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A State Feedback Vaccination Strategy Applied to a SISV Epidemic Model for Avoiding Endemic Equilibrium Points*

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Abstract— **A vaccination strategy based on the state feedback control theory is proposed. The objective is to fight against the propagation of an infectious disease within a host population. Such a propagation is modelled by means of a SISV (susceptible-infectious-susceptible-vaccinated) epidemic model with a time varying whole population and with a mortality directly associated with the disease. This model contains some free-design parameters, namely, the feedback gains of the vaccination control law. The paper analyses the positivity of such a model under the proposed vaccination strategy as well as the conditions for the existence of the equilibrium points of its normalized model. In this context, it is proved that an appropriate adjustment of the control gains avoids the existence of endemic equilibrium points in the normalized SISV model while guaranteeing the existence of a unique disease-free equilibrium point being globally asymptotically stable.**

I. Introduction

The propagation of epidemic diseases by means of mathematical models has been broadly analysed [1]. Such researches can be a starting point to elucidate the type of control strategy to be applied in order to minimise the impact of the disease within the host population. Also, the behaviour of the disease propagation under the application of a designed control strategy can be predicted by using such models. The control strategies can be of different types, namely, vaccination, quarantines, isolation in hospitals and use of antiviral drugs among others [2-6]. The epidemic models used in the literature are of different types. The most used ones are the compartmental models where the host population is split in different categories depending on its status with respect to the infection [7]. In this sense, the model can contain the categories of susceptible, exposed, infectious, recovered, vaccinated, hospitalized and so on.

The analysis of a SISV epidemic model, with a time-varying population and mortality directly associated to the disease, under a vaccination strategy based on a state feedback control law is carried out in this paper. The model takes into account that the efficiency of the vaccines can be less than 100%, i.e., only a portion of the susceptible individuals who receive a vaccine passes to the vaccinated category [8]. Furthermore, a portion of vaccinated individuals loses the immunity after an immunity period, i.e., the immunity for life of all the vaccinated individuals is not guaranteed by the vaccines [9]. The vaccination strategy provides four free design parameters. Three of them are the constant control gains, each one associated to each state variable of the model. The other one is used to switch off the vaccination action when the proportion of susceptible subpopulation is smaller than a prescribed threshold. The analysis includes the proof of the positivity of the model under the proposed vaccination strategy. Also the influence of the control gains on the dynamics of the disease transmission within the host population is studied by means of a normalized SISV model. Such a normalized model has two independent state variables, instead of the three of the original SISV model, what simplifies the analysis. The conditions for the existence of the equilibrium points of this normalized model depending on the assigned values to the control gains are analysed. In this context, *the existence of appropriate choices for the control gains guaranteeing the non-existence of endemic equilibrium (EE) points and the existence of a unique globally asymptotically stable disease-free equilibrium (DFE) point for the normalized SISV model is proved*. In this way, *the main objective of eradicating the disease propagation, while guaranteeing the persistence of the host population, can be achieved by means of the application of a vaccination strategy based on the state feedback control law with an appropriate adjustment of the control gains. A main reason is that EE points can be removed through the choice of the control gains.* Furthermore, the proportion of the vaccinated subpopulation in such a globally asymptotically stable DFE point depends on the values of the control gains. Then, the number of vaccines to be used during the vaccination campaign can be prefixed by adjusting such gains. In a practical situation, this fact can be used to choose the control gains according to the number of available vaccines while guaranteeing the unique existence of the globally asymptotically stable DFE point.

II. The SISV Epidemic Model

The SISV epidemic model splits the host population in three different categories: susceptible, infectious and vaccinated subpopulations. The transitions between these subpopulation categories are given by:

$$\begin{aligned}\dot{S}(t) &= \upsilon N(t) + \gamma I(t) - \beta\frac{S(t)I(t)}{N(t)} - \mu S(t) + p\sigma V(t) - fU(t)\\ \dot{I}(t) &= \beta\frac{S(t)I(t)}{N(t)} - (\gamma+\alpha+\mu)I(t)\\ \dot{V}(t) &= -(\mu+p\sigma)V(t) + fU(t)\end{aligned} \tag{1}$$

subject to $S(0)\geq 0$, $I(0)\geq 0$, $V(0)\geq 0$ and $S(0)+I(0)+V(0)>0$, where $S(t)$, $I(t)$ and $V(t)$ denote respectively the susceptible, infectious and vaccinated

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subpopulations at the instant t, $N(t)=S(t)+I(t)+V(t)$ denotes the whole host population and $U(t)$ is a control function to be specified later on. By summing up the equations of (1) one obtains the dynamics of the whole population given by:

$$\dot{N}(t)=(\upsilon-\mu)N(t)-\alpha I(t)\,. \tag{2}$$

The parameters of the model are all strictly positive. Namely, υ and μ denote respectively the birth and the mortality by natural causes rates of the host population. The transmission of the infection from mothers to sons/daughters is not considered so the new births are included directly in the susceptible category. The rest of the parameters are associated with the infectious disease: β denotes the infection transmission rate, γ is the recuperation rate and α is the mortality rate by causes directly related to the disease. The inverse of γ denotes the average time interval that an infectious individual stays within the infectious category before passing to the susceptible category. The transition of an individual from the susceptible category to the infectious one can happen, with certain probability, when such a susceptible individual contacts with an infectious one. In this sense, the factor $\beta I(t)/N(t)$ is the per capita probability of acquiring the infection at the instant t and the term $\beta S(t)I(t)/N(t)$ represents the total rate of transmissions of the infection at the instant t [1]. The parameter $f\in[0,1]$ denotes the vaccination efficiency, i.e., the fraction of susceptible individuals who pass to the vaccinated category after receiving a vaccine. The value $f=1$ means a 100% of efficacy in the vaccines. Finally, the parameter $p\in[0,1]$ denotes the portion of vaccinated individuals who loses the immunity and passes to the susceptible category after passing a certain time period σ^{-1}. The value $p=0$ points out that all the vaccinated individuals acquire an immunity for life after receiving a vaccine.

The function $U(t)$ works as a control signal and it denotes the number of susceptible individuals receiving a vaccine at the instant t. Such a control signal is defined by:

$$U(t)=\begin{cases}\mathrm{Max}\{0,\,k_s S(t)+k_\iota I(t)+k_v V(t)\} & \text{if } S(t)\geq\varepsilon_s N(t)>0\\ 0 & \text{otherwise}\end{cases} \tag{3}$$

where k_s, k_ι and k_v are constants, namely, the controller gains. Such a vaccination control law is based on the feedback of the variables of the SISV model while it is non-negative definite. Furthermore, the vaccination is switched off while the proportion of susceptible subpopulation is strictly smaller than a prescribed threshold $\varepsilon_s\in(0,1)$.

Theorem 1 (positivity of the SISV model). The SISV epidemic model (1) is positive under the application of the control signal (3) irrespective of the values assigned to the control gains since $S(t)\geq 0$, $I(t)\geq 0$ and $V(t)\geq 0\ \ \forall t\geq 0$ provided that $S(0)\geq 0$, $I(0)\geq 0$ and $V(0)\geq 0$.

Proof. It is omitted by space reasons. ***

III. Normalized SISV Model

A variables change lets us obtain a normalized SISV epidemic model useful to analyse the dynamics of the propagation of the disease under the proposed vaccination strategy. Such a variables change is given by:

$$s(t)=\frac{S(t)}{N(t)}\quad;\quad \iota(t)=\frac{I(t)}{N(t)}\quad;\quad v(t)=\frac{V(t)}{N(t)} \tag{4}$$

where the resulting new variables $s(t)$, $\iota(t)$ and $v(t)$ represent, respectively, the proportion of susceptible, infectious and vaccinated individuals within the host population. Note that $s(t)\in[0,1]$, $\iota(t)\in[0,1]$ and $v(t)\in[0,1]\ \ \forall t\geq 0$ is derived from Theorem 1 if a vaccination strategy based on the control law (3) is applied in the epidemic model. One obtains the following normalized SISV model:

$$\begin{aligned}\dot{\iota}(t)&=\big(\beta-(\upsilon+\gamma+\alpha)\big)\iota(t)-(\beta-\alpha)\iota^2(t)-\beta\iota(t)v(t)\\ \dot{v}(t)&=-(\upsilon+p\sigma)v(t)+\alpha\iota(t)v(t)+fu(t)\end{aligned} \tag{5}$$

where:

$$u(t)=\frac{U(t)}{N(t)}=\begin{cases}\mathrm{Max}\{0,\,k_s s(t)+k_\iota\iota(t)+k_v v(t)\} & \text{if } s(t)\geq\varepsilon_s>0\\ 0 & \text{otherwise}\end{cases} \tag{6}$$

denotes the normalized control signal and the fact that $s(t)+\iota(t)+v(t)=1\ \ \forall t\geq 0$ has been used. The following subsection analyses the equilibrium points for the model (5) under the control law (6) depending on the values of the controller gains.

Remark 1. The dynamics of the normalized SISV model (5) in absence of vaccination, i.e. with $u(t)=0\ \ \forall t\geq 0$, and an initialization with $v(0)=0$ is given by:

$$\dot{\iota}(t)=\big(\beta-(\upsilon+\gamma+\alpha)\big)\iota(t)-(\beta-\alpha)\iota^2(t) \tag{7}$$

together with $s(t)=1-\iota(t)$ and $v(t)=0\ \ \forall t\geq 0$. This model has two potential equilibrium points obtained by introducing the condition $\dot{\iota}(t)=0$ in (7). One of them is a DFE point where the proportions of the subpopulations are $s^*=1$ and $\iota^*=v^*=0$. The other one is an EE point where $s^*=(\upsilon+\gamma)/(\beta-\alpha)$, $\iota^*=\big(\beta-(\upsilon+\gamma+\alpha)\big)/(\beta-\alpha)$ and $v^*=0$. The DFE point exists irrespective of the values of the model parameters while the existence of the EE one requires that $\beta\geq\upsilon+\gamma+\alpha$ so that $\iota^*\in[0,1]$. Note that both points degenerate in one if $\beta=\upsilon+\gamma+\alpha$. Moreover, one obtains that $\dot{\iota}(t)\leq\big(\beta-(\upsilon+\gamma+\alpha)\big)\iota(t)$ if $\beta\geq\alpha$ while $\dot{\iota}(t)\leq-(\upsilon+\gamma)\iota(t)$ if $\beta\leq\alpha$ from (7) where the fact that $\iota(t)\in[0,1]\ \ \forall t\geq 0$ and then $\iota^2(t)\leq\iota(t)$ has been taken into account. As a consequence, $\iota(t)\leq\iota(0)\,e^{\lambda t}$ with either $\lambda=\beta-(\upsilon+\gamma+\alpha)$ if $\beta\geq\alpha$ or $\lambda=-(\upsilon+\gamma)$ if $\beta\leq\alpha$. Thus, the DFE point is unique and globally exponentially stable if

$\beta < \upsilon+\gamma+\alpha$. Furthermore, $\dot{\iota}(t) = -(\beta-\alpha)\iota^2(t)$ if $\beta = \upsilon+\gamma+\alpha$ from (7) and one obtains that $\iota(t) = \iota(0)\big/\big(1+(\upsilon+\gamma)\iota(0)t\big)$ by direct calculations. Then, the DFE point is globally asymptotically stable since $\iota(t) \to 0$ as $t \to \infty$ irrespective of the initial condition. Finally, the local stability of the model around the equilibrium points is given by:

$$\dot{\delta}_\iota(t) = \big(\beta-(\upsilon+\gamma+\alpha)-2(\beta-\alpha)\iota^*\big)\delta_\iota(t) \tag{8}$$

if $\beta > \upsilon+\gamma+\alpha$ where $\delta_\iota(t) = \iota(t) - \iota^*$ denotes the deviation of the proportion of infectious subpopulation with respect to such a proportion at the equilibrium point. In this context, the DFE point where $\iota^* = 0$ is locally exponentially unstable since $\delta_\iota(t) = \delta_\iota(t_0)\, e^{\lambda\cdot(t-t_0)}$, with $\lambda = \beta-(\upsilon+\gamma+\alpha) > 0$, is obtained by direct calculations from (8). The global stability of the EE point is analyzed by applying the variable change $x(t) = \iota(t) - \big(\beta-(\upsilon+\gamma+\alpha)\big)\big/(\beta-\alpha)$ to (7). Then, one obtains that $\dot{x}(t) = -\big(\beta-(\upsilon+\gamma+\alpha)\big)x(t) - (\beta-\alpha)x^2(t)$ and then $\dot{x}(t) \le -\big(\beta-(\upsilon+\gamma+\alpha)\big)x(t)$ since $\beta > \upsilon+\gamma+\alpha > \alpha$. As a consequence, $x(t) \le x(0)\, e^{\lambda t}$ with $\lambda = -\big(\beta-(\upsilon+\gamma+\alpha)\big) < 0$. It implies that $x(t) \to 0$ exponentially as $t \to \infty$ or, equivalently, $\iota(t) \to \big(\beta-(\upsilon+\gamma+\alpha)\big)\big/(\beta-\alpha)$ exponentially as $t \to \infty$ irrespective of the initial condition. Thus, the EE point is globally exponentially stable if $\beta > \upsilon+\gamma+\alpha$. A basic reproduction number defined as $R_0 = \beta/(\upsilon+\gamma+\alpha)$ can be used to summarize these stability results. In this sense, the normalized SISV model in absence of vaccination and with $v(0) = 0$ has a unique equilibrium point, namely the DFE point, which is globally exponentially stable if $R_0 < 1$. Such a model has only the DFE point which is globally asymptotically stable if $R_0 = 1$. Finally, the model has both equilibrium points if $R_0 > 1$ being the DFE point locally exponentially unstable while the EE point being globally exponentially stable. ***

The design of a vaccination strategy is of special interest when $\beta > \upsilon+\gamma+\alpha$ or, equivalently, $R_0 > 1$ to avoid the evolution of the model towards an EE point. Then, the assumption $\beta > \upsilon+\gamma+\alpha$ is considered in the following.

A. Equilibrium Points

The normalized SISV model (5) under the control law (6) asymptotically reaches an equilibrium point given by ι^*, v^* and $s^* = 1-\iota^*-v^*$ when $\dot{\iota}(t) = 0$ and $\dot{v}(t) = 0$. Then:

$$\begin{aligned} &\big(\beta-(\upsilon+\gamma+\alpha)\big)\iota^* - (\beta-\alpha)\iota^{*2} - \beta\iota^* v^* = 0 \\ &-(\upsilon+p\sigma)v^* + \alpha\iota^* v^* + fu^* = 0 \end{aligned} \tag{9}$$

where u^* denotes the value of the control signal at each potential equilibrium point. Such a value can be $u^* = k_s + (k_\iota - k_s)\iota^* + (k_v - k_s)v^* \ge 0$ or $u^* = 0$ by taking into account the control law (6). The first equation of (9) has two solutions, namely, $\iota^* = \big(\beta-(\upsilon+\gamma+\alpha)-\beta v^*\big)\big/(\beta-\alpha)$ and $\iota^* = 0$. The potential equilibrium points of the model are obtained by combining each feasible expression for u^* with both solutions for ι^* and they depend on the control gains. In this way:

(a) If $\iota^* = 0$ and $u^* = 0$ then the solution $v^* = 0$ is obtained from the second equation of (9). Then, a DFE point is obtained, namely, the point DFE1 defined by:

$$\text{DFE1:} \quad s^* = 1 \;\; ; \;\; \iota^* = 0 \;\; ; \;\; v^* = 0 \tag{10}$$

The point DFE1 exists if the control gain k_s is chosen such that $k_s \le 0$ and then $u^* = 0$. Moreover, such an existence is irrespective of the values chosen for the control gains k_ι and k_v . Such facts are deduced from (6) by taking into account that $s^* = 1 > \varepsilon_s$ at such a point.

(b) If $\iota^* = 0$ and $u^* = k_s + (k_\iota - k_s)\iota^* + (k_v - k_s)v^* \ge 0$ then the solution $v^* = fk_s\big/\big(f(k_s-k_v)+\upsilon+p\sigma\big)$ is obtained from the second equation of (9). Then, a DFE point is obtained, namely, the point DFE2 defined by:

$$\text{DFE2:} \quad s^* = \frac{\upsilon+p\sigma-fk_v}{f(k_s-k_v)+\upsilon+p\sigma} \;\; ; \;\; \iota^* = 0 \qquad v^* = \frac{fk_s}{f(k_s-k_v)+\upsilon+p\sigma} \tag{11}$$

The feasibility of the point DFE2 requires that $s^* \in [0, 1]$, $v^* \in [0, 1]$ and $u^* \ge 0$. Such conditions are simultaneously satisfied if the gains k_s and k_v are chosen so that $\varepsilon_s \le (\upsilon+p\sigma-fk_v)\big/\big(f(k_s-k_v)+\upsilon+p\sigma\big) \le 1$. This implies that the point DFE2 exists if (i) $k_s > 0$ and $k_v \le m_{v1}(k_s, \varepsilon_s) = (\upsilon+p\sigma)/f - \varepsilon_s k_s/(1-\varepsilon_s)$, (ii) $k_s < 0$ and $k_v \ge m_{v1}(k_s, \varepsilon_s)$ or (iii) $k_s = 0$ and $k_v \ne (\upsilon+p\sigma)/f$. Note that the point DFE2 is the same that DFE1 in the last case. Also, note that if $k_s = 0$ and $k_v = (\upsilon+p\sigma)/f$ then $u^* = (\upsilon+p\sigma)v^*/f$ since $\iota^* = 0$. Then, $\iota^* = 0$ with any s^* and v^* , such that $s^* + v^* = 1$, are solutions for the equations system (9). The feasibility of such equilibrium points requires that $s^* \in [\varepsilon_s, 1]$, $v^* \in [0, 1-\varepsilon_s]$ so that $u^* \ge 0$ from (6). Then, a set of DFE points defined by:

$$\text{DFE3:} \quad s^* \in [\varepsilon_s, 1] \;\; ; \;\; \iota^* = 0 \;\; ; \;\; v^* \in [0, 1-\varepsilon_s] \quad \text{such that } s^* + v^* = 1 \tag{12}$$

is obtained with such a choice of k_s and k_v irrespective of the value assigned to k_ι. Note that the point DFE1 is included in the set of points DFE3.

(c) If $\iota^* = \left(\beta - (\upsilon+\gamma+\alpha) - \beta v^*\right)\big/(\beta-\alpha)$ and $u^* = 0$ then two solutions for v^* are obtained from the second equation of (9), namely, $v^* = \left(\beta(\alpha-\upsilon) - \alpha(\gamma+\alpha) - p\sigma(\beta-\alpha)\right)\big/\alpha\beta$ and $v^* = 0$. Then, the model has two potential EE points, namely, the point EE1 defined as:

$$\text{EE1}: \quad s^* = \frac{\upsilon+\gamma}{\beta-\alpha} \quad ; \quad \iota^* = \frac{\beta-(\upsilon+\gamma+\alpha)}{\beta-\alpha} \quad ; \quad v^* = 0 \tag{13}$$

and the point EE2 given by:

$$\begin{aligned} \text{EE2}: \quad s^* &= \frac{\gamma+\alpha-p\sigma}{\beta} \quad ; \quad \iota^* = \frac{\upsilon+p\sigma}{\alpha} \\ v^* &= 1 - \frac{\beta\upsilon+\alpha(\gamma+\alpha)+p\sigma(\beta-\alpha)}{\alpha\beta} \end{aligned} \tag{14}$$

The feasibility of such points requires that $s^* \in [0, 1]$, $\iota^* \in [0, 1]$, $v^* \in [0, 1]$ and $u^* = 0$ at them. In this context, the existence of the point EE1 requires that $s^* = (\upsilon+\gamma)/(\beta-\alpha) < \varepsilon_s$ or, otherwise, that the controller gains k_s and k_ι are chosen such that $k_s(\upsilon+\gamma) + k_\iota\left(\beta-(\upsilon+\gamma+\alpha)\right) \le 0$ so that $u^* = 0$ at such a point by taking into account (6). Note that $s^* \in [0, 1]$, $\iota^* \in [0, 1]$ and $v^* \in [0, 1]$ for the point EE1 provided that $\beta > \upsilon+\gamma+\alpha$.

The existence of the point EE2 requires that:

$$\begin{aligned} &\alpha \ge \upsilon \quad ; \quad \beta \ge \alpha(\gamma+\alpha)/(\alpha-\upsilon) \\ &p \le \bar{p} = \alpha/\sigma - (\alpha\gamma+\beta\upsilon)\big/\left(\sigma(\beta-\alpha)\right) \end{aligned} \tag{15}$$

so that:

$$\begin{aligned} s^* &= \frac{\gamma+\alpha-p\sigma}{\beta} \ge \frac{\gamma+\alpha-\bar{p}\sigma}{\beta} = \frac{\upsilon+\gamma}{\beta-\alpha} > 0 \\ \iota^* &= \frac{\upsilon+p\sigma}{\alpha} \le \frac{\upsilon+\bar{p}\sigma}{\alpha} = \frac{\beta-(\upsilon+\gamma+\alpha)}{\beta-\alpha} < 1 \\ v^* &= 1 - \frac{\beta\upsilon+\alpha(\gamma+\alpha)+p\sigma(\beta-\alpha)}{\alpha\beta} \\ &\ge 1 - \frac{\beta\upsilon+\alpha(\gamma+\alpha)+\bar{p}\sigma(\beta-\alpha)}{\alpha\beta} = 0 \end{aligned} \tag{16}$$

In this way, $s^* \in (0, 1)$, $\iota^* \in (0, 1)$ and $v^* \in [0, 1)$ at the point EE2 since the model parameters are defined positive and provided that $\beta > \upsilon+\gamma+\alpha$. Furthermore, the existence of the point EE2 requires that either $\varepsilon_s > \frac{\gamma+\alpha-p\sigma}{\beta}$ so that $s^* \in [0, \varepsilon_s)$ and $u^* = 0$ at such a point by taking into account (6) or, otherwise, $\varepsilon_s \le \frac{\gamma+\alpha-p\sigma}{\beta}$ together with the controller gains k_s, k_ι and k_v chosen such that:

$$\begin{aligned} &k_s\alpha(\gamma+\alpha-p\sigma) + k_\iota\beta(\upsilon+p\sigma) + \\ &k_v\left(\beta(\alpha-\upsilon) - \alpha(\gamma+\alpha) - p\sigma(\beta-\alpha)\right) \le 0 \end{aligned} \tag{17}$$

so that $s^* \in [\varepsilon_s, 1]$ and $u^* = 0$ by taking into account (6).

(d) If $\iota^* = \left(\beta - (\upsilon+\gamma+\alpha) - \beta v^*\right)\big/(\beta-\alpha)$ or, equivalently, $v^* = \left(\beta - (\upsilon+\gamma+\alpha) - (\beta-\alpha)\iota^*\right)\big/\beta$ together with $u^* = k_s + (k_\iota - k_s)\iota^* + (k_v - k_s)v^* \ge 0$ then two potential solutions for ι^* are obtained from the second equation of (9), namely, the solutions of $\iota^{*2} + g_1(k_s, k_\iota, k_v)\iota^* + g_0(k_s, k_v) = 0$ with the coefficients $g_0(k_s, k_v)$ and $g_1(k_s, k_\iota, k_v)$, depending on the controller gains, defined as:

$$\begin{aligned} g_0 &= \frac{(\upsilon+p\sigma-fk_v)\left(\beta-(\upsilon+\gamma+\alpha)\right) - f(\upsilon+\gamma+\alpha)k_s}{\alpha(\beta-\alpha)} \\ g_1 &= \frac{f\left(\alpha(k_s-k_v)+\beta(k_v-k_\iota)\right) - \beta\upsilon + \alpha(2\upsilon+\gamma+\alpha-\beta)}{\alpha(\beta-\alpha)} - \frac{p\sigma}{\alpha} \end{aligned} \tag{18}$$

Then, the model has two potential EE points, namely, the points EE3 and EE4 defined as:

$$\begin{aligned} &\text{EE3}: \quad s^* = s_3 = 1-\iota_3-v_3 \quad ; \quad \iota^* = \iota_3 \quad ; \quad v^* = v_3 \\ &\text{EE4}: \quad s^* = s_4 = 1-\iota_4-v_4 \quad ; \quad \iota^* = \iota_4 \quad ; \quad v^* = v_4 \end{aligned} \tag{19}$$

where $v_j = \left((\beta-\alpha)(1-\iota_j) - (\upsilon+\gamma)\right)\big/\beta$ and $s_j = \left(\alpha(1-\iota_j)+\upsilon+\gamma\right)\big/\beta$ for $j \in \{3, 4\}$. The feasibility of the point EE3, respectively EE4, requires that $\iota_j \in [0, 1]$, $v_j \in [0, 1]$ and $s_j \in [\varepsilon_s, 1]$ for $j = 3$, respectively $j = 4$, for a prescribed $\varepsilon_s \in (0, 1)$ so that $u^* = u_3 \ge 0$, respectively $u^* = u_4 \ge 0$. The four conditions for the feasibility of the point EE3, respectively EE4, are jointly fulfilled if $\iota_j \in [0, \min\{q_1, q_2, q_3\}]$ where:

$$\begin{aligned} q_1 &= \frac{\beta-(\upsilon+\gamma+\alpha)}{\beta-\alpha} \quad ; \quad q_2 = \frac{\upsilon+\gamma+\alpha-\varepsilon_s\beta}{\alpha} \\ q_3 &= \frac{k_s(\upsilon+\gamma+\alpha) + k_v\left(\beta-(\upsilon+\gamma+\alpha)\right)}{\beta(k_v-k_\iota) - \alpha(k_v-k_s)} \end{aligned} \tag{20}$$

for $j = 3$, respectively $j = 4$, as it can be deduced by direct calculations and taking into account (6). Note that if the free-design control parameter ε_s is chosen such that $\varepsilon_s > (\upsilon+\gamma+\alpha)/\beta$ then $q_2 < 0$ and there is not solution for

ι_j satisfying simultaneously the four conditions $\iota_j \in [0, 1]$, $v_j \in [0, 1]$, $s_j \in [\varepsilon_s, 1]$ and $u^* = u_j \geq 0$. Then, the points EE3 and EE4 do not exist but the normalized model has at least one EE point, namely, the point EE2, in such a case.

Theorem 2 (conditions for inexistence of EE points in the normalized SISV model). The normalized SISV model (5), under the control law (6), does not have EE points if the control parameter ε_s satisfies the condition:

(c1) $0 < \varepsilon_s \leq (\upsilon+\gamma)/(\beta-\alpha)$

and the control gains k_s, k_ι and k_v simultaneously satisfy:

(c2) $k_s(\upsilon+\gamma)+k_\iota\left(\beta-(\upsilon+\gamma+\alpha)\right)>0$,

(c3) $k_s\alpha(\gamma+\alpha-p\sigma)+k_\iota\beta(\upsilon+p\sigma)+k_v\left(\beta(\alpha-\upsilon)-\alpha(\gamma+\alpha)-p\sigma(\beta-\alpha)\right)>0$ if the model parameters fulfil the conditions in (15) and

(c4) $k_\iota \geq m_{i1}(k_v)$ and $m_{s1}(k_v) \leq k_s \leq m_{s2}(k_\iota, k_v)$ where:

$$m_{i1}(k_v) = \frac{\upsilon+\gamma}{\upsilon+\gamma+\alpha}k_v + \frac{\alpha}{f} - \frac{(\upsilon+\gamma)(\upsilon+p\sigma)}{f(\upsilon+\gamma+\alpha)} + \frac{\alpha\left(\upsilon^2-\alpha^2+\gamma(2\upsilon+\gamma)\right)}{f\beta(\upsilon+\gamma+\alpha)}$$

$$m_{s1}(k_v) = \frac{\beta-(\upsilon+\gamma+\alpha)}{f(\upsilon+\gamma+\alpha)}(\upsilon+p\sigma-k_v) \quad . \quad (21)$$

$$m_{s2}(k_\iota, k_v) = \frac{\beta}{\alpha}k_\iota - \frac{\beta-\alpha}{\alpha}k_v + \frac{\beta(\upsilon-\alpha)+\alpha(\alpha-2\upsilon-\gamma)+p\sigma(\beta-\alpha)}{f\alpha}$$

As a consequence, only the points DFE1, DFE2 and DFE3 are feasible under such conditions.

Proof. The proportion of susceptible subpopulation at the point EE1 fulfils that $s^* \geq \varepsilon_s$ under the condition (c1). Then, the normalized control signal at such a point satisfies that $u^* > 0$ under the condition (c2) by taking into account (6). This fact contradicts the requirement for the existence of the point EE1. The existence of the point EE2 requires that the model parameters satisfy all the conditions in (15). Note that if, at least, one of such conditions is not fulfilled then $\iota^* \notin [0, 1]$ or $v^* \notin [0, 1]$ at the point EE2 what implies the inexistence of such a point. In this context, if $\beta < \alpha(\gamma+\alpha)/(\alpha-\upsilon)$ then $\bar{p} < 0$ so that the condition $p \leq \bar{p}$ is not fulfilled since the parameter p is non-negative by definition. Then, $p > \bar{p}$ and one obtains that $v^* < 0$ at the point EE2 by direct calculations from (14). This fact implies the non-existence of the point EE2. Moreover, if $\alpha < \upsilon$ then $\iota^* > 1$ at the point EE2 from (14) which is not compatible with the existence of such a point. On the other hand, if the model parameters fulfil the conditions in (15) then:

$$s^* = \frac{\gamma+\alpha-p\sigma}{\beta} \geq \frac{\gamma+\alpha-\bar{p}\sigma}{\beta} = \frac{\upsilon+\gamma}{\beta-\alpha} \geq \varepsilon_s \quad (22)$$

by taking into account the condition (c1). This fact together with the condition (c3) implies that $u^* > 0$ at the point EE2 by taking into account (6). This fact contradicts the requirement for the existence of such a point. Finally, the points EE3 and EE4 do not exist if the condition (c4) is satisfied. Concretely, such a condition implies that $\iota_j \notin [0, 1]$ for $j \in \{3, 4\}$ as it is proved in the following way. The proportion of infectious subpopulation at the potential equilibrium points EE3 and EE4 are the solutions of the equation $\iota_j^2 + g_1(k_s, k_\iota, k_v)\iota_j + g_0(k_s, k_v) = 0$. Note that the function $F(\iota_j) = \iota_j^2 + g_1(k_s, k_\iota, k_v)\iota_j + g_0(k_s, k_v)$, for any given values of k_s, k_ι and k_v, corresponds to a parabola which is opening to the top and its intersecting points with the abscissas axis are the solutions of $F(\iota_j) = 0$. In this context, direct calculations prove that such intersecting points are not within the domain $[0, 1]$ under the condition (c4). First, note that $F(0) = g_0(k_s, k_v)$ and $\iota_{j,min} = -g_1(k_s, k_v, k_\iota)/2$ where $\iota_{j,min}$ is the value of ι_j at which the parabola $F(\iota_j)$ reaches its minimum value. Under the condition (c4) one obtains that:

$$F(0) \leq \frac{(\upsilon+p\sigma-k_v)\left(\beta-(\upsilon+\gamma+\alpha)\right)-f(\upsilon+\gamma+\alpha)m_{s1}}{\alpha(\beta-\alpha)} = 0$$

$$\iota_{j,min} \geq \frac{f\left(\beta k_\iota-(\beta-\alpha)k_v\right)+\beta\upsilon-\alpha(2\upsilon+\gamma+\alpha-\beta)-f\alpha m_{s2}}{2\alpha(\beta-\alpha)} + \frac{p\sigma}{2\alpha} = 1$$

where the condition $k_\iota \geq m_{i1}(k_v)$ is necessary to guarantee $m_{s1}(k_v) \leq m_{s2}(k_\iota, k_v)$ and then the existence of values for k_s such that $m_{s1}(k_v) \leq k_s \leq m_{s2}(k_\iota, k_v)$ be able, as one can deduce by direct calculations. The result $\iota_{j,min} \geq 1$ implies that $F(\iota_j)$ is monotonically decreasing $\forall \iota_j \in [0, 1)$. Such a fact, together with $F(0) \leq 0$, implies that $F(\iota_j) < 0$ $\forall \iota_j \in (0, 1]$ since the parabola $F(\iota_j)$ is opening to the top. Then, $F(\iota_j) = 0$ cannot have solutions within $\iota_j \in (0, 1]$ under such a condition (c4) implying the non-existence of the points EE3 and EE4. In summary, there are not feasible solutions for EE points under the conditions established in the theorem and the result is proved. ***

B. Stability analysis of the DFE points

The following theorem analyses the local stability of the DFE points of the normalized SISV model (5) under the control law (6) depending on the assigned values for the control parameters.

Theorem 3 (local stability/instability of the DFE points of the normalized SISV model).

(i) The point DFE1 is locally exponentially unstable whenever it exists, i.e. when $k_s \leq 0$, if $\beta > \upsilon+\gamma+\alpha$.

(ii) The point DFE2 is locally exponentially stable if $k_s > 0$, $\varepsilon_s < (\upsilon+\gamma+\alpha)/\beta$ and $m_{v2}(k_s) < k_v \le m_{v1}(k_s, \varepsilon_s)$ where $m_{v2}(k_s) = (\upsilon+p\sigma)/f - (\upsilon+\gamma+\alpha)k_s/(\beta-(\upsilon+\gamma+\alpha))$. The point DFE2 is locally exponentially unstable if either (a) $k_s > 0$, $k_v < m_{v2}(k_s)$ and $k_v \le m_{v1}(k_s, \varepsilon_s)$ or (b) $k_s < 0$ and $k_v \ge m_{v1}(k_s, \varepsilon_s)$ or (c) $k_s = 0$ and $k_v \ne (\upsilon+p\sigma)/f$.

(iii) The point DFE3 is locally exponentially unstable whenever it exists, i.e. when $k_s = 0$ and $k_v = (\upsilon+p\sigma)/f$, if $v^* < 1-(\upsilon+\gamma+\alpha)/\beta$ with $v^* \in [0, 1-\varepsilon_s]$. The local stability of this point is critical if $v^* \ge 1-(\upsilon+\gamma+\alpha)/\beta$ with $\varepsilon_s \le (\upsilon+\gamma+\alpha)/\beta$ so that $v^* \in [0, 1-\varepsilon_s]$.

Proof. It is omitted by space reasons. ***

Remark 2. Assume that the free-design parameter ε_s and the control gains k_s, k_ι and k_v simultaneously satisfy the conditions of Theorem 2 so that the normalized SISV model has not EE points. Moreover, if $k_s > 0$ then the normalized SISV model only has a DFE point, namely, the point DFE2. Furthermore, if $\varepsilon_s < (\upsilon+\gamma+\alpha)/\beta$ and $m_{v2}(k_s) < k_v \le m_{v1}(k_s, \varepsilon_s)$ then such a DFE point is locally exponentially stable in view of Theorem 3. A relevant result is that the point DFE2 is globally stable under such conditions for the controller parameters from the following facts: (i) the variables of the normalized SISV epidemic model are bounded, since $s, \iota, v \in [0, 1]$ from the positivity of the original SISV model as Theorem 1 establishes, (ii) the point DFE2 is the unique equilibrium point of the normalized SISV epidemic model and (iii) such a point is locally exponentially stable. ***

IV. Simulation Results

A. SISV Epidemic Model without Vaccination

The model (1) with a control signal $U(t) = 0 \ \forall t \ge 0$ and an initial condition given by $S(0) = 990$, $I(0) = 10$ and $V(0) = 0$ is considered. In this way, the vaccination subpopulation is $V(t) = 0 \ \forall t \ge 0$ so that the SISV model is equivalent to a simple SIS model. The values for the parameters $\upsilon = 2.6301\times10^{-5}$ d^{-1}, $\mu = 2.4658\times10^{-5}$ d^{-1}, $\beta = 1.66$ d^{-1}, $\gamma = 0.4545$ d^{-1} and $\alpha = 0.001$ d^{-1}, where d^{-1} means days^{-1}, are used to obtain the time evolution of the subpopulations and that of the whole population under the influence of the infectious disease. Such values are based on the transmission of influenza in a developed country [1]. The proportions of susceptible and infectious subpopulations, i.e. the normalized subpopulations, can be obtained by using (4). Also, such subpopulations could be directly obtained by using the normalized SISV model (5) with the aforementioned values for the parameters and $u(t) = 0 \ \forall t \ge 0$. The basic reproduction number of this normalized model results $R_0 = 3.6438$ in such a situation so that its DFE point is globally unstable while its EE point is globally exponentially stable as Remark 1 points out. Fig. 1 shows the time evolution of the susceptible, infectious and whole populations. One can see that the whole population tends to the extinction because of the dominant effect of the mortality associated to the disease. As a consequence, the application of a vaccination is indispensable in order to eliminate the infection irrespective of the initial conditions or, at least, diminish its effect within the host population and, in this way, achieve the persistence of the host population.

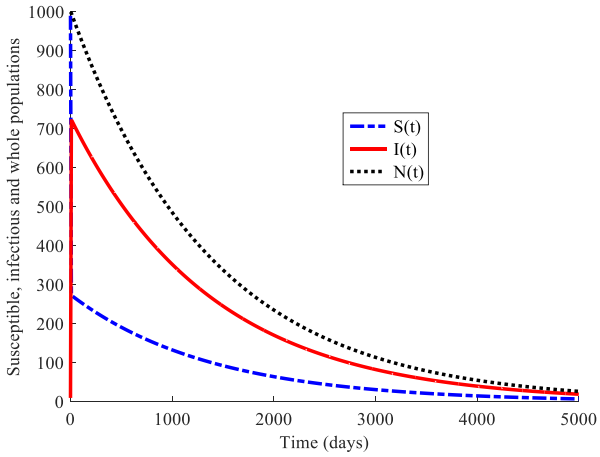

Figure 1. Susceptible, infectious and whole populations in absence of vaccination.

B. SISV Epidemic Model with Vaccination

The model (1), with the same values for the parameters and the same initial condition than those considered previously, is used under the application of a vaccination strategy based on a feedback of the model variables as that given in (3). Several situations are analysed depending on as the efficiency of the applied vaccines as the proportion of vaccinated population acquiring the immunity for life after receiving a vaccine. In this sense, three values for the parameters f and p are considered and 9 different examples are analysed by combining such values. The use of the vaccination strategy, with an appropriate choice of the free-design control parameters ε_s, k_s, k_ι and k_v is crucial to eradicate the disease while guaranteeing the persistence of the host population in all the examples. In this way, a suitable choice of the control parameters is: $\varepsilon_s = 0.01$, $k_v = -13\upsilon = -3.4192\times10^{-4}$ and $k_i = 3000\upsilon = 0.0789$ for all the examples while the value for k_s is chosen according to the assigned values for the parameters f and p in each example so that the conditions (c2), (c3) and (c4) of Theorem 2, which depend on k_s, f and p, are satisfied. In this way, all the conditions of Theorem 2 are fulfilled in all the examples and then the normalized SISV model (5) under the control law (6) only has a DFE point, namely, the point DFE2 defined in (11). Moreover, the conditions of Theorem 3 are also satisfied with such choices and then the point DFE2 is globally stable from the facts that it is locally

exponentially stable while the normalized subpopulations being bounded, see Remark 2. Other alternative ways can be used to assign the values of the control gains satisfying the conditions of Theorems 2 and 3. The used one is interesting from the fact that the smaller the gain k_s is, the smaller the cost in vaccines is when the proportion of susceptible individuals is large within the host population, as it occurs in the first days in all the examples. For such a purpose, the gain k_s is fixed to the minimum possible value for satisfying the condition of those theorems after assigning the other control parameters for each example. Concretely, 9 different examples are considered.

The efficacy of the vaccines is of 100%, i.e. $f=1$, in the first three ones with a different value for p in each one, namely $p=0$, $p=0.5$ and $p=1$. Such a parameter points out the proportion of vaccinated population acquiring the immunity for life. The $p=0$ means that all the vaccinated individuals acquire the immunity for life after receiving a vaccine, $p=0.5$ implies that one half of them acquires the immunity for life while the other half acquires a temporal immunity and they pass to the susceptible category after losing the immunity and $p=1$ means that all of them acquire a temporal immunity. Fig. 2 displays the time evolution of the subpopulations under the proposed vaccination for these three examples. The value for the control gain k_s is $k_s=38\upsilon=9.9945\times10^{-4}$ when $p=0$, $k_s=1713\upsilon=0.0451$ when $p=0.5$ and $k_s=3388\upsilon=0.0891$ when $p=1$. Fig. 3 shows the time evolution of the applied vaccines for these three examples.

The efficacy of the vaccines is of 75%, i.e. $f=0.75$, in the second three examples with a different value for p in each one, namely $p=0$, $p=0.5$ and $p=1$. Fig. 4 displays the time evolution of the subpopulations under the proposed vaccination for these three examples. The value for the control gain k_s is $k_s=50\upsilon=0.0013$ when $p=0$, $k_s=2284\upsilon=0.0601$ when $p=0.5$ and $k_s=4517\upsilon=0.1188$ when $p=1$. Fig. 5 shows the time evolution of the applied vaccines for these three examples.

The efficacy of the vaccines is of 50%, i.e. $f=0.5$, in the last three examples with a different value for p in each one, namely $p=0$, $p=0.5$ and $p=1$. Fig. 6 displays the time evolution of the subpopulations under the proposed vaccination for these three examples. The value for the control gain k_s is $k_s=75\upsilon=0.002$ when $p=0$, $k_s=3425\upsilon=0.0901$ when $p=0.5$ and $k_s=6776\upsilon=0.1782$ when $p=1$. Fig. 7 shows the time evolution of the applied vaccines for these three examples.

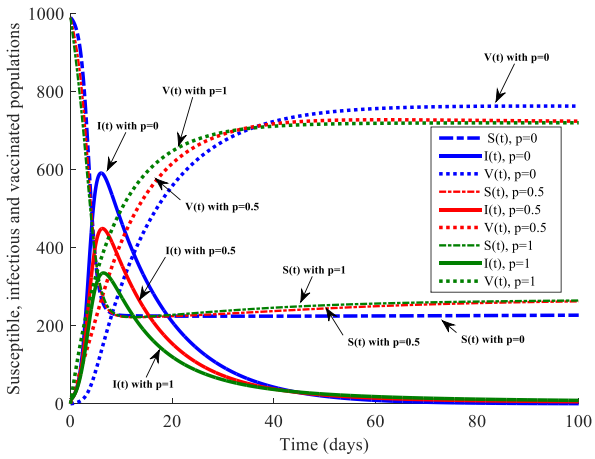

Figure 2. Susceptible, infectious and vaccinated populations with the proposed vaccination strategy if the vaccines efficacy is of 100% with different proportions of vaccinated acquiring permanent immunity.

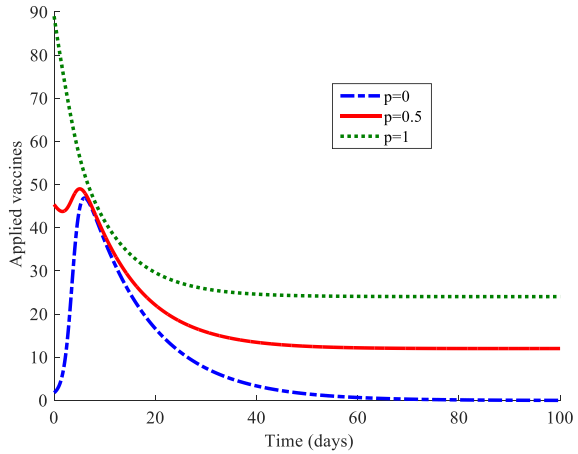

Figure 3. Applied vaccines with the proposed vaccination strategy if the vaccines efficacy is of 100% with different proportions of vaccinated acquiring permanent immunity.

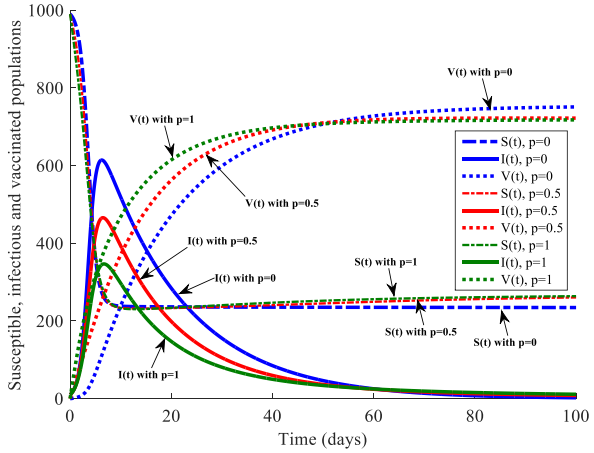

Figure 4. Susceptible, infectious and vaccinated populations with the proposed vaccination strategy if the vaccines efficacy is of 75% with different proportions of vaccinated acquiring permanent immunity.

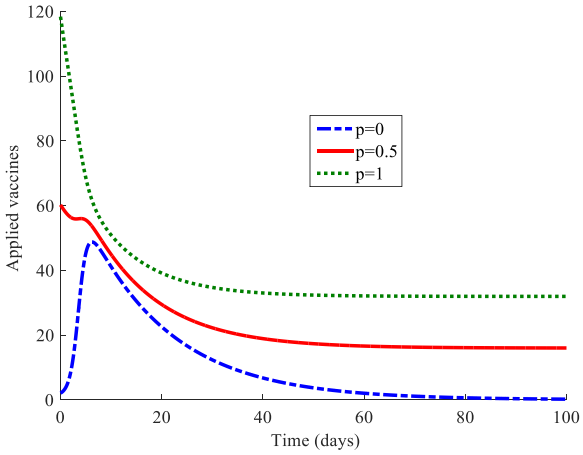

Figure 5. Applied vaccines with the proposed vaccination strategy if the vaccines efficacy is of 75% with different proportions of vaccinated acquiring permanent immunity.

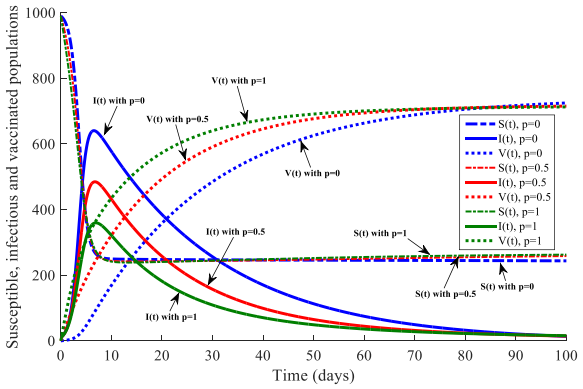

Figure 6. Susceptible, infectious and vaccinated populations with the proposed vaccination strategy if the vaccines efficacy is of 50% with different proportions of vaccinated acquiring permanent immunity.

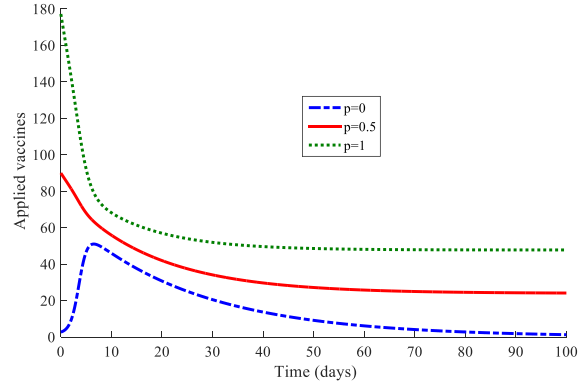

Figure 7. Applied vaccines with the proposed vaccination strategy if the vaccines efficacy is of 50% with different proportions of vaccinated acquiring permanent immunity.

The vaccination campaign has a finite period if all the vaccinated individuals acquire the immunity for life after receiving a vaccine. Obviously, the duration of the vaccination campaign increases if the efficacy of the vaccines decreases, as one can deduce from Figs. 3, 5 and 7. On the other hand, the vaccination campaign has to be kept active for all time if the immunity for life is not acquire for all the vaccinated individuals. The evolution of the applied vaccines reaches a constant value once the system converges to the point DFE2 in such cases. Such a value is the number of vaccines to be applied each day in order to maintain the disease propagation under control. Furthermore, the number of vaccines increases if p increases, i.e. the proportion of vaccinated acquiring the immunity for life decreases, and/or f decreases, i.e. the efficacy of the vaccination decreases, as one can see in Figs. 3, 5 and 7.

The number of infectious individuals reaches a maximum value and then it decreases towards zero. Such a peak value decreases if p and/or f increase. In this sense, an improvement in the efficacy of the vaccination imply a decreasing in the number of vaccines as well as in the peak of the infectious subpopulations, as one can expect. On the other hand, a larger proportion of vaccinates acquiring the immunity for life implies a smaller cost in vaccines with a larger peak in the evolution of the infectious subpopulation, as one can deduce from Figs. 2 to 7.

V. Conclusion

A vaccination strategy based on the state feedback control technique to fight against the propagation of an infectious disease is proposed in a SISV epidemic model. The main result points out that an appropriate adjustment of the control parameters can avoid the existence of endemic solutions. Such a fact is key to guarantee the eradication of the infectious disease in different situations depending on the efficacy of the vaccines as well as the proportion of individuals acquiring the immunity for life after being vaccinated. Future researches will analyze the dynamics of the controlled epidemic model subject to uncertainty in the measures needed to implement the control law.

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