COVID-19 Disease Model with Reservoir of Infection: Cleaning Surfaces and Wearing Masks Strategies

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Abstract

At the end of 2019 a new coronavirus (called SARS-COV-2) epidemic appears in China and spreads from China to the rest of the world at beginning of 2020 and caused a new disease called COVID-19. It’s well known that, COVID-19 disease spreads between humans through the air by coughing and sneezing or by contact. In this paper, we develop a mathematical SIR model which takes into account the effect of disease transmission by coughing and sneezing and the period of latency which is represented by time delays. We prove that, there is no effect of latency period on the dynamics of the propagation and transmission of the coronavirus, and for some critical value of the basic reproduction number a transcritical bifurcation may occur and the disease disappears for values smaller than this critical value and persist otherwise. In the end, we carry out some numerical simulations in order to illustrate our theoretical results. Our study confirm that, cleaning surfaces and wearing masks is a controlling strategy for limiting the propagation of COVID-19.

Keywords: SIR Model, latency period, DDE, transcritical bifurcation, basic reproduction number $R_0$.

1. Introduction and mathematical model

Coronavirus 2019—nCoV (SARS-COV-2) is one of the largest family of coronavirus, it was firstly identified in Wuhan (China) on December 2019 and spreads to the rest of the world. Till now it causes 139,816,716 infected cases and 3,002,419 death and 118,843,964.M recovered around the world and becomes a major...
global health concern. According to the World Health Organization (WHO) the disease caused by 2019–
 nCoV virus (called COVID – 19 by WHO on February 2020) spreads between humans through air by
coughing and sneezing or by contacting surfaces which containing the drops resulting by coughing and
sneezing [WHO , 2020]. Humans people who are infected with COVID – 19 have developed mild to severe
respiratory illness with symptoms including fever, cough, shortness of breath, and potentially respiratory
distress 2 – 14 days after exposure. In the last months, some authors introduce some mathematical models
to better understanding the behaviour of COVID – 19 disease and how to control its spread, for example
[Chen et al., 2020; Jia et al., 2020; Shao et al., 2020]. According to statista [statista , 2020], its well known
that the mortality rate is about 2%, the infection index is between 1.5 and 3.5, the critical cases is 6.1%
and the mortality rate is 15% for persons with age bigger than 80 years. Based on some studies given in
[worldometers , 2020], the incubation period of COVID – 19 varies between 2 and 14 days (see Table 1),
but a case with an incubation period of 27 days was reported by Hubei Province local government on
Feb. 22 and another case with an incubation period of 19 days was reported in JAMA study of five cases
published on Feb. 21 [Chinese provincial government , 2020]. Then, knowing the range of the incubation
period is very important for health authorities in order to introduce more effective quarantine systems,
for people suspected of carrying the virus, best controlling and preventing the spread of the virus. In the
following table 1, we resume the incubation period of some coronavirus family.

| Virus       | Incubation period |
|-------------|-------------------|
| SARS-COV-2  | 2-19 days or 0-24 days |
| SARS        | 2-7 days          |
| MERS        | 2-14 days         |

In the present work, we construct a mathematical COVID-19 model of type SIR, which takes into account
the effect of latency period and a reservoir of infection. Our model is given by a system of differential
equations with two delays as follows

\[
\begin{align*}
\frac{dS}{dt} &= \Lambda - \beta_sSI_r - \beta_WSW_\nu - \mu_sS \\
\frac{dI_r}{dt} &= \beta_sSI_r + \beta_WSW_\nu - (\gamma + \mu_I)I \\
\frac{dR}{dt} &= \gamma I - \mu_R R \\
\frac{dW}{dt} &= \mu_W I - \epsilon W \\
S(0) &= S_0 \geq 0, I(s) = \varphi(s), s \in [-\tau, 0], R(0) = R_0 \geq 0, W(s) = \xi(s), s \in [-\nu, 0].
\end{align*}
\]

Where S, I and R are the total number of susceptible, infected and recovered populations respectively.
W is the concentration of SARS-COV-2 virus caused by humans, resulting by coughing and sneezing. All
parameters of the model are positive and defined in Table 2. As the equation of recovered population R
depends only on infected population I, it suffices to study the following reduced system:

\[
\begin{align*}
\frac{dS}{dt} &= \Lambda - \beta_sSI_r - \beta_WSW_\nu - \mu_sS \\
\frac{dI_r}{dt} &= \beta_sSI_r + \beta_WSW_\nu - (\gamma + \mu_I)I \\
\frac{dW}{dt} &= \mu_W I - \epsilon W \\
S(0) &= S_0 \geq 0, I(s) = \varphi(s), s \in [-\tau, 0], W(s) = \xi(s), s \in [-\nu, 0].
\end{align*}
\]

The rest of the paper is organized as follows. Section 2 deals with the positivity, boundedness of solutions
and the existence of equilibria. The stability analysis without delays of the corresponding equilibria is
established in Section 3 by applying direct and indirect Lyapunov method, we prove also the occurrence
of a transcritical bifurcation for some critical value of the basic reproduction number R_0. In Section 4 and
5, we consider the model with one and two delays respectively and we study the stability of equilibrium
points. In Section 6 we prove how parameters can affect the basic reproduction number R_0 by sensitivity
analysis. Some numerical simulations are given in Section 7 in order to confirm and illustrate our analytical
results. Finally, some biological and mathematical conclusions are summarized in the last Section.
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### Parameters

| Parameter | Epidemiological interpretation |
|-----------|--------------------------------|
| \( \Lambda \) | Birth rate parameter of S population |
| \( \beta_s \) | Transmission rate from I to S |
| \( \beta_W \) | Transmission rate from W to S |
| \( \mu_s \) | Death rate of people S |
| \( \gamma \) | Recovery rate |
| \( \mu_I \) | Death rate of I population |
| \( \mu_R \) | Death rate of R population |
| \( \mu_W \) | Shedding coefficients from I to W |
| \( \frac{1}{\varepsilon} \) | Lifetime of the virus in W |
| \( \tau \) | Latency period |
| \( \nu \) | Time needed for a population S to become infected by coughing and sneezing |

### 2. Boundedness of solutions and equilibria

For system (2) to be biologically meaningful, it is necessary to show the nonnegativity and boundedness of solutions.

**Proposition 1.** The set \( \mathbb{R}^3_+ \) is positively invariant with respect to system (2) with \( (\tau = \mu = 0) \). Furthermore, all solutions of (2) are uniformly bounded in the compact subset.

\[
\Gamma = \left\{ (S, I, W) \in \mathbb{R}^3_+ : S + I \leq \frac{\Lambda}{\mu_S}, \ W \leq \frac{\mu_W \Lambda}{\varepsilon \mu_S} \right\}. \tag{3}
\]

**Proof.** From the first equation of system (2), we have

\[
S(t) = S(0) \times \exp(\int_0^t -\phi(s)ds) + \exp(\int_0^t -\phi(s)ds) \times \int_0^t \Lambda \times \exp(\int_0^u \phi(l)dl)du.
\]

Thus \( S(t) > 0, \forall t > 0 \). To establish that \( \forall t > 0, I(t) > 0, W(t) > 0 \) whenever \( I(0) > 0, W(0) > 0 \), the above arguments can not be easily implemented. We then use an alternative trick. We Consider the following sub-equations related to the time evolution of variables

\[
\begin{align*}
\frac{dI}{dt} &= \beta_s SI + \beta_W SW - (\gamma + \mu_I)I \\
\frac{dW}{dt} &= \mu_W I - \varepsilon W \\
I(0) &> 0, W(0) > 0.
\end{align*}
\tag{4}
\]

\[
\dot{X}(t) = AX(t)
\]

where \( X = \begin{pmatrix} I \\ W \end{pmatrix} \) and \( A = \begin{pmatrix} \beta_s S - (\gamma + \mu_I) & \beta_W S \\
\mu_W & -\varepsilon \end{pmatrix} \).

From the expression of \( A \), its a Metzler matrix and its exponential is positive. Then we deduce the positivity of \( I(t) \) and \( W(t) \) whenever \( I(0) > 0 \) and \( W(0) > 0 \). This proves the positively invariant property of \( \mathbb{R}^3_+ \) with respect to system (2).

Let \( N(t) = S(t) + I(t) \), then

\[
\frac{dN}{dt} = \Lambda - \mu_S S - (\gamma + \mu_I)I \leq \Lambda - \mu_S N.
\]

Hence,

\[
\limsup_{t \to \infty} N(t) \leq \frac{\Lambda}{\mu_S}.
\]
This implies that $S$ and $I$ are uniformly bounded in the region $Γ$. Furthermore, from the bound of $I$ and the last equation of (2), it follows that

$$\limsup_{t \to \infty} W(t) \leq \frac{\mu W A}{\varepsilon \mu S}.$$ 

This guarantees the boundedness of $W$. This completes the proof. \hfill \blacksquare

The equilibrium points of model (2) are obtained by solving the algebraic system obtained by cancelling all derivatives of $S(t), I(t)$ and $W(t)$. Thus:

1. The disease free equilibrium (DFE) is: $E_0 = (S_0, 0, 0) = (\frac{\Lambda}{\mu s}, 0, 0)$.

2. The endemic equilibrium is: $E_1 = (S^*, I^*, W^*)$, with its components given by

$$S^* = \frac{\varepsilon (\gamma + \mu I)}{\varepsilon \beta S + \beta W \mu W}, \quad W^* = \frac{\mu W}{\varepsilon} I^* \quad \text{and} \quad I^* = \frac{\Lambda}{\gamma + \mu I} - \frac{\varepsilon \mu S}{\varepsilon \beta S + \beta W \mu W}. \quad (5)$$

The endemic equilibrium $E_1$ lies in the positive orthant if the basic reproduction number $R_0$ of model (2) is greater than 1, it is given by:

$$R_0 = \frac{\Lambda (\varepsilon \beta S + \beta W \mu W)}{\mu s \varepsilon (\gamma + \mu I)}. \quad (6)$$

**Proposition 2.**

- If $R_0 \leq 1$, the system (2) has only the disease free equilibrium $E_0 = (S_0, 0, 0)$.
- If $R_0 > 1$, in addition to $E_0$ the system (2) has a unique endemic equilibrium $E_1 = (S^*, I^*, W^*)$ which is positive.

### 3. Model without delay

Suppose $\tau = \nu = 0$, system (2) is written as

$$\begin{align*}
\frac{dS}{dt} &= \Lambda - \beta_s SI - \beta_W SW - \mu_s S \\
\frac{dI}{dt} &= \beta_s SI + \beta_W SW - (\gamma + \mu_I) I \\
\frac{dW}{dt} &= \mu_W I - \varepsilon W.
\end{align*} \quad (7)$$

#### 3.1. Stability Analysis

The aim of this section is to analyze the stability of the two equilibria $E_0$ and $E_1$.

Linearizing system (7) around an equilibrium point $E = (S, I, W)$, we get the following Jacobian matrix:

$$J_{E=(S,I,W)} = \begin{pmatrix} -\beta_s I - \beta_W W - \mu_s S & -\beta_s S & -\beta_W W \\
\beta_s I + \beta_W W & \beta_s S - \gamma - \mu_I & \beta_W S \\
0 & \mu_W & -\varepsilon \end{pmatrix}.$$ 

Replacing $E$ by $E_0$ and calculating the characteristic equation, we have

$$\det(\lambda I - J_{E_0}) = (\lambda + \mu_s)(\lambda^2 - Tr(J_{E_0}^1)\lambda + \det(J_{E_0}^{11})) \quad (8)$$

where

$$J_{E_0}^1 = \begin{pmatrix} -\beta_s S_0 - \gamma - \mu_I & \beta_W S_0 \\
\mu_W & -\varepsilon \end{pmatrix}$$

and

$$Tr(J_{E_0}^{11}) = \beta_s S_0 - \gamma - \mu_I - \varepsilon$$
and
\[ \det(J_{1E_0}^1) = \varepsilon(-\beta S S_0 + \gamma + \mu I) - \mu W \beta W S_0 = \varepsilon(\gamma + \mu I)(1 - R_0). \]

Then, we have the following result.

**Proposition 3.** If \( R_0 < 1 \), the disease free equilibrium \( E_0 \) is asymptotically stable and it becomes unstable if \( R_0 > 1 \).

**Proof.** If \( R_0 < 1 \), then \( \text{Tr}(J_{1E_0}^1) < 0 \) and \( \det(J_{1E_0}^1) > 0 \), then the characteristic equation (8) does not admit a real strictly positive root.

If \( R_0 > 1 \), then \( \det(J_{1E_0}^1) < 0 \). So, the characteristic equation (8) has at least one positive root. □

**Proposition 4.** If \( R_0 > 1 \), the endemic equilibrium \( E_1 \) is asymptotically stable.

**Proof.** Let \( R_0 > 1 \) and we compute the characteristic equation associated to \( E_1 \). The Jacobian matrix at \( E_1 \) is given by
\[ J_{E_1}(S^*, I^*, W^*) = \begin{pmatrix} -\frac{\Lambda}{S^*} & -\beta S S^* I^* & -\beta W S^* \\ (\gamma + \mu I) I^* & -\beta W S^* W^* & \beta W S^* \\ 0 & \mu W & -\varepsilon \end{pmatrix} \]
and the characteristic equation associated to \( E_1 \) is as follows:
\[ P(\lambda) = \lambda^3 + a_2 \lambda + a_1 \lambda + a_0, \tag{9} \]
where
\[ a_2 = \varepsilon + \beta W \frac{S^* W^*}{I^*} + \frac{\Lambda}{S^*}, \]
\[ a_1 = \frac{\Lambda}{S^*} (\varepsilon + \beta W \frac{S^* W^*}{I^*}) + \beta S (\gamma + \mu I) I^* \]
\[ a_0 = (\gamma + \mu I) (\varepsilon \beta S + \mu W \beta W) I^*. \]

Note that if \( R_0 > 1 \), then the coefficients \( a_0, a_1 \) and \( a_2 \) are all strictly positive, then the polynomial \( P \) does not admit a real strictly positive root. □

**Proposition 5.** If \( R_0 > 1 \), the endemic equilibrium \( E_1 \) is globally asymptotically stable.

**Proof.** Define a Lyapunov functional as follows:
\[ V(t) = S(t) - S^* - \int_{S^*}^{S(t)} \frac{S^*}{Z} dZ + I^* \Phi \left( \frac{I(t)}{I^*} \right) + \frac{\beta W S^* W^*}{\varepsilon} \Phi \left( \frac{W(t)}{W^*} \right), \tag{10} \]
where \( \Phi(Z) = Z - 1 - \ln Z > 0 \), for \( Z > 0 \). It is obvious that \( \Phi \) attains its strict global minimum at 1 and \( \Phi(1) = 0 \). Then \( \Phi(Z) > 0 \) and the functional \( V \) is nonnegative.

For convenience, let \( \psi = \psi(t) \) for any \( \psi \in \{S, I, W\} \).

Differentiating \( V \) with respect to \( t \) along the solutions of (7), we obtain
\[ \dot{V}(t)|_{(7)} = \left( 1 - \frac{S^*}{S} \right) \frac{dS}{dt} + \left( 1 - \frac{I^*}{I} \right) \frac{dI}{dt} + \frac{\beta W S^*}{\varepsilon} \left( 1 - \frac{W^*}{W} \right) \frac{dW}{dt}. \tag{11} \]

Using \( \Lambda = \mu S S^* + (\gamma + \mu I) I^* \) and \( \mu W I^* = \varepsilon W^* \), we get
\[ \dot{V}(t)|_{(7)} = \mu S S^* \left( 1 - \frac{S^*}{S} \right) \left( 1 - \frac{S^*}{S} \right) + \beta S S^* I^* \left( 2 - \frac{S^*}{S} - \frac{S}{S^*} \right) + \beta W S^* W^* \left( 3 - \frac{S^*}{S} - \frac{SW I^*}{S^* W^* I - W^* I} \right). \tag{12} \]
Thus,
\[
\dot{V}(t) |_{(7)} = \mu S S^* (1 - \frac{S^*}{S}) (1 - \frac{S}{S^*}) - \beta_S S^* I^* \left[ \Phi \left( \frac{S}{S^*} \right) + \Phi \left( \frac{S^*}{S} \right) \right]
\]
\[- \beta_W S^* W^* \left[ \Phi \left( \frac{S}{S^*} \right) + \Phi \left( \frac{W}{W^*} \right) \right] + \Phi \left( \frac{SW^*}{S^*W^*} \right) \]
\[= \mu S S^* (1 - \frac{S}{S^*}) (1 - \frac{S^*}{S}) \]
\[- \beta_S S^* I^* \left[ 2 \Phi \left( \frac{S}{S^*} \right) + \Phi \left( \frac{S^*}{S} \right) + \Phi \left( \frac{W}{W^*} \right) + \Phi \left( \frac{SW^*}{S^*W^*} \right) \right]. \tag{13}
\]

Since \((1 - \frac{S}{S^*}) (1 - \frac{S^*}{S}) \leq 0\) and \(\Phi(Z) \geq 0\) for \(Z > 0\), we deduce that \(\dot{V}(t) |_{(7)} \leq 0\) and the equality occurs at the endemic equilibrium \(E_1\). Consequently, the global asymptotic stability of \(E_1\) follows from LaSalle’s invariance principle [LaSalle, 1976].

### 3.2. Analysis at \(R_0 = 1\)

In this section, we state and prove the following theorem which characterizes the occurrence of transcritical bifurcation at \(R_0 = 1\).

**Theorem 1.** The disease free equilibrium \(E_0\) changes its stability from stable to unstable at \(R_0 = 1\) and system (7) exhibits a transcritical bifurcation at bifurcation parameter value \(\Lambda = \Lambda^* = \frac{\mu S \varepsilon (\gamma + \mu_I)}{\varepsilon \beta_S + \beta_W \mu_I}\).

**Proof.** Linearized matrix of system (7) around \(E_0\) at the bifurcation parameter value \(\Lambda = \Lambda^* = \frac{\mu S \varepsilon (\gamma + \mu_I)}{\varepsilon \beta_S + \beta_W \mu_I}\) is given by

\[
J = \begin{pmatrix}
-\mu_S & -\beta_S \frac{\Lambda^*}{\mu_S} & -\beta_W \frac{\Lambda^*}{\mu_S} \\
0 & \beta_S \frac{\Lambda^*}{\mu_S} - \gamma - \mu_I & \beta_W \frac{\Lambda^*}{\mu_S} \\
0 & \mu_W & -\varepsilon
\end{pmatrix}.
\]

The matrix \(J\) has a simple zero eigenvalue at \(R_0 = 1\), (see characteristic equation 8) and the others eigenvalues have negative real part. At this stage the linearization techniques fail to conclude the behaviour of system (7). Center Manifold Theory is used to study the behaviour of non-hyperbolic equilibrium. Then from Theorem 1 of Castillo-Chavez and Song [Castillo-Chavez & Song, 2004] the bifurcation constants \(a_1\) and \(b_1\) are given by

\[
a_1 = \sum_{k,i,j=1}^{n} v_k w_i w_j \left( \frac{\partial^2 f_k}{\partial x_i \partial x_j} \right) |_{E_0} \tag{14}
\]

and

\[
b_1 = \sum_{k,i=1}^{n} v_k w_i \left( \frac{\partial^2 f_k}{\partial x_i \partial \Lambda^*} \right) |_{E_0}. \tag{15}
\]

A right eigenvector associated with 0 eigenvalue is \(w = \left[ -\Lambda^* \left( \frac{\beta_S \varepsilon + \beta_W \mu_I}{\mu_S \mu_W} \right), \frac{\varepsilon}{\mu_S \mu_W}, 1 \right]^T\) and the left eigenvector \(v\) satisfying \(v.w = 1\) is \(v = \left[ 0, \frac{\mu_S \mu_W + \Lambda^* (\beta_S \varepsilon + \beta_W \mu_I)}{\varepsilon}, -\Lambda^* \left( \frac{\beta_S \varepsilon + \beta_W \mu_I}{\mu_S} \right) \right] \mu_S \mu_W \mu_W \mu_W \). Algebraic calculations show that

\[
\frac{\partial^2 f_1}{\partial x_1 \partial x_2} = -\beta_S, \quad \frac{\partial^2 f_1}{\partial x_1 \partial x_3} = -\beta_W, \quad \frac{\partial^2 f_2}{\partial x_1 \partial x_2} = \beta_S, \quad \frac{\partial^2 f_2}{\partial x_1 \partial x_3} = \beta_W \quad \text{and} \quad \frac{\partial^2 f_2}{\partial x_1 \partial \Lambda^*} = \frac{\beta_W}{\mu_S}.
\]
The rest of the second derivatives appearing in the formula for $a_1$ in (14) and $b_1$ in (15) are all zero. Hence,

$$a_1 = v_2 w_1 w_2 \left( \frac{\partial^2 f_2}{\partial x_1 \partial x_2} \right) + v_2 w_1 w_3 \left( \frac{\partial^2 f_2}{\partial x_1 \partial x_3} \right)$$

$$= -\frac{\Lambda^*}{\mu_S} \left( \beta_S \epsilon + \beta_W \mu_W \right) \frac{\mu_W \mu_S + \Lambda^* (\beta_S \epsilon + \beta_W \mu_W)}{\epsilon \mu_S} \left( \frac{\epsilon S + \beta_W}{\mu_W} \right)$$

$$= -\frac{\Lambda^*}{\mu_S} \left( \beta_S \epsilon + \beta_W \mu_W \right)^2 \frac{\mu_W \mu_S + \Lambda^* (\beta_S \epsilon + \beta_W \mu_W)}{\epsilon \mu_S} < 0.$$  

$$b_1 = v_2 w_2 \left( \frac{\partial^2 f_2}{\partial x_1 \partial \Lambda^*} \right)$$

$$= \frac{\mu_W \mu_S + \Lambda^* (\beta_S \epsilon + \beta_W \mu_W)}{\epsilon \mu_S} \frac{\epsilon \beta_W}{\mu_W \mu_S} > 0.$$  

This shows that at $R_0 = 1$, disease free equilibrium $E_0$ changes stability from stable to unstable and endemic equilibrium $E_1$ exists when $R_0$ crosses the threshold value one. This emphasizes that, the system exhibits transcritical bifurcation at $R_0 = 1$.  

4. Model with one delay $\tau$

We suppose $\tau > 0$ and $\nu = 0$, system (1) is written as

$$\begin{align*}
\frac{dS}{dt} &= \Lambda - \beta_S SI - \beta_W SW - \mu_S S \\
\frac{dI}{dt} &= \beta_S SI + \beta_W SW - (\gamma + \mu_I) I \\
\frac{dW}{dt} &= \mu_W I - \epsilon W \\
S(0) &= S_0 \geq 0, I(s) = \varphi(s), s \in [-\tau, 0], W(0) = W_0 \geq 0.
\end{align*}$$  

(16)

4.1. **Local stability of disease free equilibrium**

Linearizing system (16) around the disease free equilibrium $E_0 = (S_0, 0, 0)$, we get

$$\frac{dX}{dt} = L_0X(t) + L_\tau X(t - \tau),$$

where $X$ is a vector composed by state variables $S, I, W$ and

$$L_0 = \begin{pmatrix}
-\mu_S & 0 & -\beta_W S_0 \\
0 & -\gamma - \mu_I & \beta_W S_0 \\
0 & \mu_W & -\epsilon
\end{pmatrix} \quad \text{and} \quad L_\tau = \begin{pmatrix}
0 & -\beta_S S_0 & 0 \\
0 & \beta_S S_0 & 0 \\
0 & 0 & 0
\end{pmatrix}.$$  

The associated characteristic equation is given by

$$\Delta(\lambda) = \det(\lambda I - L_0 - e^{-\lambda \tau} L_\tau)$$

$$= (\lambda + \mu_S) [P_0(\lambda) + Q_0(\lambda) e^{-\lambda \tau}]$$

$$= 0.$$  

As $\lambda_1 = -\mu_S$ is a root of the characteristic equation, so the study of stability of $E_0$ is reduced to the study of the roots of equation

$$P_0(\lambda) + Q_0(\lambda) e^{-\lambda \tau} = 0,$$

where

$$P_0(\lambda) = \lambda^2 + (\gamma + \mu_I + \epsilon) \lambda + \epsilon (\gamma + \mu_I) - \mu_W \beta_W S_0,$$

$$Q_0(\lambda) = -\beta_S S_0 \lambda - \epsilon \beta_S S_0.$$
Define $F$ by $F(y) = |P_0(iy)|^2 - |Q_0(iy)|^2$, (see [Cooke & van den Driessche, 1986]), then we have
$$F(Y) = Y^2 + b_1Y + b_0,$$
with $Y = y^2$, and
$$b_1 = (\gamma + \mu I)^2 + (\beta_S S_0)^2 + \varepsilon^2 + 2\mu_W \beta_W S_0,$$
$$b_0 = (\varepsilon(\gamma + \mu I) - \mu_W \beta_W S_0)^2 + (\varepsilon \beta_S S_0)^2.$$
Since $b_1 > 0, b_2 > 0$, the function $F$ does not admit a real strictly positive root. So there is no change in stability.

Therefore, we summarize the above discussions in the following result.

**Proposition 6.** The disease free equilibrium $E_0$ is asymptotically stable for all $\tau > 0$.

### 4.2. Local stability of endemic equilibrium

In this section, we study the local asymptotic stability of the endemic equilibrium $E_1 = (S^*, I^*, W^*)$ of system (16) considering latency period.

The linearized system of (16) around $E_1$ is given by:
$$\frac{dX}{dt} = J_0X(t) + J_\tau X(t-\tau),$$
where
$$J_0 = \begin{pmatrix} -\beta_S I^* - \beta_W W^* - \mu_S & -\beta_W S^* \\ \beta_S I^* + \beta_W W^* & -\varepsilon \\ 0 & \mu_W \end{pmatrix} \quad \text{and} \quad J_\tau = \begin{pmatrix} 0 & -\beta_S S^* & 0 \\ 0 & -\beta_S S^* & 0 \\ 0 & 0 & 0 \end{pmatrix}.$$

The associated characteristic equation to $E_1$ is as follows
$$P_1(\lambda) + Q_1(\lambda)e^{-\lambda\tau} = 0,$$
where
$$P_1(\lambda) = \lambda^3 + \alpha_2 \lambda^2 + \alpha_1 \lambda + \alpha_0,$$
$$Q_1(\lambda) = c_2 \lambda^2 + c_1 \lambda + c_0,$$
with
$$\alpha_2 = -\beta_S I^* - \beta_W W^* - \mu_S + \gamma + \mu_I + \varepsilon,$$
$$\alpha_1 = \varepsilon(\gamma + \mu I) - \mu_W \beta_W S^* + (\beta_S I^* + \beta_W W^* + \mu_S)(\gamma + \mu_I + \varepsilon),$$
$$\alpha_0 = \varepsilon(\gamma + \mu I)(\beta_S I^* + \beta_W W^* + \mu_S) - \mu_S \mu_W \beta_W S^*$$
and
$$c_2 = -\beta_S S^*,$$
$$c_1 = -(\varepsilon + \mu_S)\beta_S S^*,$$
$$c_0 = -\varepsilon \beta_S \mu_S S^*.$$

Since the endemic equilibrium $E_1$ is asymptotically stable for $\tau = 0$, (proposition (4)), by the continuity property, it’s still asymptotically stable for small $\tau > 0$ [Cooke & van den Driessche, 1986; Boese, 1998]. To obtain the switch of stability, one needs to find a purely imaginary root for some critical value of $\tau$.

Let $i\omega (\omega > 0)$ be a root of Eq. (17), then we have
$$-i\omega^3 - \alpha_2 \omega^2 + i\alpha_1 \omega + \alpha_0 - (c_2 \omega^2 - i c_1 \omega - c_0)(\cos \omega \tau - i \sin \omega \tau) = 0.$$
Separating the real and imaginary parts, we find
$$\begin{cases}
\omega^3 - \alpha_1 \omega = c_1 \omega \cos \omega \tau + (c_2 \omega^2 - c_0) \sin \omega \tau \\
\alpha_2 \omega^2 - \alpha_0 = -(c_2 \omega^2 - c_0) \cos \omega \tau + c_1 \omega \sin \omega \tau.
\end{cases}$$
Adding up the squares of both the equations, we obtain

\[ \omega^6 + B_2 \omega^4 + B_1 \omega^2 + B_0 = 0. \] (20)

Let \( z = \omega^2 \), eq. (20) becomes

\[ f(z) = z^3 + B_2 z^2 + B_1 z + B_0 = 0, \] (21)

where

\[ B_2 = \alpha_2^2 - c_2^2 - 2\alpha_1, \]
\[ B_1 = 2(\alpha_0c_2 - \alpha_0\alpha_2) - (c_1^2 - \alpha_1^2), \]
\[ B_0 = \alpha_0^2 - c_2^2. \]

Let the hypothesis:

\[ (H) : B_0 > 0, \quad B_2 > 0 \text{ and } B_2B_1 - B_0 > 0. \] (22)

**Proposition 7.** If \( R_0 > 1 \) and \( (H) \) is satisfied, then endemic equilibrium \( E_1 \) is asymptotically stable for all \( \tau > 0 \).

**Proof.** The proof is deduced from the Routh-Hurwitz stability criterion [Chebotarev & Meiman, 1949].

\[ \Box \]

5. Model with two delays \( \tau = \nu > 0 \)

We consider \( \tau = \nu > 0 \), the model (2) is written as follows

\[
\begin{align*}
\frac{dS}{dt} &= \Lambda - \beta_S SI + \beta_W SW - \mu_SS \\
\frac{dI}{dt} &= \beta_S SI - \beta_W SW - (\gamma + \mu_I)I \\
\frac{dW}{dt} &= \mu_W I - \varepsilon W \\
S(0) &= S_0 \geq 0, I(s) = \varphi(s), W(s) = \xi(s), s \in [-\tau, 0].
\end{align*}
\] (23)

5.1. **Local stability of** \( E_0 \)

Linearising the system (23) around the disease free equilibrium \( E_0 \), we get

\[ \frac{dX}{dt} = M_0 X(t) + M_\tau X(t - \tau), \]

where,

\[ M_0 = \begin{pmatrix} -\mu_S & 0 & 0 \\ 0 & -\left(\gamma + \mu_I\right) & 0 \\ 0 & \mu_W & -\varepsilon \end{pmatrix} \quad \text{and} \quad M_\tau = \begin{pmatrix} 0 & -\beta_SS_0 & -\beta_WS_0 \\ 0 & \beta_SS_0 & \beta_WS_0 \\ 0 & 0 & 0 \end{pmatrix}, \]

and the corresponding characteristic equation is as follows:

\[
det(\lambda I - M_0 - e^{-\lambda\tau} M_\tau) = (\lambda + \mu_S)[P_2(\lambda) + Q_2(\lambda)e^{-\lambda\tau}] = 0. \] (24)

Since \( \lambda_1 = -\mu_S \) is a root of the characteristic equation, so the study of stability of \( E_0 \) is reduced to the study of the roots of

\[ P_2(\lambda) + Q_2(\lambda)e^{-\lambda\tau} = 0, \] (25)

with

\[ P_2(\lambda) = \lambda^2 + (\gamma + \mu_I + \varepsilon)\lambda + \varepsilon(\gamma + \mu_I), \]
\[ Q_2(\lambda) = -S_0(\beta_S\lambda + \varepsilon\beta_S + \mu_W\beta_W). \]
Define $G$ by $G(y) = |P_2(iy)|^2 - |Q_2(iy)|^2$ and we have
\[ G(Y) = Y^2 + d_1 Y + d_0, \] (26)
with $Y = y^2$, and
\[
\begin{align*}
d_1 &= \gamma + \mu_I + \varepsilon + (S_0 \beta_S)^2 - 2 \varepsilon (\gamma + \mu_I), \\
d_0 &= \varepsilon^2 (\gamma + \mu_I)^2 - \left( \frac{\Lambda}{\mu_S} \right)^2 (\varepsilon \beta_S + \mu_W \beta_W)^2, \\
&= \varepsilon^2 (\gamma + \mu_I)^2 (1 - R_0^2).
\end{align*}
\]
Note that, if $R_0 > 1$ then $G(0) = d_0 < 0$. As the function $G$ is continuous and $\lim_{Y \to \infty} G(Y) = +\infty$. Then, equation (26) has at least one positive root, denoted by $Y_0$. Consequently, the characteristic equation (24) has a pair of purely imaginary roots $\pm iy_0$. Hence the following result.

**Proposition 8.** The disease free equilibrium $E_0$ is stable if $R_0 \leq 1$ and unstable when $R_0 > 1$ for all time delay $\tau > 0$.

### 5.2. Local stability of $E_1$

In this part, we study the local asymptotic stability of the endemic equilibrium $E_1$ of system (23). By linearising system (23) at $E_1$, we get
\[
\frac{dX}{dt} = J_0^* X(t) + J_\tau^* X(t - \tau),
\]
where
\[
J_0^* = \begin{pmatrix} -\beta_S^* - \beta_W W^* - \mu_S & 0 & 0 \\ \beta_S^* + \beta_W W^* & -(\gamma + \mu_I) & 0 \\ 0 & \mu_W & -\varepsilon \end{pmatrix} \quad \text{and} \quad J_\tau^* = \begin{pmatrix} 0 & -\beta_S S^* - \beta_W W^* \\ 0 & \beta_S S^* & \beta_W W^* \\ 0 & 0 & 0 \end{pmatrix}.
\]

We get the following corresponding characteristic equation
\[
(\lambda + \beta_S I^* + \beta_W W^* + \mu_S)[P^*(\lambda) + Q^*(\lambda)e^{-\lambda \tau}] = 0.
\] (27)

Since $\lambda = -(\beta_S I^* + \beta_W W^* + \mu_S)$ is a root of the characteristic equation, so the study of stability of $E_1$ is reduced to the study of the roots of
\[
P^*(\lambda) + Q^*(\lambda)e^{-\lambda \tau} = 0,
\] (28)
with
\[
\begin{align*}
P^*(\lambda) &= \lambda^3 + \alpha_2^* \lambda^2 + \alpha_1^* \lambda + \alpha_0^*, \\
Q^*(\lambda) &= \delta_2^* \lambda^2 + \delta_1^* \lambda + \delta_0^*,
\end{align*}
\]
where
\[
\begin{align*}
\alpha_2^* &= \beta_S I^* + \beta_W W^* + \mu_S + \gamma + \mu_I + \varepsilon, \\
\alpha_1^* &= (\beta_S I^* + \beta_W W^* + \mu_S)(\gamma + \mu_I + \varepsilon + \varepsilon (\gamma + \mu_I), \\
\alpha_0^* &= \varepsilon (\gamma + \mu_I)(\beta_S I^* + \beta_W W^* + \mu_S)
\end{align*}
\]
and
\[
\begin{align*}
\delta_2 &= -\beta_S S^*, \\
\delta_1 &= -(\beta_S S^* (\beta_S I^* + \beta_W W^* + \mu_S + \varepsilon) + \mu_W \beta_W S^*), \\
\delta_0 &= (\varepsilon - 1)\beta_S S^* (\beta_S I^* + \beta_W W^*) - \mu_S (\beta_S S^* + \mu_W \beta_W W^*).
\end{align*}
\]
Since the endemic equilibrium $E_1$ is asymptotically stable for $\tau = 0$, by the continuity property, it’s still asymptotically stable for small $\tau > 0$, (see proposition 4). To obtain the switch of stability, one needs to
find a purely imaginary root for some critical value of \( \tau \).

Let \( i \omega (\omega > 0) \) be a root of Eq. (28), then we have

\[
-i\omega^3 - \alpha_2^* \omega^2 + \alpha_1^* i \omega + \alpha_0^* + (-\delta_2 \omega^2 + \delta_1 i \omega + \delta_0)(\cos \omega \tau - i \sin \omega \tau) = 0.
\]

(29)

Separating the real and imaginary parts, we find

\[
\begin{align*}
\omega^3 - \alpha_1^* \omega &= \delta_2 \omega^2 \sin \omega \tau + \delta_1 \omega \cos \omega \tau - \delta_0 \sin \omega \tau \\
\alpha_0^* - \alpha_0^* &= -\delta_2 \omega^2 \cos \omega \tau + \delta_0 \cos \omega \tau + \delta_1 \omega \sin \omega \tau.
\end{align*}
\]

(30)

Adding up the squares of both the equations, we obtain

\[
\omega^6 + A_2^* \omega^4 + A_1^* \omega^2 + A_0^* = 0.
\]

(31)

Let \( z = \omega^2 \), then equation (31) becomes

\[
h(z) = z^3 + A_2^* z^2 + A_1^* z + A_0^* = 0,
\]

(32)

where

\[
A_2^* = (\alpha_2^*)^2 - 2\alpha_1^*,
\]

\[
= (\beta_S I^* + \beta_W W^* + \mu_S)^2 + (\gamma + \mu_I)^2 + \varepsilon^2,
\]

\[
A_1^* = (\alpha_1^*)^2 - \delta_1^2 - \delta_2^2 + 2(\delta_0 \delta_2 - \alpha_0^* \alpha_2^*),
\]

\[
A_0^* = (\alpha_0^*)^2 - \delta_0^2.
\]

Since \( A_2^* > 0 \), let the hypothesis:

\[
(H_3) : A_0^* > 0 \text{ and } A_2^* A_1^* - A_0^* > 0.
\]

(33)

**Proposition 9.** If \( R_0 > 1 \) and \( (H_3) \) is satisfied, then endemic equilibrium \( E_1 \) is asymptotically stable for all \( \tau = \nu > 0 \).

**Proof.** The proof is deduced from the Routh-Hurwitz stability criterion [Chebotarev & Meiman, 1949].

\[
\]

\[
\]

\[
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\[
\]

6. Sensitivity Analysis

As we saw in Section 2, the basic reproduction number for the COVID-19 model (2), which we propose in Section 1, is given by (6). The sensitivity analysis for the endemic threshold (6) tells us how important each parameter is to disease transmission. This information is crucial not only for experimental design, but also to data assimilation and reduction of complex models [Powell et al., 2005]. Sensitivity analysis is commonly used to determine the robustness of model predictions to parameters values, since there are usually errors in collected data and presumed parameters values. It is used to discover parameters that have a high impact on the threshold \( R_0 \) and should be targeted by intervention strategies. More accurately, sensitivity indices allows us to measure the relative changes in a variable when a parameter changes. For that purpose, we use the normalized forward sensitivity index of a variable with respect to a given parameter, which is defined as the ratio of the relative change in the variable to the relative change in the parameter. If such variable is differentiable with respect to the parameter, then the sensitivity index is defined as follows.

**Definition 6.1.** [Ngoteya & Gyekye, 2015] The normalized forward sensitivity index of \( R_0 \), which is differentiable with respect to a given parameter \( \theta \), is defined by

\[
\tau^R_0 = \frac{\partial R_0}{\partial \theta} \frac{\theta}{R_0}.
\]

The values of the sensitivity indices for parameters values of Table 4, are presented in Table 3.

Note that the sensitivity index may depend on several parameters of the system, but also can be constant, independent of any parameter. For example, \( \tau^R_0 = +1 \) means that increasing (decreasing) \( \theta \) by a given percentage increases (decreases) always \( R_0 \) by that same percentage.
a sensitive parameter should be carefully done, since a small perturbation in such parameter leads to relevant quantitative changes. On the other hand, the estimation of a parameter with a rather small value for the sensitivity index does not require as much attention to estimate, because a small perturbation in that parameter leads to small changes.

From Table 3, we conclude that the most sensitive parameters to the basic reproduction number $R_0$ of COVID-19 model (2) are $\Lambda, \beta_S, \mu_S, \mu_I$ and $\mu_W$. In concrete, an increase of the value of $\beta_S$ by (100%) will increase the basic reproduction number by 75% and this happens, in a similar way, for the parameters $\Lambda$ and $\mu_W$. In contrast, an increase of the value of $\mu_I$ by (100%) will decrease $R_0$ by 81.82% and this happens, in a similar way, for the parameter $\mu_S$.

7. Numerical simulations

In this section, we aim to provide a numerical simulation to substantiate the theoretical results established in the previous sections by using Matlab Software with the parameters given in table 4. Here, we consider $W$ be a virtual reservoir population that fits to scaling of the population amounts and that is fitting by the infected (as stated) and decreases over time if no further input occurs.

8. Conclusions

In this work, we have proposed and investigated a new model SIR that describes the transmission dynamics of Corona virus (COVID-19), which takes into account the effect of disease transmission by coughing and sneezing and the period of latency. The well-posedness of the proposed model and the stability analysis of equilibria are rigorously studied. More precisely, we have established the existence, uniqueness, non
Fig. 1. Stability of $E_0$ and non existence of $E_1$ for $\tau \geq 0$ and $\nu \geq 0$ (left) and Instability of $E_0$ and stability of $E_1$ for $\tau \geq 0$ and $\nu \geq 0$ (right).

Fig. 2. Temporal evolution of $S$, $I$ and $W$ for different values of $\Lambda$. If $\Lambda$ increases, the number $R_0$ increases which imply the increasing of the infected population and the virus concentration.

negativity, and boundedness of solutions. By using appropriate Lyapunov functional and linearisation technique, we have proved the first steady state $E_0$ is asymptotically stable when $R_0 \leq 1$, which means that the disease dies out in the population. However, when $R_0 > 1$, $E_0$ becomes unstable and the model has an endemic steady state $E_1$ which is asymptotically stable (Fig. 1). This leads to the persistence of disease in the population when $R_0 > 1$.

From figures 2, 3, 4 and 5; the best strategy to stop the propagation of $COVID-19$ disease is to decrease the concentration of coronavirus, by cleaning surfaces and make the wearing of masks mandatory for all infected people in order to limiting the transmission of the virus from infected population to surfaces and from surfaces to susceptible population.
Fig. 3. Temporal evolution of $S$, $I$ and $W$ for different values of $\beta_W$. If $\beta_W$ increases, the number $R_0$ increases which imply the increasing of infected population and virus concentration.

Fig. 4. Temporal evolution of $S$, $I$ and $W$ for different values of $\epsilon$. If $\epsilon$ increases, the number $R_0$ decreases which imply the decreasing of infected population and virus concentration.
Fig. 5. Temporal evolution of $S$, $I$ and $W$ for different values of $\mu_W$. If $\mu_W$ increases, the number $R_0$ increases which imply the increasing of infected population and virus concentration.

Then we deduce that, the strategies have the effect due to their modification of critical growth drives: i) wearing a mask reduces $\beta_S$ and $\mu_W$, and ii) cleaning surfaces reduces $\beta_W$. What could be made more clear is that it may be important to use both strategies together.

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