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Partial stability of controlled SEIR epidemic models

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Abstract— **This paper proposes to use the concept of partial stability instead of that of global stability to analyze the dynamic behavior of epidemic models. The rationale behind this is that it is only needed, from a practical point of view, to ensure the boundedness of the infectious and infected subpopulations in order to get the disease under control. Thus, the partial stability of a controlled SEIR epidemic model is studied in the vaccination-free case. It will be proved that the infectious and infected subpopulations may still be bounded when the total population increases and global stability is not achieved. In addition, a feedback-based vaccination policy is designed based on the concept of partial stability of the closed-loop system. Some numerical simulations illustrating the usefulness of the designed control law along with a comparison with previous vaccination laws complete the paper.**

I. INTRODUCTION

Mathematical models have become an important tool in analyzing the causes, dynamics and spread of epidemies. Thus, its study is crucial in order to obtain valuable knowledge of the underlying aspects of the disease. Furthermore, the analysis of mathematical models describing epidemics spreading allows us to make decisions regarding the best vaccination policies, quarantine application and so on. In this way, a large number of mathematical models have been proposed in the literature (see [1] for more information on mathematical models). In addition, many specific features regarding these models have been studied in many works such as the presence of bifurcations [2], oscillating behavior [3] and existence of waves [6], for instance. However, model stability has been by far the most important property to be studied, [2],[5],[7]-[11]. Typically, global stability is the main stability property to be analyzed, [7]-[9], [11]. Global stability is referred to the boundedness of all the variables composing the model as time goes by. Nevertheless, this approach seems to be quite conservative for the study of epidemics since a globally stable model would never capture the potential natural increase of the population which would lead to diverging subpopulations. It is also worth noting that, from an epidemic point of view, it is only needed the boundedness (and eventually the convergence to zero) of the infected and infectious subpopulations regardless the behavior of the other ones, which are not directly suffering from the disease. Thus, if global stability is required, the analysis and conditions found may not be applicable to situations where the total population grows. This is a great inconvenience since the model may be globally unstable because the susceptible or the immune diverge while no specific information is obtained for the infected and infectious which are the most important subpopulations from an epidemic point of view. A further analysis should be performed.

In this paper, the concept of partial stability, [12], is introduced and applied to a SEIR controlled epidemic model with two types of nonlinear incidence rates. Then, the partial stability of the model is defined and analyzed for the control-free (i.e. vaccination-free) case. The concept of partial stability allows us to focus on the analysis of the infected and infectious subpopulations and to analyze their boundedness regardless the behavior of the other variables, including thus a potential natural increase of the total population. Moreover, a feedback-type vaccination control law is designed in order to eradicate the illness. The control law is designed based on the partial stability frame through a Lyapunov-type adapted theorem,[12],[13]. Some simulation results showing the usefulness of the proposed approach are included and a comparison with respect other feedback-type vaccination strategies is performed.

II. MODEL DESCRIPTION AND PROBLEM FORMULATION

A. Model description

Consider the SEIR epidemic model described by the following equations:

$$\begin{aligned}\dot{S}(t) &= -\mu S(t)+\omega R(t)-g(S,E,I,R) \\ &\quad +\nu N(t)(1-V(t)) \qquad (1)\\ \dot{E}(t) &= g(S,E,I,R)-(\mu+\sigma)E(t) \qquad (2)\\ \dot{I}(t) &= -(\mu+\gamma)I(t)+\sigma E(t) \qquad (3)\\ \dot{R}(t) &= -(\mu+\omega)R(t)+\gamma I(t)+\nu N(t)V(t) \qquad (4)\end{aligned}$$

where $S(t)$, $E(t)$, $I(t)$ and $R(t)$ denote the subpopulations of susceptible, infected, infectious and immune respectively. $N(t)$ denotes the total population at each time, t (i.e. $N(t) = S(t)+E(t)+I(t)+R(t)$), $V(t)$ represents the vaccination function, μ is the rate of deaths from causes unrelated to the infection, ν denotes the natality rate and ω is the rate of losing immunity. The function $g(S,E,I,R)$ is referred to as the disease incidence rate. When $g(S,E,I,R) = g_1(S,E,I,R) = \beta\frac{S(t)I(t)}{N(t)}$, it is said to be the standard incidence rate and when $g(S,E,I,R) = g_2(S,E,I,R) = \frac{\beta S(t)I(t)}{1+\alpha S(t)}$ it is said to be the saturated incidence rate where

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β is the transmission constant (with, for instance, the total number of new infections per unit of time at time t being $\beta\frac{S(t)I(t)}{N(t)}$ for the standard incidence rate) and α is the saturation coefficient. σ^{-1} and γ^{-1} are, respectively, the average durations of the latent and infective periods. All the above parameters are assumed to be positive so as to represent a real situation. The total population dynamics at time t can be calculated by summing up all the equations (1)-(4), leading to:

$$\dot{N}(t) = (\nu - \mu)N(t) \tag{5}$$

It can be deduced from Eq. (5) that the total population is constant when $\nu = \mu$, increases when $\nu > \mu$ and decreases when $\nu < \mu$. Furthermore, the relation between μ and ν also determines the existence or not of equilibrium points as the following theorem for incidence rate g_1 shows:

Theorem 1. The equilibrium points of the system of equations (1)-(4) with incidence rate g_1 are given by:

A) If $\mu \neq \nu$ then, the only equilibrium point is given by $S^* = E^* = I^* = R^* = 0$. This point represents the case of total population extinction.
B) If $\mu = \nu$, the total population is constant, i.e. $N(t) = N = N(0) = S(0) + E(0) + I(0) + R(0)$ and there are two equilibrium points given by:
   - $S^* = N, E^* = I^* = R^* = 0$, i.e., the total population becomes susceptible.
   - $$S^* = \frac{(\mu+\sigma)(\mu+\gamma)}{\sigma\beta}N \tag{6}$$
     $$E^* = \frac{(\mu+\omega)(\mu+\gamma)(\sigma\beta - (\mu+\gamma)(\mu+\sigma))}{\sigma\beta((\mu+\sigma+\gamma)(\mu+\omega)+\gamma\sigma)}N \tag{7}$$
     $$I^* = \frac{(\mu+\omega)(\sigma\beta - (\mu+\sigma)(\mu+\gamma))}{\beta((\mu+\sigma+\gamma)(\mu+\omega)+\gamma\sigma)}N \tag{8}$$
     $$R^* = \frac{\gamma(\sigma\beta - (\mu+\sigma)(\mu+\gamma))}{\beta((\mu+\sigma+\gamma)(\mu+\omega))+\gamma\sigma)}N \tag{9}$$

Proof. Part B was proved in [16] and, therefore, it is only necessary to prove A. Thus, the equilibrium point implies, in particular, that Eq. (5) zeroes. Hence, we have:

$$\dot{N}(t) = 0 = (\nu - \mu)N(t) \tag{10}$$

Since $\nu - \mu \neq 0$, then $N(t) = 0$ is the only possibility of equilibrium which implies that $S^* = E^* = I^* = R^* = 0$.

Thus, as Eq. (5) reveals, this model is able to describe the case when the total population experiences a net natural increase. Moreover, if $\nu > \mu$ global stability of the epidemic model (1)-(4) will not hold. However, from an epidemic point of view, it is not needed all the populations to be bounded, just the infected, $E(t)$, and infectious, $I(t)$. Hence, in this paper we propose to use the concept of partial stability to study the stability of the SEIR epidemic model given by Eqs. (1)-(4) rather than the concept of global stability. An introduction to partial stability is given in the next subsection.

B. Partial stability of systems

The problem of partial stability is referred to the problem of studying the stability of a system restricted only to a part of the variables (but not all). Pioneering results in this field are due to Rumyantsev, [14], but a large number of researches have subsequently contributed to the field through years. Interested reader can consult [12],[17] for more information on the history and developments of the partial stability theory. Now, a brief introduction to the partial stability problem is provided. Consider a nonlinear dynamic system $\dot{x}(t) = f(x)$ with state vector $x(t)$ decomposed in the form:

$$x(t)^T = \begin{bmatrix} y(t)^T & z(t)^T \end{bmatrix} \tag{11}$$

in such a way that the nonlinear system can be written as:

$$\dot{y}(t) = Y(y(t), z(t)),\ \dot{z}(t) = Z(y(t), z(t)) \tag{12}$$

Consider also that the origin $x(t)^T = 0 = \begin{bmatrix} 0 & 0 \end{bmatrix}$ is an equilibrium point. If the equilibrium point is located at other position it can be placed at the origin by a coordinates transformation. Thus, the concept of partial stability reads:

Definition 1. An equilibrium position $x = 0$ of the system (12) is:

i) locally y-stable if for any $\epsilon > 0$ there exists a $\delta(\epsilon) > 0$ such that $||x_0|| < \delta$ implies $||y(t)|| < \epsilon$ for all $t \geq t_0$ with x_0 denoting the initial condition,
ii) locally asymptotically y-stable if it is locally y-stable and furthermore $y(t) \to 0$ as $t \to \infty$.
iii) globally asymptotically y-stable if the asymptotically y-stability holds for any bounded initial condition $||x_0||$.

The intuitive meaning of Definition 1 is that the partial state variables in y are bounded for all time for a bounded initial condition of the full state regardless the tendency of the variables in z. This is an important issue since it allows us to study the behavior of just a subset of all the state variables as is convenient in epidemic models. In this way, the objectives of the paper are:

a) To study the partial stability of the vaccination-free model (1)-(4) with respect to $E(t)$ and $I(t)$ according to Definition 1. For this, consider $y^T = [E\ I]$ and $z^T = [S\ R]$. In the sequel, the y-stability property for this system will be referred to as (E, I)-stability, highlighting, thus, the variables with respect to stability is considered.
b) To design a feedback-type vaccination control law guaranteeing the partial (E, I)-stability of the epidemic model and the convergence of the infected and infectious to zero, eradicating the illness.

These objectives will be raised for both types of incidence rates, g_1 and g_2, introduced in subsection II-A. The design of the vaccination control law will be based on the partial stability frame. One of the main tools to analyze the partial stability of a nonlinear system is the extension of the classical Lyapunov theorems to this concept, [12],[13].

Thus, the following Theorem 2 holds:

Theorem 2. For system (12), assume that there exists a function L satisfying:

$$L(x) \geq a(||y||) \quad (13)$$
$$\dot{L}(x) \leq 0 \quad (14)$$

where $a(\cdot)$ denotes any continuous increasing function with argument $||y(t)||$. Then, the equilibrium position $x = 0$ of the system (12) is y-stable.

The meaning of Theorem 2 is that if we use a positive definite funcion only in the variables y, the Lyapunov theorem will be able to prove the partial stability of just those variables. Thus, this result will be used in the sequel to design a novel vaccination control law.

III. PARTIAL STABILITY OF THE VACCINATION-FREE MODEL

This section is devoted to the study of the partial stability of the vaccination-free system (i.e. when $V = 0$ in Equations (1) and (4)). [15] proved that Equations (1)-(4) lead to non-negative solutions for all $t \geq 0$ if $V = 0$. It will be proved that the infected and infectious may still be bounded for all time when the total system does not exhibit a global stability property. Hence, a further insight into the dynamics of the overall system is gained. The following theorem holds:

Theorem 3. The nonlinear system (1)-(4) is (E, I)-stable for any set of positive parameters with $\alpha \geq 1$ and incidence rate g_1 or g_2 provided that $(\mu + \sigma)(\mu + \gamma) - \beta\sigma \geq 0$.
Proof. Since $N = S + E + I + R$, then $S/N \leq 1$ and $g_1(S, E, I, R) = \beta I \frac{S}{N} \leq \beta I$. In addition, since $\alpha \geq 1$ then $1 + \alpha S > S$ and $\frac{S}{1+\alpha S} < 1$. Hence, $g_2(S, E, I, R) = \beta I \frac{S}{1+\alpha S} < \beta I$. Thus, regardless the incidence rate, Eq. (2) can be upper-bounded as:

$$\dot{E}(t) \leq \beta I(t) - (\mu + \sigma)E(t) \quad (15)$$

Now, Eq. (15) and Eq. (3) form a system of linear differential inequalities whose stability can be stated through the comparison principle by analyzing the stability of the dynamics matrix:

$$A = \begin{bmatrix} -(\mu + \sigma) & \beta \\ \sigma & -(\mu + \gamma) \end{bmatrix} \quad (16)$$

whose characteristic equation is:

$$det(sI - A) = s^2 + (2\mu + \sigma + \gamma)s + (\mu + \sigma)(\mu + \gamma) - \beta\sigma \quad (17)$$

According to the Routh-Hurwitz criterion, all the coeficients in (17) must be non-negative in order to make the dynamics matrix stable. Since all the parameters of the model are assumed to be positive, $(2\mu + \sigma + \gamma)$ is trivially positive and we only need to require:

$$(\mu + \sigma)(\mu + \gamma) - \beta\sigma \geq 0 \quad (18)$$

and the theorem is proved.

Notice that the system may still be (E, I)-stable despite $\nu > \mu$, which implies that some of the other variables diverge. Furthermore, the following corollary may be obtained:
Corollary 1. The system (1)-(4) is (E, I)-stable for both incidence rates provided that all the parameters are positive, $\alpha \geq 1$ and $\mu + \gamma \geq \beta$.
Proof. Since $\mu + \sigma > \sigma$, then Eq. (18) can be satisfied if $\mu + \gamma \geq \beta$.

Corollary 1 provides an insight concerning when the epidemics is bounded despite any natural increasing of the population (global instability). Thus, μ represents the rate of deaths for causes unrelated to the infection, γ the rate at which a new infective losses its infection capacities and β the rate of new infectious. Thus, when people die soon and the latent period of being infective is small compared with the transmission velocity, the disease will not be able to become unbounded. Hence, the application of the concept of partial stability gives another insight in the analysis of the model and relates stability properties with more practical issues.

IV. DESIGN OF A VACCINATION LAW BASED ON PARTIAL STABILITY

In this section a feedback-type vaccination control law will be designed starting from the concept of partial stability. Thus, the objective of the control is not to stabilize all the variables of the system, but only (E, I)-stabilize the system regardless the other variables. For this, Lyapunov's type Theorem 2 will be used to prove:

Theorem 4. The vaccination law given by:

$$V(t) = 1 + \frac{\omega}{\nu N(t)} R(t) \quad (19)$$

(E, I)-stabilizes the system of equations (1)-(4) for any set of positive parameters and any incidence rate. Furthermore, $S(t), E(t), I(t) \to 0$ as $t \to \infty$.

To perform the proof of Theorem 4 we will need the following result:

Theorem 5. The solution of the system of Equations (1)-(4) under the vaccination law (19) satisfies $S(t), E(t), I(t), R(t) \geq 0$ for all $t \geq 0$ for any incidence rate and any set of positive parameters provided that $S(0), E(0), I(0), R(0) \geq 0$.
Proof. It is firstly proved that the total population remains non-negative for all time. Thus, from Eq. (5) we have:

$$N(t) = e^{(\nu - \mu)t} N(0) \geq 0 \quad (20)$$

since $N(0) = S(0) + E(0) + I(0) + R(0) \geq 0$. Now it will be proved that the susceptible are non-negative. For this, notice that both incidence rates g_1 and g_2 can be expressed as $g = g'(S, E, I, R) \cdot S(t)$. Thus, Eq. (1) under vaccination law (19) takes the form:

$$\dot{S}(t) = -\left(\mu + g'(S,E,I,R)\right) S(t) \tag{21}$$

Now we will proceed by contradiction. Assume that there exists a time instant t^* such that $S(t^*) < 0$. Thus, since $S(t)$ is a continuous function there must be a time instant $t_S < t^*$ such that $S(t_S) = 0$. However, Eq. (21) implies $\dot{S}(t_S) = 0$ deducing that $S(t) = 0$ for all $t \geq t_S$. Thus, this contradicts the existence of such a t^* and the susceptible cannot become negative. A case-based reasoning will be used to prove the non-negativeness of E and I. For this, recall that the incidence rates can also be expressed as $g = g^*(S,E,I,R) \cdot I(t)$, i.e. g vanishes when I vanishes. Thus, assume that there exists a time instant t_{EI} such that $E(t_{EI}) = I(t_{EI}) = 0$. Hence, Eqs. (2) and (3) imply $\dot{E}(t_{EI}) = \dot{I}(t_{EI}) = 0$ and $E(t) = I(t) = 0$ for all $t \geq t_{EI}$. This means that when both variables vanish simultaneously, they remain in zero for all time onwards. Now, define the following time instants:

$$\begin{aligned} t_E &= \{t | E(t) = 0 \wedge I(t) > 0 \wedge S(t) > 0\} &(22)\\ t_I &= \{t | I(t) = 0 \wedge E(t) > 0\} &(23)\\ t_{E2} &= \{t | E(t) = 0 \wedge I(t) > 0 \wedge S(t) = 0\} &(24)\end{aligned}$$

These time instants can be interpreted as the time instants when one variable E or I zeroes. Notice that in all cases it is supposed that the other variable is positive since as it has been proved before, if both variables vanish simultaneously, they remain in zero for the rest of the time. Thus, for t_E we have $I(t_E) > 0$ and:

$$\dot{E}(t_E) = g(S,E,I,R) = g^*(S,E,I,R) \cdot I(t_E) > 0 \tag{25}$$

implying that E does not become negative but it tends to be positive again while for t_I we have $E(t_I) > 0$ and:

$$\dot{I}(t_I) = \sigma E(t_I) > 0 \tag{26}$$

implying that I does not become negative but it tends to be positive again. Now, from the definition of t_{E2}, we have that $\dot{E}(t) = 0$ and $\dot{I}(t) = -(\mu+\gamma)I(t)$ for all $t \geq t_{E2}$ implying:

$$I(t) = e^{-(\mu+\gamma)(t-t_{E2})} I(t_{E2}) \geq 0 \tag{27}$$

for all $t \geq t_{E2}$. Hence, both variables E and I remain non-negative for all time. Finally, the explicit solution of Eq. (4) under vaccination law (19) can be written as:

$$R(t) = e^{-\mu t} R(0) + \int_0^t e^{-\mu(t-\tau)} \left[\gamma I(\tau) + \nu N(\tau)\right] d\tau \geq 0 \tag{28}$$

which is non-negative for $R(0) \geq 0$ since the rest of variables S, E, I and N have been proved to be non-negative. Thus, the theorem is proved.
Now, we can prove Theorem 4.

Proof of Theorem 4. Consider the partially positive function:

$$L(t) = S(t) + E(t) + I(t) \tag{29}$$

$L(t)$ is positive definite in S, E and I from Theorem 5 but it is not a positive definite function in the complete state $x = [S\ E\ I\ R]$. The time derivative of (29) is calculated as:

$$\begin{aligned} \dot{L}(t) &= \dot{S}(t) + \dot{E}(t) + \dot{I}(t) \\ &= -\mu S(t) + \omega R(t) + \nu N(t)(1 - V(t)) \\ &\quad -\mu E(t) - (\mu+\gamma) I(t) &(30)\end{aligned}$$

If Eq. (19) is introduced in (30) one obtains:

$$\dot{L}(t) = -\mu S(t) - \mu E(t) - (\mu+\gamma) I(t) \leq 0 \tag{31}$$

Thus, we are in conditions of applying Theorem 2, and therefore, S, E and I are bounded for all time. Furthermore, while any of the variables, S, E, I is positive, $\dot{L}(t) < 0$, implying that L decreases continually until it arrives to $S = E = I = 0$. Hence, all these variables converge to zero, proving the theorem.

Therefore, the epidemics is eradicated while the rest of variables evolve. Appreciate the rationale behind the vaccination law (19). It is designed in order to cancel the positive terms appearing in Eq. (30) so as to make the derivative of L negative semidefinite (in the complete state). Furthermore, this design technique would not be used if a Lyapunov function in the complete state (e.g. $L = S + E + I + R$) would be proposed, since the vaccination function $V(t)$ disappears when all the subpopulations are summed up. In consequence, the partial stability approach has also provided us a practical vaccination design tool. Moreover, Theorem 4 also proves the convergence of the susceptible to zero despite we are now only interested in the infected and infectious. Finally, note that the vaccination control law holds for any kind of incidence rate since its particular value is canceled when summing up the equations for $\dot{S}$ and $\dot{I}$. Therefore, the vaccination strategy holds for general nonlinear incidence rate equations.
The following section provides some simulation examples of the applicability and usefulness of the approach as well as a comparison with some previous vaccination policies.

V. SIMULATION EXAMPLES

In this section some simulation examples are presented in order to illustrate the theoretical results stated in the previous sections. Thus, the following parameters have been used for all simulations: $\mu^{-1} = 200$ days, $\sigma^{-1} = 2.2$ days, $\omega^{-1} = 15$ days, $\gamma = \sigma$, $\nu^{-1} = 150$ days, $g = g_1$ and $\beta = 1.66$. The initial conditions are given by $S(0) = 400$, $E(0) = 150$, $I(0) = 250$ and $R(0) = 200$. Notice that since ν^{-1} is smaller than μ^{-1}, then the total population increases through time. These parameters have been chosen so as to illustrate the theoretical results in a short-time simulation. The total simulation period is 50 days. Figure 1 shows the evolution of the disease in the absence of vaccination. There are two significant facts that deserve commentary. The first one is that there is a number of infected an infectious through time, i.e. the disease does not disappear in a natural way. Note that according to Theorem 1 there is not an equilibrium position, and thus, the values for the populations always change through time. The other one is that the model is not globally bounded since the total population diverges as Figure 2 confirms. Thus, a vaccination policy is applied to this model in order to make the infectious and infected vanish.

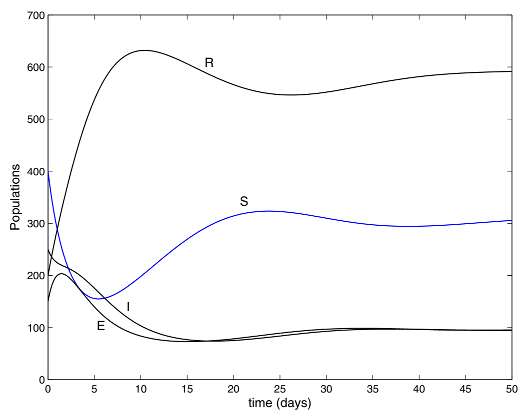

Fig. 1. Dynamics of the system without vaccination.

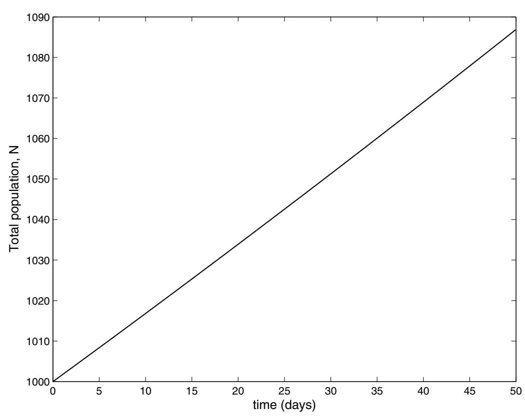

Fig. 2. Dynamics of the total population, N, without vaccination.

Figure 3 depicts the evolution of the disease when the vaccination law (19) is applied to the system. Thus, the susceptible, infected and infectious converge to zero while the total population tends to be immune, as Theorem 4 states. Figure 4 shows a zoom on the final period of the simulation. It can be verified that the infective and infectious are already zero while the susceptible tends to zero. The rate at which the susceptible tends to zero is given by μ once the infectious and infected have vanished. Since this value is in general small, then the convergence of the susceptible to zero is slow. Finally, Figure 5 shows the vaccination law.

Note that the total population is still not bounded since Figure 3 shows the immune diverging. Hence, the vaccination control law does not try to globally stabilize the system but only the variables corresponding to the epidemics, i.e. it only tries to partially stabilize the system. The next section

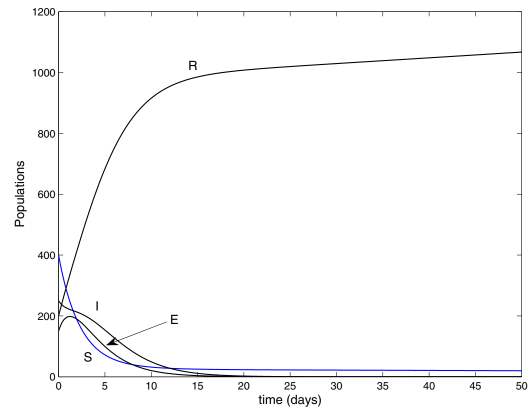

Fig. 3. Dynamics of the system with the proposed vaccination.

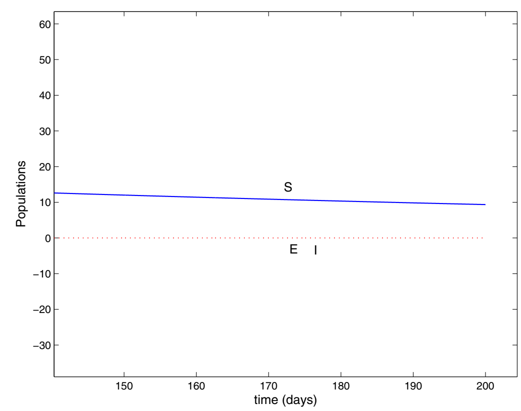

Fig. 4. Zoom on a large-term simulation of the controlled system.

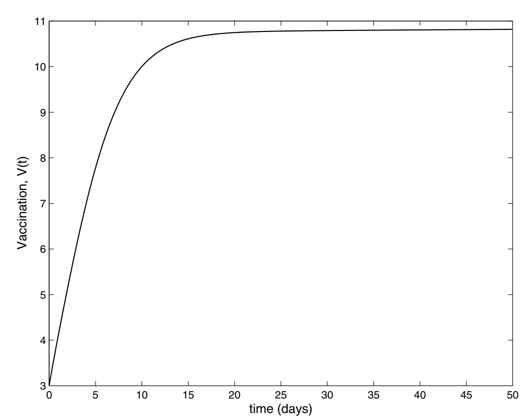

Fig. 5. Vaccination function, $V(t)$.

performs a comparison between the proposed control law and others previously suggested in the literature.

A. Comparison with other vaccination laws

This section contains a comparative study of the proposed control law with respect to others recently proposed in the literature. Three indexes will be used in order to perform the comparison:

- The total control effort (CE) defined as:

$$CE = \int_0^{T_{final}} N(t)V(t)dt \tag{32}$$

  where T_{final} denotes the final time of simulation, which in our case is 50 days.
- The maximum value of the control (MV):

$$MV = max\{V(t)|t \geq 0\} \tag{33}$$

- The time in days needed to eradicate the illness (TE):

$$TE = min\{t|I(t) < 1\} \tag{34}$$

The lower all the above values are, the better the control law is. Moreover, three control laws will be used for comparison. The one proposed in [4] and two proposed in [15] through equations (26b) and (69) introduced therein. The obtained results are condensed in Table (I).

TABLE I

COMPARISON BETWEEN DIFFERENT VACCINATION LAWS.

	CE	MV	TE
Proposed	$5.252 \cdot 10^5$	10.93	22.58
[4]	$5.371 \cdot 10^5$	11.08	22.83
[15]. Eq. (26b)	$5.407 \cdot 10^5$	11.04	22.91
[15]. Eq. (69)	$5.233 \cdot 10^5$	20.21	22.75

The free design parameters of the compared control laws have been adjusted in order to obtain a similar time to eradicate the illness, TE, among all of them. In this way, the comparison will be fair since the same performance with respect to infectious vanishing is used for all of them. The first conclusion is that all the control laws offer a similar behavior once fixed the TE parameter. The only one to offer a distinct value is the last one which possesses a larger peak value for the control law, MV. Hence, the proposed control law is slightly better than the others. However, the presented approach has the advantage that its derivation and proof of stability and convergence is much easier than those made in [4] and [15]. Hence the presented approach is more convenient than a classical global-stability frame to deal with this problem.

VI. CONCLUSIONS

This paper proposes to use the concept of partial stability rather than the global stability one to deal with the stability issues of epidemic models. The partial stability is able to provide a more meaningful analysis of the problem since it only focuses on the behavior of some of the variables (infected and infectious) instead of the complete population. Thus, it can capture the situation when a natural increase of the population occurs which would not lead to a global stability property. It has been shown that the vaccination-free SEIR model can still be partially stable even when a gobally stability property does not hold for two types of nonlinear incidence rates. In addition, a feedback-type vaccination control law has been designed from the concept of partial stability through adapted Lyapunov-type methods. The proposed technique opens a new line to design vaccination policies based on partial stability.

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