A Stochastic Differential Equations Model for Internal COVID-19 Dynamics

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Abstract. In this article, we analyze stochastic differential equations model for internal coronavirus (COVID-19) dynamics. The stochastic differential equations model are expressed using the Ito's formula. The Environmental stochasticity in this dynamical model is presented via parameters disturbance which is the standard method in the stochastic differential equations(SDEs) in the population modeling. We than prove that this model decided in this paper have a unique global positive solution because this is fundamental in any population dynamics model. The main aim of this paper, we formulate the interaction of coronavirus COVID-19 with host cells and presented the conditions required in order to the COVID-19 to die out. And this results also illustrated by computer simulation.

Keywords: COVID-19 dynamics, Angiotensin converting enzyme 2, Stochastic differential equation, Ito's formula, Computer simulation.

1. Introduction
Comprehension and studying behavior of novel coronavirus (COVID-19) has become very important because of to the large global health burden. Until 25/10/2020 almost (42512186) became newly infected with COVID-19, about (1147301) they have died since the first cases of infection appeared of COVID-19, where specified in Wuhan City, China, in December 2019.e.g., see[1] and [2]. The world data indicate that number of peoples infected by coronavirus go on to increase in spite of the fact that there are real preventive strategies. No country of the whole world have been safe from coronavirus. The pandemic is still very dynamic, increasing and varying personality as the COVID-19 is using multiple and new opportunities used for the purpose of entering the human body cells.(e.g., see[3-7]. Actually coronavirus is infiltrates nearly every aspect of life, damages the global economy, and changes both the manmade and natural environments. COVID-19 spreads by attacking the Respiratory system, as the lung being the prime site of pathology in patients with coronavirus ,the heart scores second as a target organ of sever acute respiratory syndrome COVID-19 or( SARS-COV-2). The reason may be an abundance of Angiotensin converting enzyme 2 (ACE-2) receptor in the heart, which helps the virus
get easily internalized into the cells [4,5], e.g., see Fig. 1. When the COVID-19 enters the human body contagion in this way the Coronavirus multiplies and spreads, and the severity of coronavirus infection depends on several factors, for example age, the virulence level of the individual strain of virus and chronic diseases such as diabetes, blood pressure and heart disease may affect the rate and severity of disease development. The cells with Angiotensin converting enzyme 2 than the receptors at the site of COVID-19 infectious carrier and viral replication begins within them. In this article we will rewriting the stochastic differential equations model for study and modeling the interaction of coronavirus COVID-19 with host cells [6,7]. The rest of this article is structured as follows. Section 2 introduces the mathematical definition of the SDEs as a stochastic process, Brownian motion, The exponentially stable, and the theorem about Ito's formula [8]. Section 3 describes A stochastic Differential Equations model for internal coronavirus (COVID-19) dynamics. In section 4 we prove that there exist of unique positive solution e.g., see [9,10 and 11]. In section 5 we present the conditions required in order to coronavirus COVID-19 to die out and that the person carrying the Coronavirus (COVID-19) has been cured. The Computer simulation present in section 6. Finally, section 7 is devote to the conclusion part.

Figure 1. (ACE-2) acts as the receptor for the (COVID-19) and allows it to infect the cells

2. Basic Concept of the Stochastic Differential Equations (SDEs)
In this section, mathematical definitions of SDEs are described. Additionally, it is better to explain some theorems that we use in this work.

Definition (2.1)
A stochastic process W(t) is defined as a family of random variables X(t, ω) of the variables t ∈ T and ω ∈ Ω on a common the Probability space (Ω, A, P).

Definition (2.2)
A stochastic process W(t), t ∈ [0, ∞] is called Brownian motion if the following conditions are satisfied:
1. P(W(0)=0)=1.
2. For 0 < t_0 < t_1 < ... < t_n, the increments W(t_1) - W(t_0), W(t_n) - W(t_{n-1}), all of them are independent.
3. If (t) and (h > 0) are arbitrary constant and W(t + h) - W(t) have the Gaussian distribution with variance h and mean zero. The Wiener process have the properties which is,
$E(w(t))=0$ and $\text{Var}(W(t) - W(s)) = t - s$, for any $0 \leq s \leq t$. Thus, they have stationary increments.

**Definition (2.3)**

The stochastic differential equations (SDEs) takes the form.

$$dX(t)/dt = f(X(t),t) + g(X(t),t)W(t), \quad X(t_0) = X_0, \quad t \in [t_0,T] , T > 0,$$

(2.1)

where $f(X(t),t)$ is the drift coefficients function, $g(X(t),t)$ is defined as a diffusion coefficient function. The solution to SDEs in equation (2.3) takes the form in the integral formula as follows.

$$X(t) = X_0 + \int_{t_0}^{t} f(X(s),s)ds + \int_{t_0}^{t} g(X(s),s)dW(s), \quad t \in [t_0,T],$$

(2.2)

where the first integral on the right of equation (2.4) is Riemann integral, and the second is stochastic integral.

**Theorem (2.1) (Ito's formula)**

Suppose that $X_t$ has SDE:

$$dX_t = f(X_t,t)dt + g(X_t,t)dw_t,$$

(2.3)

for $f,g \in C^{1,2} (J \times R, R)$ assume $F:J \times R \to R$ is continuous and has $\frac{\partial F}{\partial t}, \frac{\partial F}{\partial X_t}$ and $\frac{\partial^2 F}{\partial X^2_t}$ exist and are continuous, set $F = F(X_t,t)$, then $F$ has the stochastic differential.

$$dF = \frac{\partial F}{\partial t} dt + \frac{\partial F}{\partial X_t} dX_t + \frac{1}{2} \frac{\partial^2 F}{\partial X^2_t} g^2 dt,$$

$$dF(X_t,t) = \left[ \frac{\partial F}{\partial t} + \frac{\partial F}{\partial X_t} f(t) + \frac{1}{2} \frac{\partial^2 F}{\partial X^2_t} g^2 \right] dt + \frac{\partial F}{\partial X_t} gdw_t,$$

(2.4)

The last equation (2.6), is called Ito’s formula or Ito’s chain rule. Equation (2.5) is sufficiently general to denote an m-dimensional d-Brownian motion system. In the equation, $W_t = (W_{t,1}, W_{t,2}, \ldots, W_{t,d})^T$ is an ad-dimensional vector consists of $d$ independent Wiener processes and $g(X_t,t)$ is an $m \times d$ matrix.

If we labeled the columns of $g(X_t,t)$ to be as $g_1(X_t,t), g_2(X_t,t), \ldots, g_d(X_t,t)$; then, the m-dimensional d-Brownian motion system is written as, $dX_t = f(X_t,t)dt + \sum_{j=1}^{d} g_j(X_t,t)dw_t^j$. Here, the component-by-component of the Ito’s formula can be $K = 1, 2, \ldots, m$.

$$dF_k(X_t,t) = \frac{\partial F_k}{\partial t} + \sum_{i=1}^{m} f_i \frac{\partial F_k}{\partial X_i} + \frac{1}{2} \sum_{i,j=1}^{m} g_{ij} \frac{\partial^2 F_k}{\partial X_i \partial X_j} dt + \sum_{i=1}^{d} g_i \frac{\partial F_k}{\partial X_t} dW_t^i,$$

(2.5)

**Definition (2.4).**[8] The exact solution of equation (2.5). It will be exponentially stable if the following inequality satisfied.

$$\lim_{t \to \infty} \frac{1}{t} \log|x(t, t_0, x_0)| < 0,$$

(2.5)

For any $x_0 \in R^d$. 
3. A stochastic Differential Equations (SDEs) Model derivation

In this paper, we suggest a vaccine that works to reduce the interaction between infectious virus particles and healthy cells. As well as a vaccine that does not form virus particles in a proper way, and this leads to new viruses that are weak, not capable on reproduction. The rate at which viruses are cleared may be affected by various of factors including binding and entry into the host cells. Because mortality rates from virus particles and cells affected via several complex biological phenomena for this reason we think there is stochastic in this death average. This will give us important incentive to think that's what we can insertion environmental stochastic in the a kill rate of infected cells which contain receptors angiotensin converting enzyme 2 (ACE-2). So the stochastic differential equations system will be as follows.

\[
\begin{align*}
\frac{dv_1(t)}{dt} &= (\xi - \eta v_1(t) - (1-r)\alpha v_1(t)v_3(t))dt - \sigma_1 v_1(t)dW_1(t), \\
\frac{dv_2(t)}{dt} &= ((1-r)\alpha v_1(t)v_3(t) - bv_2(t))dt - \sigma_2 v_2(t)dW_2(t), \\
\frac{dv_3(t)}{dt} &= ((1-n)Mb v_2(t) - \mu v_3(t) - (1-r)\alpha v_1(t)v_3(t))dt - \sigma_3 v_3(t)dW_3(t),
\end{align*}
\]  

(3.1) (3.2) (3.3)

At this system \(W_1(t)\) and \(W_2(t)\) are independent standard wiener process or Brownian motion. If there is stochastic in parameters for example coronavirus (COVID-19) death average, it is a normal method to present environmental stochastic into the parameters in this system. You can note the intensity of the noise \(\sigma\) and the Wiener process \(W(t)\) are similar for infected and uninfected ACE-2 cells, but dissimilar for the ACE-2 host cells and COVID-19. Since although biological agents that influence all deaths average of uninfected and infected ACE-2 host cells may be probable to be very alike different biological agents affecting Coronavirus particles and ACE-2 cells. So, though there is no detailed biological data, the noise intensity is likely \(\sigma\) and the wiener process \(W(t)\) are different for infected and uninfected ACE-2 cells. As a first simplified approximation, it is reasonable to assume that these are the same. Since coronavirus particles and angiotensin converting enzyme 2 cells are abundant more dissimilar biological units it appears much extra probable that \(\sigma\) and \(W(t)\) are dissimilar among the infectious coronavirus atoms and ACE-2 cells. You should note the state with no casualty virus particles and no infected cells can described as follows \((v_1, v_2, v_3) = (\xi/\eta, 0, 0)\) it is fixed point in the deterministic model but not for random differential equations (SDEs) model. In the SDEs model the last two equations \((v_2, v_3) = (0,0)\) are still a stochastic stability, but state is different for the first equation (3.1) of the procedure which we will see advanced differs randomly about the value \(\xi/\eta\) .
### Table 1: Model States and Model parameters

| Parameter   | Description                                                                 |
|-------------|-----------------------------------------------------------------------------|
| \( \nu_1(t) \) | Represent target cells                                                      |
| \( \nu_2(t) \) | Represent infected cells                                                     |
| \( \nu_3(t) \) | Represent concentration of Coronavirus COVID-19 particles                   |
| \( (1 - r) \) | Represent the effect of the reverse transcriptase inhibitor vaccine          |
| \( (1 - n) \) | Represent protease inhibitor vaccine effect                                  |
| \( \xi \)    | It represents the production rate of uninfected cells per unit time          |
| \( \eta \)   | It represents the individual mortality rate from uninfected cells            |
| \( \alpha \) | It represents the coefficient of correlation and interaction between infectious coronavirus particles and uninfected cells |
| \( b \)      | Represents the individual mortality ratio of infected host cells             |
| \( M \)      | It represents the total number of infectious coronavirus that an infected cell would produce with no suitable vaccine. |
| \( \mu \)    | Represents the death rate per capita from infectious virus particles         |
| \( \sigma_1 \) and \( \sigma_2 \) | Represent a parameter used to model the stochastic or randomness in the evolution, which will cause local deviation from the typical (exponential) |
| \( W_1(t) \) and \( W_2(t) \) | Represent an increment Weiner process which models the randomness in the evolution |

### 4. Existence of Unique Nonnegative Solution

To show this model make sense and can be applied to explain the life cycle of the virus, we must show that, A stochastic differential equations model does not only have a single global solution, but has a unique non-passive universal solution. In this article we suppose \((\Omega, \mathcal{F}, \{\mathcal{F}_t\}_{t \geq 0}, \mathbb{P})\) to be possibility space complete with a filtration\(\mathcal{F}_t\) satisfying the normal condition (i.e., it is growing and right uninterrupted whereas \(\mathcal{F}_0\) covers all \(\mathbb{P}\)-null sets), and suppose \(W(t)\) be a constant Wiener procedure defined on the possibility space. Through this paper \(a \wedge b\) denote \(\min(a,b)\) and \(a \vee b\) denote \(\max(a,b)\).

Also suppose \(R^+_3 = \{v \in \mathbb{R}^3: v_i > 0, \text{ for all } 1 \leq i \leq 3\}\) and let \(v(t) = (v_1(t), v_2(t) \text{ and } v_3(t))\)

Let us give this lemma, before proving the main theorem

**Lemma 4.1.** The next inequality validated

\[
\mu \leq 2(\mu + 1 - \log(\mu)) - (4 - 2 \log 2), \forall \mu > 0.
\]

**Proof:** let \(\mu > 0\),

\[
f(\mu) = \mu + 2 - 2 \log \mu.
\]

\(f(\mu)\) has the minimum at \(\mu = 2\). This completes the proof

**Theorem 4.1.** Let \(0 < r, n < 1\) and assume \(\eta, \xi, b, \mu, M\) and \(\alpha\) are nonnegative constant numbers. So for all initial value \(v_0 \in R^+_3\), there exist a unique nonnegative solution \(v(t)\) to equations, \((3.1)-(3.3)\) on \(t \geq 0\) and this solution will stay in \(R^+_3\) with possibility one (i.e., \(v(t) \in R^+_3\) for all \(t \geq 0\). It is nearly certain),
proof.
Because of the coefficients numbers of the domestic Lipchitz equation is continuous, for all assumed initial value \(v_0 \in R^3_{++}\), there exist a single local solution \(v(t)\) on \(t \in [0, \tau_e)\), where \(\tau_e\) is the bang time \(8\). To proof this solution is universal, we must proof that \(\tau_e = \infty\), suppose \(k_0 \geq 0\) be big enough so that each element of \(v_0\) lies inside the set \([1/k_0, k_0]\). For each number \(k \geq k_0\), if we presented the stopping time as,

\[ \tau_k = \inf \{t \in [0, \tau_e): v_i(t) \notin \left(\frac{1}{k}, k \right) \text{ for some } i \text{ and } 1 \leq i \leq 3 \} \]

In this article we let \(\inf \emptyset = \infty\) (where \(\emptyset\) represent empty set), obviously is growing when \(k \to \infty\. Let \(\tau_{\infty} = \lim_{k \to \infty} \tau_k\), whence \(\tau_{\infty} \leq \tau_e \text{ a.s.}\).

Can show that \(\tau_{\infty} = \infty\). Then \(\tau_e = \infty\) and \(v(t) \in R^3_{++}\). For all \(t \geq 0\),

on other words this means that, to finish the proof we will show that \(\tau_{\infty} = \infty\). If this declaration is not true, so there is a couple of constant \(T > 0\) and \(\epsilon \in (0,1)\) so that \(P(\tau_{\infty} \leq T) > \epsilon\).

So there is integer number \(k_1 \geq k_0\) s.t

\[ P(\tau_k \leq T) \geq \epsilon \text{ for any } k \geq k_1 \quad (4.1) \]

Define a \(C^2\) function \(\Psi: R^3_{++} \to R_{++}\) via,

\[ \Psi(x) = \sum_{i=1}^{3} \left[v_i + 1 - \log(v_i) \right], \]

The positive of this function can be discovery from \(\mu + 1 - \log(\mu) \geq 0, \forall \mu > 0\). If we use Ito's formula we find:

\[ d\Psi(v(t)) = \left[1 - \frac{1}{v_1(t)}\right] \left(\xi - \eta v_1(t) - (1 - r)\alpha v_1(t)v_3(t)\right) \]

\[ + \left[1 - \frac{1}{v_2(t)}\right] \left((1 - r)\alpha v_1(t)v_3(t) - b v_2(t)\right) \]

\[ + \left[1 - \frac{1}{v_3(t)}\right] \left((1 - n)Mb v_2(t) - \mu x_3(t) - (1 - r)\alpha v_1(t)v_3(t) + \sigma_1^2 + \frac{\sigma_2^2}{2}\right) dt \]

\[ + \sigma_1 (2 - v_1(t) - v_2(t)) dW_1(t) + \sigma_2 (1 - v_3(t)) dW_2(t) \]
\[
\begin{aligned}
&= \left[ \xi - \eta v_1(t) - (1 - r)\alpha v_1(t) v_3(t) + (1 - r)\alpha v_1(t) v_3(t) - b v_2(t) + (1 - n)M b v_2(t) - \mu v_3(t) \\
& \quad - (1 - r)\alpha v_1(t) v_3(t) - \frac{\xi}{v_1(t)} + \eta + (1 - r)\alpha v_3(t) - \frac{(1 - r)}{v_2(t)} \alpha v_1(t) v_3(t) + b \\
& \quad - \frac{(1 - n)M b v_2(t)}{v_3(t)} + \mu + (1 - r)\alpha v_1(t) + \sigma_1^2 + \frac{\sigma_2^2}{2} \right] dt \\
& \quad + \sigma_1 (2 - v_1(t) - v_2(t)) dW_1(t) + \sigma_2 (1 - v_3(t)) dW_2(t).
\end{aligned}
\]

Hence

\[
d\Psi(v(t)) \leq \left[ \xi + \eta + b + \mu + \sigma_1^2 + \frac{\sigma_2^2}{2} + (1 - n)M b v_2(t) + (1 - r)\alpha v_3(t) + (1 - r)\alpha v_1(t) \right] dt \\
\quad + \sigma_1 (2 - v_1(t) - v_2(t)) dW_1(t) + \sigma_2 (1 - v_3(t)) dW_2(t).
\]

Assume

and \( s_2 = 2(1 - n)M b + 2(1 - r)\alpha, s_1 = \xi + \eta + b + \mu + \sigma_1^2 + \frac{\sigma_2^2}{2} \)

By using Lemma 4.1,

\( v_1 \leq 2n(v_1 + 1 - \log(v_1)) \) so \((1 - n)M b v_2(t) + (1 - r)\alpha v_3(t) + (1 - r)\alpha v_2(t) \leq s_2 \Psi(v), \)

Therefore

\[
d\Psi(v(t)) \leq (s_1 + s_2 \Psi(v)) dt + \sigma_1 (2 - v_1(t) - v_2(t)) dW_1(t) + \sigma_2 (1 - v_3(t)) dW_2(t).
\]

Subsequently

\[
d\Psi(v(t)) \leq s_3 (1 + \Psi(v)) + \sigma_1 (2 - v_1(t) - v_2(t)) dW_1(t) + \sigma_2 (1 - v_3(t)) dW_2(t),
\]

Wherever \( s_3 = \max(s_1, s_2) \). So if \( t_1 \leq T \),

\[
\int_0^{T \wedge t_1} d\Psi(v(t)) \leq \int_0^{T \wedge t_1} s_3 (1 + \Psi(v(t))) dt + \int_0^{T \wedge t_1} \sigma_1 (2 - v_1(t) - v_2(t)) dW_1(t) \\
\quad + \int_0^{T \wedge t_1} \sigma_2 (1 - v_3(t)) dW_2(t).
\]

This means that
\[ E\Psi(v(\tau_k \wedge t_1)) \leq \Psi(v_0) \\
+ E \int_0^{\tau_k \wedge t_1} s_3 \left(1 + \Psi(v(t))\right) dt \leq \Psi(v_0) + s_3 t_1 + s_3 E \int_0^{\tau_k \wedge t_1} \Psi(v(t))dt \\
\leq \Psi(v_0) + s_3 T \\
+ s_3 E \int_0^{t_1} \Psi(v(\tau_k \wedge t_1)) dt = \Psi(v_0) + s_3 T + s_3 \int_0^{t_1} E\Psi(v(\tau_k \wedge t_1)) dt. \]

By the Gromwell inequality,
\[ E\Psi(v(\tau_k \wedge T)) \leq s_4, \]

Where \[ s_4 = (\Psi(v_0) + s_3 T)e^{s_3 T}. \] (4.2)

let set \( \Omega_k = \{\tau_k \leq T\} \) for \( k \geq k_1 \) and by equation (4.1), \( P(\Omega_k) \geq \epsilon. \)

So, for any \( \omega \in \Omega_k \), there is \( i \) and \( (1 \leq i \leq 3) \) s.t \( v_i(\tau_k, \omega) \) equals \( k \) or \( \frac{1}{k} \), and henceforth \( \Psi(v(\tau_k, \omega)) \) is more than the minimum of \( k + 1 - \log(k) \)

And \( \left(\frac{1}{k} + 1 - \log \frac{1}{k}\right) = \left(\frac{1}{k}\right) + 1 + \log(k). \)

Consequently
\[ \Psi(v(\tau_k, \omega)) \geq \left[ k + 1 - \log(k) \right] \wedge \left[ \left(\frac{1}{k}\right) + 1 + \log(k) \right], \]

So from equations (4.1) and (4.2) we get
\[ s_4 \geq E[I_{\Omega_k}(\omega)\Psi(v(\tau_k, \omega))] \geq \epsilon \left[ k + 1 - \log(k) \wedge \left[ \left(\frac{1}{k}\right) + 1 + \log(k) \right] \right]. \]

Where \( I_{\Omega_k} \) is the pointer function of \( \Omega_k \), letting \( k \to \infty \) we get the contradiction \( \infty > s_4 = \infty \), so we must have \( \tau_\infty = \infty. \)

5. Asymptotic behavior

In the study the dynamical behavior of for Internal COVID-19 Dynamics, it is important for us to study and consider the conditions required in order to the coronavirus COVID-19 to die out, i.e. when \( v_2(t) \to 0 \) and \( v_3(t) \to 0 \), as \( t \to \infty. \)

**Theorem 5.1.** If the following two conditions satisfied

1. \[ 2[(1 - n)Mb - b] - \sigma_1^2 < 0, \]
2. \[ \mu \left[ (1 - n)Mb - b \right] - \mu \left[ \sigma_1^2 + 2\mu(\sigma_1^2 - 2[(1 - n)Mb - b]) \right] < 0, \]

are almost certain that is exponentially stable meaning that \( v_2(t) \) and \( v_3(t) \) will goes \( v_2(t) \) and \( v_3(t) \) to their fixed point zero exponentially with probability 1.
\textbf{Proof}. Depending on the equations (3.2) and (3.3) consider \(d(v_2(t) + v_3(t))\)

\[ d(v_2(t) + v_3(t)) = (1-r)\alpha v_1(t)v_3(t) - bv_2(t) + (1-n)Mb v_2(t) - \mu v_3(t) - (1-r)\alpha v_1(t)v_3(t) dt - \sigma_1 v_2(t)dW_1(t) - \sigma_2 v_3(t)dW_2(t) \]

Let \(v = (v_2, v_3)\) and \(\Psi(v) = \log(v_2 + v_3)\) for \(v_2, v_3 \in (0, \infty)\).

By using Ito's formula we find

\[ d\Psi(v(t)) = \left( \frac{(1-n)Mb v_2(t)}{v_2(t) + v_3(t)} - \frac{b v_2(t)}{v_2(t) + v_3(t)} - \frac{\mu v_3(t)}{v_2(t) + v_3(t)} - \frac{1}{2} \frac{\sigma_1^2 v_2^2(t)}{(v_2(t) + v_3(t))^2} \right) dt - \frac{\sigma_1 v_2(t)}{v_2(t) + v_3(t)} dW_1(t) - \frac{\sigma_2 v_3(t)}{v_2(t) + v_3(t)} dW_2(t). \]

After simplifying we find

\[ d\Psi(v(t)) = \frac{1}{2(v_2(t) + v_3(t))^2} \left( 2(v_2(t) + v_3(t))((1-n)Mb v_2(t) - bv_2(t) - \mu v_3(t)) - \sigma_1^2 v_2^2(t) - \sigma_2^2 v_3^2(t) \right) dt - \frac{\sigma_1 v_2(t)}{v_2(t) + v_3(t)} dW_1(t) - \frac{\sigma_2 v_3(t)}{v_2(t) + v_3(t)} dW_2(t) \]

We can rewrite the term

\[ (2(v_2(t) + v_3(t))((1-n)Mb v_2(t) - bv_2(t) - \mu v_3(t)) - \sigma_1^2 v_2^2(t) - \sigma_2^2 v_3^2(t) \]

In the following method

\[ \begin{pmatrix} v_2(t) \\ v_3(t) \end{pmatrix} \begin{pmatrix} 2((1-n)Mb - b) - \sigma_1^2 \\ ((1-n)Mb - b) - \mu \end{pmatrix} \begin{pmatrix} v_2(t) \\ v_3(t) \end{pmatrix} \]

So we can write \(d\Psi(v(t))\) as follows

\[ d\Psi(v(t)) = \frac{1}{2(v_2(t) + v_3(t))^2} \left( v_2(t) \begin{pmatrix} 2((1-n)Mb - b) - \sigma_1^2 \\ ((1-n)Mb - b) - \mu \end{pmatrix} \begin{pmatrix} v_2(t) \\ v_3(t) \end{pmatrix} \right) dt \]

\[ - \frac{\sigma_3 v_2(t)}{v_2(t) + v_3(t)} dW_1(t) - \frac{\sigma_2 v_3(t)}{v_2(t) + v_3(t)} dW_2(t) \]

let us consider the matrix

\[ \begin{pmatrix} 2((1-n)Mb - b) - \sigma_1^2 \\ ((1-n)Mb - b) - \mu \end{pmatrix} \begin{pmatrix} v_2(t) \\ v_3(t) \end{pmatrix} \]

Because of the last matrix is negative – determinant with main (negative) eigenvalue \(\xi_{\max}\) so:

\[ \begin{pmatrix} v_2(t) \\ v_3(t) \end{pmatrix} \begin{pmatrix} 2((1-n)Mb - b) - \sigma_1^2 \\ ((1-n)Mb - b) - \mu \end{pmatrix} \begin{pmatrix} v_2(t) \\ v_3(t) \end{pmatrix} \]

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\[ \leq \xi_{\text{max}} \left( v^2(t) + v^2(t) \right) = -|\xi_{\text{max}}| \left( v^2(t) + v^2(t) \right) \]

Therefore

\[ d\Psi(v(t)) \leq \left( -|\xi_{\text{max}}| \frac{1}{2(v_2(t) + v_3(t))^2} \left( v^2(t) + v^2(t) \right) \right) dt - \frac{\sigma_1 v_2(t)}{v_2(t) + v_3(t)} dW_1(t) - \frac{\sigma_2 v_3(t)}{v_2(t) + v_3(t)} dW_2(t), \]

(5.1)

Since \( \frac{(v^2(t) + v^2(t))}{2} \geq v_2v_3 \) we can write \(- (v^2(t) + v^2(t)) \leq - (v_2v_3)^2 \).

When we substituting this in inequality (5.1) we find

\[ d\Psi(v(t)) \leq - \frac{1}{4} |\xi_{\text{max}}| dt - \frac{\sigma_1 v_2(t)}{v_2(t) + v_3(t)} dW_1(t) - \frac{\sigma_2 v_3(t)}{v_2(t) + v_3(t)} dW_2(t), \]

\[ d(\log(v_2(t) + v_3(t)) \leq - \frac{1}{4} |\xi_{\text{max}}| dt - \frac{\sigma_1 v_2(t)}{v_2(t) + v_3(t)} dW_1(t) - \frac{\sigma_2 v_3(t)}{v_2(t) + v_3(t)} dW_2(t), \]

If we integrate the above inequality and by using the large number theorem [8], we have

\[ \lim_{t \to \infty} \sup_t \frac{1}{t} |W_i(t)| = 0 \quad \text{for } i = 1, 2 \]

We get

\[ \lim_{t \to \infty} \sup_t \frac{1}{t} \log(v_2(t) + v_3(t)) \leq - \frac{1}{4} |\xi_{\text{max}}| < 0, \]

This gives us \( v_2(t) \to 0 \) and \( v_3(t) \to 0 \), as \( t \to \infty \) This complet the proof

Note that the conditions of theorem 5.1, will always be achieved If \( \sigma_1^2 \) and \( \sigma_2^2 \) are big sufficient these situations will constantly be verified. This exciting result as it declares that if stochastic alterations are big so coronavirus COVID-19 and infected cells will at all times die out, whatsoever the extra parameter values, even though \( R_0 > 0 \). We now will concentrate on \( v_1(t) \). We'll show at the end that \( v_1(t) \) is expantially stable in supply in the sense that it stabilizes about the value \( \xi/\eta \). In order to get this we will present the new stochastic procedure \( \varphi(t) \) which is can defined via its primary Condition \( \varphi(0) = v_1(0) \) and SDE

\[ d\varphi(t) = (\xi - \eta \varphi(t)) dt - \sigma_1 \varphi(t) dW_1(t). \]

We will express that in the limit as \( t \) becomes great \( v_1(t) \) can be approximated by \( \varphi(t) \) so to proof this we will present another stochastic function \( Z_e(t) \) which is presented by the initial condition \( Z_e(t) = v_1(0) \) and SDE

\[ dZ_e(t) = (\xi - (\eta + \epsilon) Z_e(t)) dt - \sigma_1 Z_e(t) dW_1(t), \]

(5.2)
\textbf{Theorem 5.2.} If the following two conditions satisfied
\begin{enumerate}
\item \[2[(1 - n)Mb - b] - \sigma_1^2 < 0, \quad \text{and} \]
\item \[\left(\frac{(1 - n)Mb - b)}{\mu} \right)^2 < \left(\frac{\sigma_2^2 + 2\mu}{\sigma_1^2} - 2[(1 - n)Mb - b]\right)\]
\end{enumerate}
Then
\[\lim_{t \to \infty} (\varphi(t) - v_1(t)) = 0 \text{ in probability}.
\]

\section{Computer Simulations}
In this section, we will try to support our analytical resulting numerical simulation produced in theorems (5.1) and (5.2). We find by the theoretically results the coronavirus particles and the infected cells are stable and goes to zero if the two conditions submitted in theorem 5.1 are satisfied. Also we note that we can estimated $v_1(t)$ via $\varphi(t)$ where $\varphi(t)$ is the mean returning process. The computer simulation programs have been written in MATLAB by using Euler Maruyama method (EM) our results were confirmed by running them frequently and extensively examination the result.

\textbf{Example (6.1).} Let us choose the parameter values as follows $\alpha = 1 \times 10^{-9} \text{day}^{-1}$, $\xi = 10^6 \text{day}^{-1}$, $M = 1 \text{ per cell}$, $r = 0.4$, $n = 0.5$, $b = 0.5 \text{day}^{-1}$, $\eta = 0.1 \text{day}^{-1}$, and $\mu = 0.01 \text{day}^{-1}$. The initial values were $v_1(0) = 10000dm^{-1}$, $v_2(0) = 10000dm^{-1}$ and $v_3(0) = 10000dm^{-1}$ if $\sigma_1 = \sigma_2 = 1$.

We can easily prove conditions presented by theorem 5.1 are achieved as follows

The first law
\[2[(1 - 0.5)(0.5) - 0.5] - 1 = -1.5 < 0,\]

The second law
\[\left(\frac{(1 - n)Mb - b)}{\mu} \right)^2 < \left(\frac{\sigma_2^2 + 2\mu}{\sigma_1^2} - 2[(1 - n)Mb - b]\right)\]

Since $\left(\frac{(1 - n)Mb - b)}{\mu} \right)^2 = 0.076 &\left(\frac{\sigma_2^2 + 2\mu}{\sigma_1^2} - 2[(1 - n)Mb - b]\right) = 0.51$

If we take the equation (3.2)
\[\frac{dv_2(t)}{dt} = \left((1 - r)\alpha v_1(t)v_2(t) - bv_2(t)\right)dt - \sigma_1 v_2(t)dW_1(t)\]

So by using Ito's formula and substitution the parameter values, we have the solution of (equation .3.2) as
\[\exp(-0.4) t \quad v_2(t) = 10000\]

So the infected cells ($v_2(t)$) tend to zero exponentially as $t \to \infty$.

The computer simulation in Fig. 2, by using Euler Maruyama method (EM), support these results clearly. We create an algorithm for plotting Figure 2, as follows

\begin{verbatim}
rate = 0.1;
sigma = 1;
nPeriods = 63;
dt = 1 / 10;
T = nPeriods * dt;
obj = gbm(rate, sigma, 'StartState', 10000);
\end{verbatim}
\[ [X, T] = 
\text{obj.simBySolution}(n\text{Periods}, \text{'DeltaTime'}, dt, 
\text{'nTrials'}, 2, \text{'Antithetic'}, \text{true}); 
\]

\[
\text{plot}(T, X(:,:,2), \text{'red'}) 
\text{xlabel('Time (days)'), ylabel('infected cells')}
\text{legend('Stochastic')} 
\]

**Figure. 2.** Stochastic simulation the infected cells tend to zero exponentially as \( t \to \infty \)

If we take the equation (3.3)

\[
\frac{dv_3(t)}{dt} = \left((1 - n)Mb_v(t) - \mu v_3(t) - (1 - r)\alpha v_1(t)v_3(t)\right)dt - \sigma_2 v_3(t)dW_2(t), 
\]

Also by using Ito’s formula and substitution the parameter values, we have the solution of equation (3.3) as

\[
v_3(t) = 10000 \exp(-98.6) t. 
\]

So the virus particles \((v_3(t))\) tend to zero exponentially as \( t \to \infty \).

The computer simulation in Figure. 3, by using Euler Maruyama method (EM), support these result clearly.
If we take the equation (3.1)
\[
\frac{dν_1(t)}{dt} = (ξ - ην_1(t) - (1 - r)αν_1(t)ν_3(t))dt - σ_1ν_1(t)dW_1(t)
\]
So by using Ito's formula and substitution the parameter values, we have the solution of Equation (3.1) as follows,
\[
ν_1(t) = 10000 \exp(998.9)t
\]
So, the uninfected cells $ν_1(t)$ will not tend to zero exponentially when $t \to \infty$. This means that the person carrying the Coronavirus (COVID-19) has been cured. The computer simulation in Figure 4, support these result clearly.

Figure 3. Stochastic simulation the virus particles tend to zero exponentially as $t \to \infty$

Figure 4. Stochastic simulation the uninfected cells not tend to zero exponentially as $t \to \infty$
7. Conclusions
Through this article, we have studying the stochastic differential equations for describing the dynamics of coronavirus (COVID-19). We demonstrated a non-negative of the model solution. Also in this paper, we have shown that overcoming coronavirus disease is possible, if a vaccine with the same specifications as the one presented in section 3 is available and stochastic variance $\sigma_1^2$ and $\sigma_2^2$ are big sufficient this gives us $v_2(t) \to 0$ and $v_3(t) \to 0$, as $t \to \infty$, regardless of the value of other parameters. In our work we prove that the stochastic differential equation model provide extra choice to model COVID-19 dynamics, via repeating the result from deterministic state and optimizing specific of it, we showed the stochastic differential equations model adds extra dimension to the COVID-19 dynamics. It gives the dissimilar viewpoint to this specific problem and gives academics new path that they can take in their future research.

References
[1] Coronavirus world meter website. https://www.worldometers.info/coronavirus
[2] World Health organization, coronavirus disease (COVID-19). www.who.int/emergencies/diseases/novel-coronavirus-2019.
[3] Ni W, Yang X, Yang D and Bao J. 2020. Role of angiotensin–Converting enzyme 2 (ACE2) in COVID-19. Ni et al. Critical Care 24(422).
[4] Davidson AM, Wysocki J and Battle D. 2020. Interaction of SARS-CoV-2 and Other Coronavirus With ACE (Angiotensin-Converting Enzyme)-2 as Their Main Receptor. Hypertension. 76(5).
[5] Kai H and Kai M. 2020. Interactions of coronaviruses with ACE2, angiotensin II, and RAS Inhibitors—lessons from available evidence and insights into COVID-19. Hypertension Research 43(1):648–654.
[6] Samavati L and Uhal BD. 2020. ACE2 Much More Than Just a Receptor for SARS-COV-2. Front. Cell. Infect. Microbiol. 10(317).
[7] Monteil V, Kwon H, Prado P and Hagelkrus A. 2020. Inhibition of SARS-CoV-2 infections in engineered human tissues using clinical-grade soluble human ACE2. Journal pre-proof. DOI: 10.1016/j.cell.2020.04.004.
[8] Mao X. 1997. Stochastic differential Equations and Application, Horwood Publishing Limited Chi Chester.
[9] Bonhoeffer S, May RM and Nowak MA. 1997. Virus dynamics and drug therapy. Proc. Natl. Acad. Sci. USA 94.
[10] Mascio DM, Ribeiro R and Markowitz. 2004. Modeling the long-term control of viremi HIV-1 infected patients treated with antiretroviral therapy. Mathematical Biosciences. doi:10.1016/j.mbs.2003.08.003.
[11] Ding A and Wu H. 1999, Relationships between Antiviral Treatment Effects and Biphasic Viral Decay Rates in Modeling HIV Dynamics, Math. Biosci. 160(1). https://doi.org/10.1016/S0025-5564(99)00021-8.