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Public health intervention as Z–type control in a SIp behavioral vaccination model

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Abstract We apply the Z–type control, a dynamic error based control method, to an SIR–like epidemic model for childhood diseases including voluntary vaccination and human behavioral change, shortly called SIp model. The control represents the communication effort by the public health system to regulate propensity to vaccinate in order to achieve a predetermined disease prevalence. The solutions of the controlled model are compared with that of the corresponding uncontrolled model. We found that the Z–control has a stabilizing effect and, compared to the case of constant effort, it may provide a faster stabilization at a lower disease prevalence.

Keywords infectious disease · vaccination · human behavior · communication · control

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1 Introduction

In the last few years, the *epidemic models*, i. e. the mathematical models describing the transmission of infectious diseases, are becoming increasingly sophisticated tools to support decision of public health planners. This is due to the natural development of the field of Mathematical Epidemiology (ME) but is also boosted by the recent global pandemic of COVID-19 [22].

As part of these advances, there is an increasing recognition for the need to add to epidemic models a layer of complexity, which is critical to understand the mechanisms underlying infection transmission and control: the human behavior [11, 15, 21, 23].

As matter of fact, classical models of ME are based on the assumption that the adoption of containment and/or control measures, like vaccination, quarantine or social distance, are static and can be represented by constant parameters [2, 6, 16].

However, this kind of description may not correctly represent modern scenarios of self–protective actions where the private choices are determined by conflicting instances. Such actions are dynamic, because they are strictly linked to human behavior which, in turn, is influenced by the information concerning the disease status in the community.

In the seventies, V. Capasso was the first to propose a behavioral–implicit SIR model, where the contact rate was not a constant, as it was assumed until then, but described by a decreasing function of the infection prevalence I. Such a function summarizes the behavioral response of individuals to prevalence (see e.g. [7]). As pointed out by d'Onofrio and Manfredi [8, 15], the Capasso's early work laid the foundations for a new development of ME, the *behavioral epidemiology of infectious diseases* (BEID) where classical mathematical modelling of infectious diseases are integrated with tools from a variety of behavioural sciences with the inclusion of the so called *behavioural immune system of humans* [19].

In the framework of BEID, d'Onofrio and coworkers proposed in 2011 a SIR–like epidemic model for childhood diseases in which the vaccine demand is based on the imitation mechanism [9]. They found that two parameters play a very relevant role in the model dynamics: the first one is the *imitiation speed* k and the second one, say θ, is the relative risk of infection compared to the risk of vaccine adverse effects (VAE). The authors found that according to the values of the couple (k, θ) sustained oscillations may occur (the *imitation-induced oscillations*).

Later on, the model in [9] has been extended in [10] by including the influence of the *public health system* (PHS). The key assumption is that the vaccinating behavior not only is influenced by private information circulating among parents of children to be vaccinated but also by information on infection and vaccines spread by PHS to favor vaccine propensity.

In [10] the communication effort by the PHS is modeled as constant and it is shown that such effort may have a stabilizing role. However, a more realistic description of the communication effort should take into account that it depends on a range of epidemiological and economical variables, including the disease burden and the economic and human costs of immunization. Therefore, instead of assuming it as a constant, it is more suitable to assume that it is a time–dependent function to be determined by a control problem. To this aim, Buonomo et al. [5] focused on short-term horizons and determined the optimal time-profiles of the communication effort by applying the optimal control (OC) theory. They used both deterministic and stochastic algorithm to get continuous control strategies and piecewise–constant strategies, respectively.

For childhood diseases, where disease costs are much larger than vaccination costs, Buonomo et al. [5] found that the OC solution sets at its maximum for most of the policy horizon. This means that the PHS cannot further improve perceptions about the net benefit of immunization. As a consequence, a fundamental role is played by the perceived risk of infection: when the prevalence declines, this risk declines as well and this, in turn, produces a future decline in vaccine uptake.

A different control approach has been recently applied to problems in epidemiology and population dynamics: a closed loop feedback control that some recent works denote as *Z–control*. This method is mainly aimed to force one or more state variables to reach a desired state, with a given convergence rate λ (the *design parameter*).

In the literature, several applications of Z–control to problems in epidemiology and population dynamics can be found. Zhang et al. [24] used the Z-control method to the classical Lotka-Volterra model with the aim of avoiding the extinction of species and promoting their coexistence. Lacitignola et al. [14] applied the Z–control as an input or harvesting of preys in a generalized predator-prey system. They showed that the value of the design parameter needs to be carefully calibrated in order to preserve the positiveness of the solutions. However, when the method works, it has a stabilizing effect on the dynamics in the sense that the Z–controlled dynamics approach a stable value when the corresponding uncontrolled one has an oscillatory regime. Alzahrani et al. [1] proposed an eco–epidemiological model where predator and prey interacts and the prey is affected by an infectious disease. They showed that the Z–control may eliminate the chaotic behavior of solutions. Samanta [18] applied the Z–control to a planar SI-like epidemic model and showed that the disease can be controlled when the basic reproduction number R_0 of the uncontrolled system is greater than one. Lacitignola and Diele [13] considered an epidemic model for which a backward transcritical bifurcations may occur, which indicates that the disease may spread also when R_0 is less than one. Very interestingly, they show that the Z–control (represented by isolation or replacement of susceptibles) is able, under certain circumstances, to erase the backward bifurcation. Senapati et al. [20] considered a SI–like epidemic model where the control is the removal of population for isolating the susceptible population from an infected region to reduce the disease prevalence. In their result they showed that such removal help to effectively achieve predetermined values of disease prevalence.

In this paper we apply the Z–control method to the SIR–like epidemic model for childhood diseases proposed by d'Onofrio and coworkers [9]. To our knowledge, this is the first attempt to apply the Z–type control method to a behavioral epidemic model. The control represents the communication effort by the PHS to favor vaccine propensity. Once that the Z–controlled model is obtained, the main aim are: *(i)* to compare the solutions with that of the corresponding uncontrolled model and, in particular, to investigate if the Z–control is able to stabilize oscillatory regimes; *(ii)* to assess the role of the design parameter; *(iii)* to compare the strategy resulting from Z–control with that of constant communication effort.

The rest of the paper is organized as follows: in section 2 we shortly recall the SIp model introduced in [9]. Section 3 is devoted to building the SIp model with Z–control. The control is expressed in terms of parameters, state variables, design parameter and the desired prevalence I_d. The analysis of the model is presented in Section 4, which is supported by numerical simulations in Section 5. In Section 6, concluding remarks close the paper.

2 The SIp model and public health systems intervention

In this section we shortly review the SIR transmission model with voluntary vaccination introduced by d'Onofrio et al. [9, 10] and its main properties.

Consider a non–fatal disease (or the disease induced deaths may be neglected) for which a non–mandatory vaccination at birth is available. Assume that the population affected by the disease is divided, at time t, in the following mutually exclusive compartments: susceptible individuals, $S(t)$; infectious individuals, $I(t)$; vaccinated individuals, $V(t)$ and removed individuals, $R(t)$. The state variables S, I, R and V represent fractions and the disease is non–fatal so that, for all $t \geq 0$,

$$S(t) + I(t) + V(t) + R(t) = 1. \tag{2.1}$$

Let $p(t)$ be the proportion of parents of newborns who are favorable to vaccination at time t. This quantity is taken as a proxy for vaccine uptake.

The dynamics of the vaccinated fraction is linearly depending on $p(t)$ and the related balance equation is: $\dot{V} = \mu p - \mu V$. The dynamics of the removed fraction can be deduced from (2.1).

The balance equation for p is a consequence of *learning by imitation* [12] and is worth to be described in details. It is obtained from the initial expression

$$\dot{p} = k_1 \,\Delta E \, p \, (1-p)\,, \tag{2.2}$$

where k_1 is the *imitation* coefficient and ΔE is the payoff gain representing the switching between the decisions to vaccinate or not to vaccinate. The quantity ΔE is expressed as the difference between the perceived payoff of non–vaccinators and the perceived cost of vaccination. The former is given by perceived risk associated to the disease, taken as a function of the infection prevalence (say, $\theta(I)$) and the latter is given by the perceived cost of vaccination, i.e. the perceived risk of suffering from vaccine adverse events (VAE), taken as a function of the vaccine uptake (say, $\alpha(I)$). Assuming linear dependence, i.e. $\theta(I) = \theta_1 I$, and $\alpha(p) = \alpha_1 p$, one gets

$$\Delta E = \theta_1 I - \alpha_1 p.$$

From (2.2) it follows

$$\dot{p} = k_1 \,(\theta_1 I - \alpha_1 p)\; p\; (1-p)\,.$$

By setting $k = k_1 \theta_1$, $\theta = \frac{\theta_1}{\alpha_1}$ and $\alpha = \frac{1}{\theta}$, one finally obtain

$$\dot{p} = k\theta \;(1-p)\,(I - \alpha p)\; p. \tag{2.3}$$

The parameter k regulates the *imitation speed* whereas θ represents a normalized measure of the relative risk of infection compared to the risk of VAE [5].

By taking into account of (2.3), the balance equations for S, I and p are given by

$$\begin{aligned} \dot{S} &= \mu\,(1-p) - \mu S - \beta S I \\ \dot{I} &= \beta S I - (\mu+\nu)\, I \\ \dot{p} &= k\theta\,\,(1-p)\,(I-\alpha p)\,\, p. \end{aligned} \tag{2.4}$$

In (2.4), which we will refer as *SIp model*, the parameters are positive constants: μ is the death rate, (which is assumed to be identical to the birth rate); ν is the recovery rate and β is the contact rate.

Model (2.4) admits an endemic equilibrium

$$E_e = (S_e, I_e, p_e) = \left(\frac{1}{R_0}, I_e, \theta I_e\right),$$

where R_0 is the basic reproduction number of system (2.4),

$$R_0 = \frac{\beta}{\mu+\nu},$$

and

$$I_e = \frac{\mu\left(1-\frac{1}{R_0}\right)}{\mu\,(1+\theta)+\nu}.$$

d'Onofrio et al. [9] showed that the equilibrium E_e is locally asymptotically stable out of a given interval of values of k, say $[k_1, k_2]$, where k_1 and k_2 depend on θ. For values of k in $[k_1, k_2]$, sustained oscillations occur (*imitation-induced oscillations*).

Model (2.4) was later extended by d'Onofrio et al. [10] to the case where the human behavior not only changes due to imitation alone but can also change as a consequence of information provided by the public health system. In such a case, they modified the third equation of the system (2.4) as follows:

$$\dot{p} = k\theta(1-p)\left[(I-\alpha p)p + \gamma(t)\right] \tag{2.5}$$

where $\alpha = 1/\theta$ is a measure of the (relative) perceived risk of VAE (that is, of the propensity to vaccine refusal) and $\gamma(t)$ accounts for the effort of the public health system aimed at increasing vaccine uptake.

D'Onofrio et al. in [10] studied the case of a constant effort $\gamma(t) = \gamma$ (const.). Therefore the model they considered is written as

$$\begin{aligned} \dot{S} &= \mu\,(1-p) - \mu S - \beta S I \\ \dot{I} &= \beta S I - (\mu+\nu)\, I \\ \dot{p} &= k\theta(1-p)\left[(I-\alpha p)p + \gamma\right]. \end{aligned} \tag{2.6}$$

They showed that if $\gamma \geq \alpha p_c^2$, where $p_c = 1 - 1/R_0$ is the *elimination threshold* [2], then $p(t)$ tends to an equilibrium value $p^\star$ greater than the elimination threshold p_c and the prevalence $I(t)$ tends to zero. This means that the disease is eliminated. On the contrary, if $\gamma < \alpha p_c^2$, then the system admits the unique endemic equilibrium

$$E^\star = (S^\star, I^\star, p^\star) = \left(\frac{1}{R_0}, \frac{\mu}{\mu+\nu}(p^\star - p_c), p^\star\right),$$

where $p^\star$ is a positive solution of the equation

$$(I^\star - \alpha p^\star)p^\star + \gamma = 0.$$

As mentioned in the introduction, a more realistic description of the communication effort by the PHS should take into account that it depends on a range of epidemiological and economical variables. Therefore, it is more realistic to assume that the effort is a time–dependent function to be determined by a control problem.

Here, the effort $\gamma(t)$, which will be denoted $U(t)$ from now on, will be assumed to be a control input which drives the system to achieve a targeted output state, in our case, a desired level of prevalence. This will be done in the next section.

3 The SIp model with Z–control

3.1 The model

In this section we apply a Z–type control method to the behavioral vaccination model introduced in [10] with the aim of assessing what new or different dynamical properties does it reveal. As far as our knowledge this work is the first to apply the Z–type control method to a behavioral epidemiological model.

For convenience, we rewrite the model (2.6) in the following form:

$$\begin{aligned} \dot{S} &= -\mu\, p + F(S, I) \\ \dot{I} &= G(S, I) \\ \dot{p} &= H(p, I) + k\theta\ (1-p)\, U(t) \end{aligned} \tag{3.1}$$

where

$$F(S, I) = \mu - \mu S - \beta S I, \tag{3.2}$$

$$G(S, I) = \beta S I - (\mu + \nu)\, I, \tag{3.3}$$

and

$$H(p, I) = k\theta\ (1-p)\, (I - \alpha p)\ p. \tag{3.4}$$

In (3.1) the function $U(t)$ is the control input. In principle, this function can be positive or negative [18]. Since here $U(t)$ represents the action of the public health system (PHS), a positive value will correspond to an action devoted to promote a change of behavior of parents from anti-vaccine to vaccine. A negative value of $U(t)$ would correspond to the opposite action.

Clearly, if the control is applied in the perspective of an increase of vaccination coverage aimed at disease eradication, a positive control $U(t)$ is more likely to act in this model.

We will determine the time–evolution of $U(t)$ by using the Z–control method. We look for a control input that drives the system to achieve a desired value of prevalence, say $I_d(t)$.

In this paper we will consider a constant function $I_d(t) = I_d$ (const.), although the design formula in the next section is obtained in the general case $I_d(t)$ for the sake of completeness.

As already mentioned, the Z–control approach is based on the following steps. Step 1: define the error function as the difference between the actual and desired output of the model, which is expected to converge to zero exponentially. This step can repeated until we find the control variable. Step 2: find an explicit expression of the control variable in terms of state variables and parameters in the model from the error function. We follow these steps in the following subsections.

3.2 Error formula

As well explained by Zhang et al. [24] and Lacitignola and Diele [13], the basic idea of Z–control is to consider the *first error* $\nu_1(t)$, which is defined as the difference between the actual system output, say $n(t)$, and a desired value $n_d(t)$. Then, it must be imposed that the first error goes to zero exponentially, i. e. that the error satisfies the so called *first design formula* $\dot{\nu}_1 + \lambda\nu_1 = 0$, where λ gives a measure of the convergence rate (the *design parameter*). From the first design formula it should be possible to write an explicit expression for the control $U(t)$ in terms of the state variables and other parameters of the model. If this will not be possible, then the procedure must be repeated by writing the *second error*, which is defined as $\nu_2 = \dot{\nu}_1 + \lambda\nu_1$, and by writing the *second design formula* $\dot{\nu}_2 + \lambda\nu_2 = 0$ and so on.

In our case, the system output of our interest is the prevalence, i. e. $n(t) = I(t)$. We will need three error functions and will get the expression of $U(t)$ given by (3.23).
The first error is:

$$\nu_1(t) = I(t) - I_d(t), \tag{3.5}$$

where $I_d(t)$ is a target value of prevalence. The second error is:

$$\nu_2(t) = \dot{\nu}_1(t) + \lambda\nu_1(t). \tag{3.6}$$

The third error is:

$$\nu_3(t) = \dot{\nu}_2(t) + \lambda\nu_2(t), \tag{3.7}$$

and must satisfy the third design formula

$$\dot{\nu}_3(t) = -\lambda\nu_3(t), \tag{3.8}$$

where $\lambda > 0$ is the design parameter.

We now manipulate these equations to obtain an explicit expression of the first error $\nu_1(t)$. We begin by noticing that the time derivative of (3.7) and (3.6) give

$$\dot{\nu}_3 = \ddot{\nu}_2 + \lambda\dot{\nu}_2 = \dddot{\nu}_1 + 2\lambda\ddot{\nu}_1 + \lambda^2\dot{\nu}_1.$$

By using (3.8) we get

$$\dddot{\nu}_1 + 2\lambda\ddot{\nu}_1 + \lambda^2\dot{\nu}_1 = -\lambda\nu_3,$$

that is, in view of (3.7), it follows

$$\dddot{\nu}_1 + 2\lambda\ddot{\nu}_1 + \lambda^2\dot{\nu}_1 = -\lambda\left(\dot{\nu}_2 + \lambda\nu_2\right).$$

Now, by using (3.6) and its time derivative, we get the equality

$$\dddot{\nu}_1 + 2\lambda\ddot{\nu}_1 + \lambda^2\dot{\nu}_1 = -\lambda\left(\ddot{\nu}_1 + \lambda\dot{\nu}_1 + \lambda\dot{\nu}_1 + \lambda^2\nu_1\right),$$

that is,

$$\dddot{\nu}_1 + 3\lambda\ddot{\nu}_1 + 3\lambda^2\dot{\nu}_1 + \lambda^3\nu_1 = 0. \tag{3.9}$$

Now, we append to this last equation the following initial values:

$$\begin{aligned}
&\nu_1(0) = I(0) - I_d(0)\\
&\dot{\nu}_1(0) = \dot{I}(0) - \dot{I}_d(0) = (\beta S(0) - (\mu+\nu))\, I(0) - \dot{I}_d(0)\\
&\ddot{\nu}_1(0) = \ddot{I}(0) - \ddot{I}_d(0) = \beta I(0)\,(\mu(1-p(0)) - \mu S(0) - \beta S(0)I(0))\\
&\qquad\qquad\qquad\qquad\quad + (\beta S(0) - \mu - \nu)^2\, I(0) - \ddot{I}_d(0)
\end{aligned}$$

where we have used the second equation in (3.1). The characteristic equation for the third order equation in the variable m is given by $m^3+3\lambda m^2+3\lambda^2 m+\lambda^3 = 0$, that is $(m+\lambda)^3 = 0$.

Thus we get

$$\nu_1(t) = (c_1 + c_2 t + c_3 t^2)e^{-\lambda t}, \tag{3.10}$$

where c_1, c_2 and c_3 are constants. By using the initial conditions we obtain

$$\nu_1(0) = c_1 = I(0) - I_d(0). \tag{3.11}$$

From (3.10) we get:

$$\begin{aligned}
&\dot{\nu}_1(t) = (c_2 + 2c_3 t)e^{-\lambda t} - \lambda(c_1 + c_2 t + c_3 t^2)e^{-\lambda t}\\
&\Rightarrow \dot{\nu}_1(0) = c_2 - c_1\lambda = (\beta S(0) - (\mu+\nu))\, I(0) - \dot{I}_d(0),
\end{aligned}$$

and therefore

$$c_2 = c_1\lambda + (\beta S(0) - (\mu+\nu))\, I(0) - \dot{I}_d(0),$$

that is

$$c_2 = (I(0) - I_d(0))\lambda + (\beta S(0) - (\mu+\nu))\, I(0) - \dot{I}_d(0). \tag{3.12}$$

Finally, from

$$\ddot{\nu}_1(t) = 2c_3e^{-\lambda t} - \lambda(c_2 + 2c_3t)e^{-\lambda t} - \lambda[(c_2 + 2c_3t)e^{-\lambda t} - \lambda(c_1 + c_2t + c_3t^2)e^{-\lambda t}],$$

one has

$$\begin{aligned}\ddot{\nu}_1(0) &= 2c_3 - 2\lambda c_2 + \lambda^2 c_1 \\ &= \beta I(0)\left(\mu(1-p(0)) - \mu S(0) - \beta S(0)I(0)\right) + \left(\beta S(0) - \mu - \nu\right)^2 I(0) - \ddot{I}_d(0),\end{aligned}$$

that is

$$c_3 = \frac{1}{2}\left[2c_2\lambda - \lambda^2 c_1 + \beta I(0)\left(\mu(1-p(0)) - \mu S(0) - \beta S(0)I(0)\right) + \left(\beta S(0) - \mu - \nu\right)^2 I(0) - \ddot{I}_d(0)\right]. \tag{3.13}$$

By plugging (3.11), (3.12) and (3.13) into (3.10) we get the explicit expression for the error function $\nu_1(t)$ as

$$\begin{aligned}\nu_1(t) = \Big\{ & I(0) - I_d(0) + \Big[(I(0) - I_d(0))\lambda + \left(\beta S(0) - (\mu + \nu)\right) I(0)) - \dot{I}_d(0)\Big]\, t + \\ & \frac{1}{2}\Big[2c_2\lambda - \lambda^2 c_1 + \beta I(0)\left(\mu(1-p(0)) - \mu S(0) - \beta S(0)I(0)\right) \\ & + \left(\beta S(0) - \mu - \nu\right)^2 I(0) - \ddot{I}_d(0)\Big]\, t^2 \Big\}\, e^{-\lambda t}\end{aligned} \tag{3.14}$$

3.3 Closed loop feedback control

As mentioned in the previous sections, an important step in the application of the Z–control is to express the control $U(t)$ in a closed–loop feedback form, that is to find an explicit expression for the control $U(t)$ in terms of the state variables and other parameters of the model. This is the aim of this section.

From now on, we will assume that the target prevalence is a constant value $I_d(t) = I_d$ (const.).

Then equation (3.9) can be written:

$$\dddot{I} + 3\lambda\ddot{I} + 3\lambda^2\dot{I} + \lambda^3\left(I - I_d\right) = 0. \tag{3.15}$$

Now, by remembering the model (3.1) and the expressions (3.2), (3.3) and (3.4), we note that

$$\ddot{I} = G_S\dot{S} + G_I\dot{I}, \tag{3.16}$$

and

$$\dddot{I} = G_{SS}\dot{S}^2 + G_{SI}\dot{S}\dot{I} + G_S\ddot{S} + G_{IS}\dot{S}\dot{I} + G_{II}\dot{I}^2 + G_I\ddot{I}, \tag{3.17}$$

where symbols like G_S denote the partial derivative of G with respect to S.

By taking into account that: $G_{SS} = 0$, $G_{II} = 0$, $G_{SI} = G_{IS}$, it follows:

$$\dddot{I} = G_S\ddot{S} + 2G_{SI}\dot{S}\dot{I} + G_I\ddot{I}. \tag{3.18}$$

By plugging (3.16) and (3.18) into (3.15), we get:

$$G_S\ddot{S} + 2G_{SI}\dot{S}\dot{I} + G_I\ddot{I} + 3\lambda G_S\dot{S} + 3\lambda G_I\dot{I} + 3\lambda^2\dot{I} + \lambda^3(I - I_d) = 0. \quad (3.19)$$

From (3.1) we have:

$$\ddot{S} = -\mu\dot{p} + F_S\dot{S} + F_I\dot{I} = -\mu H - \mu k\theta(1-p)U(t) + F_S\dot{S} + F_I\dot{I} \quad (3.20)$$

Therefore (3.19) and (3.20) give:

$$\begin{aligned} &-\mu HG_S - \mu k\theta(1-p)G_SU(t) + F_SG_S\dot{S} + F_IG_S\dot{I} + \\ &+2G_{SI}\dot{S}\dot{I} + G_I\ddot{I} + 3\lambda G_S\dot{S} + 3\lambda G_I\dot{I} + 3\lambda^2\dot{I} + \lambda^3(I - I_d) = 0. \end{aligned} \quad (3.21)$$

From this equality we obtain the expression of the control $U(t)$ in terms of the state variables and their derivatives:

$$U(t) = \frac{-\mu HG_S + (F_SG_S + 3\lambda G_S)\dot{S} + \left(F_IG_S + 3\lambda G_I + 3\lambda^2\right)\dot{I} + 2G_{SI}\dot{S}\dot{I} + G_I\ddot{I} + \lambda^3(I - I_d)}{\mu k\theta(1-p)G_S} \quad (3.22)$$

It is also possible to get this expression solely in terms of the state variables by substituting $\dot{S}$ and $\dot{I}$ from (3.1) into (3.22):

$$\begin{aligned} U(t) = &\frac{-\mu HG_S + (F_SG_S + 3\lambda G_S + 2\beta G + G_SG_I)(-\mu p + F)}{\mu k\theta(1-p)G_S} \\ &+ \frac{\left(F_IG_S + 3\lambda G_I + G_I^2 + 3\lambda^2\right)G + \lambda^3(I - I_d)}{\mu k\theta(1-p)G_S} \end{aligned} \quad (3.23)$$

3.4 SIp model with feedback control

By inserting (3.22) in the p-equation of model (3.1) we get

$$\dot{p} = H(p, I) + k\theta\ (1-p)$$

$$\frac{-\mu HG_S + (F_SG_S + 3\lambda G_S)\dot{S} + \left(F_IG_S + 3\lambda G_I + 3\lambda^2\right)\dot{I} + 2G_{SI}\dot{S}\dot{I} + G_I\ddot{I} + \lambda^3(I - I_d)}{\mu k\theta(1-p)G_S},$$

that is

$$\begin{aligned} \dot{p} = &\frac{\mu HG_S - \mu HG_S + (F_SG_S + 3\lambda G_S)\dot{S} + \left(F_IG_S + 3\lambda G_I + 3\lambda^2\right)\dot{I}}{\mu G_S} \\ &+ \frac{2G_{SI}\dot{S}\dot{I} + G_I\ddot{I} + \lambda^3(I - I_d)}{\mu G_S}, \end{aligned} \quad (3.24)$$

and therefore:

$$\dot{p}=\frac{\left(F_S G_S+3\lambda G_S\right)\dot{S}+\left(F_I G_S+3\lambda G_I+3\lambda^2\right)\dot{I}+2G_{SI}\dot{S}\dot{I}+G_I\ddot{I}+\lambda^3(I-I_d)}{\mu G_S}. \tag{3.25}$$

We are now in a position to write the SIp model with feedback control, or *Z–controlled SIp model*:

$$\begin{aligned}
\dot{S} &= \mu\,(1-p)-\mu S-\beta SI\\
\dot{I} &= \beta SI-(\mu+\nu)\,I\\
\dot{p} &= \frac{(F_S G_S+3\lambda G_S)\dot{S}+\left(F_I G_S+3\lambda G_I+3\lambda^2\right)\dot{I}+2G_{SI}\dot{S}\dot{I}+G_I\ddot{I}+\lambda^3(I-I_d)}{\mu G_S}:=\Pi(S,I,p),
\end{aligned} \tag{3.26}$$

where

$$\begin{gathered}
F=\mu-\mu S-\beta SI;\;\; G=\beta SI-(\mu+\nu)I;\;\; F_S=-\mu-\beta I;\;\; F_I=-\beta S;\;\; G_S=\beta I\\
G_I=\beta S-(\mu+\nu)\,;\;\; G_{SI}=\beta.
\end{gathered} \tag{3.27}$$

By using equation (3.23), system (3.26) may be also written as:

$$\begin{aligned}
\dot{S} &= \mu\,(1-p)-\mu S-\beta SI\\
\dot{I} &= \beta SI-(\mu+\nu)\,I\\
\dot{p} &= \frac{(F_S G_S+3\lambda G_S+2\beta G+G_S G_I)(-\mu p+F)+\left(F_I G_S+3\lambda G_I+G_I^2+3\lambda^2\right)G+\lambda^3(I-I_d)}{\mu G_S}.
\end{aligned} \tag{3.28}$$

4 Analysis of the Z–controlled SIp model

Model (3.1) admits the unique equilibrium

$$\tilde{E}=\left(\tilde{S},\tilde{I},\tilde{p}\right)=\left(\frac{1}{R_0},\ I_d,\ \frac{1}{R_0}\left(R_0-1-\frac{\beta I_d}{\mu}\right)\right), \tag{4.1}$$

where

$$R_0=\frac{\beta}{\mu+\nu}, \tag{4.2}$$

Since $p\in[0,1]$, this equilibrium exists only if

$$I_d\leq\frac{\mu}{\beta}\left(\frac{\beta}{\mu+\nu}-1\right):=\hat{I}_d. \tag{4.3}$$

Note that this upper bound for I_d is less than the behavior–induced prevalence equilibrium, I_e, in absence of control. In fact, we have

$$\hat{I}_d<\frac{\mu(1-\frac{1}{R_0})}{\mu(1+\theta)+\nu}:=I_e. \tag{4.4}$$

In order to study the local stability analysis of $\tilde{E}$, first note that the Jacobian matrix of system (3.26) is given by

$$J\left(S,I,p\right)=\begin{pmatrix}-\mu-\beta I & -\beta S & -\mu\\ \beta I & \beta S-(\mu+\nu) & 0\\ \Pi_S & \Pi_I & \Pi_p\end{pmatrix}.$$

When evaluated at the equilibrium we have

$$J\left(\tilde{S},\tilde{I},\tilde{p}\right)=\begin{pmatrix}-\mu-\beta\tilde{I} & -\beta\tilde{S} & -\mu\\ \beta\tilde{I} & 0 & 0\\ \tilde{\Pi}_S & \tilde{\Pi}_I & \tilde{\Pi}_p\end{pmatrix},$$

where

$$\tilde{\Pi}_S=\left.\frac{\partial\Pi}{\partial S}\right|_{\tilde{E}},\quad \tilde{\Pi}_I=\left.\frac{\partial\Pi}{\partial I}\right|_{\tilde{E}},\quad \tilde{\Pi}_p=\left.\frac{\partial\Pi}{\partial p}\right|_{\tilde{E}}.$$

The characteristic equation comes from the matrix (we denote the eigenvalues as $\hat{\lambda}$, since λ is used to denote the design parameter):

$$J\left(\tilde{S},\tilde{I},\tilde{p}\right)=\begin{pmatrix}-\mu-\beta\tilde{I}-\hat{\lambda} & -\beta\tilde{S} & -\mu\\ \beta\tilde{I} & -\hat{\lambda} & 0\\ \tilde{\Pi}_S & \tilde{\Pi}_I & \tilde{\Pi}_p-\hat{\lambda}\end{pmatrix},$$

and is given by:

$$-\hat{\lambda}\left(-\mu-\beta\tilde{I}-\hat{\lambda}\right)\left(\tilde{\Pi}_p-\hat{\lambda}\right)-\mu\beta\tilde{I}\tilde{\Pi}_I-\mu\hat{\lambda}\tilde{\Pi}_S+\beta^2\tilde{S}\tilde{I}\left(\tilde{\Pi}_p-\hat{\lambda}\right)=0,$$

that is

$$\hat{\lambda}^3+\left(\mu+\beta\tilde{I}-\tilde{\Pi}_P\right)\hat{\lambda}^2+\left(-\left(\mu+\beta\tilde{I}\right)\tilde{\Pi}_P+\mu\tilde{\Pi}_S+\beta^2\tilde{S}\tilde{I}\right)\hat{\lambda}+\mu\beta\tilde{I}\tilde{\Pi}_I-\beta^2\tilde{S}\tilde{I}\tilde{\Pi}_P=0. \tag{4.5}$$

which we can write:

$$\hat{\lambda}^3+a_1\,\hat{\lambda}^2+a_2\,\hat{\lambda}+a_3=0.$$

We now look for the explicit expression of the coefficients $a_1\,a_2$ and a_3. For the sake of simplicity denote the quantity Π in (3.26) as

$$\Pi=\frac{\Lambda}{\mu G_S},$$

where

$$\Lambda=\Lambda_1+\Lambda_2+\Lambda_3+\Lambda_4,$$

and $\Lambda_1=(F_SG_S+3\lambda G_S)\,\dot{S}$, $\Lambda_2=\left(F_IG_S+3\lambda G_I+3\lambda^2\right)\dot{I}$, $\Lambda_3=2G_{SI}\dot{S}\dot{I}$, $\Lambda_4=G_I\ddot{I}+\lambda^3\left(I-I_d\right)$.
It follows

$$\Pi_S=\frac{\mu\Lambda_SG_S-\mu\Lambda G_{SS}}{(\mu G_S)^2}.$$

However $G_{SS} = 0$, therefore

$$\varPi_S = \frac{\varLambda_S}{\mu G_S}. \tag{4.6}$$

Now, the derivative of $\varLambda_1$ with respect to S gives:

$$(\varLambda_1)_S = (F_S G_S + 3\lambda\, G_S)\, F_S$$

and, in view of (3.27),

$$(\varLambda_1)_S = -(3\lambda - \mu - \beta I)(\mu + \beta I)\beta I.$$

This quantity evaluated at $\tilde{E}$ gives

$$(\varLambda_1)_S|_{\tilde{E}} = -(3\lambda - \mu - \beta I_d)(\mu + \beta I_d)\beta I_d. \tag{4.7}$$

In the same way, we get

$$(\varLambda_2)_S|_{\tilde{E}} = \beta I_d \left(3\lambda^2 - \frac{\beta^2 I_d}{R_0}\right), \quad (\varLambda_3)_S|_{\tilde{E}} = 0, \quad (\varLambda_4)_S|_{\tilde{E}} = 0. \tag{4.8}$$

Therefore, in view of (4.7) and (4.8), we get

$$\tilde{\varPi}_S = \frac{(\varLambda_1)_S|_{\tilde{E}} + (\varLambda_2)_S|_{\tilde{E}}}{\mu\beta I_d} = \frac{-(3\lambda - \mu - \beta I_d)(\mu + \beta I_d)\beta I_d + \beta I_d \left(3\lambda^2 - \frac{\beta^2 I_d}{R_0}\right)}{\mu\beta I_d} \tag{4.9}$$

Analogously, we have

$$\varPi_I = \frac{\mu \varLambda_I G_S - \mu \varLambda G_{SI}}{(\mu G_S)^2}. \tag{4.10}$$

By taking into account that

$$(\varLambda_1)_I|_{\tilde{E}} = -(3\lambda - \mu - \beta I_d)\frac{\beta^2 I_d}{R_0},\ (\varLambda_2)_I|_{\tilde{E}} = 0,\ (\varLambda_3)_I|_{\tilde{E}} = 0,\ (\varLambda_4)_I|_{\tilde{E}} = \lambda^3, \tag{4.11}$$

and from (3.27), and the fact that $(\varLambda)|_{\tilde{E}} = 0$ we have

$$\tilde{\varPi}_I = \frac{(\varLambda_1)_I|_{\tilde{E}}\, \mu\beta I_d + (\varLambda_4)_I|_{\tilde{E}}\, \mu\beta I_d - (\varLambda)|_{\tilde{E}}\, \mu\beta}{(\mu\beta I_d)^2} = \frac{-(3\lambda - \mu - \beta I_d)\frac{\beta^2 I_d}{R_0} + \lambda^3}{\mu\beta I_d}. \tag{4.12}$$

Finally, we have

$$\varPi_p = \frac{\mu \varLambda_p G_S - \mu \varLambda G_{Sp}}{(\mu G_S)^2}. \tag{4.13}$$

By taking into account that

$$(\varLambda_1)_p|_{\tilde{E}} = -\mu\beta I_d(3\lambda - \mu - \beta I_d),\ (\varLambda_2)_p|_{\tilde{E}} = 0,\ (\varLambda_3)_p|_{\tilde{E}} = 0,\ (\varLambda_4)_p|_{\tilde{E}} = 0, \tag{4.14}$$

and from (3.27) and the fact that $(\Lambda)|_{\tilde{E}} = 0$ we obtain

$$\tilde{\Pi}_p = \frac{(\Lambda_1)_p|_{\tilde{E}}\,\mu\beta I_d - (\Lambda)|_{\tilde{E}}\,\mu\beta}{(\mu\beta I_d)^2} = \frac{-\mu\beta I_d(3\lambda-\mu-\beta I_d)}{\mu\beta I_d} = -3\lambda-\mu-\beta I_d. \tag{4.15}$$

We are now in position to get the coefficients a_i, $i = 1, 2, 3$. From (4.5) and (4.15) we get

$$a_1 = \mu+\beta I_d - \tilde{\Pi}_p = \frac{(\mu+\beta I_d)\mu\beta I_d + \mu\beta I_d(3\lambda-\mu-\beta I_d)}{\mu\beta I_d} = 3\lambda.$$

From (4.5), (4.9) and (4.15) we get

$$\begin{aligned} a_2 &= -(\mu+\beta I_d)\tilde{\Pi}_p + \mu\tilde{\Pi}_S + \tfrac{\beta^2 I_d}{R_0} \\ &= (\mu+\beta I_d)(3\lambda-\mu-\beta I_d) - (\mu+\beta I_d)(3\lambda-\mu-\beta I_d) + 3\lambda^2 - \tfrac{\beta^2 I_d}{R_0} + \tfrac{\beta^2 I_d}{R_0} \\ &= 3\lambda^2. \end{aligned}$$

Finally, by using (4.5), (4.12) and (4.15) we have

$$a_3 = \mu\beta I_d\tilde{\Pi}_I - \frac{\beta^2 I_d}{R_0}\tilde{\Pi}_p = \lambda^3.$$

By applying the well known Routh–Hurwitz criterion, we get the matrix

$$RH = \begin{pmatrix} a_1 & a_3 & 0 \\ 1 & a_2 & 0 \\ 0 & a_1 & a_3 \end{pmatrix}$$

and the characteristic equation has solutions with negative real parts if and only if:

$$a_1 > 0, \quad \Delta_2 = a_1a_2 - a_3 > 0, \quad a_3\Delta_2 > 0,$$

In our case we have

$$a_1 = 3\lambda > 0, \quad \Delta_2 = a_1a_2 - a_3 = 9\lambda^3 - \lambda^3 = 8\lambda^3 > 0, \quad a_3\Delta_2 = 8\lambda^6 > 0$$

All the conditions of Routh-Hurwitz stability criterion are satisfied, therefore the equilibrium (4.1) is locally asymptotically stable.

5 Numerical simulations

In this section we perform some numerical simulations to show the dynamics of the SIp model when Z–control is applied. As a specific case, we consider a set of parameter values that produces sustained oscillations in the uncontrolled case. Then, we investigate the potential stabilizing effects due to the Z–control. We also compare the output with the case of constant control as considered in [10].

Parameter	Description	Baseline value	Ref.
μ	Birth and death rate	1/78 year^{-1}	[10]
ν	Recovery rate	1/7 day^{-1}	[10]
R_0	Basic reproduction number	15	[10]
$k\theta$	The product of imitation speed and relative risk of infection compared to the risk of VAE	40	[10]
λ	Design parameter	varying in $(0, +\infty)$	[24]
β	Transmission rate	$R_0(\mu + \nu)$	
$S(0)$	Initial fraction of susceptible population	$1.01/R_0$	
$I(0)$	Initial fraction of infectious population	$0.82{\cdot}10^{-5}$	[10]
$p(0)$	Initial vaccination coverage	0.95	[10]

Table 1 Parameters and their baseline values used for the numerical simulation. The value of $S(0)$ is slightly different than the one used in [10], where it is taken $S(0)$=$1.04/R_0$. The change is necessary in order to have a successful Z–control, as explained in Section 5.

The parameter values are listed in Table 1. The epidemiological parameter values are taken from [10], where the case of measles in Italy is considered. We also impose that the Z–control must drive the prevalence at a very low value which we take $I_d = 4 \cdot 10^{-7}$. This is just a reference value. To make an example, in the case of a population of $60 \cdot 10^6$ inhabitants (like e.g. Italy, approximately), the chosen value correspond to a prevalence level of only 24 infectious individuals.

In Figure 1 it is shown that the Z-controlled system (3.26) exhibits stable dynamics locally, as expected from the analysis. The prevalence $I(t)$ converges to the equilibrium I_d (the desired prevalence level) following the convergence of the error function to zero (Figure 1, panels (b) and (d)). It can be also seen that the vaccination coverage initially increases, as result of the epidemic peak, after that declines until the replacement of susceptibles determine a new increase until the equilibrium is reached (Figure 1, panels (a) and (c)).

In Figure 2 we compare the dynamics of the uncontrolled model (2.6), the model with constant control and the Z–controlled model for a given value of the design parameter. To this aim, the plots in Figure 2 are obtained by using a slightly modified version of model (3.1) where an *immigration term*, Imm, is included. This term represents a small constant transfer per unit of time from the susceptible to the infectious state [4,9,10]. As it is well explained in [9], such a small immigration of infectious is due to two assumptions: the first assumption is a steady flow of infectious as a consequence of international traveling; the second one is that this term mimics the possibility of infection resurgence. Furthermore, this small transfer is used to avoid the question of appropriateness of the deterministic model when infection prevalence may approach values extremely close to zero. As done in [9, 10], the value of Imm is set at one infectious individual per week in a population of $5 \cdot 10^6$ individuals.

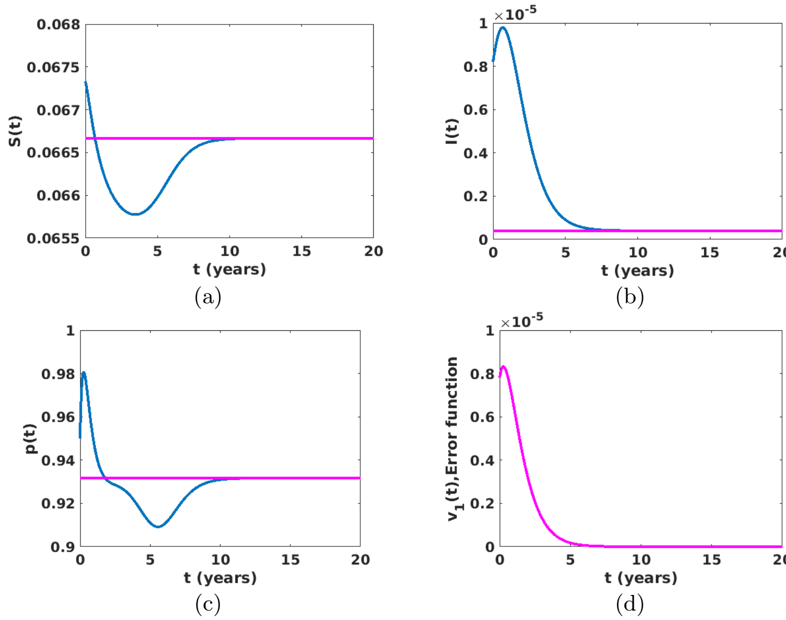


Fig. 1 Dynamics of the Z-controlled SIp model (3.26): (a) Susceptible fraction (b); Infectious fraction; (c) vaccination coverage; (d) error function. The horizontal line in panels (a), (b) and (c) represents the equilibrium value. The parameter values are as in Table 1.

Therefore, we consider the model

$$\begin{aligned}\dot{S} &= -\mu\, p + F(S,I) - Imm\\ \dot{I} &= G(S,I) + Imm\\ \dot{p} &= H(p,I) + k\theta\ (1-p)\, U(t).\end{aligned} \tag{5.1}$$

When there is no public information supplied to the model (that is, when $\gamma = 0$ in (2.6)), self–sustained oscillations can be observed with very short inter–epidemic period, see Figure 2, panels (a), (b), and (c). When a constant public intervention effort is applied, precisely $U(t) = \gamma(const.)$ and $\gamma = 1.3296 \cdot 10^{-4}$, the system shows damped oscillation with smaller epidemic peaks and a wider inter–epidemic period compared to the uncontrolled case. Therefore, a constant public effort is able to reduce, in this case, the amplitude of the oscillation and the state variables converges to an endemic equilibrium, see Figure 2, panels (d), (e), and (f). In the Z–controlled model with design parameter $\lambda = 0.0036$, the imitation–induced oscillations are removed and the dynamics is stabilized, see Figure 2, panels (g), (h), and (i). See Figure 4, panel (b) for the corresponding effort.
Note that the value of the prevalence at the equilibrium is $I_c = 156.52 \cdot 10^{-7}$ in case of constant control, which is greater than the prevalence target $I_d = 4 \cdot 10^{-7}$. The differences in the control efforts are shown in Figure

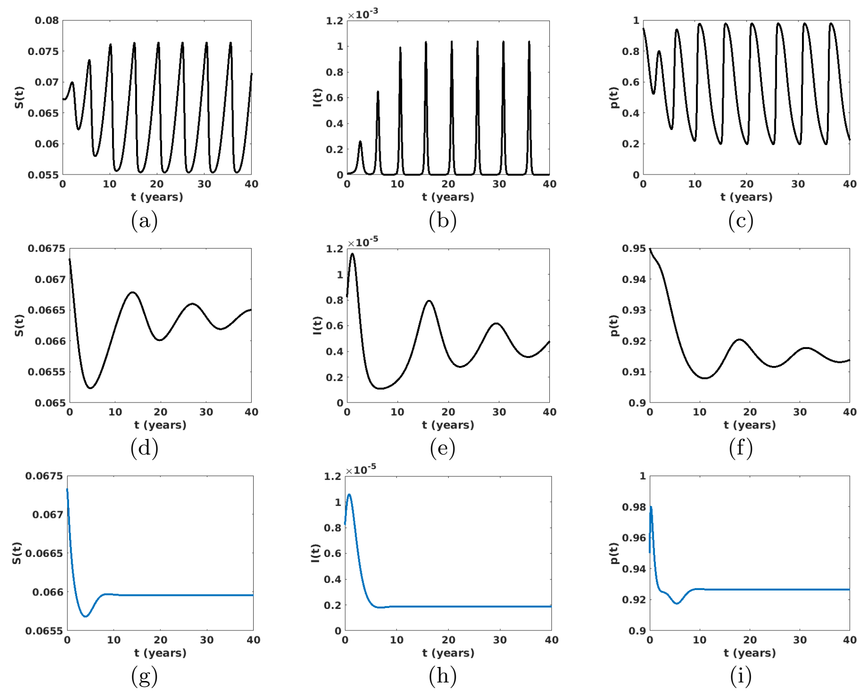


Fig. 2 First row (panels (a), (b) and (c)): dynamics of the uncontrolled model (2.4). Second row (panels (d), (e) and (f)): dynamics of the model (2.6) with $\gamma = 1.3296 \cdot 10^{-4}$. Third row (panels (g), (h) and (i)): dynamics of Z–control model with $I_d = 4 \cdot 10^{-7}$ and $\lambda = 0.0036$. Initial data and parameter values are as in Table 1.

4 panel (a): at the beginning, a large overhang is expected for the Z–control effort, which after about 2 years settles at values close to that of the constant effort case ($\gamma = 1.3296 \cdot 10^{-4}$).

In [10] the authors have shown that a constant public intervention may produce the elimination of the disease under certain circumstances. In particular, the elimination may be obtained for $\gamma = 1.5178 \cdot 10^{-4}$. In this case the vaccination coverage settles to $\overline{p} = 0.963$ (see Figure 3, panels (a), (b), and (c)). A similar behavior may be obtained by employing the Z–control with design parameter $\lambda = 0.0020$ (see Figure 3, panels (d), (e), and (f)). Of course, this feedback control does not produce elimination but allows only to reach the prevalence value I_d. However, when compared to the elimination case the vaccination coverage stabilizes to a much lower value (0.924), which could be a more convenient goal for public health planners, and the effort is also much lower (see Figure 4 panel (a)).

It must be pointed out that in the studies of epidemiological modeling incorporating the Z–control approach [13, 18] a positive control input $U(t)$ implies a removal of a portion of population from a given state. Unlike such

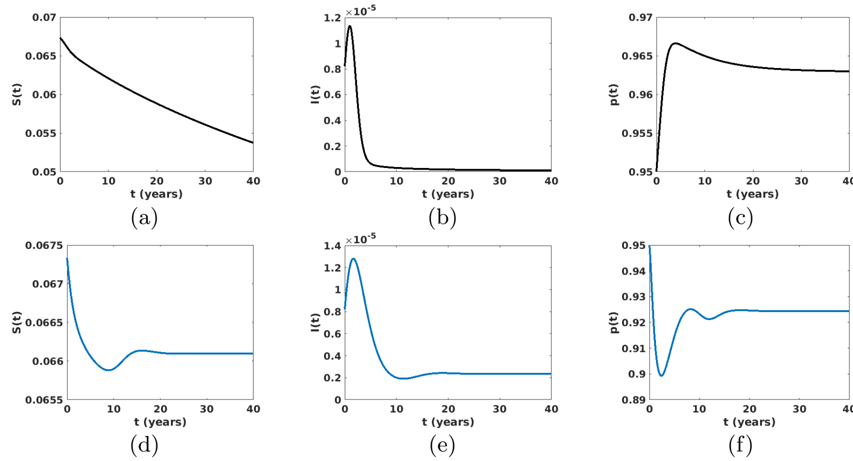


Fig. 3 First row (panels (a), (b) and (c)): dynamics of the model (2.6) with $\gamma = 1.5178e{-4}$ (elimination case). Second row (panels (d), (e) and (f)): dynamics of Z–control model with $I_d = 4 \cdot 10^{-7}$ and $\lambda = 0.0020$. Initial data and parameter values are as in Table 1.

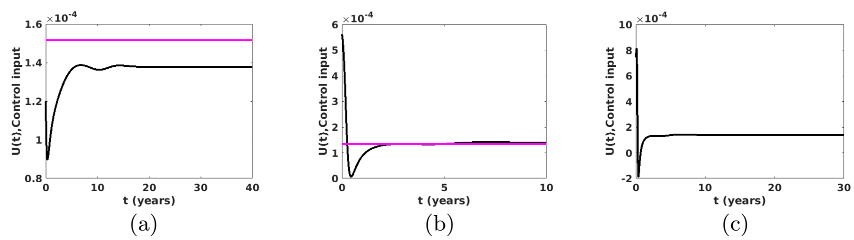
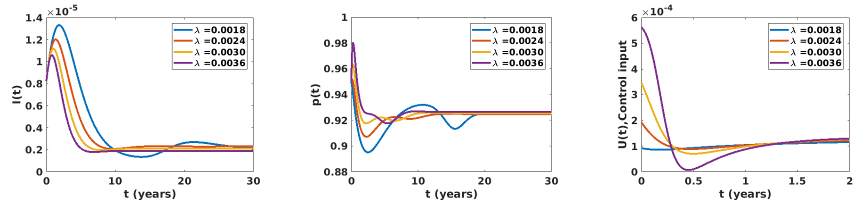


Fig. 4 The control variable, $U(t)$. Panel (a): Case $\lambda = 0.002$. The horizontal line indicates the value of the constant public effort ($\gamma = 1.5178 \cdot 10^{-4}$). Panel (b): Case $\lambda = 0.0036$. The horizontal line indicates the value of the constant public effort ($\gamma = 1.3296 \cdot 10^{-4}$). Panel (c): Case $\lambda = 0.0040$. The initial conditions and the parameter values are as in Table 1.

studies, in our case a positive $U(t)$ indicates the effort of the public health system to change the behavior of non-vaccinated population towards the decision to vaccinate and the negative $U(t)$, the effort of the public health system to change the behavior of the society in opposite. So in the process towards Z–control achievement $U(t)$ may assume negative values. This behavior is observed in our case for large design parameter, $\lambda = 0.0040$. See Figure 4, panel (c).

For the Z–control method to be biologically meaningful it is necessary that all the state variable remain positive and less than one. In our case we see that this is obtained only for a small range of values of the design parameter λ, which is estimated as $0.0018 < \lambda < 0.0045$ for the initial conditions in Table 1. On the other hand, the Z–control model needs specific set of initial



(a) (b) (c)

Fig. 5 The role of design parameter λ on the dynamics of the infected and vaccinated proportion, and on the control input.

conditions for a given value of λ to be successful [20]. For example, as far as we investigated, when the initial value is $S(0) = 1.04/R_0$, as taken in [10], the positiveness of the state variable is assured but the constrain $p \leq 1$ is not satisfied.

In the last plots it can be seen that low values of the design parameter λ (see for example the blue line in Figure 5, corresponding to $\lambda = 0.0018$) produces low PHS effort $U(t)$ (panel (c)) and an initial oscillatory transient of the vaccination coverage (panel (b)), which in turn makes the prevalence to raise with high initial peak (panel (a)). By increasing the value of λ (till to the maximum allowed, $\lambda = 0.0036$), higher efforts by public health are required initially (panel (c)), the vaccination coverage increases (panel (b)) and the first peak of prevalence decreases (panel (a)). It can be also observed that the greater the value of λ, the faster the system stabilizes.

6 Conclusions

The role of human behavior in the transmission of infectious disease cannot be neglected in many cases when control strategies by public health planners must be designed. A particular special case is that of childhood infectious diseases such as measles, mumps and pertussis where vaccine is available and administered on voluntary basis. In this case, it has been shown in [9] that sustained oscillations of the disease prevalence may occur as result of change in vaccination behavior. However, the PHS may still obtain disease elimination or steady low level of prevalence by implementing campaigns to promote vaccination behavior with constant communication effort [10].

In this paper we have assumed that the PHS action may be described by a closed loop feedback control designed according to the rules of Z–control. We found that the Z–control has a stabilizing effect and, compared to the case of constant effort, it may provide a faster stabilization at a lower disease prevalence (see Figure 2).

A special feature of the Z–control method is that the controlled model, unlike the uncontrolled system, admits a single equilibrium, which is the only attractor as we showed analytically.

Since the model refers to the spread of a disease in a human population, its biological feasibility is important. In this aspect for the given initial conditions we have estimated an intermediate value of the design parameter λ and we have observed that within this estimated range the larger the value of λ the faster the system converges to the desired state.

Another approach has been used in [17] with a method known as sliding mode control, which differs from that used here in that the method is characterized by discontinuous control structure and it may lead to high frequency oscillations called chattering. However this could be useful to explore in the future.

There are two main relevant limitations to the Z–control approach. From one hand, it can be successfully applied only for limited values of the design parameter, which could be difficult to estimate in real scenarios. On the other hand, this study does not take into account the economic implications of the PHS efforts. Optimal control theory can help in providing a deep insight to such problems [3]. A comparative study with the optimal strategies coming from optimal control problems, like in [5], will help to clarify this aspect. This will be part of our future investigation on the subject.

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