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Inverse Optimal Impulsive Control for a SIR Epidemic Model

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Abstract—Epidemiological models constitute a fundamental means to understand the behavior and evolution of infectious diseases, as well as the mechanisms of action to counteract them. If an infectious disease spreads rapidly and affects a big population, it is vital to have effective control schemes that optimize the amount of available resources to mitigate the propagation of the disease, before it becomes a risk condition for public health systems. Inspired by this idea, we consider an extension of the SIR epidemic model and study the application of an impulsive control action to this system that minimizes vaccines and treatment given to population, using an inverse optimal control (IOC) approach.

Index Terms—Inverse optimal control, impulsive control, epidemic model, seasonality.

I. INTRODUCTION

Since the appearance in 1927 of the Kermack and McKendrick's mathematical model for the spread of infectious diseases, also known as the SIR epidemic model, several epidemiological models have been proposed by a variety of researchers to explain the mechanisms of propagation for this kind of diseases. Epidemiological models have become important tools in analyzing the spread and control of infectious diseases such as measles, rubella, influenza, malaria, rabies, gonorrhea, cholera, tuberculosis and HIV/AIDS [1]-[3]. Vaccination of susceptible individuals and treatment of infectives are two commonly used methods for preventing and controlling epidemic outbreaks. If adequate and opportune control schemes, based on these two forms, are applied to infectious diseases, most of them could be driven toward eradication [4]. On the other hand, if poor and inefficient control actions are taken to bring down an infectious disease, it could remain endemic.

The aim of this letter is to demonstrate that an appropriate impulsive control action, based on the combined strategy of vaccination and treatment, can be applied to an epidemiological model to prevent the spread of an infectious disease, and even achieve its eradication. In order to do this, the rest of the paper is organized as follows. Section II introduces background information about the classical SIR epidemic model. Section III describes the control schemes applied on epidemic models to mitigate the propagation of infectious diseases at the theoretical level (two namely, *constant* and *impulsive*). In Section IV, the problem of the inverse optimal control for *impulsive dynamical systems* is addressed, because of the control action proposed for the infectious model will be impulsive-type. In Section V, the impulsive control action that will drive the treated epidemic model toward a disease-free solution is found out, optimally combining the vaccination and treatment strategies. Numerical simulations and discussion of results are offered in Section VI. And finally, some conclusions are drawn in Section VII.

II. THE SIR EPIDEMIC MODEL

In the classical SIR epidemic model, the population splits into three nonintersecting classes: individuals who are healthy but can contract the disease, called *susceptible individuals S*; individuals who have contracted the disease, and can transmit it, called *infected individuals I*; and individuals who have recovered and cannot contract the disease again due to a permanent infection-acquired immunity, called *recovered individuals R* [3]. A set of assumptions is made on this model to simplify reality: first, the infected individuals are also *infective* right after contract the disease, meaning that incubation period is short enough to be negligible; second, total population size, N, remains constant during the study period, where N = S + I + R, with each of these three classes (S, I, R) changing in time; and third, all classes are uniformly mixed. The model obtained on the above assumptions is as follows

$$S = -\beta SI,$$
 $I = \beta SI - \alpha I,$ $R = \alpha I$ (1)

It consists of a system of ordinary differential equations (ODEs) that describe the rate of change in each class. The constant β is called the *transmission rate* and it represents the probability at which susceptible individuals move to infected class after enter into contact with infective individuals;

therefore, the term βSI is the number of individuals who become infected per unit of time, and that is proportional to the amount of infectives and susceptibles (*bilinear incidence*). α is known as the *recovery rate* and it describes the percentage of infected individuals that naturally recovers per unit of time – or dies, if the disease is fatal; ergo, the term αI is the number of individuals who move from the infected class to the recovered one during the same timeframe [1], [3], [5].

The mathematical formulation of the epidemic problem above is completed given initial conditions such as $S(0) = S_0 > 0$, $I(0) = I_0 > 0$, and $R(0) = R_0 \ge 0$. Also, *S*, *I*, and *R* quantities must satisfy the normalization condition S + I + R = 1 in the previous equations [2].

Realistic infectious-disease models incorporate additional effects to the system's equations to describe specific epidemics with different properties, and also to provide a better understanding of their particular dynamics. Among the possible aspects to be included are the following: mortality, immunity, stages of infection, age structure, social and sexual mixing groups, time evolution of epidemics, etcetera [1], [2], [4]–[6].

The model presented in (1) possesses particular characteristics that describe the standard results for the propagation of infectious diseases [3]. Since $\dot{S} < 0$ and $\dot{R} > 0$ for all time, the number of susceptible individuals is always decreasing while the number of the recovered population is consistently increasing over time, independently of the initial conditions (S_0, I_0, R_0) . In consequence, S and R exhibit monotonous behaviors bounded below and above by 0 and N, respectively, until a steady state is finally reached. Conversely, the number of infected individuals I may be monotonically decreasing to zero, or may display a nonmonotone behavior by first increasing to some maximum level, and then decreasing to zero (see Fig. 1). The sudden increase in the prevalence of an infectious disease, and its subsequent decline to zero, is the classical model of an epidemic outbreak [5]. For the SIR epidemic model, the prevalence first stars to increase if $\tilde{I}(0) = (\beta S_0 - \alpha)I_0 > 0$; hence, the necessary and sufficient condition for an initial increase in the amount of infected population throughout an infectious period (epidemic scenario) is $\beta S_0 - \alpha > 0$, or $\frac{\beta S_0}{\alpha} > 1$ [5].

III. CONTROL SCHEMES

In recent years, plethora of control schemes for infectious diseases have been proposed in literature by a diversity of authors (see [2], [4], [6]–[10] and the references therein); nonetheless all of these can be grouped into two broad categories: constant and impulsive schemes. The *constant control* scheme is a conventional control strategy in which vaccination, or treatment, is regularly (continuously in time) given to a fraction $0 \le p \le 1$ of individuals in the population, in such a way that the number of susceptible and infected individuals are decreased, while the number of recovered people is increased. By contrast, *impulsive* (or *pulse*) *control* scheme is based on the strategy of applying vaccinations, or treatments, periodically to a portion p of the population in a

very short time period, compared to the time scale for the dynamics of the disease [2], [8], [9].

According to Li, Cui, Anderson, and May, time-constant control strategies lead to epidemic eradication if the proportion of the successfully vaccinated individuals is larger than a certain critical value [8], [11]; however, in some cases it could result in a population coverage as high as 95% (measles, for example), and this control scheme becomes difficult and expensive to implement. Under these circumstances, pulse control strategies offer a more tractable and efficient control scheme; theoretical results show that the pulse vaccination strategy can be distinguished from the conventional strategy in leading the disease eradication at relatively low values of vaccination [8], [13].

IV. INVERSE OPTIMAL CONTROL FOR IMPULSIVE SYSTEMS

Dynamical systems involving continuous-time dynamics and discrete (impulsive) events are called *hybrid dynamical systems*. Modern complex engineering systems with a highly interconnected and interdependent, physical and abstract, hierarchical structure, are characterized by continuous-time dynamics at the lower-level units and logical decision-making units at the higher levels of hierarchy represent some of such systems. However, hybrid systems also abound in nature and are not limited to engineering-controlled systems. For example, hybrid systems arise naturally in biology, pharmacology, economics, chemistry, among several others [14], [15].

A. Nonlinear Impulsive Dynamical Systems

Impulsive dynamical systems can be viewed as a subclass of hybrid systems and consists of three elements [13].

- i. A continuous-time differential equation, which governs the motion of the system between impulsive or resetting events;
- ii. A difference equation, which governs the way the states are instantaneously changed when a resetting event occurs; and
- iii. A criterion for determining when the states of the system are to be reset.

Thus, an impulsive dynamical system has the form

$$\dot{x}(t) = f_c(x(t)), \quad x(0) = x_0, \quad t \neq t_k,$$
 (2)

$$\Delta x(t) = f_d(x(t)), \qquad t = t_k \qquad , \qquad (3)$$

where $t \ge 0$, $x(t) \in D \subseteq \mathbb{R}^n$, *D* is an open set with $0 \in D$, $\Delta x(t) \triangleq x(t^+) - x(t)$, where $x(t^+) \triangleq x(t) + f_d(x(t)) = \lim_{\varepsilon \to 0} x(t + \varepsilon)$, $f_c: D \to \mathbb{R}^n$ is continuous, $f_d: D \to \mathbb{R}^n$ is continuous, and $\mathfrak{T} = \{t_k\}$ is the resetting set with $t_k \in \mathbb{R}$, $t_k < t_{k+1}$, $k = 1, 2, \cdots$.

It is assumed that the continuous-time dynamics $f_c(\cdot)$ is such that the solution to (2) is jointly continuous in *t* and x_0 between resetting events. The differential equation (2) is referred as the *continuous-time dynamics*; and difference equation (3), as the *resetting law*. In addition, the notation $s(t, \tau, x_0)$ denotes the solution x(t) of (2) and (3) at time equation $t \ge \tau$ with initial condition $x(\tau) = x_0$. Finally, a point $x_e \in D$ is an *equilibrium point* of (2) and (3) if and only if $s(t, \tau, x_e) = x_e$ for all $\tau \ge 0$ and $t \ge \tau$. Note that $x_e \in D$ is an equilibrium point of (2) and (3) if and only if $f_c(x_e) = 0$ and $f_d(x_e) = 0$.



Fig. 1. Comportment of infected population in the SIR epidemic model. *Top*: Behavior monotonically decreasing to zero, $\beta S_0/\alpha < 1$. *Bottom*: Nonmonotone behavior first increasing and then decreasing to zero (epidemic outbreak), $\beta S_0/\alpha > 1$.

B. Inverse Optimal Control

In order to avoid the complexity in solving the Hamilton-Jacobi-Bellman equations of an optimal control problem for nonlinear impulsive dynamical systems, Haddad, Chellaboina, and Nersesov characterized a class of globally stabilizing hybrid controllers for the *inverse optimal hybrid control problem* [13]. This minimizes some *derived* cost functional that provides flexibility in specifying the control law [13], [15].

Consider the controlled impulsive dynamical system

$$\dot{x}(t) = f_c(x(t)) + G_c(x(t))u_c(t), \ x(0) = x_0, \ t \neq t_k, (4)$$

$$\Delta x(t) = f_d(x(t)) + G_d(x(t))u_d(t), \ t = t_k$$
(5)

and hybrid performance functional

$$J(x_{0}, u_{c}(\cdot), u_{d}(\cdot)) = \int_{0}^{\infty} [L_{1c}(x(t)) + u_{c}^{T}(t)R_{2c}(x(t))u_{c}(t)]dt + \sum_{k \in \mathbb{Z}_{[0,\infty)}} [L_{1d}(x(t_{k})) + u_{d}^{T}(t_{k})R_{2d}(x(t_{k}))u_{d}(t_{k})]$$
(6)

where $u_c(\cdot)$ and $u_d(\cdot)$ are restricted to a class of admissible hybrid controls consisting of measurable functions such that $(u_c(t), u_d(t_k)) \in U_c \times U_d$ for all $t \ge 0$ and $t_k \in \mathbb{R}$, $t_k < t_{k+1}$, $k = 1, 2, \cdots$, $L_{1c}: \mathbb{R}^n \to \mathbb{R}$ and satisfies $L_{1c}(x) \ge 0$, $x \in \mathbb{R}^n$, $R_{2c}: \mathbb{R}^n \to \mathbb{P}^{m_c}$, $L_{1d}: D \to \mathbb{R}$ and satisfies $L_{1d}(x) \ge 0$, $x \in \mathbb{R}^n$, and $R_{2d}: D \to \mathbb{P}^{m_d}$. Also, assume there exists a continuously differentiable function $V: \mathbb{R}^n \to \mathbb{R}$, and functions $P_{12}: D \to \mathbb{R}^{1 \times m_d}$ and $P_2: D \to \mathbb{N}^{m_d}$ such that V(0) = 0, V(x) > 0, $x \in \mathbb{R}^n$, $x \neq 0$,

$$V'(x)\left[f_{c}(x) - \frac{1}{2}G_{c}(x)R_{2c}^{-1}(x)G_{c}^{T}(x)V'^{T}(x)\right] < 0, \quad x \neq 0, (7)$$

$$V\left(x + f_{d}(x) - \frac{1}{2}G_{d}(x)\left(R_{2d}(x) + P_{2}(x)\right)^{-1}P_{12}^{T}(x)\right) - V(x) \le 0, \quad (8)$$

$$V(x + f_{d}(x) + G_{d}(x)u_{d}) = V\left(x + f_{d}(x)\right) + P_{12}(x)u_{d} + u_{d}^{T}P_{2}(x)u_{d}, u_{d} \in R^{m_{d}}, (9)$$

where u_d is admissible, and

$$V(x) \to \infty \text{ as } \|x\| \to \infty. \tag{10}$$

Then the zero solution $x(t) \equiv 0$ of the close loop system in (4) and (5) is globally asymptotically stable with the optimal hybrid feedback control law

$$u_{c}(t) = \phi_{c}(x) = -\frac{1}{2}R_{2c}^{-1}(x)G_{c}^{T}(x)V'^{T}(x), \quad t \neq t_{k}, (11)$$

$$u_{d}(t) = \phi_{d}(x) = -\frac{1}{2}\left(\left(R_{2d}(x) + P_{2}(x)\right)^{-1}P_{12}^{T}(x)\right), \quad t = t_{k}, (12)$$

and performance functional (6), with

$$L_{1c}(x) = \phi_c^T(x)R_{2c}(x)\phi_c(x) - V'(x)f_c(x)$$

$$L_{1d}(x) = \phi_d^T(x)(R_{2d}(x) + P_2(x))\phi_d(x) - V(x + f_d(x)) + V(x).$$
(13)

is minimized in the sense that

$$J\left(x_{0},\phi_{c}\left(x(\cdot)\right),\phi_{d}\left(x(\cdot)\right)\right) = \underset{\left(u_{c}(\cdot),u_{d}(\cdot)\right)\in C\left(x_{0}\right)}{\min} J\left(x_{0},u_{c}\left(\cdot\right),u_{d}\left(\cdot\right)\right), x_{0} \in \mathbb{R}^{n}, (15)$$
$$J\left(x_{0},\phi_{c}\left(x(\cdot)\right),\phi_{d}\left(x(\cdot)\right)\right) = V(x_{0}), \qquad x_{0} \in \mathbb{R}^{n}, (16)$$

where $\phi_c: D \to U_c$, $\phi_d: D \to U_d$, and $(u_c(t), u_d(t_k)) = (\phi_c(x(t)), \phi_d(x(t_k)))$ satisfies (4) and (5) for $x(t), t \ge 0$.

Complementary explanations and formal proof for the optimal hybrid control law above can be found in literature [13].

V. INVERSE OPTIMAL IMPULSIVE CONTROL FOR AN EXTENSION OF THE SIR MODEL

A. SIR Epidemic Model with Seasonality

There exist several variations of the classical SIR model to describe epidemics evolution with different characteristics with respect to mortality, immunity, infectivity, and time horizon [4]. One of these variations proposed by Liu and Stechlinski analyzes an infectious disease model with variable transmission rate and pulse control scheme as a means to eradicate the disease [2]; this is considered in this article to develop an *inverse optimal impulsive controller*. Population is divided into three distinct compartments: the susceptible, *S*; the infected, *I*; and the removed, *R*. Each individual may have either of the following transitions $S \rightarrow I \rightarrow R$, or stay in the susceptible state

forever. Vital dynamics (births and deaths) are also included. The birth rate of the population into the susceptible class is $\mu > 0$, which equals the natural death rates in the three classes μS , μI , and μR . Thus, it is assumed that all individuals are born without the disease – i.e., there is not vertical transmission from mother to unborn child. The natural recovery rate from the disease for all individuals is $\alpha > 0$.

In this approach, the transmission rate β is treated as a timevarying parameter motivated by seasonal changes in the transmission of an infectious disease, in contrast to major part of epidemic models that assume a constant transmission rate in time for a given population [2]. Hence, the transmission rate is modelled as a switching parameter $\beta_i > 0$ with $i \in \{1, 2, ..., m\}$, and is governed by a switching rule $\sigma(t): (t_{n-1}, t_n] \rightarrow \{1, 2, \dots, m\},\$ for n = 1, 2, ..., which is a piecewise continuous function (assumed to be continuous from the left). That is, $i = \sigma(t)$ on the interval $(t_{n-1}, t_n]$, and at the switching times $t = t_n$ the parameter switches values according to the value of β_i $\sigma(t^+) \coloneqq \lim_{h \to 0^+} \sigma(t_n + h)$. The switching times satisfy $t_0 = 0 < t_1 < t_2 < \dots < t_n < \dots$, with $t_n \to \infty$ as $n \to \infty$. Denote the set of all such switching rules by *s*.

We consider the following SIR epidemic model with seasonality, and pulse control of vaccination and treatment

$$\begin{split} \dot{S} &= \mu - \beta_{i} SI - \mu S, & t \neq t_{k}, \\ \dot{I} &= \beta_{i} SI - \alpha I - \mu I, \\ \dot{R} &= \alpha I - \mu R, \\ S(t^{+}) &= (1 - p) S(t), & t = t_{k}, \\ I(t^{+}) &= (1 - p) I(t), \\ R(t^{+}) &= R(t) + p S(t) + p I(t). \end{split}$$
(17)

Variables have been normalized by the total population N, which is constant, so that the meaningful physical domain for this system is $\Omega_{SIR} = \{(S, I, R) \in \mathbb{R}^3_+ | S + I + R = 1\}$. The initial conditions are $S(0) = S_0 > 0$, $I(0) = I_0 > 0$, and $R(0) = R_0 > 0$ such that $(S_0, I_0, R_0) \in \Omega_{SIR}$.

Furthermore, consider that a portion $0 \le p \le 1$ of the infected population is successfully treated in a relatively short time period, compared to the timespan for the dynamics of the disease; and that the same portion p, but of susceptible population, is vaccinated at the same time. Then, suppose that infected individuals impulsively treated at times t_k with k = 1,2,..., and susceptible ones impulsively vaccinated, immediately enter the recovered class with permanent immunity. This is reasonable from a physical perspective, since the treatment and vaccination processes take a certainly short amount of time.

B. Inverse Optimal Impulsive Controller

Now that the SIR epidemic model with seasonality has been presented, we will set out the inverse optimal control for such system, which impulsively changes its state due to the pulse control employed to eradicate the disease.

Epidemiological model in (17) can be represented as the controlled impulsive dynamical system in (4) and (5), with the state variables *S*, *I*, and *R* impulsively changed at the resetting times $t = t_k$ with k = 1, 2, ..., and suitable feedback control law

(11,12) which solves the inverse optimal control (IOC) problem. Specifically,

$$\dot{x}(t) = f_c(x(t)) + \phi_c(x(t)), \qquad t \neq t_k, \qquad (18)$$
$$(\mu - \beta_i SI - \mu S,$$

$$f_c(x(t)) \triangleq \begin{cases} \beta_i SI - \alpha I - \mu I, \\ \alpha I - \mu R. \end{cases}$$
(19)

$$\phi_c(x(t)) = 0 \tag{(1)}$$

$$\Delta x(t) = f_d(x(t)) + \phi_d(x(t))$$

$$(\Delta S(t) = -pS(t),$$
(20)

$$f_d(x(t)) = \begin{cases} \Delta I(t) = -pI(t), & t = t_k, \\ \Delta R(t) = pS(t) + pS(t). \end{cases}$$
(21)

$$\phi_d(x(t)) = abs\left(-\frac{1}{2}\left(\left(R_{2d}(x) + P_2(x)\right)^{-1}P_{12}^T(x)\right)\right)$$
(22)

where $x(t) \triangleq [S \ I \ R]^T$, and $abs(\cdot)$ in $\phi_d(x(t))$ takes the absolute value of the function. The reason for this positivity restriction is to make the problem physically interesting (positive fractions of infected, or susceptible, population can only be considered).

Notice that, because of the impulsive control scheme proposed in (17), the continuous-time control function $\phi_c(x(t))$ equals zero in (18), i.e., there exists no constant control action for the system. Moreover, $\phi_d(x(t))$ drives now the impulsive control action in (20) to make it dependent of the system state, in such a way that a better performance is achieved. In this sense

$$\Delta x(t_k) = x(t_k^{-}) - \phi_d(x(t_k))$$
(23)
$$\Delta x(t_k) = \begin{cases} \Delta S(t_k) = S(t_k^{-}) - p_1 S(t_k^{-}), \\ \Delta I(t_k) = I(t_k^{-}) - p_2 I(t_k^{-}), \\ \Delta R(t_k) = R(t_k^{-}) + p_1 S(t_k^{-}) + p_2 I(t_k^{-}). \end{cases}$$
(24)

where p_1 and p_2 are the portions of individuals vaccinated and treated, respectively, every resetting time t_k , dictated by the inverse optimal impulsive controller (22). Finally, the condition $x(t_k^-) - \Delta x(t_k)$ establishes the initial condition x_0 for (18) after a resetting time t_k has occurred.

VI. NUMERICAL SIMULATIONS AND RESULTS

Numerical simulations of the impulsive-controlled SIR epidemic model defined in (18) and (23), with the inverse optimal control (22), are computed using MATLAB to illustrate the main theoretical results. All parameters values are chosen hypothetically owing to the unavailability of real world data but based on a realistic criterion.

First, motivated by seasonal practical application (as the spread of a winter-infectious disease, like influenza), assume that the switching for the transmission rate, β_i , is periodic, according to the next switching rule

$$\sigma(t) = \begin{cases} 1 & \text{during the winter months,} \\ 2 & \text{otherwise.} \end{cases}$$

So, if switching intervals, for each transmission rate, are defined as $\tau_n = t_n - t_{n-1}$, for n = 1, 2, ..., the period of the

switching rule above is given by $T = \tau_1 + \tau_2$, where T = 1, $\tau_1 = 0.25$, and $\tau_2 = 0.75$ years. Now, let the transmission rates take the values $\beta_1 = 9$ during the winter, and $\beta_2 = 1$ for other seasons, to emulate a scenario where the rate of incidence increases considerably over this period.

Second, as stated in section II, the condition for an epidemic to occur is that $\dot{I}(0) > 0$; consequently, for the SIR model examined the inequality $(\beta_i S_0 - \alpha - \mu)I_0 > 0$ must be satisfied, which can be reformulated as $\beta_1 S_0 / (\alpha + \mu) > 1$. Let's consider the initial conditions, and the birth and the recovery rates as follows: $S_0 = 0.7$, $I_0 = 0.3$, $R_0 = 0$, $\mu = 0.1$, and $\alpha = 0.9$. μ and α are selected to portray a moderated renewal rate of the total population in the timespan of simulation, and a strong ability to recover from the disease.

Third, suppose that $p_1 = p_2 = 0$ unless $t = t_k = kT$ with k = 1,2,..., that is, control pulses are applied every T time units, which implies executing impulsive treatment and vaccination yearly (it is assumed that there is no impulsive effect at the initial time t = 0).

Last, parameters for the impulsive control function (22) have been optimized to get the desired behavior of the system in terms of the disease's eradication. Final numerical values determined heuristically are

$$R_{2d} = \begin{bmatrix} 15 & 0 \\ 0 & 10 \end{bmatrix}, \quad P_2 = \begin{bmatrix} 1 & 0 \\ 0 & 1 \end{bmatrix}, \quad P_{12} = \begin{bmatrix} 5 & 1 \end{bmatrix}$$

Infected population I is selected as the variable that impact the contribution of the matrix P_2 to the control law (22) at every resetting time t_k , while susceptible population S affects the weights of R_{2d} and P_{12} , simultaneously. The following expressions summarizes what has been mentioned

$$\phi_d(x(t)) = \begin{bmatrix} p_1(x) \\ p_2(x) \end{bmatrix}, \quad t = t_k$$

$$\phi_d(x(t_k)) = abs \left(-\frac{1}{2} \left(\left(\begin{bmatrix} 15 & 0 \\ 0 & 10 \end{bmatrix} S(t_k) + \begin{bmatrix} 1 & 0 \\ 0 & 1 \end{bmatrix} I(t_k) \right)^{-1} \begin{bmatrix} 5 \\ 1 \end{bmatrix} S(t_k) \right) \right)$$
(25)

Simulation results in Fig. 2 show that the system converges to a periodic disease-free solution, where infected population I decreases to zero as the time approaches to infinity; this way, the infectious disease is successfully eradicated. However, we can observe that in an interval of approximately only 10 years, the system almost reaches the disease-free equilibrium. Through a tuning process of the control parameters R_{2d} , P_2 , and P_{12} , it is possible to define different times of converge for the same equilibrium point, or even to solutions where $I \neq 0$ as $t \rightarrow 0$ ∞ (endemic states). If the parameter values of the SIR epidemic model change to reflect more stringent conditions – e.g., higher β_i transmission rates –, the system will probably take longer to converge to the periodic disease-free solution, or even will converge to one of the multiple existing endemic states, in which case, values for the impulsive control function (22) must be adjusted to obtain the desired behavior.

Figure 3 illustrates how the inverse optimal controller works in estimating the number of susceptible individuals p_1

that should be vaccinated per year, and the number of individuals that should be treated p_2 , to achieve the eradication objective for the disease. The solid red lines indicate that, at the steady state of the system, vaccination strategy remains around 5.6% of the total population throughout the entire time horizon considered. Conversely, the blue graphic establishes that the percentage of people that must be treated at the end of the simulation time is close to 0%, which makes sense since the infectious disease have been eradicated from population. Again, if control parameters R_{2d} , P_2 , and P_{12} , are modified, different percentages p_1 and p_2 could result.



Fig. 2. Solution to the SIR model with seasonality and inverse optimal impulsive control. The system reaches a disease-free equilibrium.

In order to introduce a comparison between the inverse optimal impulsive control action proposed on this article and conventional control schemes employed to contain the spread of infectious diseases, we consider anew the SIR epidemic model with seasonality previously analyzed, and the parameter values used for simulation of Fig. 2. Figure 4 demonstrates the effects of constant (top graphic) and pulse (bottom) control schemes on the behavior of infected population in (17), where treatment of the infected and vaccination of the susceptible are simultaneously applied as control strategies. For the constant scheme, the control action is present all the way through the specified time period in a permanent fashion, while for the impulsive or pulse scheme, the control action takes place at only specific periodic time instants (yearly). In both traditional control schemes, the proportion of individuals to be treated or vaccinated is a *fixed fraction* of the infected or susceptible population and, to eradicate a disease, such a fraction requires to surpass a critical value which is defined by the parameters of the system, by the control scheme utilized, and by the disease itself; otherwise, the system will remain in an endemic state where the disease persists above some positive level [2]. Simulations results evidence that using similar control rates, the pulse scheme has a slightly better performance than the constant control approach, and therefore it is possible to eradicate a disease with a lower control action p with the former (and it implies a significant less amount of treatments and vaccines). Last, it is important to mention that any of the conventional control schemes requires greater control rates to have a disease-free equilibrium point compared to the inverse optimal impulsive control treated in this work, and those need to be computed in advance prior their application.



Fig. 3. Portion of total population to be vaccinated and treated at resetting times $t = t_k$.



Fig. 4. Behavior of the infected population in the SIR epidemic model with seasonality. *Top*: Constant control scheme. *Bottom*: Pulse control scheme.

VII. CONCLUSIONS

We have considered the application of an impulsive control action, based on the IOC approach, to eradicate an infectious disease on a SIR epidemic model. The proposed control scheme proves to be an effective tool to help in the containment of infectious diseases, and to contribute to their possible eradication due to it minimizes the amount of vaccines and treatments given to population to reach the desired goal of a disease-free state. Unlike the conventional constant and pulse control schemes, the inverse optimal impulsive controller does not require to know in advance the parameters of the system whereon it will act to determine an adequate control action to mitigate the spread of a disease (which supposes an advantage over the traditional control schemes presented); however, the controller considered does require knowing the evolution of the current states of the system to perform such an operation. If any of the system's states is unknown, the performance of the impulsive controller can become dependent only on the remaining states. Even though this work does not consider any particular disease for the application of the control scheme above, results provide insight of efficient ways to tackle infectious diseases in realistic situations, that could be considered by epidemic researchers and social security entities. Note that in the inverse optimal control it is not require to provide a specific cost function to find an optimal solution in the system, but is the controller itself that uncovers the function cost in terms of an observed optimal response.

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