

Research Article

Fatiha Najm, Radouane Yafia*, My Ahmed Aziz Alaoui, Abdessamad Tridane, and Lahcen Boukrim

Mathematical analysis of an epidemic model with direct and indirect transmission modes and two delays

<https://doi.org/10.1515/msds-2023-0103>

received June 6, 2023; accepted September 18, 2023

Abstract: In this article, we consider an epidemiological model in which we take into account the effects of direct and indirect transmissions. The first mode occurs through direct contact between infectious and susceptible individuals, and the second one will take place through the shedding of virus particles by infectious individuals and their acquisition by susceptible ones. We also study the effect of latency period and time needed for a susceptible person to become infected by indirect transmission mode. By considering the direct and indirect basic reproduction numbers, we define the basic reproduction number R_0 of the model, which helps us to analyze the stability of equilibria and bifurcation and determine the most sensitive parameters. In conclusion, some numerical simulations are given to confirm the analytical analysis.

Keywords: epidemic model, latency period, delay differential equation, bifurcation, sensitivity

MSC 2020: 34D05, 39B05, 92B05, 34C23, 92-10

1 Introduction and mathematical model

It is well known that all infectious epidemiological diseases are infections resulting from the emergence and transmission of diseases among a specific population, which are caused by organisms such as bacteria, viruses, fungi, or parasites, and are serious public health problems for most countries in the world. Some of the examples of infectious diseases can be found in [9,11,17,20,21,23,30,40,42,46]. Some infectious and communicable diseases can be transmitted and spread to the host population by indirect transmission [1,7,12,15,16,18,22,26–29,32,34,35,41]. In parallel to this transmission mode, we find the direct mode, which is caused by contact between infected individuals and susceptible ones. Some authors developed mathematical models to study the dynamics of infectious diseases by introducing simple or multigroup mathematical models that involve both direct and indirect transmissions. Mukandavire et al. [28,29] introduced a dynamic model of cholera in Zimbabwe, and Eisenberga et al. [12] proposed “A cholera model in a patchy environment with water and human movement.” In a few studies [13,14,25,37,38,45], multigroup models have been introduced to

* **Corresponding author: Radouane Yafia**, Department of Mathematics, Faculty of Sciences, Ibn Tofail University, Campus Universitaire, BP 133, Kenitra, Morocco, e-mail: radouane.yafia@uit.ac.ma

Fatiha Najm: Department of Mathematics, Faculty of Sciences, Ibn Tofail University, Campus Universitaire, BP 133, Kenitra, Morocco, e-mail: fatiha.najm@uit.ac.ma

My Ahmed Aziz Alaoui Normandie Univ, 76063, Le Havre, France; ULH, LMAH, F-76600 Le Havre; FR-CNRS-3335, ISCN, 25 rue Ph. Lebon, 76600 Le Havre, France, e-mail: aziz.alaoui@univ-lehavre.fr

Abdessamad Tridane: Mathematical Sciences (COS), United Arab Emirates University, P.O. Box No. 15551, Al Ain, UAE, e-mail: a-tridane@uaeu.ac.ae

Lahcen Boukrim: Department of Mathematics, Faculty of Sciences, Ibn Tofail University, Campus Universitaire, BP 133, Kenitra, Morocco

describe the transmission dynamics of infectious diseases by using ordinary differential equations, while others use delay differential equations [2,5,10,19,36,39,43,44]. Kumar and Abbas, in their study [22], used a partial differential equation to study the dynamics of an age-structured susceptible-infectious-recovered model by taking into account the or indirect contacts. In this article, we propose an epidemic mathematical model that involves delays of direct and indirect transmissions. The model is given by the following delay differential equations system:

$$\begin{cases} \frac{dS}{dt} = \Lambda - \beta_s SI_\tau - \beta_w SW_v - \mu_s S \\ \frac{dI}{dt} = \beta_s SI_\tau + \beta_w SW_v - (\gamma + \mu_I)I \\ \frac{dR}{dt} = \gamma I - \mu_R R \\ \frac{dW}{dt} = \mu_W I - \varepsilon W \\ S(0) = S_0 \geq 0, I(\theta) = \varphi(\theta), \theta \in [-\tau, 0] \\ R(0) = R_0 \geq 0, W(\theta) = \xi(\theta), \theta \in [-\nu, 0], \end{cases} \quad (1.1)$$

where all parameters are supposed to be positive and are defined as follows: Λ (resp. μ_s) is the birth (resp. death) rate of susceptible population, β_s (resp. β_w) is the direct (resp. indirect) transmission mode rate, γ is the recovery rate, μ_I (resp. μ_R) is the death rate of infectious (resp. recovered) population, μ_W is the shedding coefficient, $\frac{1}{\varepsilon}$ is the lifetime of the virus in the environment, τ is the period of latency, and ν is the time needed for susceptible individuals to become infected by indirect transmission mode.

Populations are defined as follows: S is the total number of susceptible population, I is the total number of infectious population, and R is the total number of recovered population. W is supposed to be the concentration of virus particles in the environment caused by coughing, shedding, or other methods.

As the third expression of equation (1.1)₃ depends only on the second state variable I , we reduce our study to the following model:

$$\begin{cases} \frac{dS}{dt} = \Lambda - \beta_s SI_\tau - \beta_w SW_v - \mu_s S \\ \frac{dI}{dt} = \beta_s SI_\tau + \beta_w SW_v - (\gamma + \mu_I)I \\ \frac{dW}{dt} = \mu_W I - \varepsilon W \\ S(0) = S_0 \geq 0, I(\theta) = \varphi(\theta), \theta \in [-\tau, 0], \\ W(\theta) = \xi(\theta), \theta \in [-\nu, 0]. \end{cases} \quad (1.2)$$

The organization of the current work is as follows: Section 2, is devoted to the study of the positivity and the boundedness of solutions; in Section 3, we prove the existence of the possible steady states and their stability without delays by applying the Lyapunov method and the occurrence of a transcritical bifurcation for some critical value of the basic reproduction number R_0 ; in Sections 4 and 5, we study the effect of time delays on the stability of the equilibrium points; in Section 6, we study the sensitivity analysis; in Section 7, we illustrate our results through some numerical simulations. We end our work with a conclusion.

2 Boundedness and steady states

In this section, we study the positivity and boundedness of solutions of system (1.2).

Proposition 2.1. *Suppose $\tau = \mu = 0$. Then,*

- (1) \mathbb{R}_+^3 is positively invariant under system (1.2), and
- (2) all solutions of equation (1.2) starting from a positive initial condition are uniformly bounded on the compact

$$\text{set } \Gamma, \text{ where } \Gamma = \left\{ (S, I, W) \in \mathbb{R}_+^3 : S + I \leq \frac{\Lambda}{\mu_s}, W \leq \frac{\mu_W \Lambda}{\varepsilon \mu_s} \right\}.$$

Proof. From the first expression of equation (1.2), we deduce that

$$S(t) = S(0) \times \exp\left(\int_0^t -\phi(s)ds\right) + \exp\left(\int_0^t -\phi(s)ds\right) \times \int_0^t \Lambda \times \exp\left(\int_0^u \phi(l)dl\right)du,$$

where $\phi(s) = -\beta_s I(s) - \beta_w W(s) - \mu_s$. Then, we obtain $S(t) > 0 \forall t > 0$.

To prove $I(t) > 0$ and $W(t) > 0 \forall t > 0$, with $I(0) > 0$ and $W(0) > 0$, the above process cannot be applied, hence another argument must be used. Let us consider the following sub-system of equation (1.2):

$$\begin{cases} \frac{dI}{dt} = \beta_s SI + \beta_w SW - (\gamma + \mu_I)I \\ \frac{dW}{dt} = \mu_W I - \varepsilon W \\ I(0) > 0, W(0) > 0. \end{cases} \quad (2.1)$$

Then, system (2.1) can be written as follows:

$$\dot{X}(t) = AX(t),$$

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 , we identify that it is Metzler matrix

and that its exponential is positive. Then, we deduce the positivity of I and W whenever $I(0) > 0$ and $W(0) > 0$. This shows the first point of our proposition. To prove the second point, let $N_1 = S + I$, then

$$\frac{dN_1}{dt} = \Lambda - \mu_s S - (\gamma + \mu_I)I \leq \Lambda - \mu_s N_1.$$

Hence,

$$\limsup_{t \rightarrow \infty} N_1(t) \leq \frac{\Lambda}{\mu_s}.$$

We deduce that S and I are uniformly bounded on Γ . From the boundedness of I and the third expression of equation (1.2), we obtain $\limsup_{t \rightarrow \infty} W(t) \leq \frac{\mu_W \Lambda}{\varepsilon \mu_s}$. This completes the proof. \square

The possible steady states of equation (1.2) are computed by finding solutions to the system and we have

- (1) The first equilibrium called disease-free equilibrium (DFE), which is given by $E_0 = (S_0, 0, 0) = \left(\frac{\Lambda}{\mu_s}, 0, 0\right)$.
- (2) The second equilibrium called endemic equilibrium is noted as $E_1 = (S^*, I^*, W^*)$, where $S^* = \frac{\varepsilon(\gamma + \mu_I)}{\varepsilon\beta_s + \beta_w\mu_W}$, $W^* = \frac{\mu_W}{\varepsilon} I^*$, and $I^* = \frac{\Lambda}{\gamma + \mu_I} - \frac{\varepsilon\mu_s}{\varepsilon\beta_s + \beta_w\mu_W}$. The endemic equilibrium E_1 is in the positive orthant reproduction number $R_0 > 1$ of equation (1.2), where

$$R_0 = \frac{\Lambda(\varepsilon\beta_s + \beta_w\mu_W)}{\mu_s\varepsilon(\gamma + \mu_I)} = R_{0d} + R_{0I}, \quad (2.2)$$

where $R_{0d} = \frac{\Lambda\beta_s}{\mu_s(\gamma + \mu_I)}$ represents the secondary infections caused directly by a single infective while $R_{0I} = \frac{\beta_w\mu_W}{\mu_s\varepsilon(\gamma + \mu_I)}$ represents the secondary infections caused indirectly through the environmental pathogen.

Proposition 2.2.

- If $R_0 \leq 1$, the model (1.2) has only one trivial equilibrium (DFE), i.e., $E_0 = (S_0, 0, 0)$.
- If $R_0 > 1$, the model (1.2) has two steady states: the first is the DFE, i.e., $E_0 = (S_0, 0, 0)$, and the second is the endemic equilibrium, i.e., $E_1 = (S^*, I^*, W^*)$.

3 Model without delay

Suppose $\tau = \nu = 0$, then the system (1.2) is written as follows:

$$\begin{cases} \frac{dS}{dt} = \Lambda - \beta_S SI - \beta_W SW - \mu_S S \\ \frac{dI}{dt} = \beta_S SI + \beta_W SW - (\gamma + \mu_I) I \\ \frac{dW}{dt} = \mu_W I - \varepsilon W \\ S(0) = S_0 \geq 0, \quad I(0) = I_0 \geq 0, \quad W(0) = W_0 \geq 0. \end{cases} \quad (3.1)$$

3.1 Asymptotic behavior analysis

The goal of this section is to study the stability of the possible steady states E_0 and E_1 .

By linearizing equation (3.1) around an arbitrary steady state $E = (S, I, W)$, we have the corresponding associated Jacobian matrix

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

Replacing E by E_0 and calculating the characteristic equation, we obtain

$$\det(\lambda I - J_{E_0}) = (\lambda + \mu_S)(\lambda^2 - \text{Tr}(J_{E_0}^{11})\lambda + \det(J_{E_0}^{11})), \quad (3.2)$$

where

$$J_{E_0}^{11} = \begin{pmatrix} \beta_S S_0 - \gamma - \mu_I & \beta_W S_0 \\ \mu_W & -\varepsilon \end{pmatrix},$$

$$\text{Tr}(J_{E_0}^{11}) = \beta_S S_0 - \gamma - \mu_I - \varepsilon,$$

and

$$\det(J_{E_0}^{11}) = \varepsilon(-\beta_S S_0 + \gamma + \mu_I) - \mu_W \beta_W S_0 = \varepsilon(\gamma + \mu_I)(1 - R_0).$$

Then, we obtain the following proposition.

Proposition 3.1. *If $R_0 < 1$, the trivial steady state (DFE) E_0 is asymptotically stable and unstable if $R_0 > 1$.*

Proof. If $R_0 < 1$, then $\text{Tr}(J_{E_0}^{11}) < 0$ and $\det(J_{E_0}^{11}) > 0$, as well as the characteristic equation (3.2) does not admit a real strictly positive root.

If $R_0 > 1$, then $\det(J_{E_0}^{11}) < 0$ and associated characteristic equation (3.2) has at least one positive solution. \square

Proposition 3.2. *If $R_0 > 1$, the nontrivial positive steady state (endemic equilibrium) E_1 is asymptotically stable.*

Proof. Considering $R_0 > 1$, the Jacobian matrix at E_1 is

$$J_{E^*=(S^*,I^*,W^*)} = \begin{pmatrix} -\frac{\Lambda}{S^*} & -\beta_S S^* & -\beta_W S^* \\ (\gamma + \mu_I) \frac{I^*}{S^*} & -\beta_W \frac{S^* W^*}{I^*} & \beta_W S^* \\ 0 & \mu_W & -\varepsilon \end{pmatrix},$$

and the characteristic equation associated to E_1 is as follows:

$$P(\lambda) = \lambda^3 + a_2 \lambda + a_1 \lambda + a_0, \quad (3.3)$$

where

$$\begin{aligned} a_2 &= \varepsilon + \beta_W \frac{S^* W^*}{I^*} + \frac{\Lambda}{S^*} \\ a_1 &= \frac{\Lambda}{S^*} \left(\varepsilon + \beta_W \frac{S^* W^*}{I^*} \right) + \beta_S (\gamma + \mu_I) I^* \\ a_0 &= (\gamma + \mu_I) (\varepsilon \beta_S + \mu_W \beta_W) I^*. \end{aligned}$$

All these coefficients are strictly positives if $R_0 > 1$, then the polynomial P does not admit any real strictly positive solution. \square

Proposition 3.3. *If $R_0 > 1$, the nontrivial positive steady state (endemic equilibrium) E_1 is globally asymptotically stable.*

Proof. Considering the following function

$$V(t) = S(t) - S^* - \int_{S^*}^{S(t)} \frac{S^*}{Z} dZ + I^* \Phi \left(\frac{I(t)}{I^*} \right) + \frac{\beta_W S^* W^*}{\varepsilon} \Phi \left(\frac{W(t)}{W^*} \right), \quad (3.4)$$

where $\Phi(Z) = Z - 1 - \ln Z > 0$, for $Z > 0$. Then, V defines a Lyapunov function. It is easy to see that Φ has a strict global minimum at 1 and $\Phi(1) = 0$ and $\Phi(Z) > 0$. Then, V is nonnegative function.

Noting $\psi = \psi(t)$ for any $\psi \in \{S, I, W\}$ and differentiating V along the solutions of equation (3.1) over time t , we obtain

$$\dot{V}(t)|_{(3.1)} = \left(1 - \frac{S^*}{S} \right) \frac{dS}{dt} + \left(1 - \frac{I^*}{I} \right) \frac{dI}{dt} + \frac{\beta_W S^*}{\varepsilon} \left(1 - \frac{W^*}{W} \right) \frac{dW}{dt}. \quad (3.5)$$

Using $\Lambda = \mu_S S^* + (\gamma + \mu_I) I^*$ and $\mu_W I^* = \varepsilon W^*$, we obtain

$$\begin{aligned} \dot{V}(t)|_{(3.1)} &= \mu_S S^* \left(1 - \frac{S}{S^*} \right) \left(1 - \frac{S^*}{S} \right) + \beta_S S^* I^* \left(2 - \frac{S^*}{S} - \frac{S}{S^*} \right) \\ &\quad + \beta_W S^* W^* \left(3 - \frac{S^*}{S} - \frac{S W^* I^*}{S^* W^* I^*} - \frac{W^* I^*}{W I^*} \right). \end{aligned} \quad (3.6)$$

Thus,

$$\begin{aligned} \dot{V}(t)|_{(3.1)} &= \mu_S S^* \left(1 - \frac{S}{S^*} \right) \left(1 - \frac{S^*}{S} \right) - \beta_S S^* I^* \left[\Phi \left(\frac{S^*}{S} \right) + \Phi \left(\frac{S}{S^*} \right) \right] \\ &\quad - \beta_W S^* W^* \left[\Phi \left(\frac{S^*}{S} \right) + \Phi \left(\frac{W^* I^*}{W I^*} \right) + \Phi \left(\frac{S W^* I^*}{S^* W^* I^*} \right) \right] \\ &= \mu_S S^* \left(1 - \frac{S}{S^*} \right) \left(1 - \frac{S^*}{S} \right) - \beta_S S^* I^* \left[2 \Phi \left(\frac{S^*}{S} \right) + \Phi \left(\frac{S}{S^*} \right) + \Phi \left(\frac{W^* I^*}{W I^*} \right) + \Phi \left(\frac{S W^* I^*}{S^* W^* I^*} \right) \right]. \end{aligned} \quad (3.7)$$

Since $\left(1 - \frac{S}{S^*} \right) \left(1 - \frac{S^*}{S} \right) \leq 0$ and $\Phi(Z) \geq 0$ for $Z > 0$, we deduce that $\dot{V}(t)|_{(3.1)} \leq 0$ and the equality occurs at the endemic equilibrium E_1 . Consequently, the global asymptotic stability of E_1 follows from LaSalle's invariance principle [24]. \square

3.2 Bifurcation analysis at $R_0 = 1$

In this section, we analyze and study the occurrence of a transcritical bifurcation at $R_0 = 1$.

Theorem 3.4. *The trivial steady state (DFE) E_0 of equation (3.1) changes its stability from stable to unstable when $R_0 = 1$, and a transcritical bifurcation occurs at the critical value $\Lambda = \Lambda^* = \frac{\mu_S \varepsilon (\gamma + \mu_I)}{\varepsilon \beta_S + \beta_W \mu_W}$.*

Proof. The linearized matrix of system (3.1) around E_0 at the bifurcation parameter value $\Lambda = \Lambda^* = \frac{\mu_S \varepsilon (\gamma + \mu_I)}{\varepsilon \beta_S + \beta_W \mu_W}$ is given as follows:

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

The matrix J has a simple eigenvalue equal to zero at $R_0 = 1$, (see characteristic equation 3.2) and the other eigenvalues have negative real part. As the linearization method cannot give us any information about the asymptotic behaviors of system (3.1), we will use the center manifold theory, and from Theorem 1 of the study by Castillo-Chavez and Song [4], the bifurcation elements a_1 and b_1 are defined as follows:

$$a_1 = \sum_{k,i,j=1}^n v_k w_i w_j \left(\frac{\partial^2 f_k}{\partial x_i \partial x_j} \right)_{E_0} \quad (3.8)$$

and

$$b_1 = \sum_{k,i=1}^n v_k w_i \left(\frac{\partial^2 f_k}{\partial x_i \partial \Lambda^*} \right)_{E_0}. \quad (3.9)$$

A right eigenvector associated with 0 eigenvalue is

$$w = \left[\frac{-\Lambda^* (\beta_S \varepsilon + \beta_W \mu_W)}{\mu_S}, \frac{\varepsilon}{\mu_W}, 1 \right]^T,$$

and the other eigenvector (left) v verifying $v \cdot w = 1$ is

$$v = \left[0, \frac{\mu_W \mu_S + \Lambda^* (\beta_S \varepsilon + \beta_W \mu_W)}{\varepsilon \mu_S}, \frac{-\Lambda^* (\beta_S \varepsilon + \beta_W \mu_W)}{\mu_S} \right].$$

A direct computation shows that

$$\frac{\partial^2 f_1}{\partial x_1 \partial x_2} = -\beta_S, \quad \frac{\partial^2 f_1}{\partial x_1 \partial x_3} = -\beta_W, \quad \frac{\partial^2 f_2}{\partial x_1 \partial x_2} = \beta_S, \quad \frac{\partial^2 f_1}{\partial x_1 \partial x_3} = \beta_W, \quad \text{and} \quad \frac{\partial^2 f_2}{\partial x_1 \partial \Lambda^*} = \frac{\beta_W}{\mu_S}.$$

The second derivatives in equations (3.8) and (3.9) are equal to zero. Then,

$$\begin{aligned} a_1 &= v_2 w_1 w_2 \left(\frac{\partial^2 f_2}{\partial x_1 \partial x_2} \right) + v_2 w_1 w_3 \left(\frac{\partial^2 f_2}{\partial x_1 \partial x_3} \right) \\ &= \frac{-\Lambda^* (\beta_S \varepsilon + \beta_W \mu_W)}{\mu_S} \left(\frac{\beta_S \varepsilon + \beta_W \mu_W}{\mu_W} \right) \frac{\mu_W \mu_S + \Lambda^* (\beta_S \varepsilon + \beta_W \mu_W)}{\varepsilon \mu_S} \left(\frac{\varepsilon \beta_S}{\mu_W} + \beta_W \right) \\ &= \frac{-\Lambda^* (\beta_S \varepsilon + \beta_W \mu_W)^2}{\mu_S} \frac{\mu_W \mu_S + \Lambda^* (\beta_S \varepsilon + \beta_W \mu_W)}{\varepsilon \mu_S} < 0. \\ b_1 &= v_2 w_2 \left(\frac{\partial^2 f_2}{\partial x_1 \partial \Lambda^*} \right) \\ &= \frac{\mu_W \mu_S + \Lambda^* (\beta_S \varepsilon + \beta_W \mu_W)}{\varepsilon \mu_S} \frac{\varepsilon \beta_W}{\mu_W \mu_S} > 0. \end{aligned}$$

This proves that at $R_0 = 1$, the trivial steady state (DFE) E_0 changes its stability from stable to unstable, and the nontrivial steady state (endemic equilibrium) E_1 always exists at $R_0 = 1$. Then, a transcritical bifurcation occurs at the critical value $R_0 = 1$. \square

4 Model with one delay τ

If $\tau > 0$ and $\nu = 0$, then system (1.1) is written as follows:

$$\begin{cases} \frac{dS}{dt} = \Lambda - \beta_S S I_\tau - \beta_W S W - \mu_S S \\ \frac{dI}{dt} = \beta_S S I_\tau + \beta_W S W - (\gamma + \mu_I) I \\ \frac{dW}{dt} = \mu_W I - \varepsilon W \\ S(0) = S_0 \geq 0, I(s) = \varphi(s), s \in [-\tau, 0], W(0) = W_0 \geq 0. \end{cases} \quad (4.1)$$

4.1 Stability of trivial equilibrium (DFE)

By linearizing system (4.1) around $E_0 = (S_0, 0, 0)$, we obtain

$$\frac{dX}{dt} = L_0 X + L_\tau X_\tau,$$

where

$$X = \begin{pmatrix} S \\ I \\ W \end{pmatrix}, \quad L_0 = \begin{pmatrix} -\mu_S & 0 & -\beta_W S_0 \\ 0 & -(\mu_I + \gamma) & \beta_W S_0 \\ 0 & \mu_W & -\varepsilon \end{pmatrix} \quad \text{and} \quad L_\tau = \begin{pmatrix} 0 & -\beta_S S_0 & 0 \\ 0 & \beta_S S_0 & 0 \\ 0 & 0 & 0 \end{pmatrix}.$$

Then, we obtain the following associated characteristic equation:

$$\Delta(\lambda) = \det(\lambda I - L_0 - e^{-\lambda\tau} L_\tau) = (\lambda + \mu_S)[P_0(\lambda) + Q_0(\lambda)e^{-\lambda\tau}] = 0.$$

As $\lambda_1 = -\mu_S$ is a root of the characteristic equation, the stability of E_0 is deduced from the study of the following equation:

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

where

$$\begin{aligned} P_0(\lambda) &= \lambda^2 + (\gamma + \mu_I + \varepsilon)\lambda + \varepsilon(\gamma + \mu_I) - \mu_W \beta_W S_0, \\ Q_0(\lambda) &= -\beta_S S_0 \lambda - \varepsilon \beta_S S_0. \end{aligned}$$

Define F by $F(y) = |P_0(iy)|^2 - |Q_0(iy)|^2$ (see [8]), then we have

$$F(Y) = Y^2 + b_1 Y + b_0,$$

with $Y = y^2$, and

$$\begin{aligned} b_1 &= (\gamma + \mu_I)^2 + (\beta_S S_0)^2 + \varepsilon^2 + 2\mu_W \beta_W S_0, \\ b_0 &= (\varepsilon(\gamma + \mu_I) - \mu_W \beta_W S_0)^2 + (\varepsilon \beta_S S_0)^2. \end{aligned}$$

Since $b_1 > 0$ and $b_2 > 0$, 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 proposition:

Proposition 4.1. *The trivial steady state (DFE) E_0 is asymptotically stable for all time delays $\tau > 0$.*

4.2 Stability of nontrivial steady state

Next, we study the stability of the nontrivial positive steady state (endemic equilibrium) $E_1 = (S^*, I^*, W^*)$ of system (4.1) by considering the latency period.

The linearized system of equation (4.1) around E_1 is given as follows:

$$\frac{dX}{dt} = J_0 X + J_\tau X_\tau,$$

where

$$X = \begin{pmatrix} S \\ I \\ W \end{pmatrix}, \quad J_0 = \begin{pmatrix} -\beta_S I^* - \beta_W W^* - \mu_S & 0 & -\beta_W S^* \\ \beta_S I^* + \beta_W W^* & -(\mu_I + \gamma) & \beta_W S^* \\ 0 & \mu_W & -\varepsilon \end{pmatrix}, \quad \text{and} \quad J_\tau = \begin{pmatrix} 0 & -\beta_S S^* & 0 \\ 0 & \beta_S S^* & 0 \\ 0 & 0 & 0 \end{pmatrix}.$$

Then, we obtain the following associated characteristic equation to E_1

$$P_1(\lambda) + Q_1(\lambda)e^{-\lambda\tau} = 0, \quad (4.2)$$

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

$$c_2 = -\beta_S S^*, \\ c_1 = -(\varepsilon + \mu_S) \beta_S S^*, \\ c_0 = -\varepsilon \beta_S \mu_S S^*.$$

As the nontrivial steady state (endemic equilibrium) E_1 is asymptotically stable for $\tau = 0$ (see Proposition 3.2), it is still asymptotically stable for small time delays $\tau > 0$ [3,8] or for all time delays $\tau > 0$. Then, one needs to find the switch of stability, which requires finding a root of the characteristic equation with a purely imaginary part for some critical value τ_c of time delay τ .

Let us replace λ by $i\omega$ ($\omega > 0$) in equation (4.2), then we obtain

$$-i\omega^3 - \alpha_2 \omega^2 + i\alpha_1 \omega + \alpha_0 - (c_2 \omega^2 - i c_1 \omega - c_0)(\cos \omega \tau - i \sin \omega \tau) = 0. \quad (4.3)$$

By separating the real and imaginary parts, we have

$$\begin{cases} \omega^3 - \alpha_1 \omega = c_1 \omega \cos \omega \tau + (c_2 \omega^2 - c_0) \sin \omega \tau \\ \alpha_2 \omega^2 - \alpha_0 = -(c_2 \omega^2 - c_0) \cos \omega \tau + c_1 \omega \sin \omega \tau. \end{cases} \quad (4.4)$$

A direct computation gives us

$$\omega^6 + B_2\omega^4 + B_1\omega^2 + B_0 = 0. \quad (4.5)$$

Let $z = \omega^2$, then equation (4.5) becomes

$$f(z) = z^3 + B_2z^2 + B_1z + B_0 = 0, \quad (4.6)$$

where

$$\begin{aligned} B_2 &= \alpha_2^2 - c_2^2 - 2\alpha_1, \\ B_1 &= 2(c_0c_2 - \alpha_0\alpha_2) - (c_1^2 - \alpha_1^2), \\ B_0 &= \alpha_0^2 - c_0^2. \end{aligned}$$

Let the hypothesis

$$(H) : B_0 > 0, \quad B_2 > 0 \quad \text{and} \quad B_2B_1 - B_0 > 0. \quad (4.7)$$

Proposition 4.2. *If $R_0 > 1$ and (H) are satisfied, then the nontrivial steady state (endemic equilibrium) E_1 is asymptotically stable for all time delays $\tau > 0$.*

Proof. The proof is based on the well-known Routh-Hurwitz stability criterion [6]. □

5 Model with two delays $\tau = \nu > 0$

We consider $\tau = \nu > 0$, then the model (1.2) is written as follows:

$$\begin{cases} \frac{dS}{dt} = \Lambda - \beta_S SI_\tau - \beta_W SW_\tau - \mu_S S \\ \frac{dI}{dt} = \beta_S SI_\tau + \beta_W SW_\tau - (\gamma + \mu_I) I \\ \frac{dW}{dt} = \mu_W I - \varepsilon W \\ S(0) = S_0 \geq 0, I(s) = \varphi(s), W(s) = \xi(s), s \in [-\tau, 0]. \end{cases} \quad (5.1)$$

5.1 Local stability of E_0

Linearizing the system (5.1) around the DFE E_0 , we obtain

$$\frac{dX}{dt} = M_0 X + M_\tau X_\tau,$$

where

$$X = \begin{pmatrix} S \\ I \\ W \end{pmatrix}, \quad M_0 = \begin{pmatrix} -\mu_S & 0 & 0 \\ 0 & -(\gamma + \mu_I) & 0 \\ 0 & \mu_W & -\varepsilon \end{pmatrix}, \quad \text{and} \quad M_\tau = \begin{pmatrix} 0 & -\beta_S S_0 & -\beta_W S_0 \\ 0 & \beta_S S_0 & \beta_W S_0 \\ 0 & 0 & 0 \end{pmatrix},$$

and the corresponding characteristic equation is

$$\det(\lambda I - M_0 - e^{-\lambda\tau} M_\tau) = (\lambda + \mu_S)[P_2(\lambda) + Q_2(\lambda)e^{-\lambda\tau}] = 0. \quad (5.2)$$

Since $\lambda_1 = -\mu_S$ is a solution of the characteristic equation, then the stability of E_0 is deduced from

$$P_2(\lambda) + Q_2(\lambda)e^{-\lambda\tau} = 0, \quad (5.3)$$

with

$$\begin{aligned} P_2(\lambda) &= \lambda^2 + (\gamma + \mu_I + \varepsilon)\lambda + \varepsilon(\gamma + \mu_I), \\ Q_2(\lambda) &= -S_0(\beta_S\lambda + \varepsilon\beta_S + \mu_W\beta_W). \end{aligned}$$

Define G_2 by $G_2(y) = |P_2(iy)|^2 - |Q_2(iy)|^2$, and we have

$$G_2(Y) = Y^2 + d_1Y + d_0, \quad (5.4)$$

with $Y = y^2$, and

$$\begin{aligned} d_1 &= \gamma + \mu_I + \varepsilon + (S_0\beta_S)^2 - 2\varepsilon(\gamma + \mu_I), \\ d_0 &= \varepsilon^2(\gamma + \mu_I)^2 - \left(\frac{\Lambda}{\mu_S}\right)^2 (\varepsilon\beta_S + \mu_W\beta_W)^2, \\ &= \varepsilon^2(\gamma + \mu_I)^2(1 - R_0^2). \end{aligned}$$

Note that if $R_0 > 1$, then $G_2(0) = d_0 < 0$. As the function G_2 is continuous and $\lim_{Y \rightarrow \infty} G_2(Y) = +\infty$, then equation (5.4) has at least one positive root, denoted by Y_0 . Consequently, equation (5.2) has two conjugate roots with a purely imaginary part $\pm iy_0$. Hence, we obtain the following proposition.

Proposition 5.1. *The trivial steady state (DFE) E_0 is stable if $R_0 \leq 1$ and unstable for all time delays $\tau > 0$ when $R_0 > 1$.*

5.2 Local stability of E_1

In this section, we analyze the asymptotic behaviors of the nontrivial steady state (endemic equilibrium) E_1 .

Proposition 5.2. *If $R_0 > 1$ and (H_3) are satisfied, then the nontrivial steady state (endemic equilibrium) E_1 is asymptotically stable for all time delays $\tau = \nu > 0$.*

Proof. By linearizing system (5.1) at E_1 , we obtain

$$\frac{dX}{dt} = J_0^*X + J_\tau^*X_\tau,$$

where

$$X = \begin{pmatrix} S \\ I \\ W \end{pmatrix}, \quad J_0^* = \begin{pmatrix} -\beta_S I^* - \beta_W W^* - \mu_S & 0 & 0 \\ \beta_S I^* + \beta_W W^* & -(\gamma + \mu_I) & 0 \\ 0 & \mu_W & -\varepsilon \end{pmatrix}, \quad \text{and} \quad J_\tau^* = \begin{pmatrix} 0 & -\beta_S S^* & -\beta_W S^* \\ 0 & \beta_S S^* & \beta_W S^* \\ 0 & 0 & 0 \end{pmatrix}.$$

We obtain the following characteristic equation:

$$P^*(\lambda) + Q^*(\lambda)e^{-\lambda\tau} = 0, \quad (5.5)$$

with

$$\begin{aligned} P^*(\lambda) &= \lambda^3 + \alpha_2^*\lambda^2 + \alpha_1^*\lambda + \alpha_0^*, \\ Q^*(\lambda) &= \delta_2^*\lambda^2 + \delta_1^*\lambda + \delta_0^*, \end{aligned}$$

where

Table 1: Estimation of parameters

Parameter	Value
Λ	0–0.7
β_S	0.09
β_W	0–0.7
μ_S	0.03
γ	0.02
μ_I	0.09
μ_W	0.03
ε	0–0.5
τ	Positive value
ν	Positive value

$$\alpha_2^* = \beta_S I^* + \beta_W W^* + \mu_S + \gamma + \mu_I + \varepsilon,$$

$$\alpha_1^* = (\mu_S + \beta_S I^* + \beta_W W^*)(\varepsilon + \gamma + \mu_I) + \varepsilon(\mu_I + \gamma),$$

$$\alpha_0^* = \varepsilon(\mu_I + \gamma)(\beta_S I^* + \beta_W W^* + \mu_S),$$

Table 2: Sensitivity index of R_0

Parameter	Sensitivity index	Index at parameters value
Λ	+1	+1
β_S	$\frac{\varepsilon\beta_S}{\varepsilon\beta_S + \beta_W\mu_W}$	0.7500
β_W	$\frac{\mu_W\beta_W}{\varepsilon\beta_S + \beta_W\mu_W}$	0.25
μ_S	-1	-1
γ	$-\frac{\gamma}{\gamma + \mu_I}$	-0.1818
μ_I	$-\frac{\mu_I}{\gamma + \mu_I}$	-0.8182
μ_W	$\frac{\beta_W}{\varepsilon\beta_S + \beta_W\mu_W}$	8.3333
ε	$\frac{\varepsilon\beta_S}{\varepsilon\beta_S + \beta_W\mu_W} \frac{\gamma}{\gamma + \mu_I}$	0.5682

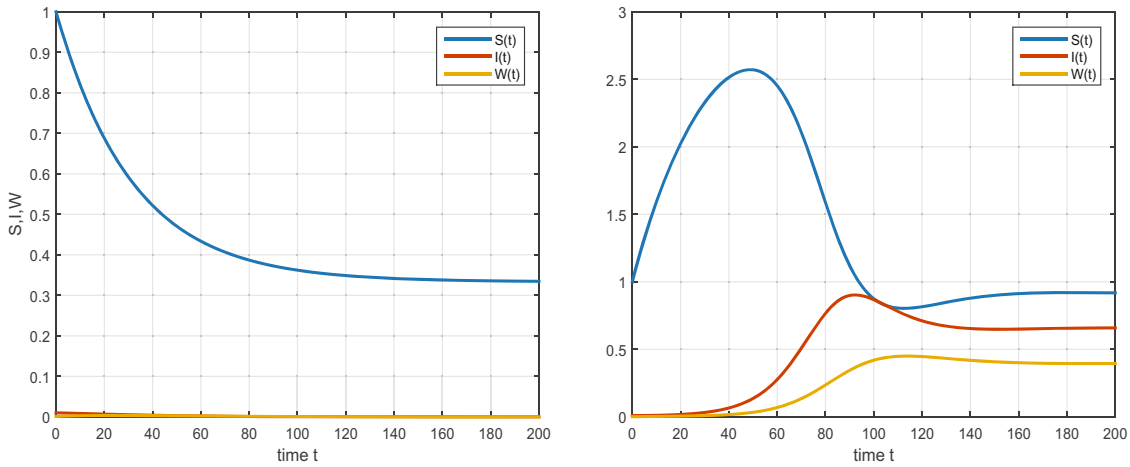


Figure 1: Stability of E_0 and nonexistence of E_1 for $\tau \geq 0$ and $\nu \geq 0$ (left). Instability of E_0 and stability of E_1 for $\tau \geq 0$ and $\nu \geq 0$ (right).

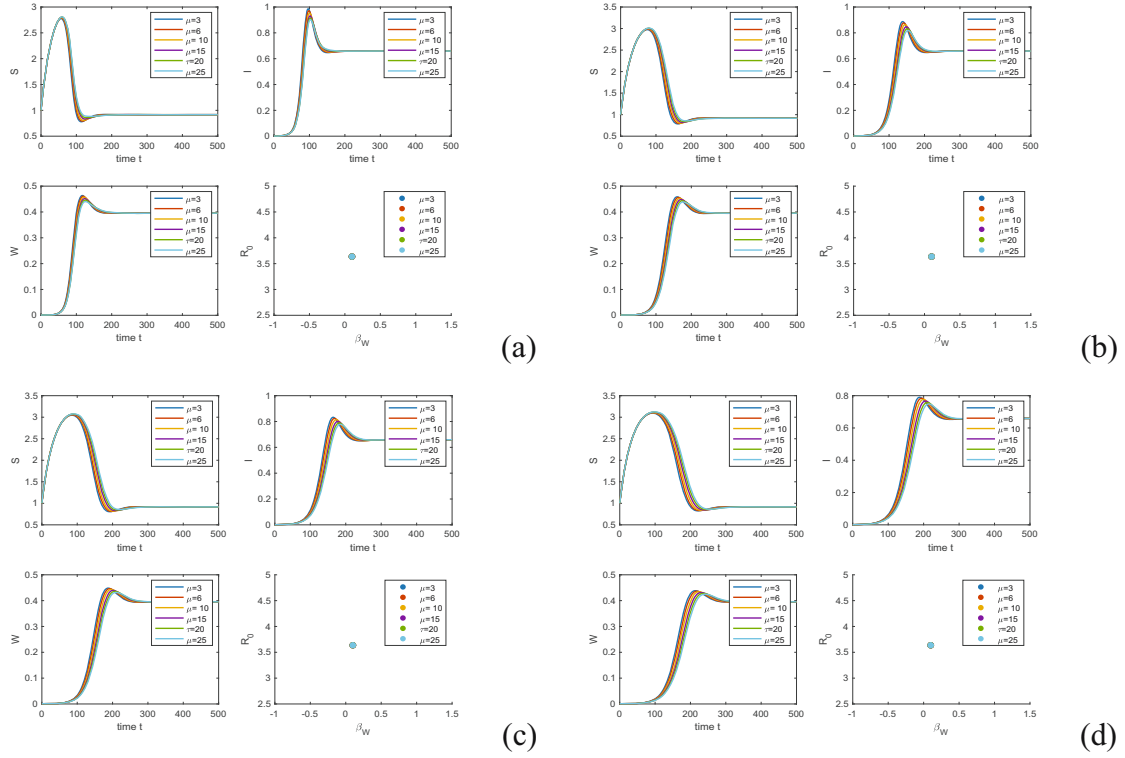


Figure 2: Temporal evolution of S , I , and W for different values of time delay τ : (a) $\mu = 3$, (b) $\mu = 10$, (c) $\mu = 15$, and (d) $\mu = 20$. If τ increases, the number R_0 is constant, and the endemic equilibrium remains asymptotically stable for all $\tau > 0$.

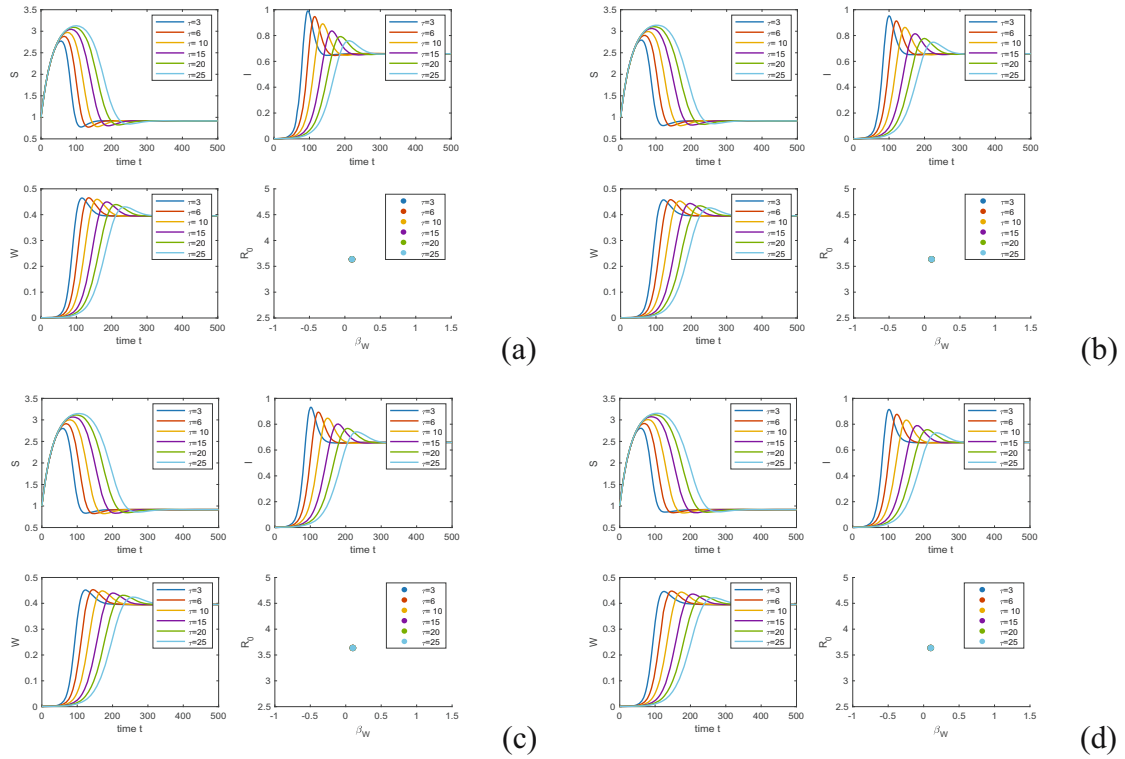


Figure 3: Temporal evolution of S , I , and W for different values of time delay μ : (a) $\tau = 3$, (b) $\tau = 10$, (c) $\tau = 15$, and (d) $\tau = 20$. If Δ increases, the number R_0 is constant, and the endemic equilibrium remains asymptotically stable for all $\mu > 0$.

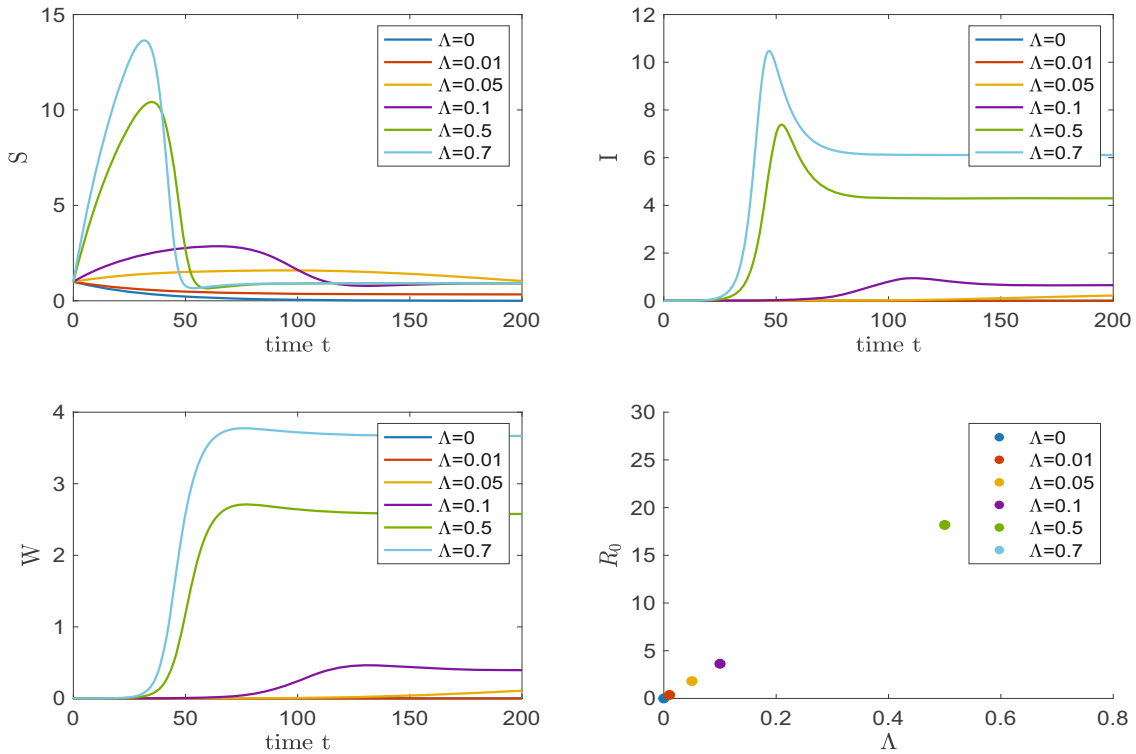


Figure 4: Temporal evolution of S , I , and W for different values of Λ . If Λ increases, the number R_0 increases, which implies an increase in the infectious population and the virus concentration.

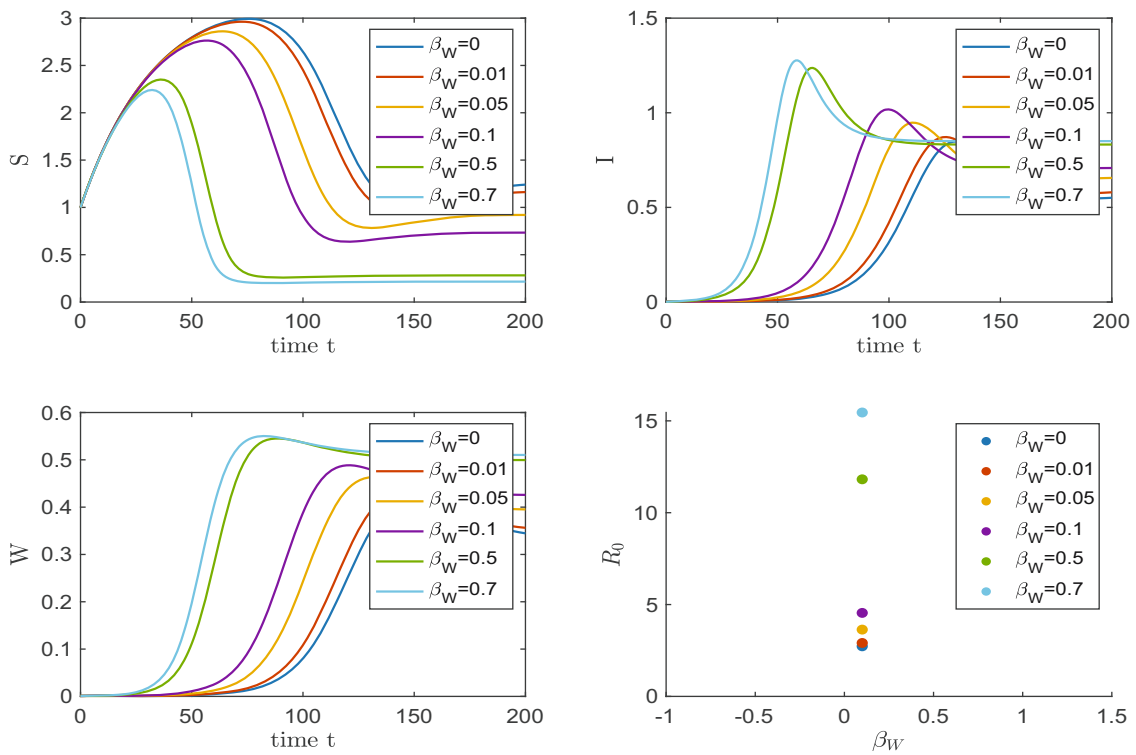


Figure 5: Temporal evolution of S , I , and W for different values of β_W . If β_W increases, the number R_0 increases, which implies an increase in the infectious population and the virus concentration.

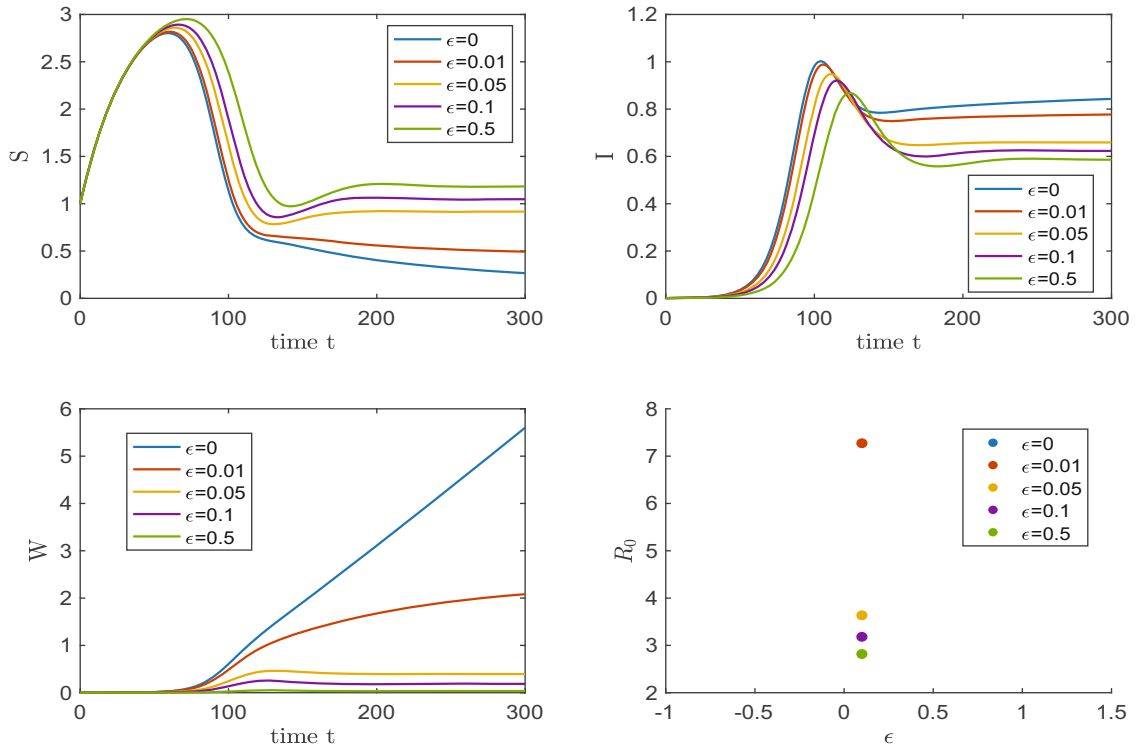


Figure 6: Temporal evolution of S , I , and W for different values of ϵ . If ϵ increases, the number R_0 decreases, which implies a decrease in the infectious population and the virus concentration.

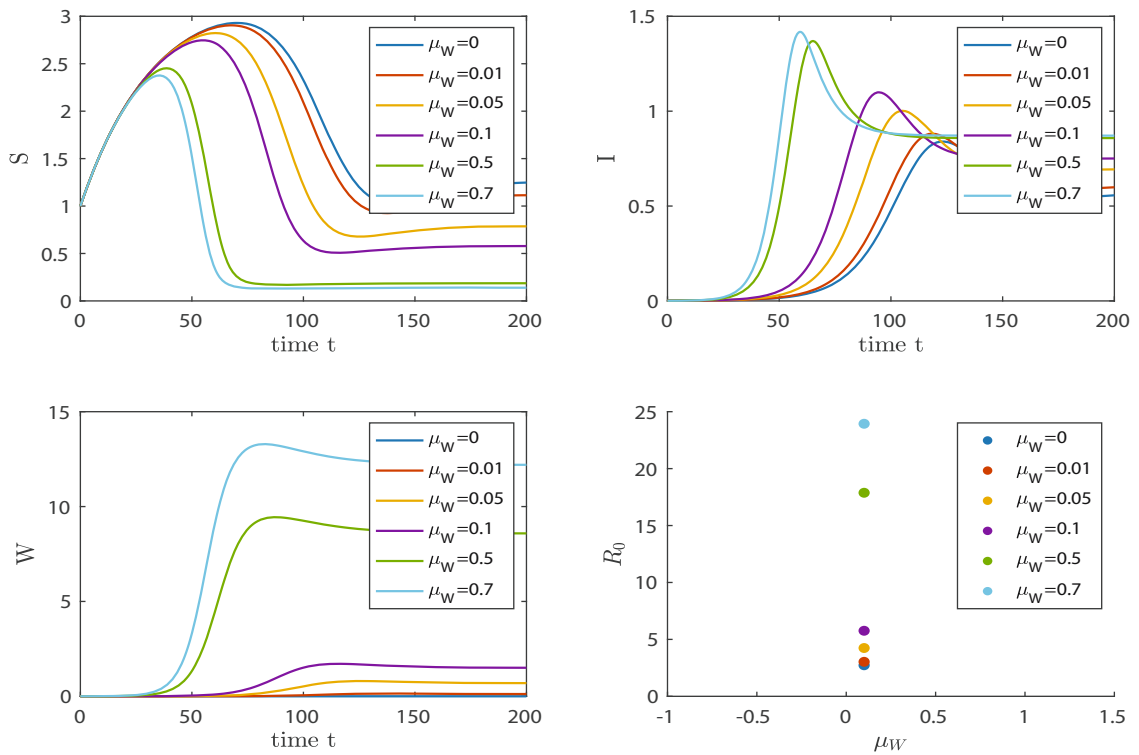


Figure 7: Temporal evolution of S , I , and W for different values of μ_W . If μ_W increases, the number R_0 increases, which implies an increase in the infected population and the virus concentration.

and

$$\begin{aligned}\delta_2 &= -\beta_S S^*, \\ \delta_1 &= -(\beta_S S^*(\beta_S I^* + \beta_W W^* + \mu_S + \varepsilon) + \mu_W \beta_W S^*), \\ \delta_0 &= (\varepsilon - 1)\beta_S S^*(\beta_S I^* + \beta_W W^*) - \mu_S(\beta_S S^* + \mu_W \beta_W W^*).\end{aligned}$$

Since the nontrivial steady state (endemic equilibrium) E_1 is asymptotically stable for $\tau = 0$, from Proposition 3.2 and by the continuity property, it is still asymptotically stable for small $\tau > 0$ or for all $\tau > 0$. To study the switch of stability, let $i\omega(\omega > 0)$ be a root of equation (5.5), then we obtain the following equation:

$$-i\omega^3 - \alpha_2^* \omega^2 + \alpha_1^* i\omega + \alpha_0^* + (-\delta_2 \omega^2 + \delta_1 i\omega + \delta_0)(\cos \omega\tau - i \sin \omega\tau) = 0. \quad (5.6)$$

By separating the real and imaginary parts, we obtain

$$\begin{cases} \omega^3 - \alpha_1^* \omega = \delta_2 \omega^2 \sin \omega\tau + \delta_1 \omega \cos \omega\tau - \delta_0 \sin \omega\tau \\ \alpha_2^* \omega^2 - \alpha_0^* = -\delta_2 \omega^2 \cos \omega\tau + \delta_0 \cos \omega\tau + \delta_1 \omega \sin \omega\tau. \end{cases} \quad (5.7)$$

It is easy to have

$$\omega^6 + A_2^* \omega^4 + A_1^* \omega^2 + A_0^* = 0. \quad (5.8)$$

Let $z = \omega^2$, then equation (5.8) becomes

$$h(z) = z^3 + A_2^* z^2 + A_1^* z + A_0^* = 0, \quad (5.9)$$

where

$$\begin{aligned}A_2^* &= (\alpha_2^*)^2 - 2\alpha_1^*, \\ &= (\beta_S I^* + \beta_W W^* + \mu_S)^2 + (\gamma + \mu_I)^2 + \varepsilon^2, \\ A_1^* &= (\alpha_1^*)^2 - \delta_1^2 - \delta_2^2 + 2(\delta_0 \delta_2 - \alpha_0^* \alpha_2^*), \\ A_0^* &= (\alpha_0^*)^2 - \delta_0^2.\end{aligned}$$

Since $A_2^* > 0$, we consider the following hypotheses:

$$(H_3) : A_0^* > 0 \quad \text{and} \quad A_2^* A_1^* - A_0^* > 0. \quad (5.10)$$

From the hypothesis (H_3) and the Routh-Hurwitz stability criterion [6], equation (5.9) has no positive root. Therefore, all roots of the characteristic equation (5.5) stay in the left half complex plane for all $\tau = \nu > 0$. This completes the proof. \square

6 Sensitivity analysis

The sensitivity analysis for the basic reproduction number [equation (2.2)] gives us information about the influence of each parameter on the transmission and the spreading of the disease [33]. It is also used to detect and determine which parameter has high impact on the epidemiological threshold R_0 and will be targeted by controlling strategies. To do that, one needs to use the normalized sensitivity index of R_0 with respect to a given parameter model, which is defined as follows.

Definition 6.1. [31] The normalized sensitivity index of R_0 , which is differentiable to a given parameter model θ , is given as follows:

$$Y_\theta^{R_0} = \frac{\partial R_0}{\partial \theta} \frac{\theta}{R_0}.$$

The sensitivity indices for each parameter model defined in Table 1 are summarized in Table 2.

From Table 2, we can detect the parameters that have a significant impact on the basic reproduction number R_0 and are classified as follows: Λ , β_S , μ_S , μ_I , and μ_W . In a simple manner, an increase of the parameter β_S by (100%) will increase R_0 by 75%, and this can be applied to the other parameters of the model.

7 Numerical simulations

In this section, and via MATLAB software, we give some numerical simulations to illustrate our analytical results. The used parameters and related values are summarized in Table 1. Here, we assume that W is of the same scale as the infectious population and decreases over time if no further input occurs (Figure 1).

Remark 7.1. As the study of the characteristic equation with two different delays becomes is more complicated, numerical simulations show that the asymptotic behavior of equilibria remains the same for $\tau \neq \nu$, as illustrated in Figures 2 and 3.

8 Conclusion

In order to study the role of indirect transmission, we proposed a mathematical model that takes into account the effects of four arguments: direct and indirect transmissions and their transmission delays from infectious individuals and the environment to susceptible individuals. We proved the positivity and boundedness of solutions and the asymptotic behaviors of the possible steady state. We also proved the occurrence of a transcritical bifurcation at the critical value of the basic reproduction number $R_0 = 1$. To our best knowledge, only a few articles have treated this situation. From the sensitivity analysis part and Figures 4–7, we conclude that the best strategy to stop the propagation of infectious diseases is to decrease the concentration of environmental viruses.

Funding information: This research was supported by National Center for Scientific and Technical Research (CNRST) (Cov/2020/102).

Conflict of interest: The authors state that there are no conflicts of interest.

Data availability statement: The datasets generated during and/or analysed during the current study are available from the corresponding author on reasonable request.

References

- [1] B. Alnseba, B. Chahrazed, and M. Pierre, *A model for ovine brucellosis incorporating direct and indirect transmission*, J. Biol. Dyn. **4** (2010), 2–11.
- [2] I. Al-Darabsah and Y. Yuan, *A time-delayed epidemic model for Ebola disease transmission*. Appl. Math. Comput. **290** (2016), 307–325.
- [3] F. G. Boese, *Stability with respect to the delay: on a paper of K. L. Cooke and P. van den Driessche*. J. Math. Anal. App. **228** (1998), no. 2, 293–321.
- [4] C. Castillo-Chavez and B. Song, *Dynamical models of tuberculosis and their applications. The reemergence of tuberculosis*. Math. Biosci. Eng. **1** (2004), no. 2, 361–404.
- [5] A. Cheddour and A. Elazzouzi, *Optimal feedback control for a class of infinite dimensional semilinear systems with distributed delay*, Syst. Control Lett. **179** (2023), 105600.
- [6] N. G. Chebotarev and N. N. Meiman, *The Routh-Hurwitz problem for polynomials and entire functions*, Trudy Mat. Inst. Steklov **26** (1949), 3–31.
- [7] C. T. Codeço, *Endemic and epidemic dynamics of cholera: The role of the aquatic reservoir*, BMC Infect. Dis. **1** (2001), no. 1, 1–4.

- [8] K. L. Cooke and P. van den Driessche, *On the zeroes of some transcendental equations*, Funkcial. Ekvac. **29** (1986), 77–90.
- [9] M. J. Corbel, *Brucellosis in Humans and Animals*, World Health Organization, Philippines, 2006.
- [10] S. Dickson, S. Padmasekaran, G. E. Chatzarakis, and S. L. Panetos, *SQIRV model for Omicron variant with time delay*, Aust. J. Math. Anal. Appl., **19** (2022), no. 2, 16666.
- [11] S. F. Dowell, *Seasonal variation in host susceptibility and cycles of certain infectious diseases*, Emerg. Infect. Dis. **7** (2001), 369–374.
- [12] M. C. Eisenberga, Z. S. Shuai, J. H. Tien, and P. van den Driessche, *A cholera model in a patchy environment with water and human movement*, Math. Biosci. **246** (2013), 105–112.
- [13] H. Guo, M. Y. Li, and Z. Shuai, *Global stability of the endemic equilibrium of multigroup SIR epidemic models*, Can. Appl. Math. Q. **14** (2006), 259–284.
- [14] H. Guo, M. Y. Li, and Z. Shuai, *A graph-theoretic approach to the method of global Lyapunov functions*, Proc. Amer. Math. Soc. **136** (2008), 2793–2802.
- [15] Q. Hou, X. D. Sun, J. Zhang, Y. J. Liu, Y. M. Wang, and Z. Jin, *Modeling the transmission dynamics of brucellosis in inner Mongolia autonomous region, China*, Math. Biosci. **242** (2013), 51–58.
- [16] Q. Hou, X. D. Sun, Y. M. Wang, B. X. Huang, and Z. Jin, *Global properties of a general dynamic model for animal diseases: A case study of brucellosis and tuberculosis transmission*, J. Math. Anal. Appl. **414** (2014), 424–433.
- [17] C. B. Jonsson, L. T. M. Figueiredo, and O. Vapalahti, *A global perspective on hantavirus ecology, epidemiology, and disease*, Clin. Microbiol. Rev. **23** (2010), no. 2, 412.
- [18] E. R. Kallio, J. Klingstro, E. Gustafsson, T. Manni, A. Vaheri, H. Henttonen, et al., *Prolonged survival of Puumala hantavirus outside the host: Evidence for indirect transmission via the environment*, J. Gen. Virol. **87** (2006), 2127–2134.
- [19] S. Kayan, H. Merdan, R. Yafia, and S. Goktepe, *Bifurcation analysis of a modified tumor-immune system interaction model involving time delay*, Math. Modell. Natural Phenomena **12** (2017), no. 5, 120–145.
- [20] E. Kenah, D. L. Chao, L. Matrajt, M. E. Halloran, and I. M. Longini Jr., *The global transmission and control of influenza*, PLoS ONE **6** (2011), no. 5, e19515.
- [21] A. Kiszewski, A. Mellinger, A. Spielman, P. Malaney, S. E. Sachs, and J. Sachs, *A global index representing the stability of malaria transmission*, Am. J. Trop. Med. Hyg. **70** (2004), no. 5, 486–498.
- [22] M. Kumar, S. Abbas, and Age-Structured SIR model for the spread of infectious diseases through indirect contacts, Mediterr. J. Math. **19** (2022), 14.
- [23] J. L. Kyle and E. Harris, *Global spread and persistence of dengue*, Annu. Rev. Microbiol. **62** (2008), 71–92.
- [24] J. P. LaSalle, *The stability of dynamical systems*, Society for Industrial and Applied Mathematics, Philadelphia, Pa., 1976.
- [25] M. Y. Li and Z. Shuai, *Global-stability problem for coupled systems of differential equations on networks*, J. Differential Equations **248** (2010), 1–20.
- [26] M. T. Li, G. Q. Sun, Y. F. Wu, J. Zhang, and Z. Jin, *Transmission dynamics of a multigroup brucellosis model with mixed cross infection in public farm*, Appl. Math. Comput. **237** (2014), 582–594.
- [27] M. T. Li, G. Q. Sun, J. Zhang, Z. Jin, X. D. Sun, Y. M. Wang, et al., *Transmission dynamics and control for a brucellosis model in Hinggan League of Inner Mongolia, China*, Math. Biosci. Eng. **11** (2014), 1115–1137.
- [28] Z. Mukandavire, S. Liao, J. Wang, H. Gaff, D. L. Smith, and J. G. Morris Jr., *Estimating the reproductive numbers for the 2008–2009 cholera outbreaks in Zimbabwe*, Proc. Natl. Acad. Sci. USA **108** (2011), 8767–8772.
- [29] Z. Mukandavire, D. L. Smith, and J. G. Morris Jr., *Cholera in Haiti: Reproductive numbers and vaccination coverage estimates*, Sci. Rep. **3** (2013), 997.
- [30] A. Mutreja, D. W. Kim, N. R. Thomson, T. R. Connor, J. H. Lee, S. Kariuki, et al., *Evidence for several waves of global transmission in the seventh cholera pandemic*, Nature **477** (2011), 462–465.
- [31] F. N. Ngoteya and Y. N. Gyekye, *Sensitivity analysis of parameters in a competition model*, Appl. Comput. Math. **4** (2015), no. 5, 363–368.
- [32] J. J. Paul Tian and J. Wang, *Global stability for cholera epidemic models*, Math. Biosci. **232** (2011), 31–41.
- [33] D. R. Powell, J. Fair, R. J. Le Claire, L. M. Moore, and D. Thompson, *Sensitivity analysis of an infectious disease model*, in: *Proceedings of the International System Dynamics Conference*, Boston, Mass, USA, 2005.
- [34] F. Sauvage, M. Langlais, N. G. Yoccoz, and D. Pontier, *Modelling hantavirus in fluctuating populations of bank voles: The role of indirect transmission on virus persistence*, J. Anim. Ecol. **72** (2003), 1–13.
- [35] Z. S. Shuai and P. van den Driessche, *Global dynamics of cholera models with differential infectivity*, Math. Biosci. **234** (2011), 118–126.
- [36] S. Soulaïmani and A. Kaddar, *Analysis and optimal control of a fractional order SEIR epidemic model with general incidence and vaccination*, in: IEEE Access, vol. 11, 2023, pp. 81995–82002.
- [37] R. Sun, *Global stability of the endemic equilibrium of multigroup SIR models with nonlinear incidence*, Comput. Math. Appl. **60** (2010), 2286–2291.
- [38] R. Sun and J. Shi, *Global stability of multigroup epidemic model with group mixing and nonlinear incidence rates*, Appl. Math. Comput. **218** (2011), 280–286.
- [39] H. Talibi Alaoui and R. Yafia, *Stability and Hopf bifurcation in an approachable haematopoietic stem cells model*, Math. Biosci. **206** (2007), no. 2, 176–184.
- [40] G. R. Thomson, W. Vosloo, and A. D. S. Bastos, *Foot and mouth disease in wildlife*, Virus Res. **91** (2003), 145–161.
- [41] J. H. Tien and D. J. D. Earn, *Multiple transmission pathways and disease dynamics in a waterborne pathogen model*, Bull. Math. Biol. **72** (2010), 1506.

- [42] L. W. Woods, *Adenoviral diseases*, in: E. S. Williams, I. K. Barker (Eds.), *Infectious Diseases of Wildlife Mammals*, 3rd edn., Manson, London, 2001, pp. 202–212.
- [43] R. Yafia, *Hopf bifurcation in a delayed model for tumor-immune system competition with negative immune response*, *Discrete Dyn Nat Soc.* 2006 (2006), 95296.
- [44] R. Yafia, *Dynamics and numerical simulations in a production and development of red blood cells model with one delay*, *Commun. Nonlinear Sci. Numer. Simulat.* **14** (2009), no. 2, 582–592.
- [45] Z. Yuan and L. Wang, *Global stability of epidemiological models with group mixing and nonlinear incidence rates*, *Nonlinear Anal. RWA* **11** (2010), 995–1004.
- [46] J. Zhang, Z. Jin, G. Q. Sun, X. D. Sun, Y. M. Wang, and B. X. Huang, *Determination of original infection source of H7N9 avian influenza by dynamical model*, *Sci. Rep.* **4** (2014), 4846.