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Research article

Global proprieties of a delayed epidemic model with partial susceptible protection

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Abstract: In the case of an epidemic, the government (or population itself) can use protection for reducing the epidemic. This research investigates the global dynamics of a delayed epidemic model with partial susceptible protection. A threshold dynamics is obtained in terms of the basic reproduction number, where for $R_0 < 1$ the infection will extinct from the population. But, for $R_0 > 1$ it has been shown that the disease will persist, and the unique positive equilibrium is globally asymptotically stable. The principal purpose of this research is to determine a relation between the isolation rate and the basic reproduction number in such a way we can eliminate the infection from the population. Moreover, we will determine the minimal protection force to eliminate the infection for the population. A comparative analysis with the classical SIR model is provided. The results are supported by some numerical illustrations with their epidemiological relevance.

Keywords: Modified SIR model; distributed delay; protection

1. Introduction

Predicting the outbreak of contagious diseases becomes one of the attractive topics in modern mathematical biology due to its importance and its huge influence. Our world witness a huge and rapid de-

velopment that leads to the appearance of numerous new infectious diseases as Ebola, HIV, COVID-19, which makes it a necessity of applying different and new ways of public health interventions.

In the case of the appearance of a new disease in a specific region, one of the first measures is to reduce the transmission by applying a restriction/confinement (full or partial), which helps sometimes in eliminating the infection. This method is effective in the case of the small region of the spread of the disease. This tool proved its effectiveness in reducing the spread of Ebola in Congo [1], and reducing the increase of the newly discovered COVID-19 disease, where it gave the researchers sufficient time to seek a new vaccine/treatment and saving millions of lives. The isolation of the susceptible persons (or those who have a high risk for mortality due to this infection) can reduce the transmission of the infection, even can lead to eliminating the infection. This measure can be applied directly by the government through measures of restriction (partial or full), where for the partial restriction, the government can allow for example the workers to move to the city or can do a restriction for a specific category of population limited by age. Also, the isolation can be performed by the population itself motivated by the fear of being infected. This case can happen for the case of infectious diseases with a high risk of mortality as Ebola.

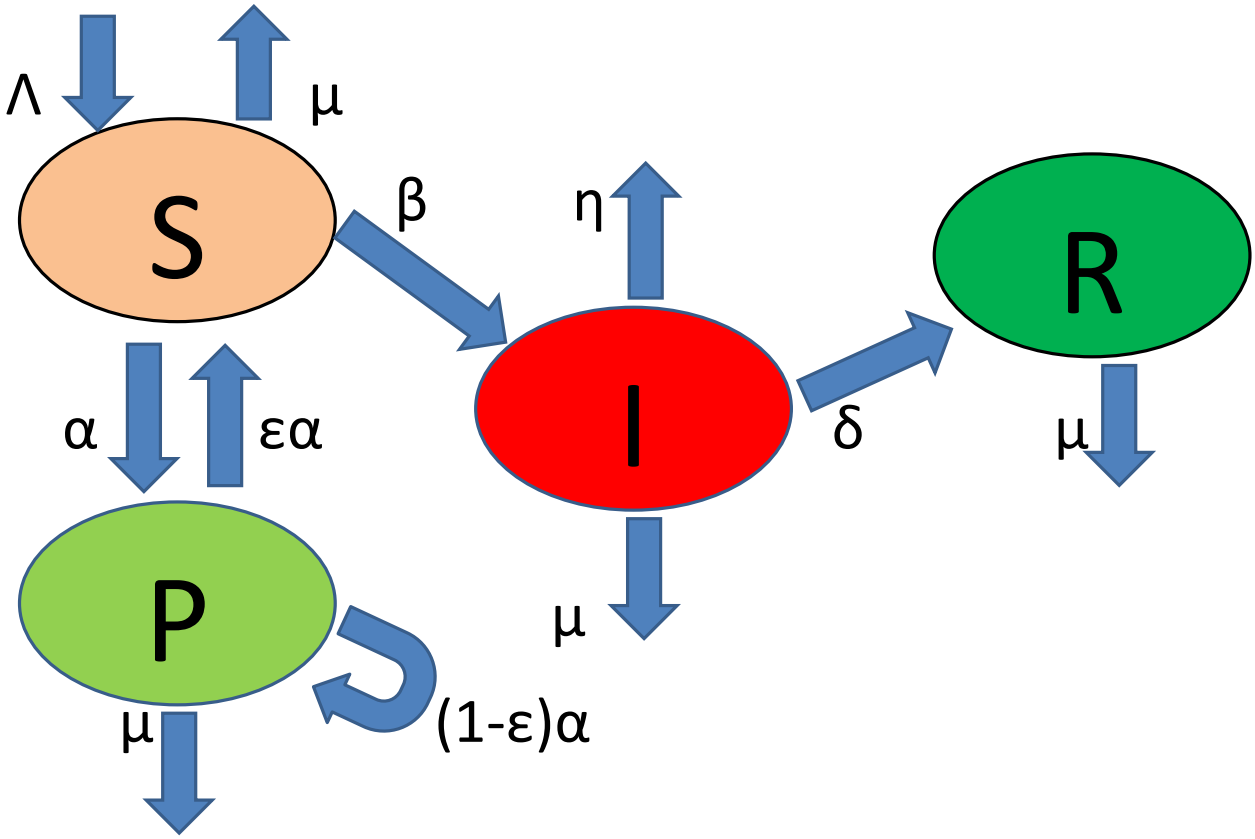

Figure 1. Flux of system (1.1).

The isolation is only one side of numerous sides of the protection strategy. There are numerous

methods for protecting the susceptible ones as vaccination which is more used. Vaccination is one of the best lines to defend against some infectious diseases. As researchers gain a better understanding of the causes of infection, the number of contagious diseases that can be prevented using vaccination continues growing. Many vaccines are provided in childhood. But adults still need routine vaccinations to avoid certain infections, such as flu Medicines and tetanus. Some drugs provide short-term protection against certain germs. For example, taking an antiparasitic medicine can prevent you from getting infected with malaria in the case of traveling or living in a high-risk area. Also, using protection materials that stand against being infected, for example using masks, washing hands which proved its usefulness in our recent fight against the pandemic of COVID-19 disease [2, 3, 4, 5] beside these see [6, 7, 8].

In the scientific field, numerous scholars used mathematical models for protecting the susceptible populations, where the main interest was on analyzing the effect of vaccination (mostly imperfect vaccination) we cite for instance the papers [9, 10, 11, 12]. The basic reproduction number (which is denoted by R_0) is the principal tool that has been used by scientists to predict the speed of the spread of disease in the population sample. Generally, for $R_0 < 1$ the infection will extinct from the population, and for $R_0 > 1$, the epidemic will persist in the population. In the case of the outbreak of an epidemic ($R_0 > 1$), the protection can help in reducing the infection. The main question is to determine the minimal protection force in such a way as to reduce R_0 below 1. Therefore, our present research is set to try to respond to the following questions:

- Can the susceptible partial to reduce the epidemic until extinction?. If yes, what it the minimal force required?
- What it the importance of the duration of this protection?

For responding to these questions we formulate a mathematical model. Based on some assumptions on the protected category. We assume that the protection is not permanent, it will last for a duration of σ. In this duration, the protected person is not able to get infected (due to getting vaccinated or this person wears a mask or simply has been isolated). After finishing the protection duration (σ) there is a proportion $1-\epsilon$ of persons in a protected zone that renew the protection (uses a second vaccine or continue using masks and protection materials), and the proportion ϵ of the protected category will become susceptible again and has a possibility for being infected. Based on these assumptions we formulate our mathematical model as:

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda - \beta S(t)I(t) - (\mu+\alpha)S(t) + \epsilon\alpha \exp^{-\mu\sigma} S(t-\sigma),\\
\frac{dP}{dt} &= \alpha S(t) + (1-\epsilon)\alpha e^{-\mu\sigma} S(t-\sigma) - \alpha e^{-\mu\sigma} S(t-\sigma) - \mu P(t),\\
\frac{dI}{dt} &= \beta S(t)I(t) - (\delta+\mu+\eta)I(t),\\
\frac{dR}{dt} &= \delta I(t) - \mu R(t),
\end{aligned} \tag{1.1}$$

where $S(t)$, $P(t)$, $I(t)$, $R(t)$ are respectively the densities of the susceptible individuals, protected individuals, infected persons, and recovered persons at time t. Λ is entering flux into S-class per unit of time, μ is the natural death coefficient. The rate β is the probability (per unit time) for transmission of the infection. δ is recovering coefficient. η is infection related death rate. α is the susceptible protection rate per unit of time. ϵ is the probability of quitting the isolation. The term $\epsilon\alpha e^{-\mu\sigma} S(t-\sigma)$ stands for the density of individuals that entered into the protected class and finished its protection duration at time t and leaves the isolation and becomes a susceptible again. The probability of surviving until t

is $e^{-\mu\sigma}$. The term $(1-\epsilon)\alpha e^{-\mu\sigma}S(t-\sigma)$ highlights the density of the protected persons that renew the protection as second vaccination after finishing the first vaccination. The dynamical flux of the model (1.1) is highlighted in Figure 1. After some simplification we get the system

$$\begin{array}{l} \frac{dS}{dt} = \Lambda - \beta S(t)I(t) - (\mu+\alpha)S(t) + \epsilon\alpha e^{-\mu\sigma}S(t-\sigma), \\ \frac{dP}{dt} = \alpha S(t) - \epsilon\alpha e^{-\mu\sigma}S(t-\sigma) - \mu P(t), \\ \frac{dI}{dt} = \beta S(t)I(t) - (\delta+\mu+\eta)I(t), \\ \frac{dR}{dt} = \delta I(t) - \mu R(t). \end{array} \tag{1.2}$$

The initial conditions are written in the form

$$S(\sigma) = \phi_1(\sigma), \;\; P(\sigma) = \phi_2, \;\; I(\sigma) = \phi_3, \;\; R(\sigma) = \phi_4, \;\; \sigma \in [-\sigma, 0], \tag{1.3}$$

with $\phi = (\phi_1, \phi_2, \phi_3, \phi_4) \in \mathbb{C}([-\sigma, 0], \mathbb{R}_+) \times \mathbb{R}_+ \times \mathbb{R}_+ \times \mathbb{R}_+$, we also suppose that $\phi_1 \not\equiv 0$.

To mention that the delay term $\epsilon\alpha e^{-\mu\sigma}S(t-\sigma)$ can be replaced by a a constant term $\epsilon\alpha P(t)$. This last cannot provide any information about the duration of protection, which is highly important mostly in the case of vaccination, also the isolation, where many countries get some serious economic losses due to the lock down and isolation, which proves the importance of the delay term instead of the constant term.

Our main interest is to determine the global proprieties of (1.2), where we will show that the behavior of this system is completely deduced by comparing R_0 with 1, where for $R_0 < 1$ the infection will extinct from the population, and for $R_0 > 1$ the infection will persist. Moreover, and motivated by the epidemiological background, we will suppose that the infection will spread in the absence of the public health intervention (protection), which can be indicated in our model by replacing α with 0, wherein this case our model will provide the results of the classical SIR model. Indeed, we will suppose that $R_0^{SIR} > 1$, where R_0^{SIR} is the corresponding SIR model basic reproduction number. In this case, our role is to provide the minimal protection force denoted $\alpha_{\min}$ that will lead to the extinction of the infection for our system (2.1). Moreover, in [13] it is obtained that the protection can generate a Hopf bifurcation, wherein in our case we will show that even in the presence of time delay we will have a threshold dynamics. For more reading concerning the threshold dynamics for delayed systems we refer as example [14, 15, 16, 17]. Based on the above-mentioned perspectives, we organize our research in the following structure:

In section 2, we provide some preliminary results regarding the well-posedness of the system (2.1) and providing the equilibrium points of (1.2), and R_0. Next, we will prove the global stability of the disease-free equilibrium for $R_0 < 1$. Section 4 is set to show the global stability of the endemic equilibrium for $R_0 > 1$. In section 5, we will determine the required public health intervention for reducing the value of R_0 below one, and studying the influence of the protection-related coefficient on the behavior of the solution. The discussion section is set to explain the required possible measures for reducing the infection and which public health intervention measure is more proper.

2. Preliminaries and existence and uniqueness of positive equilibrium

In this section, we show some results regarding the well-posedness of the solution of (1.2). We start with the following theorem:

Theorem 2.1. *Assume that* $(S(t), P(t), I(t), R(t))$ *is the solution of* (1.2)*, hence,* $S(t) > 0$, $P(t) > 0$, $I(t) > 0$, $R(t) > 0$ *for all finite* $t \geq 0$*. Moreover, we consider the following set*

$$\Omega = \left\{(S, P, I, R),\ \ S \geq 0,\ \ P \geq 0,\ \ I \geq 0,\ \ R \geq 0,\ \ S + P + I + R \leq \frac{\Lambda}{\mu}\right\},$$

positively invariant set.

Proof. We proceed a similar procedure as in [18]. We assume that $S(t) = 0$, then $S'(t) = \Lambda + \epsilon\alpha e^{-\mu\sigma} S(t - \sigma) > 0$, which means that $S(t) > 0$ for all $t \geq 0$. the second equation of (1.2) yields $I(t) = I(0)e^{\int_0^t S(\kappa) - (\mu+\eta+\delta)d\kappa} > 0$. The positivity of I implies the positivity of R. Now focusing on the second part of the proof. We suppose that $N(t) = S(t) + P(t) + I(t) + R(t)$, hence the sum of the three equation of (1.2) yields $N'(t) = \Lambda - \mu N(t) - \eta I(t)$, then $N(t) \leq \left(N(0) - \frac{\Lambda}{S_0}\right) e^{-\mu t} + \frac{\Lambda}{S_0} \leq max\{\frac{\Lambda}{\mu}\}$, which means that $S(t)$, $P(t)$, $I(t)$, $R(t)$ remain bounded, then the solution always exist for all $t \geq 0$ and remain in Ω.

□

Clearly, the first and the third equations are independent of P and R, and these last are respectively determined by S, and I. Hence, we can omit the P and R equations from the model (1.2). Hence, the dynamics of (1.2) can be deduced by analyzing the system:

$$\begin{array}{l} \frac{dS}{dt} = \Lambda - \beta S(t)I(t) - (\mu + \alpha)S(t) + \epsilon\alpha e^{-\mu\sigma} S(t - \sigma), \\ \frac{dI}{dt} = \beta S(t)I(t) - (\delta + \mu + \eta)I(t). \end{array} \tag{2.1}$$

The corresponding basic reproduction number is:

$$R_0 = \frac{\beta\Lambda}{(\alpha + \mu - \alpha\epsilon e^{-\mu\sigma})(\mu + \delta + \eta)}.$$

It is not hard to show that (2.1) has the disease free equilibrium (DFE) $E_0 = (S_0, 0)$ which always exists, and for $R_0 > 1$ there exist a unique endemic equilibrium (EE) $E^* = (S_*, I_*)$, where

$$S_0 = \frac{\Lambda}{\alpha + \mu - \alpha\epsilon e^{-\mu\sigma}},\ \ S_* = \frac{\mu + \delta + \eta}{\beta},\ \ I_* = \frac{(\alpha + \mu - \alpha\epsilon e^{-\mu\sigma})(R_0 - 1)}{\beta}.$$

remark This value is obtained using next generation matrix. Further, it can be obtained by using local stability of the DFE or the existence condition of EE.

3. Global stability of the disease free equilibrium

In this section, we employ a Lyapunov function to obtain the global stability of the DFE E_0 for $R_0 < 1$. The obtained results are resumed in the following theorem:

Theorem 3.1. E_0 *is globally asymptotically stable for* $R_0 < 1$ *and unstable for* $R_0 > 1$.

Proof. At first, we show the local stability of the DFE for $R_0 > 1$. The local stability can be deduce by analyzing the roots of the following characteristic equation

$$(\lambda - \beta(S_0 - S_*))(\lambda + (\mu + \alpha) - \epsilon\alpha e^{-\mu\sigma} e^{-\lambda\sigma}) = 0, \tag{3.1}$$

Obviously, if $S_0 > S_*$ (which equivalent to $R_0 > 1$) then E_0 is unstable. Now, we suppose that $R_0 < 1$, hence, the first root of (3.1) is $\lambda = \beta(S_0 - S_*) < 0$. The others are the root of

$$f(\lambda) = \lambda + \mu + \alpha - \epsilon\alpha e^{-\mu\sigma} e^{-\lambda\sigma},$$

we seek for roots of the form $\lambda = x + iy$ with $x, y \in \mathbb{R}$ and $x > 0$. Hence:

$$\begin{aligned} |\epsilon\alpha e^{-\mu\sigma} e^{-\lambda\sigma}| &= \epsilon\alpha e^{-\mu\sigma} e^{-x}, \\ &< \epsilon\alpha e^{-\mu\sigma}, \\ &< \mu + \alpha, \\ &< |\lambda + \mu + \alpha|, \end{aligned}$$

which is impossible. Thus, for $R_0 < 1$, E_0 is locally asymptotically stable. To complete the proof of Theorem 3.1, we need to prove the global attraction for $R_0 < 1$ using Lyapunov function. We consider the Lyapunov function $V(I(t)) = \frac{1}{2}I^2(t)$. Hence, for values of $t \geq \sigma$ such that $V(I(t+s)) = V(I(t))$ with $s \in [-\sigma, 0]$, we calculate $V'(t)$ along (2.1) in the following manner

$$\begin{aligned} V'(t) &= \beta S(t) I^2(t) - (\mu + \eta + \delta) I^2(t), \\ &\leq \beta S_0 I^2(t) - (\mu + \eta + \delta) I^2(t), \\ &\leq \beta I^2(t) S^* (R_0 - 1), \\ &\leq 0. \end{aligned}$$

For $R_0 < 1$ and Lyapunov-Razumikhin type theorem (see as example [19]), we deduce that $I(t)$ tends to 0 as t tends to ∞. Using system (2.1), we deduce that $S(t)$ goes to S_0 as t tends to ∞. The proof is achieved. □

4. Global stability of the endemic equilibrium

In this section, we employ a Lyapunov function to obtain the global stability of E_* whenever exists. The obtained results are resumed in the following theorem:

Theorem 4.1. *E_* is always globally asymptotically stable whenever exists for any value of time delay σ.*

Proof. At first, we study the local stability of E_* for $R_0 > 1$. The local stability can be deduce by analyzing the roots of the characteristic equation

$$\begin{vmatrix} \lambda + \beta I_* + (\mu + \alpha) - \epsilon\alpha e^{-\mu\sigma} e^{-\lambda\sigma} & \beta S_* \\ -\beta I_* & \lambda \end{vmatrix} = 0,$$

which equivalent to:

$$\Phi_\sigma(\lambda) = 0, \tag{4.1}$$

with

$$\Phi_\sigma(\lambda) = \lambda^2 + \lambda \left[\beta I_* + \mu + \alpha - \epsilon\alpha e^{-\mu\sigma} e^{-\lambda\sigma} \right] + \beta^2 S_* I_*.$$

Clearly, $\Phi_\sigma(0) = \beta^2 S_* I_* > 0$, thus $\lambda = 0$ is not a solution of $\Phi_\sigma(\lambda) = 0$. For $\sigma = 0$, $\Phi_0(\lambda)$ is expressed as:

$$\Phi_0(\lambda) = \lambda^2 + \lambda (\beta I_* + \mu + \alpha - \epsilon\alpha) + \beta^2 S_* I_*.$$

Obviously, $\Phi_0(\lambda) = 0$ has two roots with negative real part. It is well known that delay can generate instability of a stable equilibrium, hence, we seek for the roots of the form iy, $y > 0$. Thus, $\Phi_\sigma(\lambda) = 0$ can be expressed as:

$$0 = -y^2 + iy\left[\beta I_* + \mu + \alpha - \epsilon\alpha e^{-\mu\sigma}(\cos(y\sigma) - i\sin(y\sigma))\right] + \beta^2 S_* I_*.$$

Which equivalent to

$$\begin{cases} \epsilon\alpha e^{-\mu\sigma} y\sin(y\sigma) &= -y^2 + \beta^2 S_* I_*, \\ \epsilon\alpha e^{-\mu\sigma} y\cos(y\sigma) &= (\beta I_* + \mu + \alpha)\, y. \end{cases} \tag{4.2}$$

By squaring the two sides of the equations of the system (4.2), then we add them, to obtain

$$\epsilon^2\alpha^2 e^{-2\mu\sigma} y^2 = (-y^2 + \beta^2 S_* I_*)^2 + (\beta I_* + \mu + \alpha)^2 y^2, \tag{4.3}$$

can be written as

$$(y^2)^2 + A_0 y^2 + (\beta^2 S_* I_*)^2 = 0, \tag{4.4}$$

with

$$\begin{aligned} A_0 &= -2\beta^2 S_* I_* + (\beta I_* + \mu + \alpha)^2 - \epsilon^2\alpha^2 e^{-2\mu\sigma}, \\ &= -2\beta^2 S_* I_* + \left(\tfrac{\Lambda}{S_*} + \epsilon\alpha e^{-\mu\sigma}\right)^2 - \epsilon^2\alpha^2 e^{-2\mu\sigma}, \\ &= -2\beta^2 S_* I_* + 2\tfrac{\Lambda}{S_*}\epsilon\alpha e^{-\mu\sigma} + \left(\tfrac{\Lambda}{S_*}\right)^2. \end{aligned}$$

For having positive roots of (4.4) in y^2, we must guarantees the positivity of the following quantity:

$$\begin{aligned} A_1 &= A_0^2 - 4(\beta^2 S_* I_*)^2, \\ &= \left[-2\beta^2 S_* I_* + 2\tfrac{\Lambda}{S_*}\epsilon\alpha e^{-\mu\sigma} + \left(\tfrac{\Lambda}{S_*}\right)^2\right]^2 - 4(\beta^2 S_* I_*)^2, \\ &= \left(-2\beta^2 S_* I_* + A_0\right)\left[2\tfrac{\Lambda}{S_*}\epsilon\alpha e^{-\mu\sigma} + \left(\tfrac{\Lambda}{S_*}\right)^2\right]. \end{aligned}$$

For having real roots of (4.4) in y^2 we must have $A_0 > 2\beta^2 S_* I_* > 0$. This means $A_0 > 0$ hence all the coefficient of eq. (4.4), which leads to deduce that (4.4) has no positive roots in y^2. Thus, EE is locally asymptotically stable. Next, we show the global attraction using Lyapunov function. We consider the following function:

$$V(t) = V_1(t) + V_2(t), \tag{4.5}$$

with

$$V_1(t) = S_* g\left(\tfrac{S(t)}{S_*}\right) + I_* g\left(\tfrac{I(t)}{I_*}\right), \quad V_2(t) = \epsilon\alpha S_* e^{-\mu\sigma}\int_0^\sigma g\left(\tfrac{S(t-\theta)}{S_*}\right) d\theta,$$

where g is Volterra function $g(w) = w - 1 - \ln w$, $w \in \mathbb{R}^+$. Note that $V(t)$ is nonnegative defined and has a global minimum at the positive equilibrium (S_*, I_*). The time derivative of $V(t)$ is:

$$\begin{aligned} \tfrac{dV_1}{dt} &= \left(1 - \tfrac{S_*}{S(t)}\right)\tfrac{dS}{dt} + \left(1 - \tfrac{I_*}{I(t)}\right)\tfrac{dI}{dt}, \\ &= \left(1 - \tfrac{S_*}{S(t)}\right)(\Lambda - \beta S(t)I(t) - (\mu + \alpha)S(t) + \epsilon\alpha e^{-\mu\sigma} S(t-\sigma)) + \left(1 - \tfrac{I_*}{I(t)}\right)(\beta S(t)I(t) - (\mu + \delta + \eta)I(t)). \end{aligned}$$

Using the equilibrium propriety $\Lambda = \beta S_* I_* + (\mu + \alpha - \epsilon\alpha e^{-\mu\sigma})S_*$ we get

$$\begin{aligned}
\frac{dV_1}{dt} &= \left(1 - \frac{S_*}{S(t)}\right)(\beta S_* I_* + (\mu + \alpha - \epsilon\alpha e^{-\mu\sigma})S_* - \beta S(t)I(t) - (\mu + \alpha)S(t) + \epsilon\alpha e^{-\mu\sigma} S(t-\sigma)) \\
&\quad + \left(1 - \frac{I_*}{I(t)}\right)(\beta S(t)I(t) - (\mu + \eta + \delta)I(t)), \\
&= -\frac{\mu}{S(t)}(S(t) - S_*)^2 + \beta S_* I_* + \alpha S_* - \epsilon e^{-\mu\sigma}\alpha S_* - \alpha S(t) - \beta S(t)I(t) + \beta S_* I(t) \\
&\quad + \epsilon\alpha e^{-\mu\sigma} S(t-\sigma) - \beta S_* I_* \frac{S_*}{S(t)} - \alpha S_* \frac{S_*}{S(t)} + \epsilon e^{-\mu\sigma}\alpha S_* \frac{S_*}{S(t)} \\
&\quad + \alpha S_* - \epsilon\alpha \frac{S_*}{S(t)} e^{-\mu\sigma} S(t-\sigma) + \beta S(t)I(t) - \beta S_* I(t) - \beta S(t) I_* + \beta S_* I_*, \\
&= -\frac{\mu}{S(t)}(S(t) - S_*)^2 + \beta S_* I_* \left(2 - \frac{S(t)}{S_*} - \frac{S_*}{S(t)}\right) + \alpha S_* \left(2 - \frac{S(t)}{S_*} - \frac{S_*}{S(t)}\right) \\
&\quad + \epsilon\alpha S_* e^{-\mu\sigma} \left(-\frac{S(t-\sigma)}{S(t)} - 1 + \frac{S_*}{S(t)} + \frac{S(t-\sigma)}{S_*}\right).
\end{aligned}$$

Now we compute $\frac{dV_2}{dt}$

$$\begin{aligned}
\frac{dV_2}{dt} &= \frac{d}{dt}\alpha e^{-\mu\sigma} S_* \int_0^\sigma g\left(\frac{S(t-\theta)}{S_*}\right) d\theta d\sigma, \\
&= S_* \alpha e^{-\mu\sigma} \frac{d}{dt} \int_0^\sigma g\left(\frac{S(t-\theta)}{S_*}\right) d\theta.
\end{aligned}$$

Note that

$$\begin{aligned}
\frac{d}{dt}\int_0^\sigma g\left(\frac{S(t-\theta)}{S_*}\right) d\theta &= \int_0^\sigma \frac{d}{dt} g\left(\frac{S(t-\theta)}{S_*}\right) d\theta, \\
&= -\int_0^\sigma \frac{d}{d\theta} g\left(\frac{S(t-\theta)}{S_*}\right) d\theta, \\
&= -g\left(\frac{S(t-\theta)}{S_*}\right)\Big|_0^\sigma, \\
&= -\frac{S(t-\sigma)}{S_*} + \frac{S(t)}{S_*} + \ln\left(\frac{S(t-\sigma)}{S(t)}\right),
\end{aligned}$$

thus, we get

$$\begin{aligned}
\frac{dV_2}{dt} &= S_* \epsilon\alpha e^{-\mu\sigma} \left(-\frac{S(t-\sigma)}{S_*} + \frac{S(t)}{S_*} + \ln\left(\frac{S(t-\sigma)}{S(t)}\right)\right), \\
&= -\alpha\epsilon e^{-\mu\sigma} S(t-\sigma) + \alpha\epsilon e^{-\mu\sigma} S(t) + S_* \alpha\epsilon e^{-\mu\sigma} \ln\left(\frac{S(t-\sigma)}{S(t)}\right).
\end{aligned}$$

Now, we sum $\frac{dV_1}{dt}$ and $\frac{dV_2}{dt}$, we get:

$$\begin{aligned}
\frac{dV}{dt} &= \frac{dV_1}{dt} + \frac{dV_2}{dt}, \\
&= -\frac{\mu}{S(t)}(S(t) - S_*)^2 + \beta S_* I_* \left(2 - \frac{S(t)}{S_*} - \frac{S_*}{S(t)}\right) + \alpha S_* \left(2 - \frac{S(t)}{S_*} - \frac{S_*}{S(t)}\right) \\
&\quad + \epsilon\alpha S_* \alpha e^{-\mu\sigma} \left(-\frac{S(t-\sigma)}{S(t)} - 1 + \frac{S_*}{S(t)} + \frac{S(t-\sigma)}{S_*}\right) - \epsilon\alpha e^{-\mu\sigma} S(t-\sigma) + \alpha\epsilon e^{-\mu\sigma} S(t) \\
&\quad + S_* \alpha\epsilon e^{-\mu\sigma} \ln\left(\frac{S(t-\sigma)}{S(t)}\right), \\
&= -\frac{\mu}{S(t)}(S(t) - S_*)^2 + (\beta S_* I_* + \alpha S_*)\left(2 - \frac{S(t)}{S_*} - \frac{S_*}{S(t)}\right) + \epsilon\alpha S_* e^{-\mu\sigma} \left(-\frac{S(t-\sigma)}{S(t)} - 1 + \frac{S_*}{S(t)} + \frac{S(t-\sigma)}{S_*}\right)
\end{aligned}$$

Then we get

$$\begin{aligned}
\frac{dV}{dt} &= -\frac{\mu}{S(t)}(S(t) - S_*)^2 + \beta S_* I_* \left(2 - \frac{S(t)}{S_*} - \frac{S_*}{S(t)}\right) + \alpha S_* \left(2 - \frac{S(t)}{S_*} - \frac{S_*}{S(t)}\right) \\
&\quad + \epsilon\alpha S_* e^{-\mu\sigma} \left(-2 + \frac{S(t)}{S_*} + \frac{S_*}{S(t)}\right) \\
&\quad + \epsilon\alpha S_* e^{-\mu\sigma} \left(1 + \ln\frac{S(t-\sigma)}{S(t)} - \frac{S(t-\sigma)}{S(t)}\right), \\
&= -\frac{\mu}{S(t)}(S(t) - S_*)^2 - (\beta S_* I_* + \alpha S_* - \epsilon\alpha S_* e^{-\mu\sigma})\left(2 - \frac{S(t)}{S_*} - \frac{S_*}{S(t)}\right) - \epsilon\alpha S_* e^{-\mu\sigma} g\left(\frac{S(t-\sigma)}{S(t)}\right).
\end{aligned}$$

Note that

$$\begin{aligned} \beta S_* I_* + \alpha S_* - \epsilon \alpha S_* e^{-\mu\sigma} &= \beta S_* I_* + \alpha S_* (1 - \epsilon e^{-\mu\sigma}), \\ &> 0. \end{aligned}$$

Thus $\frac{dV}{dt} \leq 0$.

We let Γ be the largest invariant subset of $\{(S(t), I(t)) | \frac{dV(t)}{dt} = 0\}$. Now, let us determine the set Γ. Note that $\frac{dV(t)}{dt} = 0$, leads to $S(t) = S_*$, by replacing this result into 1st eq. of (2.1) we get $I(t) = I_*$ for all t. Hence, $\Gamma = \{E^*\}$. By the help of Theorem 1.2 and the LaSalle invariance principle [10], every solution of (2.1) goes to E^*. The proof is achieved. □

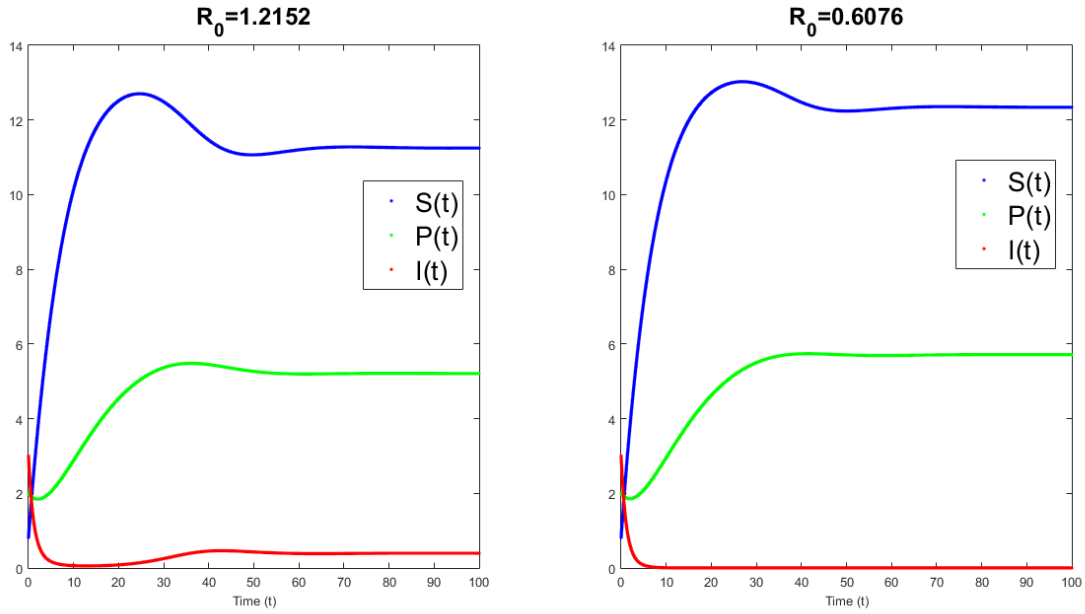

Figure 2. The global dynamics of the system (1.2) in two cases $R_0 < 1$ and $R_0 > 1$ for the values:
$\lambda = 2$, $\mu = 0.1$, $\beta = 0.08$, $\eta = 0.7$, $\delta = 0.1$, $\alpha = 0.05$, $\epsilon = 0.1$, and the initial conditions $S(\theta) = 1 + 0.2\cos(\theta)$, $\theta \in [-\sigma, 0]$, $I(0) = 3$, $P(0) = 2$, where for the left hand figure we use $\beta = 0.08$ and the right hand figure $\beta = 0.04$

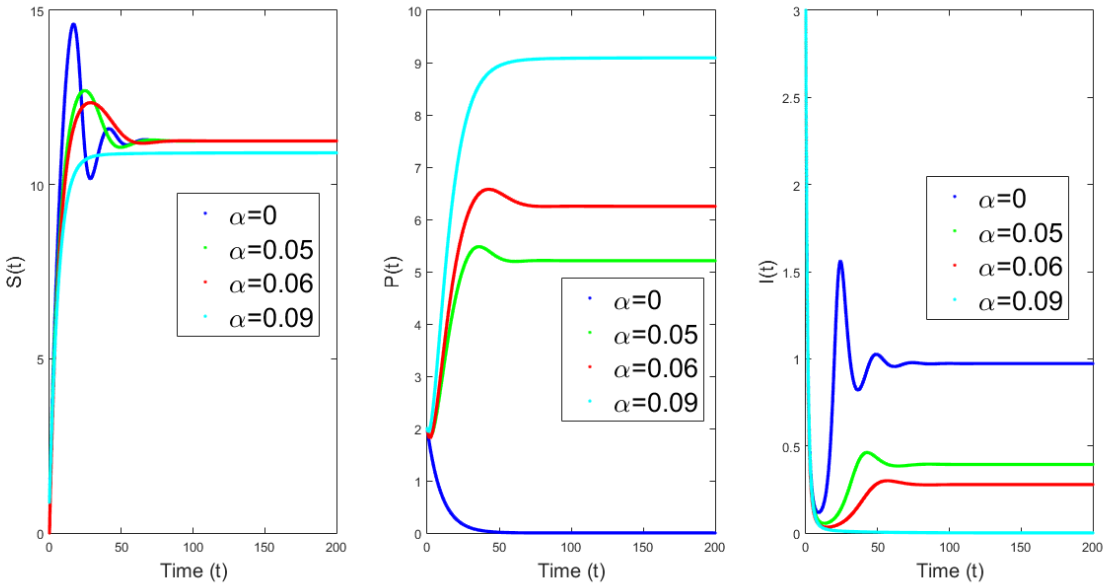

Figure 3. The impact of α on the final size of the populations for the values: $\lambda = 2$, $\mu = 0.1$, $\beta = 0.08$, $\eta = 0.7$, $\delta = 0.1$, $\epsilon = 0.1$, and the initial conditions $S(\theta) = 1 + 0.2\cos(\theta)$, $\theta \in [-\sigma, 0]$, $I(0) = 3$, $P(0) = 2$, and multi values of α. Note that the minimal protection force is $\alpha_{\min} = 0.0840$.

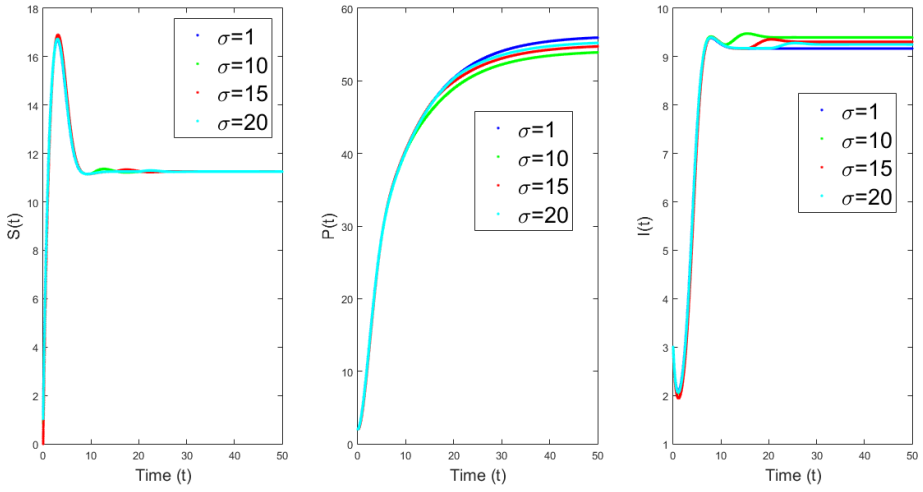

Figure 4. The impact of σ on the final size of the populations for the values: $\lambda = 15$, $\mu = 0.1$, $\beta = 0.08$, $\eta = 0.7$, $\delta = 0.1$, $\epsilon = 0.1$, $\alpha = 0.5$, and the initial conditions $S(\theta) = 1 + 0.2\cos(\theta)$, $\theta \in [-\sigma, 0]$, $I(0) = 3$, $P(0) = 2$, and multi values of σ.

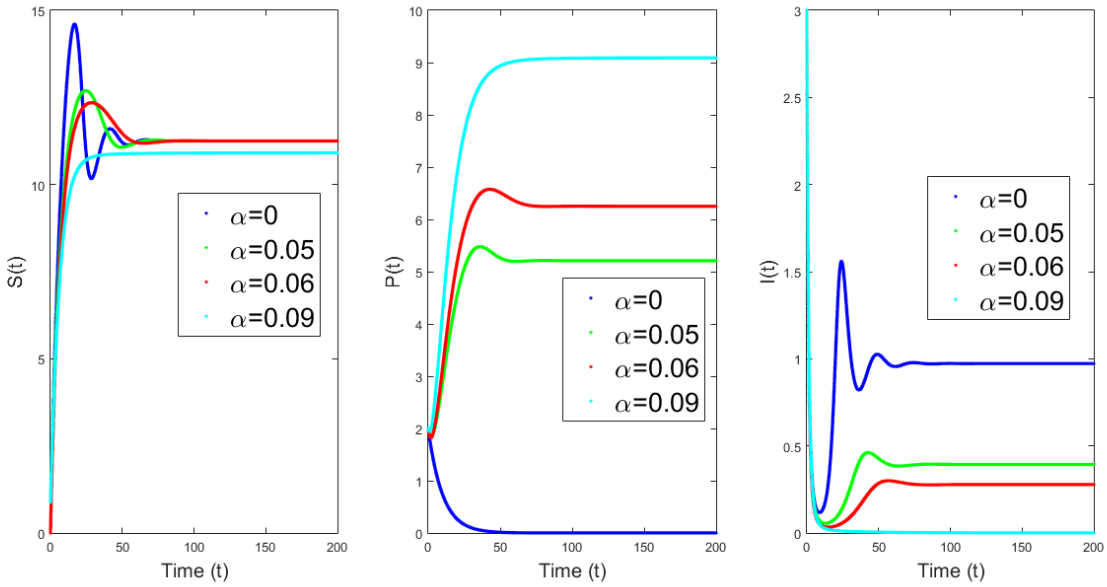

Figure 5. The impact of ϵ on the final size of the populations for the values:
$\lambda = 15, \mu = 0.1, \beta = 0.08, \eta = 0.7, \delta = 0.1, \sigma = 10, \alpha = 0.5$, and the initial conditions $S(\theta) = 1 + 0.2\cos(\theta), \quad \theta \in [-\sigma, 0]$, $I(0) = 3$, $P(0) = 2$, and multi values of σ.

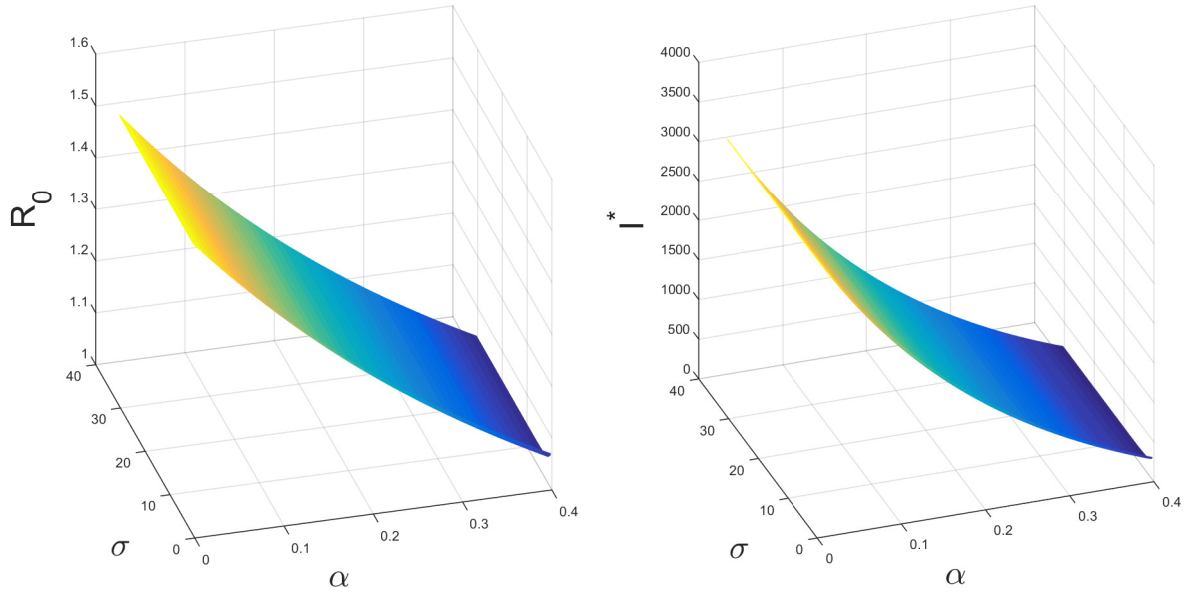

Figure 6. The impact of σ and α on the final size of the infected population and R_0 for the values:
$\lambda = 25, \mu = 0.8, \beta = 0.08, \eta = 0.7, \delta = 0.1, \epsilon = 0.1$.

5. Required public health intervention

In this section, we are interested in discussing the effect of protection, and its influence on reducing the infection in the population. We denote by R_0^{SIR} is the basic reproduction number for the classical SIR model, where $R_0^{SIR} = \frac{\beta\Lambda}{\mu(\mu+\delta+\eta)}$. The main goal is to suppose that $R_0^{SIR} > 1$ in the case of the absence of the protection (means $\alpha = 0$), and determining the sufficient protection force for reaching $R_0 < 1$. Also, for $\alpha > 0$ we have:

$$\begin{aligned} R_0 &= \frac{\beta\Lambda}{(\alpha+\mu-\alpha\epsilon e^{-\mu\sigma})(\mu+\eta+\delta)}, \\ &= R_0^{SIR}\frac{\mu}{\alpha+\mu-\alpha\epsilon e^{-\mu\sigma}}, \\ &< R_0^{SIR} \end{aligned}$$

Hence, the protection will help in reducing the speed of the outbreak of the disease. Further, we study the sensitivity of R_0 with respect to α, σ, and $1-\epsilon$. By a straightforward computation we get:

$$\begin{aligned} \frac{\partial R_0}{\partial \alpha} &= -\frac{(1-\epsilon e^{-\mu\sigma})R_0}{(\alpha+\mu-\alpha\epsilon e^{-\mu\sigma})} < 0, \\ \frac{\partial R_0}{\partial \sigma} &= \frac{-\alpha\epsilon\mu e^{-\mu\sigma}}{(\alpha+\mu-\alpha\epsilon e^{-\mu\sigma})}R_0 < 0, \\ \frac{\partial R_0}{\partial (1-\epsilon)} &= -\frac{\partial R_0}{\partial \epsilon} = -\frac{(\alpha e^{-\mu\sigma})R_0}{(\alpha+\mu-\alpha\epsilon e^{-\mu\sigma})} < 0, \end{aligned}$$

hence, we conclude that α, σ, and $1-\epsilon$ have a respectively negative impact, positive impact, negative impact on the value of R_0. The negative (resp. positive) impact means that the value of R_0 decreases (resp. increase) when the variable goes larger. For epidemiological perspectives, we presume that $R_0^{SIR} > 1$, this means that the public health interventions are needed, so, we need to determine the minimal protection effort for reducing R_0 below 1. It has been proved previously that $R_0 = R_0^{SIR}\frac{\mu}{\alpha+\mu-\alpha\epsilon e^{-\mu\sigma}}$, which is needed to be reduced below 1. This means that

$$R_0^{SIR}\frac{\mu}{\alpha+\mu-\alpha\epsilon e^{-\mu\sigma}} < 1.$$

Using $R_0^{SIR} > 1$, we get

$$\alpha > \alpha_{\min} := \frac{\mu(R_0^{SIR}-1)}{1-\epsilon e^{-\mu\sigma}}.$$

Sometimes, it is hard for some governments to achieve the required protection force $\alpha = \alpha_{\min}$ to reduce the value of R_0 below 1. In this case, these countries can reduce R_0 until an adequate value (larger than 1). Note that in this case, we get the global stability of E_*. Our next objective is to determine the sensitivity of EE with respect to α, σ, and $1-\epsilon$. Note that in this case, we presume that $R_0 > 1$ (for guaranteeing the existence of the E_*). By a straightforward calculation, we get

$$\frac{\partial S_*}{\partial \alpha} = 0, \quad \frac{\partial S_*}{\partial \sigma} = 0, \quad \frac{\partial S_*}{\partial (1-\epsilon)} = 0,$$

hence α, σ, and $1-\epsilon$ have no influence on the final size (in the case of $R_0 > 1$) of the susceptible population. Similarly, the derivative of I_* with respect to the previous variable is

$$\begin{aligned}\frac{\partial I_*}{\partial \alpha} &= \tfrac{1}{\beta}\left((1-\epsilon e^{-\mu\sigma})(R_0-1)+(\alpha+\mu-\alpha\epsilon e^{-\mu\sigma})\tfrac{\partial R_0}{\partial \alpha}\right),\\ &= -\tfrac{1}{\beta}(1-\epsilon e^{-\mu\sigma})<0.\end{aligned}$$

Next we calculate

$$\begin{aligned}\frac{\partial I_*}{\partial \sigma} &= \tfrac{1}{\beta}\left(\mu\alpha\epsilon e^{-\mu\sigma}(R_0-1)+(\alpha+\mu-\alpha\epsilon e^{-\mu\sigma})\tfrac{\partial R_0}{\partial \sigma}\right),\\ &= -\mu\epsilon\alpha e^{-\mu\sigma}<0\end{aligned}$$

Finally, we calculate the last derivative:

$$\begin{aligned}\frac{\partial I_*}{\partial(1-\epsilon)} &= -\frac{\partial I_*}{\partial \epsilon}\\ &= -\frac{1}{\beta}\alpha e^{-\mu\sigma}<0.\end{aligned}$$

Now, passing on the epidemiological meaning of the above-mentioned calculations. In fact, for $R_0 > 1$, we obtained that I_* is decreasing in α, hence we conclude that augmenting the quantity of the protected susceptible will lead to a decrease of the final size of the infected population, and α has no impact on the final size of the susceptible population (due to $\dfrac{\partial S_*}{\partial \alpha} = 0$). Similarly, we get that increasing the density of re-protected individuals will decrease also the final size of the infected population I_* and no impact is noted on S_*. For the time delay σ, it has also a negative impact on the final size of the infected population as it is been highlighted in Figure 4. Hence the considered parameters α, σ, $(1-\epsilon)$ reduce the speed of the spread of the infectious disease, and it can be considered as a control of the outbreak of the disease, and it can be used as a public health intervention. The principal result of this section is obtaining the minimal effort for reducing R_0 below 1, which is expressed by $\alpha = \alpha_{\min}$. This result can be seen clearly through Figure 3, where for $\alpha = 0.09 > \alpha_{\min} = 0.084$ we get the extinction of the disease, which is the desired result. For more explanation, we provide the detailed discussion on the figures:

Figure 2: In this figure we illustrated the principal results of theorems 3.1 and 4.1, where for $R_0 < 1$ the disease free equilibrium is globally stable, and for $R_0 > 1$ the endemic equilibrium is globally asymptotically stable.

Figure 3: Here, the impact of the protection force on the spread of the disease. More precisely, by augmenting the value of the protection rate α the infection will reduce remarkably, and can lead to the extinction of the disease (which has been notice for $\alpha = 0.09$). This shows the huge importance role of the susceptible protection in the public health intervention.

Figure 4: In this figure, the impact of the duration of the susceptible protection on the spread of the infection. More precisely, by augmenting the value of the protection rate σ we can remark that the infection will vary, and cannot lead to the extinction of the disease as it is been remarked for the rate α. This shows that the duration of the protection will influence the outbreak of the disease but will not help in getting rid of it.

Figure 5: In this figure, the impact of ϵ on the spread of the infection. More precisely, by augmenting the value of the protection rate ϵ we can remark that the infection will reduce, and cannot lead to the extinction. However, the final size of the susceptible population is not concerned by this reduce, where ϵ has no influence on the susceptible final size. This shows that the re-protection rate will influence the

outbreak of the disease but will not help in getting rid of it.
Figure 6: Here, we investigated the influence of some parameters on the basic reproduction number, where it is remarked that the rate α has the most remarkable influence on the spread of the disease, which leads us to deduce that the protection force α is the best manner for controlling the epidemic.

6. Concluding and remarks

We dealt in this research with a delayed epidemic model with susceptible protection. Our interest is to provide a comparative analysis between the classical SIR epidemic model and our model (1.2) for distinguishing the influence of protection on the outbreak of the disease, and showing if the protection can be used as public health intervention or not. It has been shown that (2.1) has a threshold dynamics in terms of R_0, where for $R_0 < 1$, DFE is globally stable, and for $R_0 > 1$, E_* is globally stable (see Figure 2). These results are proved to be used in the principal part of our research (section 5). We presumed that in the absence of the protection ($\alpha = 0$) the infection persists in the population (which means that $R_0^{SIR} > 1$). The main purpose of the government is to use minimal protection effort denoted $\alpha_{\min}$ in such a way the corresponding basic reproduction number to the model (1.2) becomes less than 1 ($R_0 < 1$). Indeed, the minimal protection force is $\alpha_{\min} := \dfrac{\mu(R_0^{SIR} - 1)}{1 - \epsilon e^{-\mu\sigma}}$ which it has a direct relationship with the R_0^{SIR}. To mention that increasing the R_0^{SIR} will lead to an increase in the required force of protection $\alpha_{\min}$ for guaranteeing the stability of the DFE (means $R_0 < 1$).

Moreover, using Figure 4, and Figure 5 it has been noticed that the reproduction rate $1 - \epsilon$ and the duration of the protection has a small impact on the dynamics of the solution comparing with the protection forceα. This means that the government should focus on augmenting the protection forceα instead of the reproduction proportion. Furthermore, using Figure 6 we conclude that α and σ have a huge impact on R_0 and final size of the infected persons (I^*), and have a high possibility for reducing the speed of the epidemic or even stop it.

Also, the recent studies proved that it is probable for a recovered person to become a susceptible person again (as COVID-19 disease), which can be modeled by a SIRS epidemic model. So, the role of protection in the case of the SIRS epidemic model is an important subject of interest of the upcoming works, where we will provide it extensive attention.

Conflict of interest

There is no conflict of interest between the authors.

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Conflict of interest

The authors declare there is no conflict of interest.

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