Mathematical analysis of a COVID-19 Epidemic model with latency period

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Abstract

In the present paper, we develop a delayed differential equations epidemic model to study the transmission dynamics of the COVID-19 by introducing a latent period into susceptible, and infectious individuals in incidence rate. We compute the basic reproduction rate of the model and study its effects on the disease diffusion. Thus, we show that the basic reproduction $\mathcal{R}_0$ is the threshold dynamics between the persistence and the extinction of the disease. More precisely, we prove, through Lyapunov direct method, that the disease-free equilibrium is globally asymptotically stable if $\mathcal{R}_0 \leq 1$, whereas the disease is persistent if $\mathcal{R}_0 > 1$. We also discuss the impact of the delay by comparing the model with delay and the model without delay. Furthermore, numerical simulations are carried out to illustrate the theoretical results.

1 Introduction

COVID-19 is a respiratory disease caused by the SARS-COV-2 virus that has spread among humans, mainly in China, since December 2019. Fever, cough, shortness of breath, and breathing difficulties are initial symptoms of this infection [5, 18]. The transmission of the disease occurs through direct, indirect, or close contact with infected people through infected secretions. The number of the confirmed cases with COVID-19 is increasing rapidly worldwide, especially in the United States and in Europe [14, 7]. One major cause of the quick spread of the disease is the lack of information and awareness about the virus during the early stages of infection. Thus, a difficulty to control COVID-19 is link to variation of incubation period, since it has been shown that the infected human is contagious during this period. Since, we lack of proper treatment or vaccines, Isolating the infected individuals

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in special quarantine cells has been implemented in most of the countries [16, 3, 15]. Despite of this prevention, COVID-19 still persists with high level death rate.

Epidemiological models in mathematics have been recognized as valuable tools in analysing the dynamics of infectious diseases. Mathematical models and computer simulations are used in comparing, planning, implementing, evaluating, and optimizing various detection, prevention, therapy, and control programs that are crucial in informing public health intervention policies. Concerning the mathematical modelling of COVID-19, different type of models have been formulated in order predict the disease transmission [17, 12, 11, 19]. However, as we said above, the time period of incubation plays an important role in understanding the COVID-19 dynamics transmission. So, it will be more realistic to incorporate that parameter in model. In that direction, P. Magal *et al.*, [10] have used a delay differential equation to studied the impact of this parameter in the model; but a rigorous mathematical analysis of the model has not been done.

In This paper, we focus on the impact of latency period of COVID-19 transmission. Thus, our model is formulated as a delay differential equation (DDE) with recruitment of susceptible individuals [13, 6].

The paper is organized as follows. In section 2, we formulate the model. In section 3, we introduce the basic reproduction number of the model and show that it is a threshold dynamics between the persistence and the extension of the disease. Hence, we prove that if $\mathcal{R}_0$ is less than one, the disease free equilibrium is globally asymptotically stable. If $\mathcal{R}_0$ is greater than 1, under certain conditions, the endemic equilibrium is globally asymptotically stable. In section 4, we compare the DDE and ODE models. Numerical simulations are performed to illustrate our theoretical results in section 5. Finally, a conclusion is drawn in section 6.

2 Model Formulation

Based on the development and epidemiological characteristics of COVID-19 infection, the SEIR model is appropriate to study the dynamic of this disease. In the presence of the disease, the population is partitioned into five sub-populations as susceptible individuals (S), asymptomatic infectious individuals (I), hospitalized symptomatic infectious individuals (H), unreported symptomatic infectious individuals (U) and recovered individuals (R). Indeed, at any time, the susceptible population is increased by the recruitment of individuals into the population at a rate Λ. The flux of Susceptible individuals that may acquire infection, following effective contact with infectious individuals (I or U), is

$$\lambda(t) = \beta S(t)I(t) + \beta S(t)U(t). \tag{1}$$

However, in the real situation, there may be a lag between the time Susceptible humans are contacted by the infectious individuals and the time the contacted individuals become infectious. Let τ be that period. Then, the flux of asymptomatic infectious individuals is described by

$$\lambda(t-\tau) = \beta S(t-\tau)I(t-\tau)e^{-\mu\tau} + \beta S(t-\tau)U(t-\tau)\big)e^{-\mu\tau}, \tag{2}$$

where $e^{-\mu\tau}$ represents the probability of survival of a human through the period τ. The flux of individuals leaving the class I is $\nu I(t)$; and we suppose that a fraction f are reported and a fraction $1-f$ are unreported. Humans leave the population through natural death rate μ and the disease-induced death rate, δ.

In order to avoid excessive use of parentheses in some of the later calculations, we use the following notations:

$$S(t) = S,\ I(t) = I,\ U(t) = U,\ H(t) = H,\ R(t) = R,$$
$$I(t-\tau) = I_\tau,\ U(t-\tau) = U_\tau,\ S(t-\tau) = S_\tau.$$

The paramters of the model are given in Table (1)

Symbols	**Biological descriptions**
Λ	Constant recruitment for humans
μ	Human natural death rate
f	Fraction of asymptomatic infectious that become reported symptomatic infectious
δ	Disease-induced death rate for unreported symptomatic infectious individuals
β	Transmission rate
$\nu_1 = f\nu$	Rate at which asymptomatic infectious become reported symptomatic
$\nu_2 = (1-f)\nu$	Rate at which asymptomatic infectious become unreported symptomatic
$1/\eta$	Average time symptomatic infectious have symptoms

Table 1: Parameters of the model.

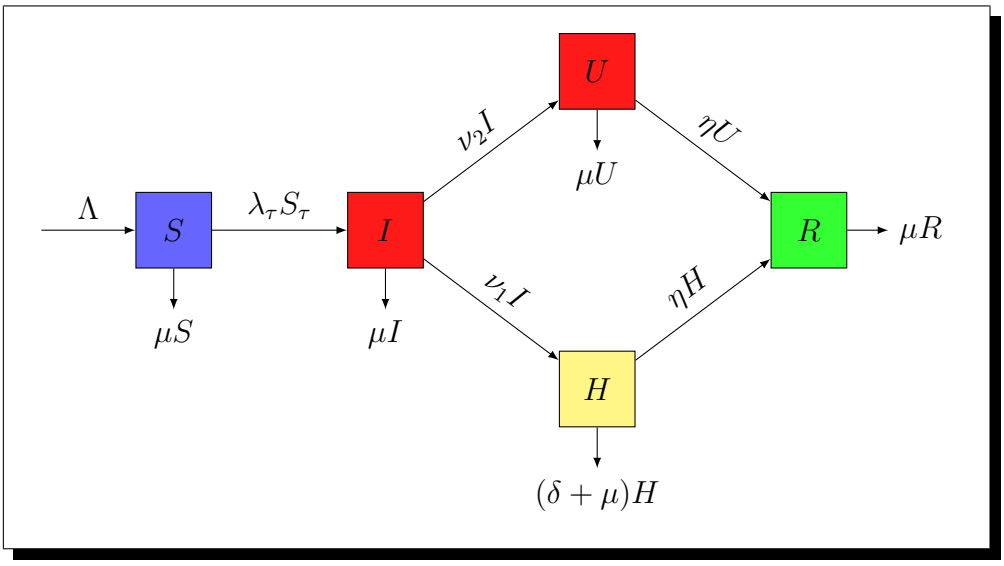


Figure 1: Diagram of the model

From Figure 1, the number of individuals which survive from recruitment into one class, the next,

is defined by the following Retarded Functional Differential Equation (RFDE):

$$\begin{cases} \dot{S}(t) = \Lambda - \beta S(t)(I(t) + U(t)) - \mu S(t), \\ \dot{I}(t) = \beta S(t-\tau)\big(I(t-\tau) + U(t-\tau)\big)e^{-\mu\tau} - (\nu + \mu)I(t) \\ \dot{U}(t) = \nu_2 I(t) - (\eta + \mu)U(t), \\ \dot{H}(t) = \nu_1 I(t) - (\eta + \delta + \mu)H(t), \\ \dot{R}(t) = \eta(U(t) + H(t)) - \mu R(t). \end{cases} \tag{3}$$

The initial condition is assumed to be on the form :

$$S_0 = \varphi_1, \quad I_0 = \varphi_2, \quad U_0 = \varphi_3, \quad H_0 = \varphi_4, \quad R_0 = \varphi_5, \tag{4}$$

where $\varphi_i \in \mathcal{C}([-\tau, 0], \mathbb{R}^+), \forall i = 1, 2, ..., 5$. $\mathcal{C}([-\tau, 0], \mathbb{R}_+)$ is the Banach space of continuous functions mapping the interval $[-\tau, 0]$ into $\mathbb{R}^+$ endowed with the sup-norm, such that $\varphi_1(0) > 0$ and $\varphi_2(0) > 0$. The existence and uniqueness of the solutions then follow from standard results in the theory of delay differential equations (see, [4]).

3 Mathematical analysis of the model

3.1 Properties of the model

Lemma 3.1. *For any $\varphi \in \mathcal{C}([-\tau, 0], \mathbb{R}^5_+)$, system (3) has a unique non-negative solution through φ. Moreover, the solution is bounded.*

Proof. For any $\varphi \in \mathcal{C}([-\tau, 0], \mathbb{R}^5_+)$, define

$$f(\varphi) := \begin{pmatrix} \Lambda - \beta\varphi_1(0)(\varphi_2(0) + \varphi_3(0)), \\ \beta\varphi_1(-\tau)\big(\varphi_2(-\tau) + \varphi_3(-\tau)\big)e^{-\mu\tau} - \nu\varphi_2(0) \\ \nu_2\varphi_2(0)) - \eta\varphi_3(0), \\ \nu_1\varphi_2(0) - (\eta + \delta)\varphi_4(0), \\ \eta(\varphi_3(0) + \varphi_4(0)). \end{pmatrix} \tag{5}$$

Note that f is continuous, and locally lipschitzian. Hence from Theorem 2.3 in [4], (3) has a unique solution through $(0, \varphi)$ on its maximal interval $[0, T_{\max}[$ of existence. Now let us show that the solution is non-negative. Let

$$\ell(t) = \min\{S(t), I(t), U(t), H(t), R(t)\}.$$

Suppose that there exists $\bar{t} \in [0, T_{\max}[$ such that $\ell(\bar{t}) < 0$ and $\ell(t) \geq 0$ for all $t \in [0, \bar{t}[$.

If $\ell(\bar{t}) = S(\bar{t})$, Since $I(t) \geq 0$ for all $t \in [0, \bar{t}[$, from the first equation of system (3) it yields that

$$\dot{S}(t) > -S(t)\big[\beta(I(t) + U(t)) + \mu\big], \ \forall t \in [0, \bar{t}[. \tag{6}$$

Then,

$$S(\bar{t}) > S(0)\exp\left(-\int_0^{\bar{t}}(\beta I(s)+\beta U(s)+\mu)ds\right) \geq 0, \tag{7}$$

which contradicts the fact that $S(\bar{t}) < 0$.

If $\ell(\bar{t}) = I(\bar{t})$, Since S_τ, I_τ and U_τ are positive, from second equation of model (3), we have

$$\dot{I}(t) > -(\nu+\mu)I(t), \ \forall t \in [0,\bar{t}[. \tag{8}$$

Then,

$$I(\bar{t}) > I(0)e^{-(\nu+\mu)} \geq 0, \tag{9}$$

which leads to a contradiction.

Similar contradiction can be obtained if $\ell(\bar{t}) = U(\bar{t})$, $\ell(\bar{t}) = H(\bar{t})$ and $\ell(\bar{t}) = R(\bar{t})$. Therefore, solutions of system (3) with conditions $S(0) \geq 0$, $I(0) \geq 0$, $U(0) \geq 0$, $H(0) \geq 0$ and $R(0) \geq 0$ are non-negative.

Moreover, at any time $t \geq 0$, we have:

$$\dot{S} = \Lambda - \beta S(I+U) - \mu S \leq \Lambda - \mu S.$$

Let us consider the following ordinary differential equation :

$$\dot{S}_1 = \Lambda - \mu S_1,$$

with general solution:

$$S_1(t) = \frac{\Lambda}{\mu} + \left(S(0) - \frac{\Lambda}{\mu}\right)e^{-\mu t}.$$

Thanks to standard comparison theorem [8] we have: $\limsup_{t\to+\infty} S(t) \leq \frac{\Lambda}{\mu}$. Similarly, we show that $I(t), U(t), H(t)$ and $R(t)$ are bounded. It then follows that $T_{\max} = +\infty$ and solutions exist globally.

Remark 1. In the rest of the paper we discuss system (3) in the following closed set :

$$\Omega := \left\{\varphi \in \mathcal{C} : \|\varphi_1\| \leq \frac{\Lambda}{\mu}, \varphi_i \geq 0\right\}.$$

It is easy to show that Ω is positively invariant with respect to (3).

□

3.2 Basic reproduction number and Existence of equilibria

The basic reproduction number is an indicator of COVID-19 transmission. It is a threshold dynamics which predicts the disease invasion. It can be defined as a average number of new free infectious individual derived from a single infectious human introduced into an entirely susceptible population. Moreover, by simple calculation, we prove that model (3) has a unique free-infected equilibrium $\mathcal{E}^0$ defined by

$$\mathcal{E}^0 = (S^0, 0, 0, 0, 0), \quad \text{with} \quad S^0 = \frac{\Lambda}{\mu}.$$

The basic reproduction number (3) can be computed as follows [10, 20]:

$$\mathcal{R}_0 = \sqrt{\frac{\beta S^0}{\nu+\mu}\left(1+\frac{\nu_2}{\eta+\mu}\right)e^{-\mu\tau}}.$$

Lemma 3.2. *If $\mathcal{R}_0 > 1$ system (3), has an unique infected equilibrium $\mathcal{E}^* = (S^*, I^*, U^*, H^*, R^*)$.*

Proof. At equilibrium, system (3), satisfies:

$$\begin{aligned}
0 &= \Lambda - \beta S^*(I^* + U^*) - \mu S^*, \\
0 &= \beta S^*\big(I^* + U^*\big)e^{-\mu\tau} - (\nu + \mu)I^*, \\
0 &= \nu_2 I^* - (\eta + \mu)U^*, \\
0 &= \nu_1 I^* - (\eta + \delta + \mu)H^*, \\
0 &= \eta(U^* + H^*) - \mu R^*.
\end{aligned} \tag{10}$$

Let

$$\begin{aligned}
&A_1 = S^0, \quad A_2 = \frac{\nu + \mu}{\mu}e^{\mu\tau}, \quad A_3 = \frac{\nu_2}{\eta + \mu} \\
&A_4 = \frac{\nu_1}{\eta + \mu + \delta}, \quad A_5 = \frac{\eta}{\mu}(A_3 + A_4).
\end{aligned}$$

Solving system (10), we have:

$$S^* = A_1 - A_2 I^*, \quad U^* = A_3 I^*, \quad H^* = A_4 I^*, \quad R^* = A_5 I^* \tag{11}$$

where I^* satisfies the following quadratic equation:

$$x^2 - bx = 0; \quad \text{with} \quad b = \frac{A_1}{A_2}\left(1 - \frac{1}{\mathcal{R}_0^2}\right). \tag{12}$$

Thus, if $\mathcal{R}_0 > 1$, then equation (12) has an unique positive solution $x^* = b$. Therefore, system (3) has an unique infected equilibrium, defined by

$$\mathcal{E}^* = (S^*, I^*, U^*, H^*, R^*),$$

with

$$S^* = \frac{S^0}{\mathcal{R}_0^2}, \quad U^* = A_3 I^*, \quad H^* = A_4 I^*, \quad R^* = A_5 I^*, \quad I^* = \frac{A_1}{A_2}\left(1 - \frac{1}{\mathcal{R}_0^2}\right).$$

□

3.3 Local stability of free infected equilibrium

Theorem 3.3. *If $\mathcal{R}_0 < 1$, then the infected free equilibrium $\mathcal{E}^0$ is locally asymptotically stable.*

Proof. By linearising the system (3) at $\mathcal{E}^0$, we obtain the following system

$$\begin{cases}
\dot{S}(t) = -\beta S^0(I(t) + U(t)) - \mu S(t), \\
\dot{I}(t) = \beta S^0\big(I_\tau + U_\tau\big)e^{-\mu\tau} - (\nu + \mu)I(t) \\
\dot{U}(t) = \nu_2 I(t) - (\eta + \mu)U(t), \\
\dot{H}(t) = \nu_1 I(t) - (\eta + \delta + \mu)H(t), \\
\dot{R}(t) = \eta(U(t) + H(t)) - \mu R(t).
\end{cases} \tag{13}$$

Let

$$J_0 = \begin{pmatrix} -\mu & -\beta S^0 & -\beta S^0 & 0 & 0 \\ 0 & 0 & -a & 0 & 0 \\ 0 & \nu_2 & -d_1 & 0 & 0 \\ 0 & \nu_1 & 0 & -d_2 & 0 \\ 0 & 0 & \eta & \eta & -\mu \end{pmatrix}, \; J_{-\tau} = \begin{pmatrix} 0 & 0 & 0 & 0 & 0 \\ 0 & Q_1(\tau) & Q_1(\tau) & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix},$$

with

$$a = \nu + \mu; \quad d_1 = \eta + \mu; \quad d_2 = \eta + \mu + \delta; \quad Q_1(\tau) = \beta S^0 e^{-(\mu+\lambda)\tau}.$$

Then, we have :

$$\Delta(\lambda) = \det(\lambda I_5 - J_0 - e^{-\lambda.\tau} J_{-\tau}) = \begin{vmatrix} \mu+\lambda & \beta S^0 & \beta S^0 & 0 & 0 \\ 0 & Q_1(\tau)+a+\lambda & Q_1(\tau) & 0 & 0 \\ 0 & -\nu_2 & d_1+\lambda & 0 & 0 \\ 0 & -\nu_1 & 0 & d_2+\lambda & 0 \\ 0 & 0 & -\eta & -\eta & \mu+\lambda \end{vmatrix}$$

By simple calculation, we have the following characteristic equation:

$$(\lambda+\mu)^2(\lambda+d_2)P(\lambda) = 0 \tag{14}$$

with

$$P(\lambda) = \lambda^2 + (a + d_1 - Q_2(\tau))\lambda + ad_1 - (d_1 + \nu_2)Q_2(\tau) \tag{15}$$

We shall study the distribution of the roots of the transcendental equation (14). Denote $\lambda = r(\tau) + i\omega(\tau)$, the eigenvalue of equation (14) where r et ω depend on the delay τ, [2]. Moreover, when the delay increases, roots can only possibly enter the right half plane by crossing the imaginary axis in the complex plane [1].

(i) If $\tau = 0$ and $\mathcal{R}_0 < 1$, then thanks to all roots of equation

$$P(\lambda) = \lambda^2 + (a + d_1 - Q_2(0))\lambda + ad_1(1 - \mathcal{R}_0^2) = 0$$

have negative real part.

(ii) Suppose that there exists $\tau_0 > 0$ such that $\lambda = i\omega(\tau_0)$, $\omega > 0$, is a purely imaginary root of (14). Thus, we have the following equation:

$$\omega^4 + (a^2 + d_1^2 - q_1^2)\omega^2 + (ad_1)^2 - (q_1 q_2)^2 = 0, \tag{16}$$

with

$$q_1 = \beta S^0 e^{-\mu\tau}; \quad q_2 = d_1 + \nu_2.$$

Let $y = \omega^2$. Then, equation:

$$y^2 + (a^2 + d_1^2 - q_1^2)y + (ad_1)^2 - (q_1 q_2)^2 = 0,$$

has no positive root if $\mathcal{R}_0 < 1$. That is a contradiction.

□

3.3.1 Global stability of steady states

In this section, we analyze the global stability of equilibria by the use of Lyapunov functions.

Theorem 3.4. *If $\mathcal{R}_0 \leq 1$, then $\mathcal{E}^0$ is globally asymptotically stable.*

Proof. We consider the following Lyapunov function:

$$\mathcal{L}_1(S,I,U) = V_1(S,I,U) + \beta \int_0^\tau S_s(I_s + U_s)ds, \tag{17}$$

where

$$V_1(S,I,U) = S - S^0 - S^0 \ln\left(\frac{S}{S^0}\right) + \mathcal{R}_0^2 I e^{\mu\tau} + \frac{\beta S^0}{(\eta+\mu)}U. \tag{18}$$

Note that:

- $\mathcal{L}_1(S^0,0,0) = 0$ and $\mathcal{L}_1(S,I,U) > 0$ if $(S,I,U) \neq (S^0,0,0)$.
- Otherwise, the time derivative of $\mathcal{L}_1$ along the solution of (3) is given by :

$$\dot{\mathcal{L}}_1(S,I,U) = \dot{V}_1(S,I,U) + \beta\Big(S(I+U) - S_\tau\big(I_\tau + U_\tau\big)\Big),$$

with

$$\begin{aligned}\dot{V}_1(S,I,U) &= \left(\frac{S-S^0}{S}\right)\dot{S} + \mathcal{R}_0^2 \dot{I} e^{\mu\tau} + \frac{\beta S^0}{(\eta+\mu)}\dot{U},\\ &= -\mu\frac{(S-S^0)^2}{S} - \beta S(I+U) + \beta S^0(I+U) + \beta\mathcal{R}_0^2 S_\tau\big(I_\tau+U_\tau\big)\\ &\quad -\beta S^0(I+U).\end{aligned}$$

Then, we have:

$$\dot{\mathcal{L}}_1(S,I,U) = -\mu\frac{(S-S^0)^2}{S} + \beta S_\tau\big(I_\tau+U_\tau\big)(\mathcal{R}_0^2 - 1) \leq 0, \quad \text{if } \mathcal{R}_0 \leq 1. \tag{19}$$

- If $S = S^0$, $I = 0$ and $U = 0$, then $\dot{\mathcal{L}}_1(S,I,U) = 0$.

therefore, the largest compact invariant set contained in $\{(S,I,U) \in \Omega : \dot{\mathcal{L}}_1(S,I,U) = 0\}$ is the singleton $\{\mathcal{E}^0\}$. Hence, thanks to LaSalle's invariant principle [9], $\mathcal{E}^0$ is globally asymptotically stable. □

Theorem 3.5. *If $\mathcal{R}_0 > 1$ and $1 \leq \dfrac{I+U}{I^*+U^*} \leq \dfrac{U}{U^*}$, then $\mathcal{E}^*$ is globally asymptotically stable.*

Proof. We consider the following lyapunov function:

$$\mathcal{L}_2(S,I,U) = V_2(S,I,U) + \int_0^\tau G\left(\frac{S_s(I_s+U_s)}{S^*(I^*+U^*)}\right)ds. \tag{20}$$

with

$$V_2 = G\left(\frac{S}{S^*}\right) + a_2 G\left(\frac{I}{I^*}\right) + a_3 G\left(\frac{U}{U^*}\right), \tag{21}$$

$$a_1 = \frac{1}{\beta(I^*+U^*)}, \ a_2 = \frac{I^* e^{\mu\tau}}{\beta(I^*+U^*)}, \ a_3 = \frac{U^*}{\nu_2 I^*} \text{ and } G(x) = x - 1 - \ln x. \tag{22}$$

- $\mathcal{L}_2(S^*, I^*, U^*) = 0$ and $\mathcal{L}_2(S, I, U) > 0$ if $(S, I, U) \neq (S^*, I^*, U^*)$.
- Otherwise, the time derivative of $\mathcal{L}_2$ along the solution of (3) is given by:

$$\dot{\mathcal{L}}_2(S, I, U) = \dot{V}_2(S, I, U) + G\left(\frac{S(I+U)}{S^*(I^*+U^*)}\right) - G\left(\frac{S_\tau(I_\tau+U_\tau)}{S^*(I^*+U^*)}\right), \tag{23}$$

with

$$\dot{V}_2(S, I, U) = a_1\frac{\dot{S}}{S^*}\left(1-\frac{S^*}{S}\right) + a_2\frac{\dot{I}}{I^*}\left(1-\frac{I^*}{I}\right) + a_3\frac{\dot{U}}{U^*}\left(1-\frac{U^*}{U}\right).$$

Thus, we have:

$$a_1\frac{\dot{S}}{S^*}\left(1-\frac{S^*}{S}\right) = -a_1\mu\frac{(S-S^*)^2}{SS^*} + 1 + \frac{I+U}{I^*+U^*} - \frac{S(I+U)}{S^*(I^*+U^*)} - \frac{S^*}{S}, \tag{24}$$

$$a_2\frac{\dot{I}}{I^*}\left(1-\frac{I^*}{I}\right) = \frac{S_\tau(I_\tau+U_\tau)}{S^*(I^*+U^*)} - \frac{I}{I^*} - \frac{S_\tau(I_\tau+U_\tau)}{S^*(I^*+U^*)}.\frac{I^*}{I} + 1, \tag{25}$$

$$a_3\frac{\dot{U}}{U^*}\left(1-\frac{U^*}{U}\right) = \frac{I}{I^*} - \frac{I}{I^*}\frac{U^*}{U} - \frac{U}{U^*} + 1. \tag{26}$$

Adding (24)−(26), It then follows that:

$$\begin{aligned}\dot{\mathcal{L}}_2(S, I, U) = {} & -a_1\mu\frac{(S-S^*)^2}{SS^*} + 1 + \frac{I+U}{I^*+U^*} - \frac{S(I+U)}{S^*(I^*+U^*)} - \frac{S^*}{S} + \frac{S_\tau(I_\tau+U_\tau)}{S^*(I^*+U^*)} \\ & - \frac{S_\tau(I_\tau+U_\tau)}{S^*(I^*+U^*)}.\frac{I^*}{I} + 1 - \frac{I}{I^*}\frac{U^*}{U} - \frac{U}{U^*} + 1 + G\left(\frac{S(I+U)}{S^*(I^*+U^*)}\right) \\ & - G\left(\frac{S_\tau(I_\tau+U_\tau)}{S^*(I^*+U^*)}\right)\end{aligned}$$

let

$$x = \frac{S}{S^*},\ y = \frac{I}{I^*},\ z = \frac{I+U}{I^*+U^*},\ z_\tau = \frac{I_\tau+U_\tau}{I^*+U^*},\ w = \frac{U}{U^*}.$$

It yields that:

$$\dot{\mathcal{L}}_2(S, I, U) = -a_1\mu\frac{(S-S^*)^2}{SS^*} + z + 1 - \frac{1}{x} - \frac{x_\tau}{y}z_\tau + 1 - \frac{y}{w} - w + 1 + \ln(x_\tau z_\tau) - \ln(xz)$$

by adding and subtracting the quantity $\ln(yw)$, we obtain:

$$\dot{\mathcal{L}}_2(S, I, U) = -a_1\mu\frac{(S-S^*)^2}{SS^*} - G\left(\frac{1}{x}\right) - G\left(\frac{x_\tau}{y}z_\tau\right) - G\left(\frac{y}{w}\right) - G(w) + G(z).$$

- If $S = S^*$, $I = I^*$ and $U = U^*$, then $\dot{\mathcal{L}}_2(S, I, U) = 0$. Therefore, the largest compact invariant set contained in $\{(S, I, U) \in \Omega : \dot{\mathcal{L}}_2(S, I, U) = 0\}$ is the singleton $\{\mathcal{E}^*\}$. Hence, thanks to LaSalle's invariant principle [9], $\mathcal{E}^*$ is globally asymptotically stable.

□

4 Comparison with model without time delay

Setting $\tau = 0$, that means the system (3) without delay, then we obtain we following ordinary differential equation (ODE):

$$\begin{cases} \dot{S}(t) = \Lambda - \beta S(t)(I(t) + U(t)) - \mu S(t), \\ \dot{I}(t) = \beta S(t)\big(I(t) + U(t)\big) - (\nu + \mu) I(t) \\ \dot{U}(t) = \nu_2 I(t) - (\eta + \mu) U(t), \\ \dot{H}(t) = \nu_1 I(t) - (\eta + \delta + \mu) H(t), \\ \dot{R}(t) = \eta(U(t) + H(t)) - \mu R(t). \end{cases} \tag{27}$$

Moreover, using the method of Van den Driessche and Watmough [20], we compute the basic reproduction ratio of model (27), as follows:

$$\hat{\mathcal{R}}_0 = \sqrt{\frac{\beta S^0}{\nu + \mu}\left(1 + \frac{\nu_2}{\eta + \mu}\right)}. \tag{28}$$

Moreover, model (27) has the two similar equilibrium $\mathcal{E}^0$ and $\mathcal{E}^*$ (if $\hat{\mathcal{R}}_0 > 1$). We have the following results:

Corollary 1. *Let* $1 \leq \dfrac{I + U}{I^* + U^*} \leq \dfrac{U}{U^*}$.

i) If $\hat{\mathcal{R}}_0 \leq 1$, *then the disease-free equilibrium* $\mathcal{E}^0$ *is locally and globally asymptotically stable.*

ii) If $\hat{\mathcal{R}}_0 > 1$, *then the endemic equilibrium* $\mathcal{E}^*$ *is locally and globally asymptotically stable.*

Proof. In the case of ODE model ($\tau = 0$), The lyapunov functional, $\mathcal{L}_1$ and $\mathcal{L}_2$ can be rewritten as follows :

$$\mathcal{L}_1(S, I, U) = S - S^0 - S^0 \ln\left(\frac{S}{S^0}\right) + \mathcal{R}_0^2 I + \frac{\beta S^0}{(\eta + \mu)} U \tag{29}$$

$$\mathcal{L}_2(S, I, U) = G\left(\frac{S}{S^*}\right) + a_2 G\left(\frac{I}{I^*}\right) + a_3 G\left(\frac{U}{U^*}\right) \tag{30}$$

□

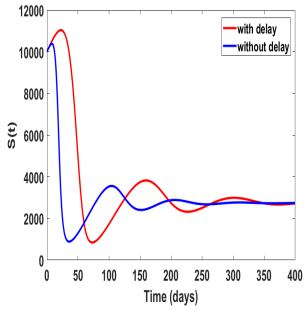
5 Numerical simulations

In this section, we perform some numerical simulations to support our theoretical analysis. we use the MATLAB technical computing software with the fourth-order Runge-Kutta method. Values of parameters are listed in Table 2.

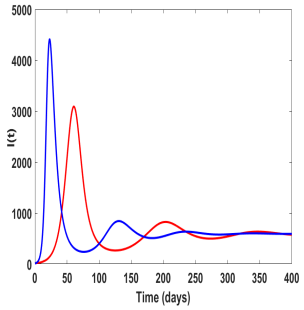
In the both Figure (2) and (3), blue solids lines represent the solution of ODE model that arise when there is no delay; and red solids lines are the solutions of RFDE model that arise when there is a delay. Our simulations indicate the persistence of COVID-19 for each model. Since $\mathcal{R}_0 > 1$, solutions converge globally to the endemic equilibrium $\mathcal{E}^*$ which illustrate the theoretical result of

Parameters	Values	Sources	Dimenssions
Λ	90	estimated	/day
μ	0.00152	[12]	/day
f	0.8	[10]	/day
δ	0.1	[19]	/day
β	0.000044	[12]	/day
ν	$1/7 - 1/4$	[10]	/day
η	$1/7 - 1/4$	[10]	/day

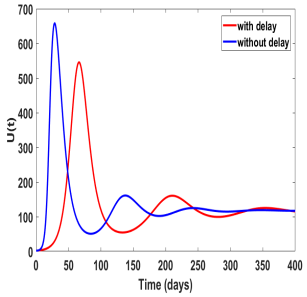
Table 2: Values of parameters



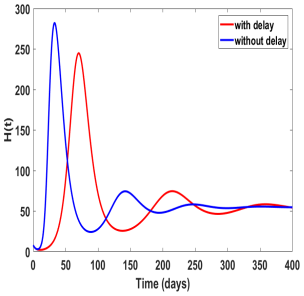
(a) Susceptible individuals



(b) asymptomatic infectious individuals



(c) reported symptomatic infectious individuals

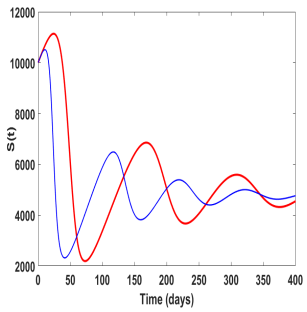


(d) Hospitalized symptomatic infectious individuals

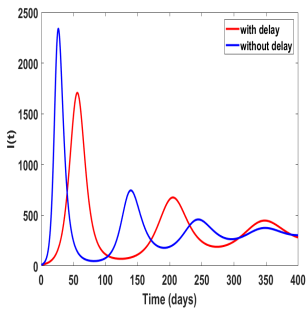
Figure 2: Graphs of symptomatic infectious individuals (Unreported and reported individuals) for $\tau = 7, \nu = 1/7,\ \eta = 1/7,\ S_0 = 10000,\ I_0 = 10,\ U_0 = 2,\ H_0 = 8,\ R_0 = 1$ and $\mathcal{R}_0 = 4.62$.

Theorem 3.5. Moreover, it is important to notice that RFDE model and ODE models show epidemics peaks. However, the corresponding time at which the peaks of ODE model occurs is different to the RFDE model.

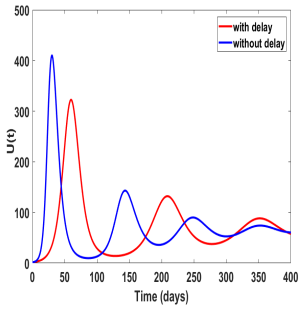
Remark 2. According to our numerical results, the latency period τ has a large impact on the dynamics of COVID-19 transmission and prediction. Thus, ODE model can underestimate the prediction date of the peaks of the disease. Besides, it easy to show that fixing all parameters, the larger τ is, the smaller the basic reproduction number, $\mathcal{R}_0$ becomes. So, realistic model must take into account this parameter.



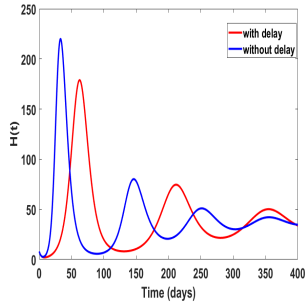
(a) Susceptible individuals



(b) asymptomatic infectious individuals



(c) reported symptomatic infectious individuals



(d) Hospitalized symptomatic infectious individuals

Figure 3: Graphs of symptomatic infectious individuals (Unreported and reported individuals) for $\tau = 4, \nu = 1/4,\ \eta = 1/4,\ S_0 = 10000,\ I_0 = 10,\ U_0 = 2,\ H_0 = 8,\ R_0 = 1$ and $\mathcal{R}_0 = 3.51$.

6 Conclusion

In this paper we have presented a mathematical model of COVID-19 transmission with latency period of incubation. The basic reproduction number has been computed, and we have shown that it is the threshold parameter between the persistence and the extinction of the disease. It emerges from our study that under some conditions, the disease-free equilibrium is globally stable if $\mathcal{R}_0 \leq 1$, whereas the disease is persistent if $\mathcal{R}_0 > 1$.

Furthermore, we have shown that the latency period, τ has significant effect on the disease transmission dynamics. More precisely, the length of the maturation period determines how fast or how slow the disease will progress within an area. From a biological viewpoint, this period play a positive role in the virus infection process in order to eliminate disease. Sufficiently large delay makes the virus progress slower, and the virus is controlled. This gives us some suggestions on new drugs to prolong the time of the latent period in order to reduce the disease transmission.

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Competing interests

The authors declare that they have no competing interests.

Authors'contributions

All authors carried out the paper. All author read and approved the final manuscript.

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