambda}{\mu}]$ for every $t \in [0, T \wedge \tau_\epsilon]$ a.s.

So, we have the following inequalities:

$$(S(t))^2 \leq \left(\frac{\lambda}{\mu}\right)^2$$

and,

$$(I_a(t) + I_s(t)) \leq \left(\frac{2\lambda}{\mu}\right).$$

Let us, put

$$k = 4\mu + \gamma_1 + \gamma_2 + \left(\frac{\sigma\lambda}{\mu}\right)^2 + \frac{2\tau\lambda}{\mu} + 2\left(\frac{\sigma\lambda}{\mu}\right)^2.$$

In view of (12), we have

$$dV(X(t)) \leq kdt + \sigma(I_a(t) + I_s(t) - 2S(t))dB(t).$$

By using the comparison theorem,

$$V(X(t)) - V(X(0)) \leq kt + \int_0^t (\sigma(I_a(u) + I_s(u) - 2S(u)))\,dB(u) \;\; a.s$$

for all $t \in [0, T \wedge \tau_\epsilon]$.

In particular at the point $T \wedge \tau_\epsilon$ this inequality remains true.

Now taking the expectation of both parts of the above inequality and using the fact that

$$\left(\int_0^t (\sigma(I_a(u) + I_s(u) - 2S(u)))\, dB_u\right)_{t\geq 0}$$

is a mean zero process, we deduce that for all $T \geq 0$

$$\mathbb{E}[V(X(T \wedge \tau_\epsilon))] \leq V(X(0)) + \mathbb{E}[k(T \wedge \tau_\epsilon)] \leq kT + V(X(0)) \tag{13}$$

Furthermore, in view of (11), we have $V(X(T \wedge \tau_\epsilon) \geq 0$ thus,

$$\begin{aligned} \mathbb{E}[V(X(T \wedge \tau_\epsilon))] &= \mathbb{E}[V(X(T \wedge \tau_\epsilon))\chi_{\{\tau_\epsilon \leq T\}}] + \mathbb{E}[V(X(T \wedge \tau_\epsilon))\chi_{\{\tau_\epsilon > T\}}] \\ &\geq \mathbb{E}[V(X(\tau_\epsilon))\chi_{\{\tau_\epsilon \leq T\}}]. \end{aligned}$$

By continuity there is some component of $X(\tau_\epsilon)$ equal to ϵ,
therefore $V(X(\tau_\epsilon)) \geq -ln\left(\frac{\mu\epsilon}{\lambda}\right)$.
So, we have

$$\mathbb{E}[V(X(T \wedge \tau_\epsilon))] \geq -ln\left(\frac{\mu\epsilon}{\lambda}\right)\mathbb{P}(\tau_\epsilon \leq T). \tag{14}$$

By combining (13) and (14) we obtain for all $T \geq 0$

$$\mathbb{P}(\tau_\epsilon \leq T) \leq -\frac{kT + V(X(0))}{ln\left(\frac{\mu\epsilon}{\lambda}\right)}.$$

By letting ϵ goes to zero, we derive that for all $T \geq 0$, $P(\tau \leq T) = 0$. Hence $\mathbb{P}(\tau = \infty) = 1$. As $\tau_e \geq \tau$, we have $\tau_e = \tau = \infty$ a.s .

Theorem 3.3 If $\mathcal{R}_0 < 1$ then $(I_a(t), I_s(t))_{t\geq 0}$ converge almost surely exponentially to $(0, 0)$.

Proof

Let $(S(0), I_a(0), I_s(0), R(0)) \in \Delta$. Consider function

$$V_3 = \ln\left(\frac{1}{\mu + \gamma_1} I_a + \frac{1}{\mu + \gamma_2} I_s\right)$$

By Itô's formula, we have

$$dV_3(t) = LV_3(t)dt + \sigma S(t)^2 dB(t)$$

where,

$$\begin{aligned} LV_3(t) &= \frac{1}{\frac{1}{\mu+\gamma_1} I_a + \frac{1}{\mu+\gamma_2} I_s}\left[\left(\frac{\tau S}{\mu + \gamma_1} - 1\right) I_a + \left(\frac{\tau S}{\mu + \gamma_2} - 1\right) I_s\right] \\ &- \left[\frac{\sigma^2 S^2}{2\left(\frac{1}{\mu+\gamma_1} I_a + \frac{1}{\mu+\gamma_2} I_s\right)^2}\left(\frac{1}{\mu + \gamma_1} I_a^2 + \frac{1}{\mu + \gamma_2} I_s^2\right)\right] \end{aligned}$$

Since the last term is negative and $S(t) \leq \frac{\lambda}{\mu}, \forall t \geq 0$
we have,

$$\begin{aligned} LV_3(t) &\leq \frac{1}{\frac{1}{\mu+\gamma_1} I_a + \frac{1}{\mu+\gamma_2} I_s} \times \left[\left(\frac{\tau\lambda}{\mu(\mu + \gamma_1)} - 1\right) I_a + \left(\frac{\tau\lambda}{\mu(\mu + \gamma_2)} - 1\right) I_s\right] \\ &= \frac{1}{\frac{1}{\mu+\gamma_1} I_a + \frac{1}{\mu+\gamma_2} I_s} \times \left[-\left(1 - \frac{\tau\lambda}{\mu(\mu + \gamma_1)}\right) I_a - \left(1 - \frac{\tau\lambda}{\mu(\mu + \gamma_2)}\right) I_s\right] \end{aligned}$$

Let $\omega = \min\left\{(\mu+\gamma_1)\left(1-\frac{\tau\lambda}{\mu(\mu+\gamma_1)}\right);(\mu+\gamma_2)\left(1-\frac{\tau\lambda}{\mu(\mu+\gamma_2)}\right)\right\}$ Therefore,

$$LV_3(t) \leq \frac{1}{\frac{1}{\mu+\gamma_1}I_a + \frac{1}{\mu+\gamma_2}I_s}\left[-\frac{\omega}{\mu+\gamma_1}I_a - \frac{\omega}{\mu+\gamma_2}I_s\right] \leq -\omega$$

By integration we check

$$\begin{aligned}\ln\left(\frac{1}{\mu+\gamma_1}I_a(t)+\frac{1}{\mu+\gamma_2}I_s(t)\right) &\leq \ln\left(\frac{1}{\mu+\gamma_1}I_a(0)+\frac{1}{\mu+\gamma_2}I_s(0)\right)\\ &- \omega t + \sigma\int_0^t S^2(u)dB(u)\end{aligned} \tag{15}$$

$(S(t))^2$ is bounded, then by the strong law of large number for local martingales we have

$$\lim\frac{1}{t}\sigma\int_0^t S^2(u)dB(u) = 0 \tag{16}$$

From (15) and (16) we have,

$$\limsup\frac{1}{t}\ln\left(\frac{1}{\mu+\gamma_1}I_a(t)+\frac{1}{\mu+\gamma_2}I_s(t)\right) \leq -\omega < 0.$$

This completes the proof.

Theorem 3.4

If $\mathcal{R}_0 < 1$ and

$$\sigma^2 < \min\left\{2\left(\frac{\mu}{\lambda}\right)^2(1-\mathcal{R}_0)(\mu+\gamma_1); 2\left(\frac{\mu}{\lambda}\right)^2(1-\mathcal{R}_0)(\mu+\gamma_2)\right\} \tag{17}$$

then

$$\limsup\frac{1}{t}\mathbb{E}\int_0^t\left[\theta\left(\frac{\lambda}{\mu}-S(u)\right)^2+\frac{1}{2}I_a^2(u)+\frac{1}{2}I_s^2(u)+\theta R^2(u)\right]du \leq 0$$

Proof Choose ϵ as in (8) and constant θ verify:

$$0 < \theta < \min\{\zeta_1\ ;\zeta_2\} \tag{18}$$

where

$$\zeta_i = \frac{2(\mu+\gamma_i)(1-\mathcal{R}_0^{(i)})\mu^2-(\sigma\lambda)^2}{\mu(\tau\lambda+\mu\gamma_i)+2\epsilon(\sigma\lambda)^2}\epsilon, \quad i\in\{1;2\}$$

Consider the same function as in the proof of the Theorem 3.1

$$V_4(\tilde{S}, I_a, I_s, R) = \theta\tilde{S}^2 + \frac{1}{2}I_a^2 + \frac{1}{2}I_s^2 + \theta R^2$$

By Itô's formula, we have

$$dV_4(t) = LV_4(t)dt + H(t)dB(t)$$

where $H(t) = \sigma(2\theta+1)S(t)(I_a(t)+I_s(t))$ and

$$\begin{aligned}LV_4(t) &= -2\theta\mu\tilde{S}^2 - 2\theta\mu R^2 - (\mu+\gamma_1)(1-\mathcal{R}_0^{(1)})I_a^2 - (\mu+\gamma_2)(1-\mathcal{R}_0^{(2)})I_s^2\\ &+ \frac{2\theta\lambda\tau}{\mu}\tilde{S}I_a + \frac{2\theta\lambda\tau}{\mu}\tilde{S}I_s + 2\theta\gamma_1 RI_a + 2\theta\gamma_2 RI_s - 2\tau\theta\tilde{S}^2(I_a+I_s) - \tau\tilde{S}I_a^2 - \tau\tilde{S}I_s^2\\ &+ \left(\frac{\sigma\lambda}{\mu}\right)^2\left(2\theta+\frac{1}{2}\right)(I_a^2+I_s^2)\end{aligned}$$

Using the fact that $0 \leq S \leq \frac{\lambda}{\mu}$ and inegalitie

$(a+b)^2 \leq 2a^2+2b^2$, we have

$$LV_4 \leq \Gamma_1\tilde{S}^2 + \Gamma_2 I_a^2 + \Gamma_3 I_s^2 + \Gamma_4 R^2$$

where,

$$\Gamma_1 = 2\theta\left(-\mu + \frac{\tau\lambda}{\mu}\epsilon\right) < 0 \quad refer \quad to \quad (8)$$

$$\Gamma_2 = -(\mu+\gamma_1)(1-\mathcal{R}_0^{(1)}) + \frac{\theta}{\epsilon}\left(\frac{\tau\lambda+\mu\gamma_1}{\mu}\right) + \left(\frac{\sigma\lambda}{\mu}\right)^2\left(2\theta+\frac{1}{2}\right) < 0$$

$$\Gamma_3 = -(\mu+\gamma_2)(1-\mathcal{R}_0^{(2)}) + \frac{\theta}{\epsilon}\left(\frac{\tau\lambda+\mu\gamma_2}{\mu}\right) + \left(\frac{\sigma\lambda}{\mu}\right)^2\left(2\theta+\frac{1}{2}\right) < 0$$

$$\Gamma_4 = \theta(-2\mu + (\gamma_1+\gamma_2)\epsilon) < 0 \quad according \quad to \quad (8)$$

Using (17) and (18) we verify that $\Gamma_2 < 0$ and $\Gamma_3 < 0$ This completes the proof.

5. Simulation and Discussions

To illustrate the various theoretical results presented above, the systems (1) and (4) were simulated for various sets of parameters. Figures 1 to 4 illustrate the deterministic model (1), and Figures 5 and 6 depict the stochastic model (4).

Figures 1 and 2 illustrate the dynamical behavior of the $SIaIsR$ model described by the deterministic system (1), when $\mathcal{R}_0 < 1$.

Table 1. Estimated parameters of figure 1 and 2

Parameter	figure 1	figure 2
τ	0.00296	0.00009
γ_1	0.7	0.7
γ_2	0.9	0.9
μ	0.4	0.4
λ	50	1000
N	10000	15000
I_a0	1500	3000
I_s0	2000	3000
$R0$	7	70

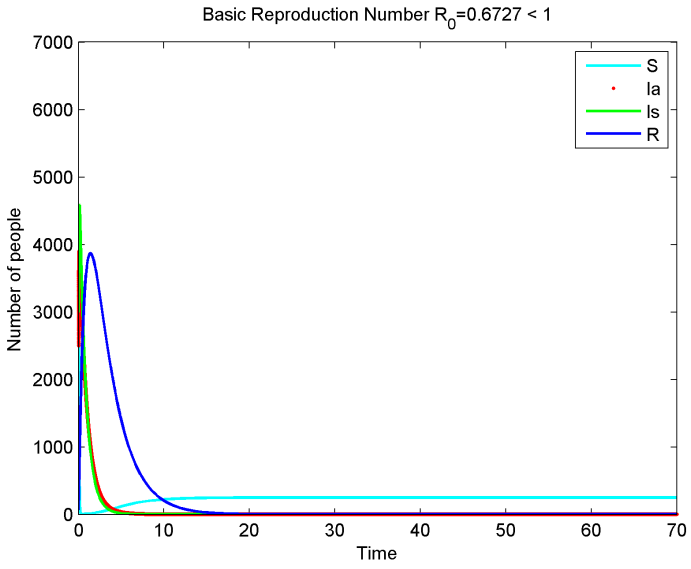

Figure 1. Deterministic trajectories of $SIaIsR$ epidemics model

The Theorem 3.1 is confirmed by the observation of the global stability of the Disease-Free Equilibrium (DFE) in this context. As a result, the number of symptomatic infected individuals decreases rapidly towards zero, while the number of asymptomatic infected individuals increases slightly and then decreases to zero as well.

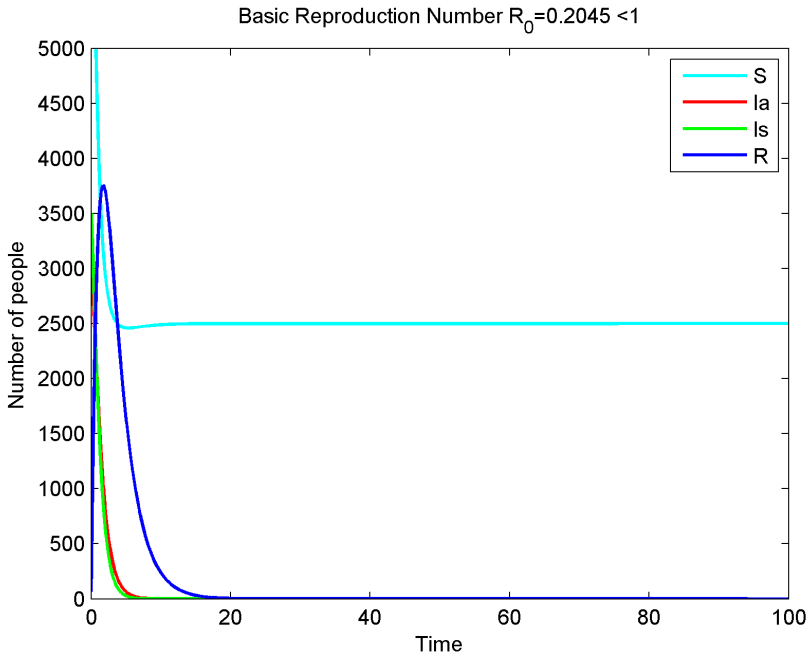

Figure 2. Deterministic trajectories of $SIaIsR$ epidemics model

We also observe the global stability of the Disease-Free Equilibrium (DFE). In this case, the recovery rates are higher than those presented in Figure 1, leading to a rapid decrease of symptomatic and asymptomatic infections towards zero.

Figures 3 and 4 illustrate the dynamical behavior of the $SIaIsR$ model described by the deterministic system (5), when $\mathcal{R}_0 > 1$. In both cases, we observe an instability of the Disease-Free Equilibrium (DFE).

Table 2. Estimated parameters of figure 3 and 4

Parameter	figure 3	figure 4
τ	0.007	0.14
γ_1	0.07	0.9
γ_2	0.7	0.9
μ	0.3	0.2
λ	800	800
N	10000	10000
I_a0	4500	3500
I_s0	2000	2500
$R0$	70	70

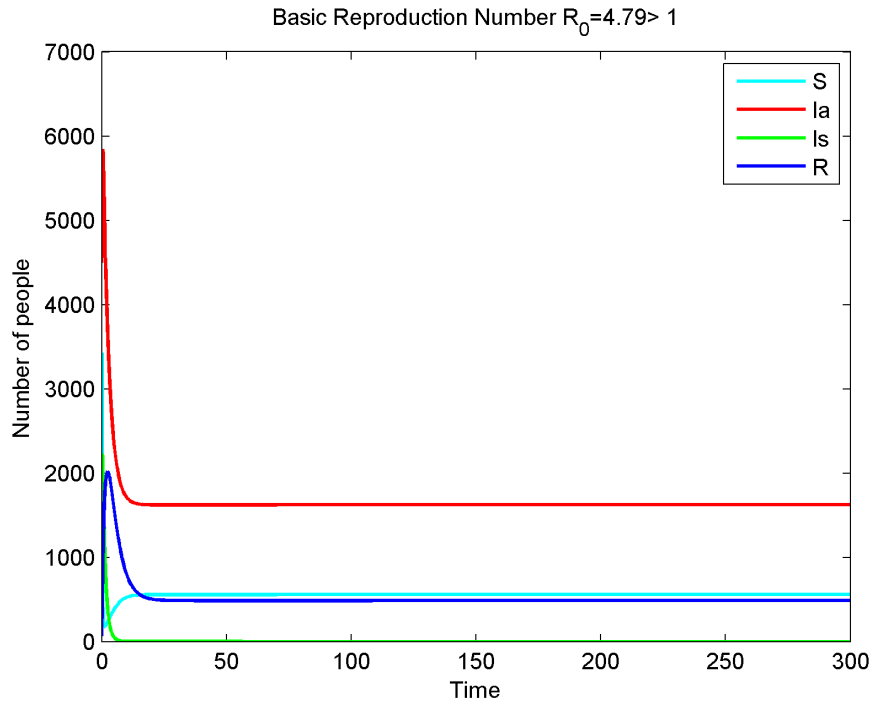

Figure 3. Deterministic trajectories of $SIaIsR$ epidemics model
The recovery rates are distinct, and we observe stability of a single endemic equilibrium ($I_a^* \neq 0$ and $I_s^* = 0$).

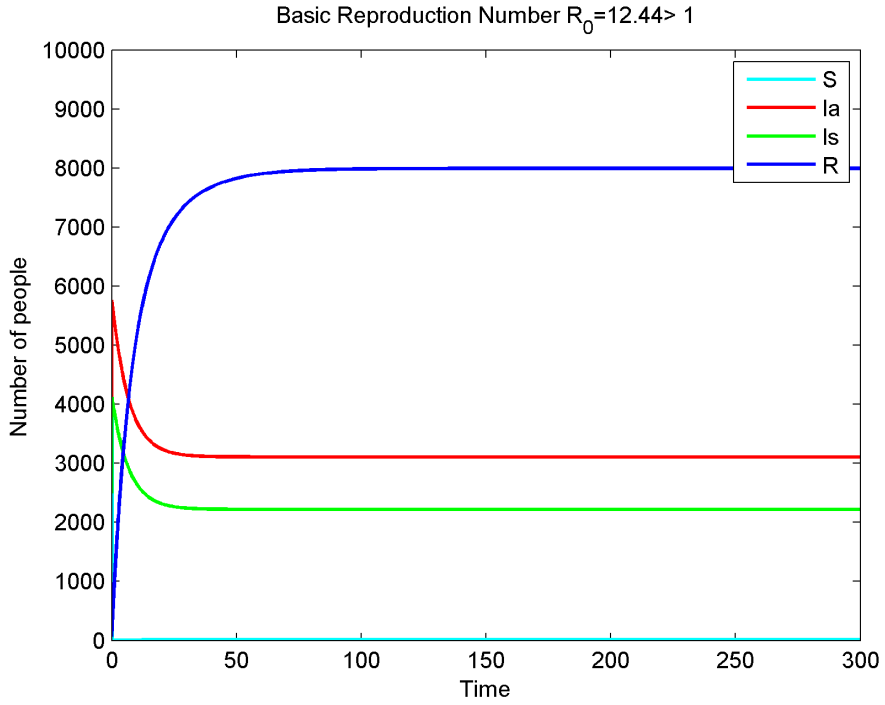

Figure 4. Deterministic trajectories of $SIaIsR$ epidemics model

When the recovery rates are the same, we have a classic SIR model with the stability of an endemic equilibrium state.

Table 3. Estimated parameters of figure 5 and 6

Parameter	figure 5	figure 6
τ	0.003	0.0003
γ_1	0.6	0.7
γ_2	0.7	0.9
μ	0.1	0.2
λ	40	540
N	7000	15000
I_a0	1500	3500
I_s0	100	3000
$R0$	7	90

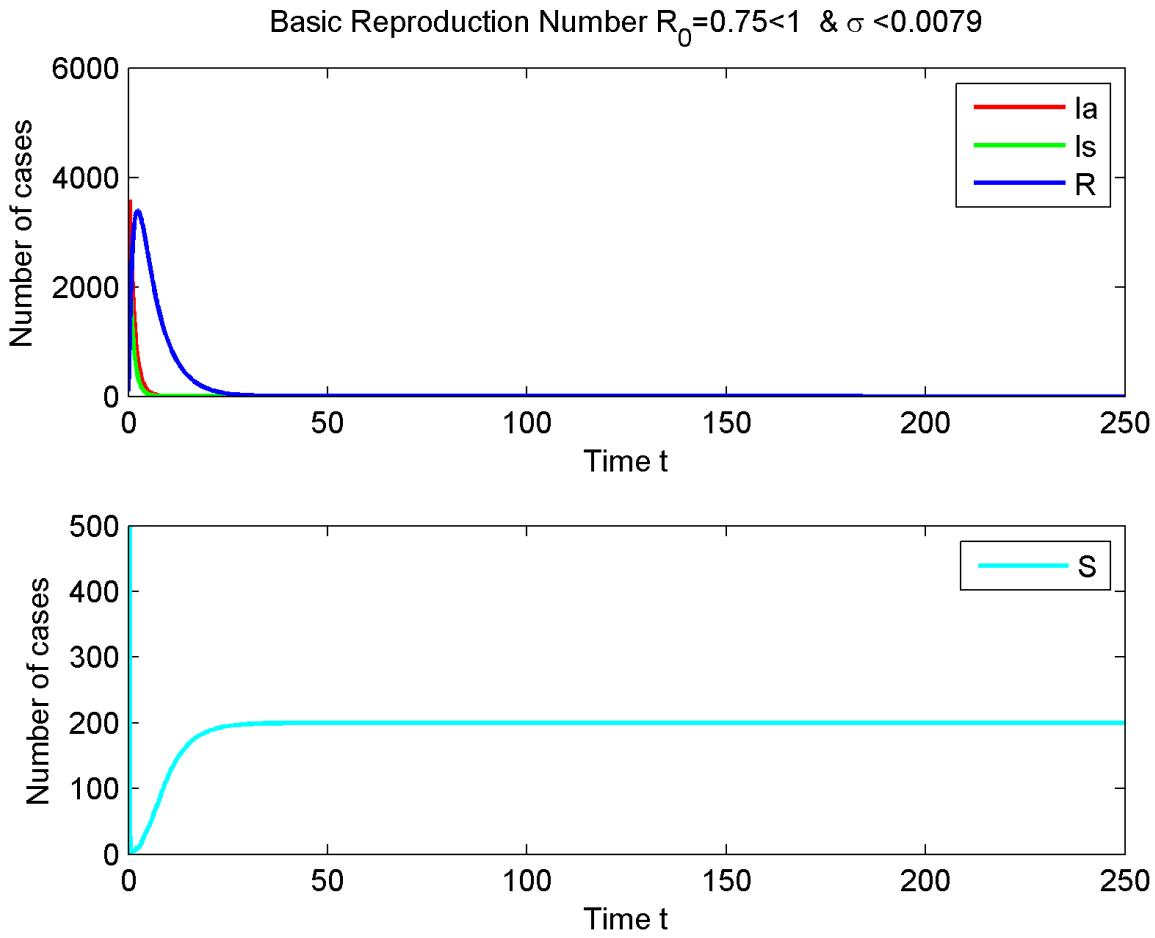

Figure 5. Stochastic trajectories of $SIaIsR$ epidemics model

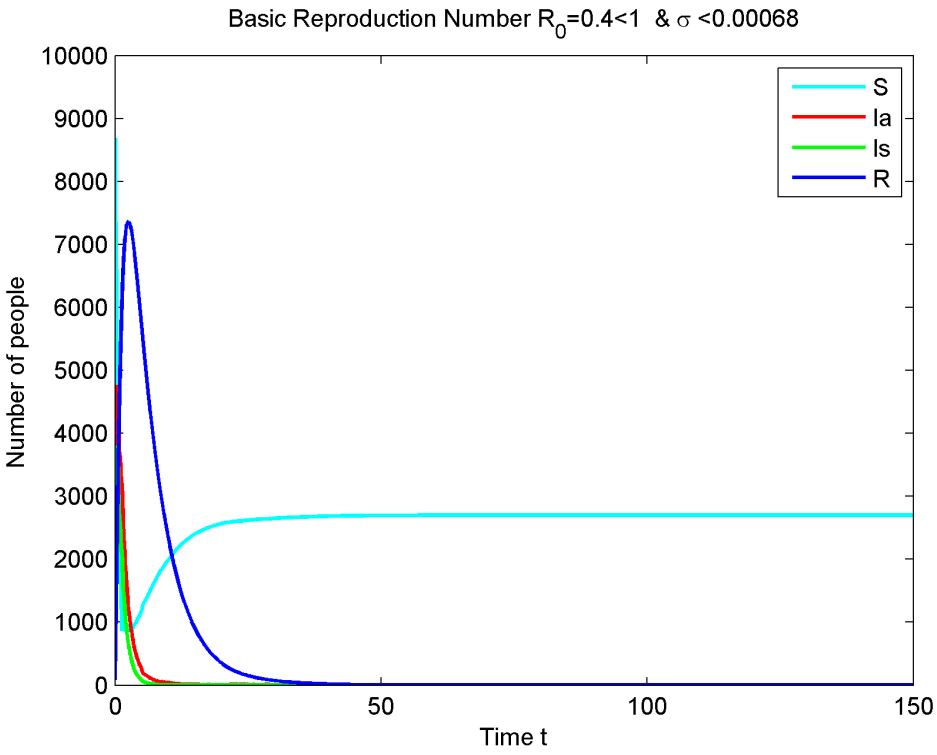

Figure 6. Stochastic trajectories of $SIaIsR$ epidemics model

Figures 5 and 6 illustrate the cases where the intensity of noise σ satisfies the conditions (17) of Theorem 3.4 . It is observed that the disease-free equilibrium state is stochastically asymptotically stable.

6. Conclusion

One primary objective of mathematical epidemiology is to comprehend how to control or eradicate diseases. Mathematical models are extensively employed in the investigation of ecological and epidemiological phenomena. One of the key challenges in studying epidemic behavior is the analysis of the model's steady states and their stability.

In this paper, we studied an epidemiological model with 2 compartments of infected individuals in both deterministic and stochastic cases. This model is suitable for studying Covid-19, which involves both symptomatic and asymptomatic infected individuals.

In the deterministic case, if the basic reproduction number is less than 1, then the disease-free equilibrium state is globally asymptotically stable, indicating disease eradication. However, if the basic reproduction number is greater than 1, the disease persists, as illustrated in Figures 3 and 4. In the stochastic case, we demonstrated that a small perturbation in the contact rate ensures global asymptotic stability of the disease-free equilibrium state. At two levels, our model extends the work of Liu et al. (Liu et al., 2020). This involves incorporating the recruitment of susceptible populations and introducing a stochastic version of the model. Our findings stem from a qualitative study (stability of disease-free equilibrium). Disease control conditions have been identified based on the basic reproduction number and the intensity of the perturbation. However, such a result does not hold with high noise intensity, which could be a subject for future investigation.

References

Cai, Y., Kang, Y., & Wang, W. (2017). A stochastic SIRS epidemic model with nonlinear incidence rate. *Applied Mathematics and Computation, 305*, 221- 240.

Chen, T., & Li, Z. (2022). Dynamics and stationary distribution of a stochastic SIRS epidemic model with a general incidence and immunity. *Boundary Value Problems,* (1), 85. https://doi.org/10.1186/s13661-022-01668-0

Coulibaly, M., & N'zi, M. (2021). A Stochastic Model with Jumps for the COVID-19 Epidem-ic in the Greater Abidjan

Region during Public Health Measures. *J Infect Dis Epidemiol, 7*, 196.

Ikeda, N., & Watanabe, S. (1977). A comparison theorem for solutions of stochastic differential equations and its applications. Retrieved from https://projecteuclid.org/journalArticle/Download-urlid=ojm

Ikram, R., Khan, A., Zahri, M., Saeed, A., Yavuz, M., & Kumam, P. (2022). Extinction and stationary distribution of a stochastic COVID-19 epidemic model with time-delay. *Computers in Biology and Medicine, 141*, 105-115.

Kaddar, A., Abta, A., & Alaoui, H. T. (2011). A comparison of delayed SIR and SEIR epidemic models. *Nonlinear Analysis: Modelling and Control, 16*(2), 181-190.

Kermack, W. O., & McKendrick, A. G. (1927). A contribution to the mathematical theory of epidemics. Proceedings of the royal society of london. *Series A, Containing papers of a mathematical and physical character, 115*(772), 700-721.

Korobeinikov, A., & Wake, G. C. (2002). Lyapunov functions and global stability for SIR, SIRS, and SIS epidemiological models. *Applied Mathematics Letters, 15*(8), 955-960.

Lahrouz, A., Omari, L., & Kiouach, D. (2011). Global analysis of a deterministic and stochastic nonlinear SIRS epidemic model. *Nonlinear Analysis: Modelling and Control, 16*(1), 59-76.

Liu, Z., Magal, P., Seydi, O., & Webb, G. (2020). Predicting the cumulative number of cases for the COVID-19 epidemic in China from early data (arXiv:2002.12298). arXiv. http://arxiv.org/abs/2002.12298

Lyapunov, A. M. (1992). The general problem of the stability of motion. *International Journal of Control, 55*(3), 531-534. https://doi.org/10.1080/00207179208934253

N'zi, M., & Kanga, G. (2016). Global analysis of a deterministic and stochastic nonlinear SIRS epidemic model with saturated incidence rate. *Random Operators and Stochastic Equations, 24*(1), 65-77. https://doi.org/10.1515/rose-2016-0005

N'zi, M., & Tano, J. (2017). Deterministic and stochastic stability of an SIRS epidemic model with a saturated incidence rate. *Random Operators and Stochastic Equations, 25*(1), 11-26. https://doi.org/10.1515/rose-2017-0002

Seidler, J. (1991). Brownian motion and stochastic calculus?: Ioannis Karatzas and Steven E. Shreve: Graduate Texts in Mathematics 113, Springer-Verlag, New York and Berlin 1988, xxiii + 470 pp., ISBN 0-387-96535-1. *Acta Applicandae Mathematicae, 24*(2), 197-200. https://doi.org/10.1007/BF00046894

Van den Driessche, P., & Watmough, J. (2002). Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission. *Mathematical biosciences, 180*(1-2), 29-48.

Waechter, C. (2021). Clinical and paraclinical features of COVID-19, virological diagnosis. NPG Neurologie-Psychiatrie-Geriatrie.
Retrieved from https://pesquisa.bvsalud.org/global-literature-on-novel-coronavirus-2019-ncov/resource/pt/covidwho-1328779

Zhang, X., & Wang, K. (2014). Stochastic SEIR model with jumps. *Applied Mathematics and Computation, 239*, 133-143.

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Authors contributions

Dr. Jacques TANO and Dr. Gerard KANGA were responsible for study design and revising. . Dr Jacques TANO drafted the manuscript and revised it. All authors read and approved the final manuscript and they contributed equally to the study.

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Data sharing statement

No additional data are available.

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