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Comparison Between Deterministic and Stochastic Model for Interaction (COVID-19) With Host Cells in Humans

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Abstract:

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In this paper, the deterministic and the stochastic models are proposed to study the interaction of the Coronavirus (COVID-19) with host cells inside the human body. In the deterministic model, the value of the basic reproduction number R_0 determines the persistence or extinction of the COVID-19. If $R_0 < 1$, one infected cell will transmit the virus to less than one cell, as a result, the person carrying the Coronavirus will get rid of the disease .If $R_0 > 1$, the infected cell will be able to infect all cells that contain ACE receptors. The stochastic model proves that if $\alpha_1 \& \alpha_2$ are sufficiently large then $\alpha_1 \& \alpha_2$ maybe give us ultimate disease extinction although $R_0 > 1$, and this facts also proved by computer simulation.

Keywords: Basic reproduction number, Computer simulation, COVID-19 dynamics, Stochastic model.

Introduction:

Coronavirus (COVID-19) is deadly and infectious which attacks and weakens cells containing the angiotensin- converting enzyme 2 (ACE-2) receptors, An example of these cells that the novel coronavirus disease attacks are the intestinal cells, myocardial cells, renal tubes, male reproductive cells, gallbladder, lungs, bronchi, and nasal mucosa. For this reason, the respiratory system is the first target organ, the heart scores second as an organ targeted by the Coronavirus. Until 13/2/2021 the total number of infected with COVID-19 around the world about (109156020) and almost (2406571) they have died since the first case by COVID-19 which appeared at the end of 2019 in Wuhan, China^{1,2}. Despite massive development in technology and medical equipment, Scientists still can't find a complete cure for the COVID-19 virus. To control the epidemic it is important to understand the dynamical behavior of COVID-19 and how its interaction with host cells in humans $^{3-5}$. Mathematical models were developed to understand the dynamics of viral infections. The large amount of scientific research done on the model of the interaction of Coronavirus with host cells in humans has been largely restricted to ordinary differential equations

(ODEs) $^{5-8}$. In our article will present a stochastic differential equations model for interaction (COVID-19) with host cells in humans. Several reasons motivated us to use stochastic differential equations models instead of deterministic equations. Real-life is random, not deterministic, especially when modeling the phenomenon of the spread of the Coronavirus for example internal COVID-19 dynamics. This is because target cells that contain ACE-2 receptors that interacting with coronavirus particles In the same environmental conditions but give different outputs. This article presents the influence of introducing stochasticity on the deterministic mathematical model. The new method in mathematical modeling gave us more accurate results than deterministic differential equations models, because employment the stochastic differential equations model many times can build up a distribution of the predicted results, such as total numbers of infected cells with the coronavirus at time t, whereas the deterministic differential equations model will introduce to us just one expected value⁹⁻¹². The paper is organized as follows. Firstly present the deterministic model and Stochastic model for interaction coronavirus with cells that contain ACE-2 receptors. Secondly, present the conditions required for persistence or extinction of COVID-19 and how the injured recovered with the virus. Then the main results present. finally, the conclusions and references are listed.

The Mathematical Model for Interaction (COVID-19) With Host Cells in Humans 1 Deterministic Model

1.Deterministic Model

Various mathematical models have been used to comprehend activity and movement coronavirus inside the human body. The simpler version includes three types: The dynamics of healthy target cells T(t), The infected cells dynamics I(t), and the dynamics of coronavirus particles C(t). They can be described by using the following set of (ODEs).

$$\frac{dT(t)}{dt} = \mathbf{G} - nT(t) - \sigma T(t)C(t), \qquad 1$$

$$\frac{dI(t)}{dt} = \sigma T(t)C(t) - aI(t), \qquad 2$$
$$\frac{dC(t)}{dC(t)} = Sol(t) - SO(t) - \sigma T(t)C(t) \qquad 2$$

$$\frac{dC(t)}{dt} = SaI(t) - \beta C(t) - \sigma T(t)C(t).$$
 3

If there is a vaccine that prevents the coronavirus particles from attaching to healthy host cells. In addition to a vaccine that prevents the compilation of the virus particles correctly and this leads to new COVID-19 that are weakly, and unable to reproduce, so the set of the deterministic model (1,2 and 3) has the following form.

$$\frac{dT(t)}{dt} = G - nT(t) - (1 - v)\sigma T(t)C(t), \qquad 4$$

$$\frac{dI(t)}{dt} = (1 - v)\sigma T(t)C(t) - aI(t), \qquad 5$$

$$\frac{dC(t)}{dt} = (1 - \gamma)SaI(t) - \beta C(t)$$
$$- (1 - \gamma)\sigma T(t)C(t)$$

 $-(1-v)\sigma T(t)C(t)$. 6 Before infection(I = 0, C = 0), uninfected target cells(T) are at the equilibrium $T_0 = G/n$.

To estimate the infection, let use the basic reproductive number denoted by (R_0) which is the total expected number of secondary infections produced by the infected cell. If $R_0 < 1$ this means any infected cell will transmission of infection to less than one cell and this give as the virus is cleared out .otherwise if $R_0 > 1$ so each infected cell produces on averages more than one new infected cells and in this case, the infection grows and the disease can invade the all cells, for model 4 – 6 the basic reproduction number will be as follows:

$$R_0 = \frac{(1-\nu)\sigma GS(1-\gamma)}{(1-\nu)\sigma GS(1-\gamma)}$$

 $\frac{n_0}{(n\beta+\sigma G(1-v))}$ **2. Stochastic model**

The clearance rate of COVID-19 particles may be affected via several important factors for example binding and entry into cells that contain receptors (ACE-2). since death rates from COVID-19

particles and target cells depend on many complex natural and biological phenomena, This made scientists believe that there is randomness in this death rate. This gives us important motivation to believe that's we can insertion the stochastic in the deaths rate of infected cells and COVID-19 particles. So the new system 4- 6 in the stochastic model will be as follows¹³⁻¹⁶.

$$dT(t) = (G - nT(t) - (1 - v)\sigma T(t)C(t))dt + \alpha_1 T(t)dW_1(t), 7 dI(t) = ((1 - v)\sigma T(t)C(t) - aI(t))dt + \alpha_1 I(t)dW_1(t) 8$$

 $+ \alpha_1 I(t) dW_1(t), \qquad 8$ $dC(t) = ((1 - \gamma) SaI(t) - \beta C(t) - (1 - \gamma) \sigma T(t) C(t)) dt + \alpha_2 C(t) dW_2(t). \qquad 9$

The parameters in the system 1 - 9 are expressed in Table 1.

Table 1. The parameters in the system 1 – 9.	
T(t)	Are cells that contain receptors ACE-
	2 or target cells
I(t)	Represent cells containing
	coronavirus particles after infection
C(t)	Represent COVID-19 particles
(1 - v)	Represent probability of vaccinated
()	cells that prevents interaction between
	coronavirus particles and target cells
$(1 - \gamma)$	Represent probability of vaccinated
$(\mathbf{I} \mathbf{\gamma})$	cells that prevents the compilation of
	· · · · · · · · · · · · · · · · · · ·
	protease inhibitor vaccine.
G	It represents the number of healthy
	cells that the body produces per unit
	of time
n	Represents the rate of death of
	uninfected cells per unit of time
σ	It represents the transmission
Ũ	coefficient of the virus into the host
	cells
а	Represents the death rate of infected
a	cells
S	Are the total number of coronavirus
3	
	produced from infected cells in the
0	absence of a vaccine
β	It represents the rate of removal of
	COVID-19 particles per unit of time
α_1 and α_2	Are parameters used to model the
	stochastic in the evolution,
$W_1(t)$ and $W_2(t)$	It represents independent standard
	Brownian motions which model the
	stochastic
ACE-2	Angiotensin-converting enzyme 2

Asymptotic behavior of the Stochastic model

To comprehend the Stochastic model behavior for interaction coronavirus with host cells in humans, it must for us study and present the conditions required for the elimination of the coronavirus, and heal the infected in the virus. i.e. when $\lim_{t\to\infty} I(t) = 0$, $\lim_{t\to\infty} C(t) = 0$, **Theorem 1**. When these conditions are satisfied (1) $2[(1-\gamma)Sa - a] - \alpha_1^2 < 0$, (2) $[((1-\gamma)Sa - a) - \beta]^2 < (\alpha_2^2 + 2\beta)(\alpha_1^2 - 2[(1-\gamma)Sa - a])$. This give $\lim_{t\to\infty} I(t) = 0$, & $\lim_{t\to\infty} C(t) = 0$, in another meaning I(t) and C(t) will goes to their fixed point exponentially with Probability 1. **Proof**. By using the Eqs 8,9 consider d(I(t) + C(t)) $d(I(t) + C(t)) = (1 - v)\sigma T(t)C(t) - aI(t) + I(t) + I(t))$

 $(1 - \gamma)SaI(t) - \beta C(t) - (1 - \nu)\sigma T(t)C(t) - \alpha_1(t) + \alpha_2 C(t) - \alpha_1 I(t) dW_1(t) - \alpha_2 C(t) dW_2(t).$ Let r = (I(t), C(t)) and $\Psi(r) = \log(I(t) + C(t))$ for $I(t), C(t) \in (0, \infty)$. Via using Ito's formula will find

$$\begin{split} d\Psi(r(t)) &= \left(\frac{(1-\gamma)SaI(t)}{I(t)+C(t)} - \frac{aI(t)}{I(t)+C(t)} \\ &- \frac{\beta C(t)}{I(t)+C(t)} - \frac{1}{2}\frac{\alpha_1^2 I^2(t)}{(I(t)+C(t))^2} \\ &- \frac{1}{2}\frac{\alpha_2^2 C^2(t)}{(I(t)+C(t))^2}\right) dt \\ &- \frac{\alpha_1 I(t)}{I(t)+C(t)} dW_1(t) \\ &- \frac{\alpha_2 C(t)}{I(t)+C(t)} dW_2(t). \end{split}$$
After simplifying will find
$$d\Psi(r(t)) &= \frac{1}{2(I(t)+C(t))^2} \left(2(I(t)+C(t))\right) \left((1-\gamma)SaI(t) - aI(t) - \beta C(t)\right) - \alpha_1^2 I^2(t) - \alpha_2^2 C^2(t)\right) dt - \frac{\alpha_1 I(t)}{I(t)+C(t)} dW_1(t) - \frac{\alpha_2 C(t)}{I(t)+C(t)} dW_2(t) \,. \end{split}$$
When rewrite the term
$$(2(I(t)+C(t)))((1-\gamma)SaI(t)-aI(t) - \beta C(t)) - \alpha_1^2 I^2(t) - \alpha_2^2 C^2(t)), \end{split}$$

In the following method

$$(I(t) \quad C(t)) \begin{pmatrix} 2((1-\gamma)Sa-a) - \alpha_1^2 & ((1-\gamma)Sa-a) - \beta \\ ((1-\gamma)Sa-a) - \beta & -2\beta - \alpha_2^2 \end{pmatrix} \begin{pmatrix} I(t) \\ C(t) \end{pmatrix},$$

So let us write $d\Psi(r(t)$ as follows
 $d\Psi(r(t)) = \frac{1}{2(I(t) + C(t))^2} \{ (I(t) \quad C(t)) \begin{pmatrix} 2((1-\gamma)Sa-a) - \alpha_1^2 & ((1-\gamma)Sa-a) - \beta \\ ((1-\gamma)Sa-a) - \beta & -2\beta - \alpha_2^2 \end{pmatrix} \begin{pmatrix} I(t) \\ C(t) \end{pmatrix} \} dt$

$$-\frac{\alpha_1 I(t)}{I(t) + C(t)} dW_1(t) - \frac{\alpha_2 C(t)}{I(t) + C(t)} dW_2(t).$$

let us consider the matrix
$$\binom{2((1-\gamma)Sa-a) - \alpha_1^2}{((1-\gamma)Sa-a) - \beta} - 2\beta - \alpha_2^2}{(1-\gamma)Sa-a) - \beta}$$

Because the above matrix is not positive – determinant with main eigenvalue is negative G_{max} so:

$$(I(t) \quad C(t)) \begin{pmatrix} 2((1-\gamma)Sa-a) - \alpha_1^2 & ((1-\gamma)Sa-a) - \beta \\ ((1-\gamma)Sa-a) - \beta & -2\beta - \alpha_2^2 \end{pmatrix} \begin{pmatrix} I(t) \\ C(t) \end{pmatrix}$$

$$\leq G_{max} (l^2(t) + C^2(t))$$

= $-|G_{max}| (l^2(t) + C^2(t)) .$

Therefore

$$d\Psi(r(t)) \leq \left(-|G_{max}| \frac{1}{2(I(t) + C(t))^2} (I^2(t) + C^2(t))\right) dt - \frac{\alpha_1 I(t)}{I(t) + C(t)} dW_1(t) - \frac{\alpha_2 C(t)}{I(t) + C(t)} dW_2(t) . \quad 10$$

Since $\frac{\left(I^{2}(t)+C^{2}(t)\right)}{2} \ge I(t)C(t) \text{ will write}$ $-\left(I^{2}(t)+C^{2}(t)\right) \le -\frac{\left(I+C\right)^{2}}{2}.$ When substituting this in inequality 10 will find $d\Psi(r(t)) \le -\frac{1}{4}|G_{max}|dt - \frac{\alpha_{1}I(t)}{I(t)+C(t)}dW_{1}(t)$ $-\frac{\alpha_{2}C(t)}{I(t)+C(t)}dW_{2}(t),$ $d(\log(I(t)+C(t)) \le -\frac{1}{4}|G_{max}|dt - \frac{\alpha_{1}I(t)}{I(t)+C(t)}dW_{1}(t) - \frac{\alpha_{2}C(t)}{I(t)+C(t)}dW_{2}(t),$ By integrate the last inequality and by use the large number theorem¹⁷, will find

$$\limsup_{t \to \infty} \frac{1}{t} |W_i(t)| = 0, \text{ for } i = 1,2$$

Will get $\limsup_{t \to \infty} \frac{1}{t} \log (I(t) + C(t)) \leq -\frac{1}{4} |G_{max}| < 0.$ This gives us

 $\lim_{t\to\infty} I(t) = 0$, and $\lim_{t\to\infty} C(t) = 0$.

So the conditions of Theorem1 will always be achieved when α_1^2 & α_2^2 are sufficiently large, then α_1^2 & α_2^2 give us ultimate disease extinction although $R_0 > 1$. We now concentrate on T(t) ,and will prove that how T(t) is expansively stable in distribution about the expected value G/n. To make this possible will present the stochastic process $\varphi(t)$ which may be defined by its primary condition $\varphi(0) = T(0)$ and (SDE)

 $d\varphi(t) = (G - n\varphi(t))dt - \alpha_1\varphi(t)dW_1(t)$

We have to show that at the end as t becomes large T(t) can be approached by $\varphi(t)$ so $\lim_{t\to\infty}(\varphi(t) - T(t) = 0$,

To proof this we'll present another function $Z_{\epsilon}(t)$ which defined by the condition $Z_{\epsilon}(t) = T(0)$ and (SDE).

$$dZ_{\epsilon}(t) = \left(G - (n + \epsilon)Z_{\epsilon}(t)\right)dt - \alpha_1 Z_{\epsilon}(t)dW_1(t).$$
 11

Theorem.2. When these two conditions are satisfied¹³.

(1) $2[(1-\gamma)Sa - a] - \alpha_1^2 < 0,$ (2) $[((1-\gamma)Sa - a) - \beta]^2 < (\alpha_2^2 + 2\beta)(\alpha_1^2 - 2[(1-\gamma)Sa - a])$. This gives $\lim_{t \to \infty} (\varphi(t) - T(t)) = 0.$

Main results:

In this part of the article, we'll prove the analytical results obtained from theories (1 and 2)by using computer simulations. Note that by the theoretically results I(t) &C(t) are exponentially stable and $\lim_{t\to\infty} I(t) = 0$, & $\lim_{t\to\infty} C(t) = 0$,

If the two conditions presented in

Theorem 1 are met, although $R_0 > 1$. Also can find the value of T(t) by $\phi(t)$ where $\phi(t)$ is the average return process . Note the computer simulation program was written using Matlab by the Euler method and the outputs were verified through run them extensively and repeatedly.

Example 1: If choose the values of the parameters as:

 $\sigma = 1 \times 10^{-12} \text{day}^{-1}, \text{G} = 10^{10} \text{day}^{-1}, \text{S} = 1 \text{ per cell}, v = 0.5, \gamma = 0.5, a = 0.6 \text{ day}^{-1}, n = 1 \text{day}^{-1}, \text{and } \beta = 1 \text{day}^{-1}. \text{ The initial values were}$ T(0) = 1000000dm⁻¹, I(0) = 1000000dm⁻¹. if $\alpha_1 = \alpha_2 = 1$

To prove the conditions presented by Theorem 1 are satisfied let us take the first condition

2[(1 - 0.5)(1)(0.6) - 0.6] - 1 = -1.6 < 0, The second condition

$$[((1-\gamma)Sa-a)-\beta]^2 < (\alpha_2^2+2\beta)(\alpha_1^2 - 2[(1-\gamma)Sa-a])$$

Since

$$\begin{split} & [((1-\gamma)Sa-a)-\beta]^2 = 1.69 \,\& (\alpha_2^2+2\beta)(\alpha_1^2-2[(1-\gamma)Sa-a]) = 4.8 \\ & R_0 = \frac{(1-v)\sigma GS(1-\gamma)}{(n\beta+\sigma G(1-v)} = \frac{0.5*0.01*0.5}{(1*1+0.01*0.5)} = \frac{0.0025}{1.005} = \\ & 0.0024876. \end{split}$$

So by substitution the strange strang

 $\alpha_1 I(t) dW_1(t)$,

and solve the resulting equation by apply Ito's formula¹⁴, which will find the stochastic solution as $I(t) = 10^6 e^{(-0.6)t}$ and the deterministic solution $I(t) = 10^6 e^{(-0.1)t}$. So the infected cells I(t) go to zero exponentially in case $t \to \infty$,

The computer simulation programs Fig. 1, by using MATLAB, support these results clearly

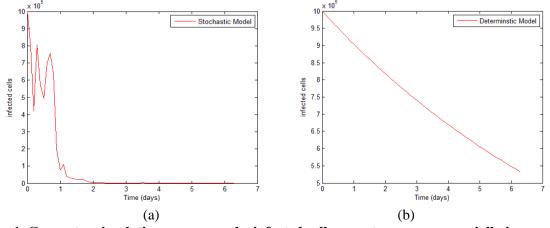


Figure 1. Computer simulation programs the infected cells goes to zero exponentially in case $t \to \infty$ (a)Stochastic model (b) Deterministic model when $R_0 < 1$,

solution C(t) =

computer

When taking the Eq.9 $dC(t) = ((1 - \gamma)SaI(t) - \beta C(t) - (1 - v)\sigma T(t)C(t))dt + \alpha_2 C(t)dW_2(t),$

Also by substituting the parameter values in Eq.9 and solve the resulting equation by using Ito's formula, will find the stochastic solution of Eq.9 as:

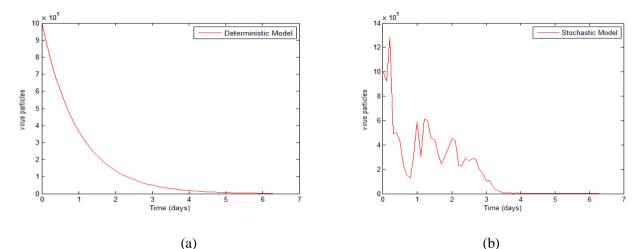


Figure 2. Computer simulation programs the virus particles goes to zero exponentially in case $t \to \infty$ (a) Deterministic model when $R_0 < 1$, (b) stochastic model

when taking the Eq.7 $dT(t) = (G - nT(t) - (1 - v)\sigma T(t)C(t))dt + \alpha_1 T(t)dW_1(t),$

So by substitution, the parameter values in Eq. 7 and by using Ito's formula¹⁴, will find the stochastic solution of Eq.7as follows, T(t) =

10⁶e^{999998t} and deterministic solution will be as :

 $C(t) = 10^6 e^{-1.42t}$ and deterministc

method (EM), support these results clearly.

exponentially in case $t \rightarrow \infty$. The

 $10^6 e^{-0.7t}$, so the virus particles C(t) goes to zero

simulation in Fig. 2, by using the Euler Maruyama

 $T(t) = 10^6 e^{999998.5t}$, So the healthy cells T(t) will not go to zero exponentially in case $t \rightarrow \infty$. This means the person with the virus has recovered. The simulation programs in Fig. 3, support these results.

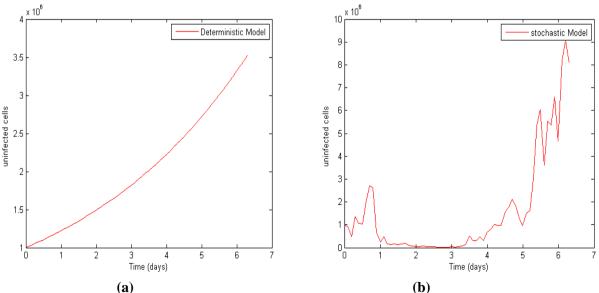
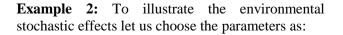


Figure 3. Computer simulation programs the healthy cells do not go to zero exponentially in case $t \to \infty(a)$ Deterministic model when $R_0 < 1$, (b) stochastic model.



 $\sigma = 1 \times 10^{-8} day^{-1}$, $G = 10^{6} day^{-1}$, S = 2 per cell, v = 0.4, $\gamma = 0.3$, $a = 0.5 day^{-1}$, $n = 0.5 day^{$

0.1day⁻¹, and $\beta = 0.01$ day⁻¹. The initial values were T(0) = 10000dm⁻¹, I(0) = 10000dm⁻¹ and C(0) = 10000dm⁻¹. if $\alpha_1 =$ 1 & $\alpha_2 = 1.2$ To prove the conditions presented by Theorem 1 are satisfied let us take the first condition 2[(0.7)(2)(0.5) - 0.5] - 1 = -0.6 < 0, The second condition [((1 - γ)Sa - a) - β]² < ($\alpha_2^2 + 2\beta$)(α_1^2

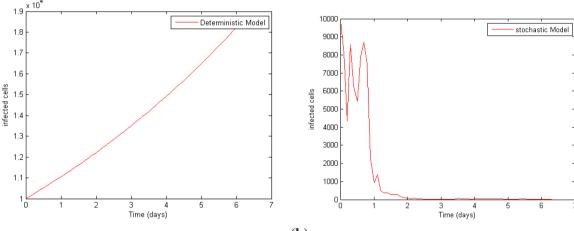
$$-2[(1-\gamma)Sa - a])$$

Since
$$\begin{split} & [\left((1-\gamma)Sa-a\right)-\beta]^2=0.0361\,\&(\alpha_2^2+2\beta)(\alpha_1^2-2[(1-\gamma)Sa-a])=0.876\\ & R_0=\frac{0.6*10^{-8}*10^6*2*0.7}{0.1*0.01+10^{-8}*10^6*0.6}=1.2>1,\\ & \text{When taking the Eq.8}: dI(t)=\left((1-\nu)\sigma T(t)C(t)-aI(t)\right)dt+\alpha_1I(t)dW_1(t), \end{split}$$

So by substitution the parameter values in Eq.8 and by using Ito's formula¹⁴, will find the stochastic solution as : $I(t) = 10^4 e^{-0.4t}$ and the deterministic solution as $I(t) = 10^4 e^{0.1t}$

So the infected cells (I(t)) go to zero exponentially in case $t \rightarrow \infty$ in stochastic model but not

in deterministic model. The simulation programs in Fig. 4, by using MATLAB, support these results.



(a)

(b)

Figure 4. Computer simulation programs infected cells not go to zero exponentially in case $t \to \infty$ In (a) Deterministic model when $R_0 > 1$, but tend to zero exponentially as $t \to \infty$ in(b) stochastic model

When taking the Eq.9 $dC(t) = ((1 - \gamma)SaI(t) - \beta C(t) - (1 - \nu)\sigma T(t)C(t))dt + \alpha_2 C(t)dW_2(t),$

Also by substitution, the parameter values in Eq.9 and by using Ito's formula¹⁴, will find the stochastic solution of Eq.9

Is $C(t) = 10^4 e^{-0.03006t}$ and deterministic solution equal to $C(t) = 10^4 e^{0.68994t}$, so the virus particles C(t) goes to zero exponentially in case $t \rightarrow \infty$

in stochastic model but not in deterministic model. The computer simulation programs Fig.5, support these results.

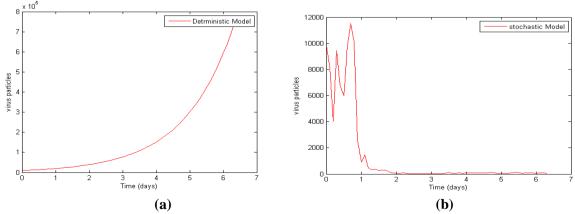


Figure 5. Computer simulation programs the virus particles do not go to zero exponentially in case $t \to \infty$, In(a) Deterministic model when $R_0 > 1$, but the virus particles goes to zero exponentially in case $t \to \infty$ In(b) stochastic model.

when taking the Eq.7 $dT(t) = (G - nT(t) - (1 - v)\sigma T(t)C(t))dt + \alpha_1 T(t)dW_1(t)$

So by substitution, the parameter values in Eq.7 and by using Ito's formula, will find the stochastic solution of Eq.7 as $T(t) = 10^4 e^{999999.3t}$, and the deterministic solution is $T(t) = 10^4 e^{999999.8t}$.

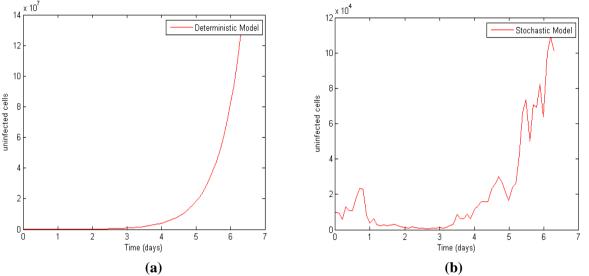


Figure 6. Computer simulation programs the uninfected cells does not go to zero exponentially as $t \to \infty$ (a) Deterministic model when $R_0 > 1$, (b) stochastic model.

Conclusions:

This paper introduced environmental stochasticity into the deterministic model also explored the properties of COVID-19 and how the disease spreads inside the human body, through its interaction with cells that contain receptors for the ACE-2. As well in this paper, we construct the basic reproduction number R₀ and conditions required for extinction or persistence COVID-19. In general in the deterministic model if $R_0 < 1$, the disease will die out the injured person will be cured .If $R_0 > 1$ the disease will persist. In stochastic model prove that anyone infected with the Coronavirus can recover if the stochastic variance α_1^2 and α_2^2 are big sufficient this gives us $\lim_{t\to\infty} I(t) =$ $0, \lim_{t \to \infty} C(t) = 0 \text{ and } \lim_{t \to \infty} T(t) =$

$$\frac{G}{2}$$
 although $R_0 > 1$.

The computer simulation Figures 1, 2, 3, 4, 5 and 6, support these results.

Authors' declaration:

- Conflicts of Interest: None.
- We hereby confirm that all the Figures and Tables in the manuscript are mine ours. Besides, the Figures and images, which are not mine ours, have been given the permission for republication attached with the manuscript.

Authors' contributions statement:

S.N. A. A. presented the main idea of the research and A.M. K applied the idea of the research from a mathematical point of view as well as using Mat lab. Both researchers presented a summary of the research as well as the main conclusions.

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مقارنة بين النموذج الحتمي والتصادفي لتفاعل (كوفيد-19) مع الخلايا المضيفة في البشر

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الخلاصة :

في هذا البحث ، تم اقتراح النموذج الحتمي والعشوائي لدراسة تفاعل فيروس كورونا (كوفيد -19) مع الخلايا المضيفة داخل جسم الانسان في النموذج الحتمي تحدد قيمة رقم الاستنساخ الأساسي استمرار أو انقراض كوفيد-19. إذا كان رقم الاستنساخ الأساسي أقل من واحد فأن خلية واحدة مصابة ستصيب أقل من خلية واحدة ،وهذا يعني ان الشخص الذي يحمل فيروس كورونا قد تم شفاؤه. إذا كان رقم الاستنساخ الاساسي اكبر من واحد ستكون الخلية المصابة قادرة على القضاء على مجموعة الخلايا المستهدفة في النموذج ال أنه إذا كانت المعلمات التصادفية كبيرة بدرجة كافية فأن هذه المعلمات التصادفية تعطينا الانقراض الذي يدمل فيروس كورونا قد تم شفاؤه. إذا كان رقم الاستنساخ الاساسي اكبر من واحد وقد تم ألبات هذه المعلمات التصادفية تعطينا الانقراض النهائي للمرض على الرغم من رقم انه إذا كانت المعلمات التصادفية حيرة بدرجة كافية فأن هذه المعلمات التصادفية تعطينا الانقراض النهائي للمرض على الرغم من رقم

الكلمات المفتاحية: رقم الاستنساخ الأساسي، محاكاة الكمبيوتر، حركة كوفيد-19، النموذج التصادفي