INFORMATICA, 2011, Vol. 22, No. 3, 339–370

On the Equilibrium Points, Boundedness and Positivity of a Sveirs Epidemic Model under Constant Regular Constrained Vaccination

Manuel De la SEN[1], Asier IBEAS[2], Santiago ALONSO-QUESADA[1], Raul NISTAL[1]

[1]*Institute of Research and Development of Processes, Faculty of Science and Technology, University of the Basque Country*
PO. Box 644-Bilbao, Spain
Also with Department of Electricity and Electronics, Faculty of Science and Technology, University of the Basque Country
PO. Box 644-Bilbao, Spain
[2]*Department of Telecommunications and Systems Engineering, Universitat Autònoma de Barcelona*
08193-Bellaterra, Barcelona, Spain
e-mail: manuel.delasen@ehu.es, santi@we.lc.ehu.es, raul.nistal@gmail.com, asier.ibeas@uab.cat

Received: December 2010; accepted: May 2011

Abstract. This paper discusses the disease-free and endemic equilibrium points of a SVEIRS propagation disease model which potentially involves a regular constant vaccination. The positivity of such a model is also discussed as well as the boundedness of the total and partial populations. The model takes also into consideration the natural population growing and the mortality associate to the disease as well as the lost of immunity of newborns. It is assumed that there are two finite delays affecting to the susceptible, recovered, exposed and infected population dynamics.

Keywords: epidemic models, equilibrium points, SEIRS epidemic models, SVEIRS epidemic models, positivity, vaccination control.

1. Introduction

Important control problems nowadays related to Life Sciences are the control of ecological models like, for instance, those of population evolution as, for instance, Beverton–Holt model, Hassell model, Ricker model etc. via the online adjustment of the species environment carrying capacity, that of the population growth or that of the regulated harvesting quota as well as the disease propagation via vaccination control (De la Sen, 2008a, 2008b; De la Sen and Alonso-Quesada, 2008b, 2008c, 2009, 2010a). In a set of papers, several variants and generalizations of the Beverton–Holt model (standard time-invariant, time-varying parameterized, generalized model or modified generalized model) have been investigated at the levels of stability, cycle-oscillatory behavior, permanence

and control through the manipulation of the carrying capacity. See, for instance, De la Sen (2008a, 2008b), De la Sen and Alonso-Quesada (2008b, 2008c, 2009). The design of related control actions has been proved to be important in those papers at the levels, for instance, of aquaculture exploitation or plague fighting. On the other hand, the literature about epidemic mathematical models is exhaustive in many books and papers. A non-exhaustive list of references is given in this manuscript, cf. (D'Amico *et al.*, 2011; Ertuk and Momani, 2008; Gao *et al.*, 2008; Keeling and Rohani, 2008; Khan, Krishnan and Al-Khodh, 2003; Khan *et al.*, 2009; Mollison, 2008; Mukhopadhyay and Bhattacharyya, 2007; Ortega *et al.*, 2003; Piccardi and Lazzaris, 1998; Song *et al.*, 2009; Yildirim and Cherruault, 2009; Zhang and Teng, 2008; Zhang *et al.*, 2009). See also the references listed therein. The sets of models include the most basic ones (Mollison, 2008; Keeling and Rohani, 2008), namely:

- SI-models where not removed-by – immunity population is assumed. In other words, only susceptible and infected populations are assumed.
- SIR-models, which include susceptible, infected and removed-by – immunity populations.
- SEIR-models where the infected populations is split into two ones (namely, the "infected" which incubate the disease but do not still have any disease symptoms and the " infectious" or "infective" which do exhibit the external disease symptoms).

The three above models have two possible major variants, namely, the so-called "pseudo-mass action models", where the total population is not taken into account as a relevant disease contagious factor or disease transmission power, and the so-called "true-mass action models", where the total population is more realistically considered as being an inverse factor of the disease transmission rates. There are other many variants of the above models, for instance, including vaccination of different kinds: constant (Yildirim and Cherruault, 2009) impulsive (Song *et al.*, 2009; Yu *et al.*, 2010; Boichuk *et al.*, 2010), discrete-time etc., incorporating point or distributed delays (Song *et al.*, 2009; Zhang *et al.*, 2009), oscillatory behaviors (Mukhopadhyay and Bhattacharyya, 2007) etc. On the other hand, variants of such models become considerably simpler for the disease transmission among plants (Mollison, 2008; Keeling and Rohani, 2008). Some generalizations involve the use of a mixed regular continuous-time/impulsive vaccination control strategies for generalized time-varying epidemic model which is subject to point and distributed either constant or time-varying delays (Song *et al.*, 2009; Zhang *et al.*, 2009; Barreiro and Banos, 2010; De la Sen, 2007a, 2007b). Other well-known types of epidemic models are the so-called SVEIRS epidemic models which incorporate the dynamics of a vaccinated population and the "infected" population without external symptoms of the SEIR-type models is replaced with an "exposed" population subject to a certain dynamics (De la Sen *et al.*, 2010b; Jiang *et al.*, 2009; Song *et al.*, 2009). Thus, in the context of SVEIRS models, the infected and infectious populations of the SEIR models are joined in a single "infected" population $I(t)$ while there is an exposed population $E(t)$ present in the model. In this paper, we focus on the existence and some properties of disease-free and endemic equilibrium points of a SVEIRS model subject to an eventual constant regular vaccination rather than to an impulsive vaccination type. Some issues about boundedness

and positivity of the model are also investigated. The following SVEIRS epidemic model, of a modified true-mass action type, with regular constant vaccination is considered:

$$\dot{S}(t) = b(1 - S(t)) - \beta \frac{S(t)I(t)}{1 + \eta S(t)} + \gamma I(t - \omega)e^{-b\omega} + \nu(1 - V_c)N(t), \tag{1.1}$$

$$\dot{V}(t) = -\frac{\delta\beta V(t)I(t)}{1 + \eta V(t)} - (\gamma_1 + b)V(t) + \nu V_c N(t), \tag{1.2}$$

$$E(t) = \beta \int_{t-\omega}^{t} \frac{S(u)I(u)}{1 + \eta S(u)} + \frac{\delta V(u)I(u)}{1 + \eta V(u)} \mathrm{e}^{-b(t-u)}\, \mathrm{d}u, \tag{1.3}$$

$$\begin{aligned} \dot{I}(t) = {} & \beta e^{-b\tau} \left(\frac{S(t - \tau)I(t - \tau)}{1 + \eta S(t - \tau)} + \frac{\delta V(t - \tau)I(t - \tau)}{1 + \eta V(t - \tau)} \right) \\ & - \gamma + b + \alpha)I(t), \end{aligned} \tag{1.4}$$

$$\dot{R}(t) = -bR(t) + \gamma_1 V(t) + \gamma\big(I()t - I(t - \omega)\mathrm{e}^{-b\omega}\big), \tag{1.5}$$

where $S,\ V,\ E,\ I$ and R are, respectively, the susceptible, vaccinated, exposed, infected (or infective or infectious) and recovered (or removed-by-immunity) partial populations, $N(t)$ is the total population being the sum of the above ones, $V_c \in [0, 1]$ is a constant vaccination action. There are potential latent and immune periods denoted by τ and ω, respectively, which are internal delays in the dynamic epidemic model (1.1)–(1.5), b is the natural birth rate and death rate of the population. The parameter $\nu < b$ takes into account a vaccination action on newborns which decreases the incremental susceptible population through time, γ_1 is the average rate for vaccines to obtain immunity and move into recovered population, β (disease transmission constant) and $\delta\beta$ are, respectively, average numbers for contacts of an infective with a susceptible and an infective with a vaccinated individual per unit of time (Jiang *et al.*, 2009; Song *et al.*, 2009). The periodic impulsive, rather than regular, vaccination action proposed in Jiang *et al.* (2009) can be got from (1.1)–(1.5) with $V_c = 0$ and a regular impulsive vaccination period $T > 0$ consisting of a culling action on the susceptible plus the corresponding increase of the vaccinated population. Impulsive vaccination has also been recently investigated in De la Sen *et al.* (2010b) concerning generalized SEIR epidemic models involving time-varying delays and presence of infected and infectious population thresholds. It has to be pointed out that the epidemic model delays, representing in particular latent and immune periods, parameterize the epidemic model apart from the role they play through the delayed model state in the dynamics and thus in the trajectory solution. This phenomenon is not very common in standard time-delay systems where delays do not play usually a relevant role in the parameterizations but only in the state-trajectory solution through the delayed state dynamics (De la Sen, 2003; De la Sen and Luo, 2004; Luo *et al.*, 1997). It has to be pointed out that the use of mathematical models supported by electronics instrumentation is also very relevant for the study of biological process such as models of blood circulation because of its facility for discretized implementation of real testing experiments (see, for instance, Maciulis *et al.*, 2009). It has also to be pointed out that epidemic models are not controllable in the sense that all the populations cannot

be simultaneously governed (Nieto and Tisdell, 2010). Therefore, the main vaccination objective is to reduce the infected population as faster and as close to zero as possible (De la Sen and Alonso-Quesada, 2010b; De la Sen *et. al.* 2010b). This paper investigates the disease-free and endemic equilibrium points, their local stability properties as well as the positivity and boundedness properties of the state-trajectory solutions under optional constant constrained vaccination.

2. The Disease-Free Equilibrium Point

The potential existence of a disease-free equilibrium point is now discussed which asymptotically removes the disease if $\nu < b$.

PROPOSITION 1. Assume that $\nu < b$. Then the disease-free equilibrium point $E^* = I^* = 0$ fulfils

$$R^* = \frac{\nu\gamma_1 V_c N^*}{b(\gamma_1+b)} = \frac{\nu\gamma_1 V_c}{(b-\nu)(\gamma_1+b)} = \gamma_1 \frac{(b-\nu(1-V_c))N^* - b}{(\gamma_1+b)b},$$
$$V^* = \frac{\nu V_c N^*}{\gamma_1+b} = \frac{(b-\nu(1-V_c))N^* - b}{\gamma_1+b},$$
$$S^* = 1 + \frac{\nu N^*(1-V_c)}{b} = 1 + \frac{\nu(1-V_c)}{b-\nu},$$

with $N^* = \frac{b}{b-\nu}$ so that $V^* + R^* = \frac{\nu V_c N^*}{b} = \frac{\nu V_c}{b-\nu}$.

Two particular disease-free equilibrium points are $S^* = N^* = \frac{b}{b-\nu}$, $E^* = I^* = V^* = R^* = 0$ if $V_c = 0$, and $S^* = 1$, $V^* = \frac{\nu N^*}{\gamma_1+b} = \frac{\nu b}{(\gamma_1+b)(b-\nu)}$, $R^* = \frac{\nu\gamma_1}{(\gamma_1+b)(b-\nu)}$, $E^* = I^* = 0$ if $V_c = 1$.

If $\nu \geqslant b$ then there is no disease-free equilibrium point.

Proof. The equilibrium points are calculated by zeroing (1.1), (1.2), (1.4) and (1.5) and making (1.3) identical to a disease-free equilibrium value E^* what leads to:

$$b - \left(b + \frac{\beta I^*}{1+\eta S^*}\right) S^* + \gamma I^* e^{-b\omega} + \nu N^*(1-V_c) = 0, \tag{2.1}$$
$$-\left(\frac{\delta\beta I^*}{1+\eta V^*} + \gamma_1 + b\right) V^* + \nu N^* V_c = 0, \tag{2.2}$$
$$E^* = \frac{\beta}{b}\left(1 - e^{-b\omega}\right)\left(\frac{S^*}{1+\eta S^*} + \frac{\delta V^*}{1+\eta V^*}\right) I^*, \tag{2.3}$$
$$\beta \mathrm{e}^{-b\tau}\left(\frac{S^*}{1+\eta S^*} + \frac{\delta V^*}{1+\eta V^*}\right) I^* - (\gamma + b + \alpha) I^* = 0, \tag{2.4}$$
$$\gamma_1 V^* - bR^* + \gamma\left(1 - e^{-b\omega}\right) I^* = 0. \tag{2.5}$$

Thus, the disease-free equilibrium point satisfies the constraints:

$$E^* = I^* = 0, \tag{2.6}$$

$$b(1 - S^*) + \nu N^*(1 - V_c) = 0 \quad \Rightarrow \quad S^* = 1 + \frac{\nu N^*(1 - V_c)}{b}, \tag{2.7}$$

$$\gamma_1 V^* - bR^* = 0 \quad \Rightarrow \quad V^* = \frac{bR^*}{\gamma_1}, \tag{2.8}$$

$$-(\gamma_1 + b)V^* + \nu N^* V_c = 0 \quad \Rightarrow \quad V^* = \frac{\nu N^* V_c}{\gamma_1 + b} = \frac{bR^*}{\gamma_1}, \tag{2.9}$$

$$N^* = S^* + V^* + R^* = 1 + \frac{\nu N^*(1 - V_c)}{b} + \left(1 + \frac{b}{\gamma_1}\right)R^*, \tag{2.10}$$

$$= 1 + \frac{\nu N^*(1 - V_c)}{b} + \frac{\nu N^* V_c}{b} = \frac{b + \nu N*}{b} \quad \Rightarrow \quad N^* = \frac{b}{b - \nu}, \tag{2.11}$$

provided that $\nu < b$.

The proof follows directly from the above equations.

REMARK 1. Note from (2.4) that the identity $\frac{S^*}{1+\eta S^*} + \frac{\delta V^*}{1+\eta V^*} = \frac{(\gamma+b+\alpha)\mathrm{e}^{b\tau}}{\beta}$ has always to be fulfilled by endemic equilibrium points, if any, but non-necessarily by disease-free equilibrium points for which $I^* = 0$. Note also that if $\gamma_1 = b$ then $R^* = V^* = \frac{\nu V_c N^*}{2b} = \frac{\nu V_c}{2(b-\nu)}$. Note also that if $\nu = 0$, as in the particular case of impulsive-free SVEIRS model obtained from that discussed in Jiang *et al.* (2009) and Song *et al.* (2009) then the disease-free equilibrium satisfies $E^* = V^* = I^* = R^* = 0,\ N^* = S^* = 1$. In such a case, the model can be ran out with population normalized to unity. Note that that the recovered population increases at the equilibrium as the vaccination increases while the susceptible one decreases.

Note that the exposed population at the equilibrium defined by (1.3) can be equivalently described by a differential equation obtained by applying the Leibniz differentiation rule under the integral symbol to yield:

$$\dot{\tilde{E}}(t) = -b\tilde{E}(t) + \beta\left(\frac{S^*}{1 + \eta S^*} + \frac{\delta V^*}{1 + \eta V^*}\right)\left(\tilde{I}(t) - \tilde{I}(t - \omega)\mathrm{e}^{-b\omega}\right). \tag{2.12}$$

Note also from the equalities of Proposition 1 that

$$\frac{S^*}{1 + \eta S^*} + \frac{\delta V^*}{1 + \eta V^*} = K^* := \frac{b - \nu V_c}{(1 + \eta)b - \nu(1 + \eta V_c)} + \frac{\delta \nu b V_c}{(\gamma_1 + b)(b - \nu) + \eta \nu b V_c}. \tag{2.13}$$

Also, since $\max(S^*, V^*) \leqslant N^* = \frac{b}{b-\nu}$, the following relation (2.14) follows irrespective of the vaccination V_c provided that the transmission constant is sufficiently small satisfying $\beta = (\gamma + b + \alpha - \varepsilon_\beta)e^{b\tau}\frac{b(1+\eta)-\nu}{b(1+\delta)} \leqslant (\gamma + b + \alpha)e^{b\tau}\frac{b(1+\eta)-\nu}{b(1+\delta)}$ for some real constant $0 \leqslant \varepsilon_\beta < \gamma + b + \alpha$:

$$\begin{aligned}\frac{S^*}{1+\eta S^*} + \frac{\delta V^*}{1+\eta V^*} &\leqslant \frac{1+\delta}{N^{*-1}+\eta} = \frac{b(1+\delta)}{b(1+\eta)-\nu} \\ &= \frac{(\gamma + b + \alpha - \varepsilon_\beta)\mathrm{e}^{b\tau}}{\beta}. \end{aligned} \tag{2.14}$$

The local stability of the disease-free equilibrium point independent of the sizes of the delays τ and ω is discussed in the sequel for the particular case of sufficiently small β satisfying (2.14) and for the general case (2.13). Also, the local asymptotic stability of the disease-free equilibrium point is guaranteed by that of the linearized incremental system about it. The linearized model about the equilibrium becomes to be defined from (1.1)–(1.2), (2.12) and (1.4)–(1.5) by the state vector $\tilde{x}(t) := (\tilde{S}(t),\ \tilde{V}(t),\ \tilde{E}(t),\ \tilde{I}(t),\ \tilde{R}(t))^T$ which satisfies the differential system:

$$\dot{\tilde{x}}(t) = A_0^*\tilde{x}(t) + A_\tau^*\tilde{x}(t-\tau) + A_\omega^*\tilde{x}(t-\omega); \quad \tilde{x}(0) = \tilde{x}_0, \tag{2.15}$$

where

$$\begin{aligned} A_0^* &= A_{0d}^* + \tilde{A}_0^* \\ &:= \begin{bmatrix} \nu(1-V_c)-b & \nu(1-V_c) & \nu(1-V_c) & \nu(1-V_c) - \frac{\beta S^*}{1+\eta S^*} & \nu(1-V_c) \\ \nu V_c & \nu V_c - (\gamma_1 + b) & \nu V_c & \nu V_c - \frac{\delta\beta V^*}{1+\eta V^*} & \nu V_c \\ 0 & 0 & -b & \beta\left(\frac{S^*}{1+\eta S^*} + \frac{\delta V^*}{1+\eta V^*}\right) & 0 \\ 0 & 0 & 0 & -(\gamma + b + \alpha) & 0 \\ 0 & \gamma_1 & 0 & \gamma & -b \end{bmatrix} \end{aligned} \tag{2.16}$$

$$= \begin{bmatrix} \nu(1-V_c)-b & \nu(1-V_c) & \nu(1-V_c) & \nu(1-V_c) - \frac{\beta(b+\nu(1-V_c)N^*)}{b+\eta(b+\nu(1-V_c)N^*)}\nu(1-V_c) \\ \nu V_c & \nu V_c - (\gamma_1 + b) & \nu V_c & \nu V_c - \frac{\delta\beta\nu V_c N^*}{\gamma_1 + b + \eta\nu V_c N^*}\nu \quad V_c \\ 0 & 0 & -b & (\gamma + b + \alpha - \bar{\varepsilon}_\beta)\mathrm{e}^{b\tau} \quad 0 \\ 0 & 0 & 0 & -(\gamma + b + \alpha) \quad 0 \\ 0 & \gamma_1 & 0 & \gamma \quad -b \end{bmatrix}. \tag{2.17}$$

for sufficiently small transmission constant if (2.14) holds for some positive real constant $\bar{\varepsilon}_\beta > \varepsilon_\beta$ where the diagonal and non-diagonal matrix additive decomposition of A_0^* is given from (2.17) by

$$A_{0d}^* := \mathrm{Diag}(\nu(1-V_c) - b, \nu V_c - (\gamma_1 + b), -b, -(\gamma + b + \alpha), -b). \tag{2.18}$$

$\tilde{A}_0^* := A_0^* - A_{0\mathrm{d}}^*$ obtained from (2.17)–(2.18), so that its off-diagonal part is identical to that of A_{0d}^* while the diagonal is identically zero, and the matrices A_τ^* and A_ω^* are

entry-wise defined by:

$$\begin{array}{ll}(A_\tau^*)_{44} = \gamma + b + \alpha - \bar{\varepsilon}_\beta, & (A_\omega^*)_{14} = \gamma e^{-b\omega}, \\ (A_\omega^*)_{34} = -(\gamma + b + \alpha - \bar{\varepsilon}_\beta)e^{b(\tau-\omega)}, & (A_\omega^*)_{54} = -\gamma e^{-b\omega},\end{array} \tag{2.19}$$

with all the remaining entries being zero. The following inequalities apply for equivalent norms of vectors and square matrices M of dimension or, respectively, order n:

$$n^{-1}\|M\|_2 \leqslant n^{-1/2}\|M\|_\infty \leqslant \|M\|_2 \leqslant n^{1/2}\|M\|_1 \leqslant n\|M\|_2. \tag{2.20}$$

Thus, one gets from the above inequalities (2.20) that

$$\begin{aligned}\|A_\tau^*\|_2 + \|A_\omega^*\|_2 &\leqslant \sqrt{5}(\|A_\tau^*\|_\infty + \|A_\omega^*\|_\infty) \\ &\leqslant \sqrt{5}(\gamma + b + \alpha)\max(1, e^{b(\tau-\omega)}) \leqslant \bar{\gamma}.\end{aligned} \tag{2.21}$$

where

$$\bar{\gamma} = \begin{cases} \sqrt{5}(\gamma + b + \alpha), & \text{if } \tau \leqslant \omega, \\ \sqrt{5}(\gamma + b + \alpha)e^{b(\tau-\omega)}, & \text{if } \tau > \omega. \end{cases} \tag{2.22}$$

Note from (2.22) that $\sqrt{5}(\gamma+b+\alpha)e^{b(\tau-\omega)} \leqslant b-b_0$ for a given b and any given positive real constant $b_0 < b$ if $(\gamma + b + \alpha)$ and $(\tau - \omega)$, if positive, are small enough such that, equivalently,

$$-\infty \leqslant \frac{1}{2}\ln 5 + \ln(\gamma + b + \alpha) + b(\tau - \omega) \leqslant \ln(b - b_0). \tag{2.23}$$

Thus, one gets from (2.21)–(2.23)

$$\|A_\tau^*\|_2 + \|A_\omega^*\|_2 \leqslant \bar{\gamma} \leqslant b - b_0. \tag{2.24}$$

On the other hand, we can use from L'Hopital rule the following limit relations in the entries (1, 4) and (2, 4) of $\tilde{A}_0^*$:

$$\begin{array}{l}\dfrac{\beta(b + \nu(1 - V_c)N^*)}{b + \eta(b + \nu(1 - V_c)N^*)} \to \dfrac{\beta}{1 + \eta}; \\ \dfrac{\delta\beta\nu V_c N^*}{\gamma_1 + b + \eta\nu V_c N^*} \to 0 \quad \text{as } b \to \infty,\end{array} \tag{2.25}$$

if the remaining parameters remain finite and then $N^* = S^* = 1$ and $E^* = I^* = V^* = R^* = 0$ from Proposition 1. By continuity with respect to parameters, for any sufficiently large $M \in \mathbf{R}_+, \exists\varepsilon_{1,2} = \varepsilon_{1,2}(M) \in \mathbf{R}_+$ with $\varepsilon_{1,2} \to 0$ as $t \to \infty$ such that for $b \geqslant M$:

$$\frac{\beta(b + \nu(1 - V_c)N^*)}{b + \eta(b + \nu(1 - V_c)N^*)} \leqslant \frac{\beta + \varepsilon_1}{1 + \eta}; \quad \frac{\delta\beta\nu V_c N^*}{\gamma_1 + b + \eta\nu V_c N^*} \leqslant \varepsilon_2, \tag{2.26}$$

and, one gets for $\tilde{A}_0^*$ being obtained from (2.16)–(2.18),

$$|\tilde{A}_0^*| = \begin{bmatrix} 0 & \nu(1-V_c) & \nu(1-V_c) & |\nu(1-V_c) - \frac{\beta+\varepsilon_1}{1+\eta}| & \nu(1-V_c) \\ \nu V_c & 0 & \nu V_c & |\nu V_c - \varepsilon_2| & \nu V_c \\ 0 & 0 & 0 & (\gamma+b+\alpha-\bar{\varepsilon}_\beta)\mathrm{e}^{b\tau} & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & \gamma_1 & 0 & \gamma & 0 \end{bmatrix}, \tag{2.27}$$

and for the parameter b being large enough such that it satisfies:

$$b \geqslant \max\left(\frac{1}{\tau}\max\left(\ln\frac{\gamma+\gamma_1}{\gamma+b+\alpha}, \ln\frac{4\max(1,\nu)}{\gamma+b+\alpha}\right), b_a\right), \tag{2.28}$$

with b_a being some existing real positive constant, depending on the vaccination constant V_c, such that $\nu(1-V_c) \geqslant \frac{\beta+\varepsilon_1}{1+\eta}$, it follows from inspection of (2.26)–(2.27) that $\|\tilde{A}_0^*\|_\infty \leqslant (\gamma+b+\alpha)\mathrm{e}^{b\tau}$. Using again (2.20)–(2.21), it follows that the following close constraint to (2.23):

$$\begin{aligned} -\infty &\leqslant \frac{1}{2}\ln 5 + \ln(\gamma+b+\alpha) + b(\tau-\omega) \\ &\leqslant \frac{1}{2}\ln 5 + \ln(\gamma+b+\alpha) + b\tau + \ln(1+e^{-b\omega}) \leqslant \ln(b-b_0), \end{aligned} \tag{2.29}$$

guarantees

$$\begin{aligned} &\|A_\tau^*\|_2 + \|A_\omega^*\|_2 + \|\tilde{A}_0^*\|_2 \leqslant \sqrt{5}\big(\|A_\tau^*\|_\infty + \|A_\omega^*\|_\infty + \|\tilde{A}_0^*\|_\infty\big) \\ &\quad\leqslant \sqrt{5}(\gamma+b+\alpha)\big(\max\big(1, e^{b(\tau-\omega)}\big) + e^{b\tau}\big) \leqslant \bar{\gamma}_1, \end{aligned} \tag{2.30}$$

where

$$\bar{\gamma}_1(>\bar{\gamma}) = \begin{cases} \sqrt{5}(\gamma+b+\alpha)(1+\mathrm{e}^{b\tau}), & \text{if } \tau \leqslant \omega, \\ \sqrt{5}(\gamma+b+\alpha)(\mathrm{e}^{b\tau}(1+e^{-b\omega})), & \text{if } \tau > \omega. \end{cases} \tag{2.31}$$

On the other hand, note that the linearized system (2.15)–(2.19) is asymptotically stable if and only if

$$\begin{aligned} &\det(sI - A_{0d}^* - \tilde{A}_0^* - \tilde{A}_\tau^*\mathrm{e}^{-\tau s} - \tilde{A}_\omega^* e^{-\omega s}) \neq 0; \\ &\quad \forall s \in \mathbf{C}_{0+} := \{s \in \mathbf{C}\colon \mathrm{Re}\, s \geqslant 0\}, \end{aligned} \tag{2.32}$$

which is guaranteed under the two conditions below:

(1) $\det(sI - A_{0d}^*) \neq 0, \forall s \in \mathbf{C}_{0+}$, equivalently, A_{0d}^* is a stability matrix
(2) The ℓ_2-matrix measure $\mu_2(A_{0d}^*)$ of (A_{0d}^*) is negative, and, furthermore, the following constraint holds

$$\bar{\gamma}_1 \leqslant b - \max\big(|\gamma_1 - \nu V_c|, \nu(1-V_c)\big),$$

which guarantees the above stability condition 2 via (2.30)–(2.31) if β is sufficiently small to satisfy (2.14) and, furthermore,

$$\begin{aligned}\big\|\tilde{A}_0^*\big\|_2 + \big\|\tilde{A}_\tau^*\big\|_2 + \big\|\tilde{A}_\omega^*\big\|_2 &\leqslant \sqrt{5}\big(\gamma + b + \alpha\big)\big(\max\big(1, e^{b(\tau-\omega)}\big) + e^{b\tau}\big)\\ &\leqslant \bar{\gamma}_1 < \big|\mu_2\big(A_{0d}^*\big)\big| = \frac{1}{2}\big|\lambda_{\max}\big(A_{0d}^* + A_{0d}^{*^T}\big)\big| = \big|\lambda_{\max}\big(A_{0d}^*\big)\big|\\ &= b - \max(|\gamma_1 - \nu V_c|, \nu(1 - V_c)). \end{aligned} \tag{2.33}$$

The following result is proven from Proposition 1, the above asymptotic stability conditions for the linearized incremental system about the disease-free equilibrium point, which implies the local asymptotic stability of the nonlinear one (1.1)–(1.5) about the equilibrium point, and the related former discussion for β being small enough fulfilling (2.14).

PROPOSITION 2. Assume that $\beta \leqslant (\gamma + b + \alpha)e^{b\tau}\frac{b(1+\eta)-\nu}{b(1+\delta)}$. Then it exists a sufficiently large $b > \max(|\gamma_1 - \nu V_c|, \nu(1 - V_c))$ such that the disease-free equilibrium point is locally asymptotically stable for any constant vaccination $V_c \in [0, 1]$ and a sufficiently small amount $(\gamma + b + \alpha)$, a sufficiently small delay τ and a sufficiently small difference delay $(\tau - \omega)$ (this being applicable if $\tau > \omega$) such that (2.33) holds.

An alternative result to Proposition 2 concerned with the asymptotic stability of the linearized SVEIRS model (and then the local asymptotic of that of the nonlinear SVEIR model) around the disease-free equilibrium for sufficiently small delays based on their parameterized quotient is given and proven in Appendix B. The result and its proof are based on an existence theorem of the first destabilizing delay and the use of the Jacobian matrix of the linearized system about the disease-free equilibrium. A more general related result can be obtained from (2.13), rather than from (2.14), without involving any "a priori" constraint on the transmission constant. By using (2.13), the following changes appear in the parameterization (2.16)–(2.19) of the linearized system about the disease free equilibrium with the auxiliary real constant K^* being defined in (2.13):

$$(\tilde{A}_{0d}^*)_{34} = \beta K^*, \qquad (A_\tau^*)_{44} = \beta \mathrm{e}^{-b\tau} K^*, \qquad (A_\omega^*)_{34} = -\beta \mathrm{e}^{-b\omega} K^*. \tag{2.34}$$

The basic relation (2.30) used for stability independent of the delays in Proposition 2 becomes accordingly modified as follows:

$$\begin{aligned}\big\|A_\tau^*\big\|_2 + \big\|A_\omega^*\big\|_2 + \big\|\tilde{A}_0^*\big\|_2 &\leqslant \sqrt{5}\big(\big\|A_\tau^*\big\|_\infty + \big\|A_\omega^*\big\|_\infty + \big\|\tilde{A}_0^*\big\|_\infty\big)\\ &\leqslant \sqrt{5}\big(\beta\big(1 + \mathrm{e}^{-b\tau}\big)K^* + e^{-b\omega}\max\big(\gamma, \beta K^*\big)\big) \qquad (2.35a)\\ &\leqslant \sqrt{5}\left(\frac{2\beta}{1+\eta} + \max\left(\gamma, \frac{\beta}{1+\eta}\right)\right) \leqslant \bar{\gamma}_1 \quad \text{as } b \to \infty, \qquad (2.35b)\end{aligned}$$

where (2.35a) holds for any positive parameter b and (2.35b) holds as such a parameter tends to infinity and also for a sufficiently large parameter b since $K^* \to \frac{1}{1+\eta} < 1$ as

$b \to \infty$ from (2.13). Thus, for a sufficiently large $b_M \in \mathbf{R}_+$ and $b \geqslant b_M$, $\bar{\gamma}_1$ may be taken as follows:

$$\bar{\gamma}_1 = \sqrt{5}\max\left(\frac{2\beta}{1+\eta} + \gamma, \frac{3\beta}{1+\eta}\right), \tag{2.36}$$

and the former stability sufficient condition (2.33), derived from (2.14), is modified as follows for the general case from (2.13):

$$\begin{aligned} &\left\|\tilde{A}_0^*\right\|_2 + \left\|\tilde{A}_\tau^*\right\|_2 + \left\|\tilde{A}_\omega^*\right\|_2 \leqslant \sqrt{5}\left(\frac{2\beta}{1+\eta} + \max\left(\gamma, \frac{\beta}{1+\eta}\right)\right) \leqslant \bar{\gamma}_1 \\ &\quad < b - \max(|\gamma_1 - \nu V_c|, \nu(1-V_c)). \end{aligned} \tag{2.37}$$

PROPOSITION 3. Assume that $b > \max(b_M,\ \max(|\gamma_1 - \nu V_c|,\ \nu(1-V_c)))$ and (2.37) holds. Then it exists a sufficiently large $b_M \in \mathbf{R}_+$ such that the disease-free equilibrium point is locally asymptotically stable for any constant vaccination $V_c \in [0,1]$ such that (2.37) holds.

Note that the statement of Propositions 2–3 guarantee the local stability of the disease-free equilibrium point under its existence condition of Proposition 1 requiring $\nu < b$.

3. The Existence of Endemic Equilibrium Points and Some Characterizations

The existence of endemic equilibrium points which keep alive the disease propagation is now discussed:

PROPOSITION 4. Assume that $\omega > 0$. Then, the following properties hold:

(i) Assume $\beta \geqslant \frac{\eta e^{b\tau}(\gamma+b+\alpha)}{1+\delta}$ for $V_c > 0$ and $\beta \geqslant \eta e^{b\tau}(\gamma+b+\alpha)$ for $V_c = 0$. It exists at least one endemic equilibrium point at which the susceptible, vaccinated, infected, exposed and recovered populations are positive and the vaccinated population is zero if and only if $V_c = 0$ (i.e., in the absence of vaccination action). Furthermore, such an equilibrium point satisfies the constraints:

$$\begin{aligned} &E^* = \frac{\beta}{b}\left(1 - e^{-b\omega}\right)\left(\frac{S^*}{1+\eta S^*} + \frac{\delta V^*}{1+\eta V^*}\right) I^* > 0, \\ &\min\left(S^* + \delta V^*, \frac{1+\delta}{\eta}\right) \geqslant \frac{S^*}{1+\eta S^*} + \frac{\delta V^*}{1+\eta V^*} = \frac{e^{b\tau}(\gamma+b+\alpha)}{\beta} > 0, \\ &R^* = \frac{\gamma_1 V^* + \gamma(1-e^{-b\omega})I^*}{b} \geqslant \frac{\gamma(1-e^{-b\omega})I^*}{b} > 0. \end{aligned}$$

(ii) If the transmission constant is small enough satisfying $\beta < \bar{\beta} := \frac{\eta e^{b\tau}(\gamma+b+\alpha)}{1+\delta}$ for $V_c > 0$ and $\beta < \eta e^{b\tau}(\gamma+b+\alpha)$ for $V_c = 0$ then there is no reachable endemic equilibrium point.

Proof. The endemic equilibrium point is calculated as follows:

$$\beta \mathrm{e}^{-b\tau}\left(\frac{S^*}{1+\eta S^*}+\frac{\delta V^*}{1+\eta V^*}\right)-(\gamma+b+\alpha)=0, \tag{3.1}$$

$$E^*=\frac{\beta}{b}\big(1-e^{-b\omega}\big)\left(\frac{S^*}{1+\eta S^*}+\frac{\delta V^*}{1+\eta V^*}\right)I^*>0 \tag{3.2}$$

with

$$E^*>0, \qquad I^*>0, \tag{3.3}$$

$$\frac{S^*}{1+\eta S^*}+\frac{\delta V^*}{1+\eta V^*}=\frac{e^{b\tau}(\gamma+b+\alpha)}{\beta}>0 \tag{3.4}$$

(since, otherwise, the above disease-free equilibrium point would be being considered).

$S*>0$ since, otherwise, the following contradiction would follow:

$$0<b+\gamma I^* e^{-b\omega}+\nu N^*(1-V_c)=0, \tag{3.5}$$

$V*=0$ if and only if $V_c=0$, since otherwise for $V_c>0$ and $V*=0$, it would follow that $\nu N^* V_c=0$ which is only possible in the disease-free equilibrium point if the total population is extinguished what is a contradiction at the endemic point.

$$R^*=\frac{\gamma_1 V^*+\gamma(1-e^{-b\omega})I^*}{b}\geqslant\frac{\gamma(1-e^{-b\omega})I^*}{b}>0 \quad \text{for } \omega\neq 0. \tag{3.6}$$

Property (i) has been proven. Property (ii) follows from the fact that the second separate condition for the endemic equilibrium point in Property (i) fails if

$$\frac{1}{1+\eta}<\frac{e^{b\tau}(\gamma+b+\alpha)}{\beta} \quad \text{for } V_c=0,$$

$$\frac{1+\delta}{1+\eta}<\frac{e^{b\tau}(\gamma+b+\alpha)}{\beta} \quad \text{for } V_c>0,$$

since $S*=V*=0$ is impossible at the endemic equilibrium point from such a second condition of Property (i). Hence, the proof of Property (ii).

REMARK 2. Note that if $\omega=0$ then it follows from (1.3) and (2.3) that $E(t)=E^*=0$; $\forall t\in\mathbf{R}_{0+}$ so that the SVEIRS model (1.1)–(1.5) becomes a simpler SVIRS one without specification of the exposed population dynamics.

REMARK 3. Note that, under the constraints in Proposition 4 (ii) for $\alpha_S^{-1}+\alpha_V^{-1}+\alpha_E^{-1}+\alpha_I^{-1}+\alpha_R^{-1}=1$, if there is no reachable endemic equilibrium point because $\beta<\bar{\beta}$ then the solution trajectory of (1.1)–(1.5) can only either converge to the disease-free equilibrium point provided that it is at least locally asymptotically stable or to be bounded converging or not to an oscillatory solution or to diverge to an unbounded total population

depending on the values of the parameterization of the model (1.1)–(1.5). Note that the endemic free transmission constant upper-bound $\bar{\beta}$ increases as η, τ and $(\gamma + b + \alpha)$ increase and also as δ decreases.

If $V_c > 0$ then it follows from Proposition 4 that there exist positive constants $\alpha_S, \alpha_V, \alpha_E, \alpha_I$ and α_R satisfying $\alpha_S^{-1} + \alpha_V^{-1} + \alpha_E^{-1} + \alpha_I^{-1} + \alpha_R^{-1} = 1$ such that the endemic equilibrium points, if any, satisfy the constraints:

$$N^* = \alpha_S S^* = \alpha_V V^* = \alpha_{\mathrm{E}} E^* = \alpha_{\mathrm{I}} I^* = \alpha_{\mathrm{R}} R^*, \tag{3.7}$$

so that, one gets from (3.6) that

$$R^* = \frac{\gamma_1/\alpha_V + \gamma(1 - e^{-b\omega})/\alpha_I}{b} \alpha_R R^* = \frac{\gamma_1 \alpha_I + \gamma(1 - e^{-b\omega})\alpha_V}{b\alpha_I \alpha_V} \alpha_R R^*, \tag{3.8}$$

$$\begin{aligned} \frac{\beta}{b}\big(1 - e^{-b\omega}\big)\frac{1+\delta}{1+\eta} &\leqslant E^*/I^* = \alpha_I/\alpha_E \\ &= \frac{\beta}{b}\big(1 - e^{-b\omega}\big)\left(\frac{S^*}{1+\eta S^*} + \frac{\delta V^*}{1+\eta V^*}\right) \\ &\leqslant \frac{\beta}{b}\big(1 - e^{-b\omega}\big)\frac{1+\delta}{\eta}. \end{aligned} \tag{3.9}$$

if $\min(S^*, V^*) \geqslant 1$, otherwise, only the upper-bounding constraint holds strictly in (3.9). Moreover, (2.1) and (3.1) may be equivalently written, respectively, as

$$b - \left(b + \frac{\beta\alpha_S S^*}{\alpha_I(1+\eta S^*)}\right)S^* + \gamma\frac{\alpha_S}{\alpha_I} S^* e^{-b\omega} + \nu\alpha_S S^*(1 - V_c) = 0, \tag{3.10}$$

$$\frac{\alpha_V V^*}{\alpha_S + \alpha_V \eta V^*} + \frac{\delta V^*}{1+\eta V^*} = \frac{e^{b\tau}(\gamma + b + \alpha)}{\beta}. \tag{3.11}$$

Equation (3.8) is equivalent, since $R* > 0$ at the endemic equilibrium point, to

$$\frac{\gamma_1 \alpha_I \alpha_R + \gamma(1 - e^{-b\omega})\alpha_V \alpha_R}{b\alpha_I \alpha_V} = 1. \tag{3.12}$$

Equation (3.10) is equivalent to

$$\begin{aligned} &\big[\alpha_S \eta\big(\nu\alpha_I(1 - V_c) + \gamma e^{-b\omega}\big) + \beta\alpha_S - b\alpha_I\eta\big]{S^*}^2 \\ &\quad + \big[\alpha_S\big(\gamma e^{-b\omega} + \nu\alpha_I(1 - V_c)\big) + b\alpha_I(\eta - 1)\big]S^* + b\alpha_I = 0. \end{aligned} \tag{3.13}$$

Equation (3.13) is an algebraic equation of real coefficients of the form $a{S^*}^2 + dS^* + c = 0$ with $c > 0$. Such an equation has two positive real roots if $a > 0,\ d < 0$ and $d^2 \geqslant 4ac$ and one positive real root if $a < 0$ and $d > 0$. Thus, since there is a nonzero susceptible

population at an endemic equilibrium point then either (3.14)–(3.16) below hold:

$$\alpha_S\eta\big(\nu\alpha_I(1-V_c)+\gamma e^{-b\omega}\big)+\beta\alpha_S > b\alpha_I\eta, \tag{3.14}$$

$$\alpha_S\big(\gamma e^{-b\omega}+\nu\alpha_I(1-V_c)\big) < b\alpha_I(1-\eta) \quad \text{provided that } \eta<1, \tag{3.15}$$

$$\begin{aligned}&\big[\alpha_S\big(\gamma e^{-b\omega}+\nu\alpha_I(1-V_c)\big)+b\alpha_I(\eta-1)\big]^2\\ &\quad\geqslant 4b\alpha_I\big[\alpha_S\eta\big(\nu\alpha_I(1-V_c)+\gamma e^{-b\omega}\big)+\beta\alpha_S-b\alpha_I\eta\big],\end{aligned} \tag{3.16}$$

or, alternatively,

$$\begin{aligned}\beta &< \frac{\alpha_I}{\alpha_S}b\eta-\big(\nu\alpha_I(1-V_c)+\gamma e^{-b\omega}\big)\eta\\ &= \frac{\eta}{I^*}\big[bS^*-\big(\nu N^*(1-V_c)+\gamma e^{-b\omega}\big)\big],\end{aligned} \tag{3.17}$$

and

$$b<\frac{\alpha_S(\gamma e^{-b\omega}+\nu\alpha_I(1-V_c))}{\alpha_I(1-\eta)}=\frac{\gamma e^{-b\omega}I^*+\nu N^*(1-V_c)}{S^*(1-\eta)}, \tag{3.18}$$

with $\eta<1$ hold. On the other hand, (3.11) is equivalent to

$$\begin{aligned}&\alpha_V\beta_0\big(1+\eta V^*\big)V^*+\delta\beta_0V^*\big(\alpha_S+\eta\alpha_VV^*\big)\\ &\quad=\big(1+\eta V^*\big)(\alpha_S+\eta\alpha_VV^*\big),\end{aligned} \tag{3.19}$$

where $\beta_0:=\frac{\beta}{e^{b\tau}(\gamma+b+\alpha)}$ so that (3.19) is of the form

$$\begin{aligned}&aV^{*^2}+dV^*+c\equiv\eta\big(\eta-(1+\delta)\beta_0\big)\alpha_VV^{*^2}\\ &\quad+\big(\alpha_V(\eta-\beta_0)+(\eta-\delta\beta_0)\alpha_S\big)V^*+\alpha_S=0.\end{aligned} \tag{3.20}$$

Now, a close reasoning to that used for the susceptible endemic equilibrium component is applied to (3.20) to construct the subsequent reasoning for a potential nonzero vaccinated population at most two possibly existing endemic equilibrium points. Note that either

$$\alpha_V\eta\big(\eta-(1+\delta)\beta_0\big)>0 \iff \beta_0<\frac{\eta}{1+\delta}, \tag{3.21}$$

$$\begin{aligned}&\alpha_V(\eta-\beta_0)+\alpha_S(\eta-\delta\beta_0)=\eta(\alpha_V+\alpha_s)-\beta_0(\alpha_V+\delta\alpha_S)<0\\ &\quad\iff\left(\beta_0>\eta\frac{\alpha_V+\alpha_S}{\alpha_V+\delta\alpha_S}\iff\beta>\eta\frac{\alpha_V+\alpha_S}{\alpha_V+\delta\alpha_S}(\gamma+b+\alpha)e^{b\tau}\right),\end{aligned} \tag{3.22}$$

and

$$\big(\alpha_V(\eta-\beta_0)+(\eta-\delta\beta_0)\alpha_s\big)^2>4\big(\eta-(1+\delta)\beta_0\big)\eta\alpha_V\alpha_S, \tag{3.23}$$

or, alternatively,

$$\alpha_V\eta\big(\eta-(1+\delta)\beta_0\big)<0 \iff \beta_0>\frac{\eta}{1+\delta}, \tag{3.24}$$

$$\alpha_V(\eta-\beta_0)+\alpha_S(\eta-\delta\beta_0)>0$$
$$\iff \left(\beta_0<\eta\frac{\alpha_V+\alpha_S}{\alpha_V+\delta\alpha_S} \iff \beta<\eta\frac{\alpha_V+\alpha_S}{\alpha_V+\delta\alpha_S}(\gamma+b+\alpha)e^{b\tau}\right). \tag{3.25}$$

However, note that (3.21)–(3.23) imply that

$$\eta\frac{\alpha_V+\alpha_S}{\alpha_V+\delta\alpha_S}<\beta_0=\frac{\beta}{e^{b\tau}(\gamma+b+\alpha)}<\frac{\eta}{1+\delta},$$

which is well-posed if and only if $\delta<-\alpha_S/\alpha_V<0$ which contradicts the positivity of the parameter δ. As a result, only the alternative constraints (3.24)–(3.25) need to be considered with a non-zero vaccinated population at the endemic equilibrium point which is always the case under a nonzero regular constant vaccination $V_c\leqslant 1$.

The above discussion concerning the endemic equilibrium point is summarized as follows:

PROPOSITION 5. Assume that $V_c\in(0,1]$ and that $\beta\geqslant(\gamma+b+\alpha)e^{b\tau}\frac{\eta}{1+\delta}$ so that $N^*=\alpha_S S^*=\alpha_V V^*=\alpha_{\mathrm{E}}E^*=\alpha_{\mathrm{I}}I^*=\alpha_{\mathrm{R}}R^*$ for some positive constants $\alpha_S,\alpha_V,\alpha_E,\alpha_I$ and α_R. Then, it exists at least one endemic equilibrium point, and at most two endemic equilibrium points, with all the corresponding partial populations being positive and the following parametrical constraints hold:

$$\alpha_S^{-1}+\alpha_V^{-1}+\alpha_E^{-1}+\alpha_I^{-1}+\alpha_R^{-1}=1, \quad \alpha_I/\alpha_E\leqslant\frac{\beta}{b}\big(1-e^{-b\omega}\big)\frac{1+\delta}{\eta}.$$

Also, the constants α_S,α_I and α_V satisfy either (3.14)–(3.16), or (3.17)–(3.18), and (3.24)–(3.25).

REMARK 4. Note that if $\min(S^*,V^*)\geqslant 1$ then

$$(\gamma+b+\alpha)e^{b\tau}\frac{\eta}{1+\delta}\leqslant\beta\leqslant(\gamma+b+\alpha)e^{b\tau}\frac{1+\eta}{1+\delta}. \tag{3.26}$$

This implies that the coefficient a in (3.20) is non-positive. If $a=0$ then

$$V^*=\frac{\alpha_s}{|\alpha_V(\eta-\beta_0)+(\eta-\delta\beta_0)\alpha_s|}>0,$$

if $\alpha_V(\eta-\beta_0)+(\eta-\delta\beta_0)\alpha_s<0$. This implies that $\beta_0>\eta\frac{\alpha_V+\alpha_S}{\alpha_V+\delta\alpha_S}$ which is compatible with (3.26) if

$$\beta\geqslant(\gamma+b+\alpha)e^{b\tau}\eta\max\left(\frac{1}{1+\delta},\frac{\alpha_V+\alpha_S}{\alpha_V+\delta\alpha_S}\right), \tag{3.27}$$

and $\eta \leqslant \frac{\alpha_V+\alpha_S}{\alpha_V+\delta\alpha_S}$ so that $\eta\frac{\alpha_V+\alpha_S}{\alpha_V+\delta\alpha_S} \leqslant \frac{1+\eta}{1+\delta}$. On the other hand, if $a < 0$ then $\beta < \eta\frac{\alpha_V+\alpha_S}{\alpha_V+\delta\alpha_S}e^{b\tau}(\gamma+b+\alpha)$ from (3.25) which is coherent with (3.26) if

$$\beta \leqslant (\gamma+b+\alpha)e^{b\tau}\min\left(\frac{1+\eta}{1+\delta}, \eta\frac{\alpha_V+\alpha_S}{\alpha_V+\delta\alpha_S}\right), \tag{3.28}$$

since $\frac{1}{1+\delta} \leqslant \frac{\alpha_V+\alpha_S}{\alpha_V+\delta\alpha_S}$ for any $\delta > 0,\ \min(\alpha_V, \alpha_S) > 1$.

The existence of a unique endemic equilibrium point under zero vaccination is dealt with in Appendix C.

4. About Infection Propagation, the Uniform Boundedness of the Total Population and the Positivity of the Partial Populations

This section discuses briefly the monotone increase of the infected population and the boundedness of the total population as well as the positivity of the model:

PROPOSITION 6. If the infection propagates through $(t-\tau, t)$ with the infected population being non-decreasing then

$$\frac{S(\sigma)}{1+\eta S(\sigma)} + \frac{\delta V(\sigma)}{1+\eta V(\sigma)} \geqslant \frac{\gamma+b+\alpha}{\beta}e^{b\sigma}; \quad \forall\sigma \in (t^*-2\tau, t^*-\tau).$$

Proof. Note from (1.4) that for $t \in (t^*-2\tau, t^*)$

$$\dot{I}(t) > 0 \iff \frac{I(t)}{I(t-\tau)} < \frac{\beta e^{-b\tau}}{\gamma+b+\alpha}\left(\frac{S(t-\tau)}{1+\eta S(t-\tau)} + \frac{\delta V(t-\tau)}{1+\eta V(t-\tau)}\right),$$

and if, furthermore, $I(t) \geqslant I(t-\tau)$ for $t \in (t^*-\tau, t^*)$, thus

$$1 \leqslant \frac{I(t)}{I(t-\tau)} < \frac{\beta e^{-b\tau}}{\gamma+b+\alpha}\left(\frac{S(t-\tau)}{1+\eta S(t-\tau)} + \frac{\delta V(t-\tau)}{1+\eta V(t-\tau)}\right).$$

Now, rewrite (1.3) in differential equivalent form by using Leibniz's rule as follows:

$$\begin{aligned}\dot{E}(t) = {} & -bE(t) + \beta\left[\left(\frac{S(t)I(t)}{1+\eta S(t)} + \frac{\delta V(t)I(t)}{1+\eta V(t)}\right)\right. \\ & \left. - \left(\frac{S(t-\omega)I(t-\omega)}{1+\eta S(t-\omega)} + \frac{\delta V(t-\omega)I(t-\omega)}{1+\eta V(t-\omega)}\right)\mathrm{e}^{-b\omega}\right].\end{aligned} \tag{4.1}$$

PROPOSITION 7. Assume that $\nu < b$. Then, the following properties hold provided that the SVEIR epidemic model (1.1)–(1.5) has non-negative solution trajectories of all the partial populations for all time:

(i) Assume furthermore that $\psi := (e^{\nu\tau} + \frac{\beta(1+\delta)(1-e^{-(b-\nu)\tau})}{\eta(b-\nu)})\mathrm{e}^{-b\tau} < 1$. Then, the total population is uniformly bounded for all time, irrespective of the susceptible and vaccinated populations, for any bounded initial conditions and $\limsup_{t\to\infty} N(t) \leqslant \frac{1-e^{-(b-\nu)\tau}}{b-\nu}(1-\psi)^{-1} < \infty$.

(ii) Assume that the transmission constant is large enough satisfying $\beta \geqslant \frac{1}{1+\delta}\cdot \sup_{t\in\mathbf{R}_{0+}}(\frac{b\eta(1+\eta)}{\eta\mathrm{e}^{-b\omega}I(t-\omega)-(1+\eta)e^{-b\tau}I(t-\tau)})$ subject to $\frac{\eta}{1+\eta} > e^{b(\omega-\tau)}$ and $\omega < \tau$. Then $N\colon \mathbf{R}_{0+} \to \mathbf{R}_{0+}$ is monotone decreasing and of negative exponential order so that the total population exponentially extinguishes as a result.

Proof. Consider the SVEIRS model in differential form described by (1.1), (1.2), (1.4), (1.5) and (4.1). Summing up the five equations, one gets directly:

$$\begin{aligned}\dot{N}(t) &= (\nu-b)N(t) + b - \alpha I(t)\\ &\quad + \beta\left[\left(\frac{S(t-\tau)I(t-\tau)}{1+\eta S(t-\tau)} + \frac{\delta V(t-\tau)I(t-\tau)}{1+\eta V(t-\tau)}\right)e^{-b\tau}\right.\\ &\quad \left. - \left(\frac{S(t-\omega)I(t-\omega)}{1+\eta S(t-\omega)} + \frac{\delta V(t-\omega)I(t-\omega)}{1+\eta V(t-\omega)}\right)\mathrm{e}^{-b\omega}\right]. \qquad (4.2)\\ &\leqslant (\nu-b)N(t) + b + \beta\left(\frac{S(t-\tau)I(t-\tau)}{1+\eta S(t-\tau)} + \frac{\delta V(t-\tau)I(t-\tau)}{1+\eta V(t-\tau)}\right)e^{-b\tau}\\ &\leqslant (\nu-b)N(t) + b + \beta\frac{1+\delta}{\eta}e^{-b\tau}I(t-\tau)\\ &\leqslant (\nu-b)N(t) + b + \beta\frac{1+\delta}{\eta}e^{-b\tau}N(t-\tau). \qquad (4.3)\end{aligned}$$

since $\frac{S(t)}{1+\eta S(t)} + \frac{\delta V(t)}{1+\eta V(t)} \leqslant \frac{1+\delta}{\eta}$; $\forall t \in \mathbf{R}_{0+}$. Then, $N(t) \leqslant \psi \sup_{t-\tau\leqslant\sigma\leqslant t} N(\sigma) + \frac{b(1-e^{-(b-\nu)\tau})}{b-\nu} < \infty; \forall t \in \mathbf{R}_{0+.}$, and Property (i) follows since $\psi < 1$. Two cases are now discussed separately concerning the proof of Property (ii):

(a) Note that if the solution trajectory is positive subject to $\min(S(t),\ V(t)) \geqslant 1$ (equivalently if $\max(S^{-1}(t),\ V^{-1}(t)) \leqslant 1$) then

$$0 < \frac{1+\delta}{1+\eta} \leqslant \frac{S(t)}{1+\eta S(t)} + \frac{\delta V(t)}{1+\eta V(t)} \leqslant \frac{1+\delta}{\eta}, \qquad (4.4)$$

so that one gets from (4.2):

$$\begin{aligned}\dot{N}(t) &\leqslant (\nu-b)N(t) - \alpha I(t)\\ &\quad + \left(b - \beta\left[\frac{1+\delta}{1+\eta}I(t-\omega)\mathrm{e}^{-b\omega} - \frac{1+\delta}{\eta}I(t-\tau)e^{-b\tau}\right]\right)\\ &\leqslant -(b-\nu)N(t) - \alpha I(t) \leqslant -(b-\nu)N(t) < 0. \qquad (4.5)\end{aligned}$$

if $N(t) > 0$ since $b > \nu$ and $N(t) = 0$ if and only if $N(t) = I(t) = 0$ since $\beta \geqslant \frac{1}{1+\delta}(\frac{b\eta(1+\eta)}{\eta e^{-b\omega}I(t-\omega)-(1+\eta)I(t-\tau)e^{-b\tau}}) > 0$ provided that $\frac{\eta}{1+\eta} > e^{b(\omega-\tau)}$ with $\omega < \tau$. Then, $N(t) \leqslant e^{-(b-\nu)t}N(0) < N(t'); \forall t, t'(< t) \in \mathbf{R}_{0+}$.

(b) If $\max(S(t), V(t)) \leqslant 1$ (equivalently, if $\min(S^{-1}(t), V^{-1}(t)) \geqslant 1$) then

$$0 \leqslant \frac{S(t)}{1+\eta S(t)} + \frac{\delta V(t)}{1+\eta V(t)} \leqslant \frac{1+\delta}{1+\eta} \leqslant \frac{1+\delta}{\eta}.$$

so that (4.5) still holds and the same conclusion arises. Thus, Property (ii) is proven.

A brief discussion about positivity is summarized in the next result:

PROPOSITION 8. Assume that $V_c \in [0, 1]$. Then, the SVEIRS epidemic model (1.1)–(1.5) is positive in the sense that no partial population is negative at any time if its initial conditions are non-negative and the vaccinated population exceeds a certain minimum measurable threshold in the event that the recovered population is zero as follows: $V(t) \geqslant \max(\frac{\gamma}{\gamma_1}(I(t-\omega)e^{-b\omega} - I(t)), 0)$ if $R(t) = 0$. The susceptible, vaccinated, exposed and infected populations are nonnegative for all time irrespective of the above constraint. If, in addition, Proposition 7 (i) holds then all the partial populations of the SVEIRS model are uniformly bounded for all time.

Proof. First note that all the partial populations are defined by continuous-time differentiable functions from (1.1)–(1.5). Then, if any partial population is negative, it is zero at some previous time instant. Assume that $S(\sigma) \geqslant 0$ for $\sigma < t$ and $S(t) = 0$ at some time instant t. Then from (1.1):

$$\dot{S}(t) = b + \gamma I(t-\omega)e^{-b\omega} + \nu(1-V_c)N(t) \geqslant 0; \quad \forall V_c \in [0, 1].$$

Thus, $S(t^+) \geqslant 0$. As a result, S(t) cannot reach negative values at any time instant. Assume that V(σ) $\geqslant 0$ for $\sigma < t$ and V(t)=0 at some time instant t. Then, $\dot{V}(t) = \nu V_c N(t) \geqslant 0$ from (1.2) so that $V(t^+) \geqslant 0$. As a result, V (t) cannot reach negative values at any time. $E(t) \geqslant 0$ for any time instant t from (1.3). Assume that $I(\sigma) \geqslant 0$ for $\sigma < t$ and $I(t) = 0$ at some time instant t. Then, $\dot{I}(t) \geqslant 0$ from (1.4). As a result, I(t) cannot reach negative values at any time. Finally, assume that $R(\sigma) \geqslant 0$ for $\sigma < t$ and $R(t) = 0$ at some time instant t. Thus, $\dot{R}(t) = \gamma_1 V(t) + \gamma(I(t) - I(t-\omega)e^{-b\omega}) \geqslant 0$ from (1.5) if $V(t) \geqslant \max(\frac{\gamma}{\gamma_1}(I(t-\omega)e^{-b\omega} - I(t)), 0)$. Thus, if $V(t) \geqslant \max(\frac{\gamma}{\gamma_1}(I(t-\omega)e^{-b\omega} - I(t)), 0)$ when $R(t) = 0$ then all the partial populations are uniformly bounded, since they are nonnegative and the total population $N(t)$ is uniformly bounded from Proposition 7(i).

5. Simulation Results

This Section contains some simulation examples which are concerned with the existence and alllocation of disease-free and endemic equilibrium points. The objective of these

examples is to numerically show the potential existence of both types of equilibrium points and that the calculated values for their coordinates are given by the presented expressions. The particular values for the equilibrium points as well as the time taken by the model to converge to them depend on the specific choice of the parameter values which correspond to the particular disease under study and the species being considered in the epidemic model. For a different parameterization, these values would be different. Also, simulations have been run for a long time interval in order to show that the reached steady-state values are true equilibrium points. The parameters of the epidemic model are: $b = 0.05$, $\gamma = 0.005$, $1/\gamma_1 = 15$ days, $\beta_1 = \beta/2$, $\delta = \beta_1/\beta$, $\tau = 6$ days, $\eta = 0.5$, $\alpha = 0.005$ and $\omega = 10$ days. It can be pointed that in the case that the parameters for a particular epidemic model are unknown they can be estimated from the analysis of population data by using, for instance, either statistical methods, see for instance, Zutautaite-Seputiene *et al.* (2010), Bougeard *et al.* (2011), or heuristic methods, as for instance in Dzemyda and Sakalauskas (2011), or adaptive methods by using either batch or recursive parametrical estimation algorithms. See, for instance, Ibeas and De la Sen (2004), Pupeikis(2010) being updated from collected real measured data on the partial populations through time.

5.1. *Disease-Free Equilibrium Point*

Consider now $\beta = 0.0166$ and $\nu = 0.2b$. The two particular cases corresponding to $V_c = 0$ and $V_c = 1$ in Section 2 will be studied separately. Thus, the following simulations have been obtained for the SVEIR system (1.1)–(1.5) and $V_c = 0$.

Figure 2 shows a zoom on the equilibrium point reached by the model in Fig. 1.

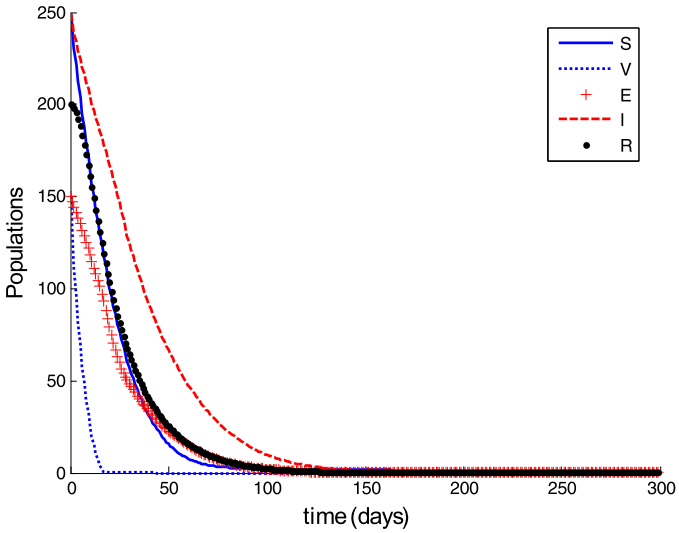

Fig. 1. Solution trajectories for $V_c = 0$.

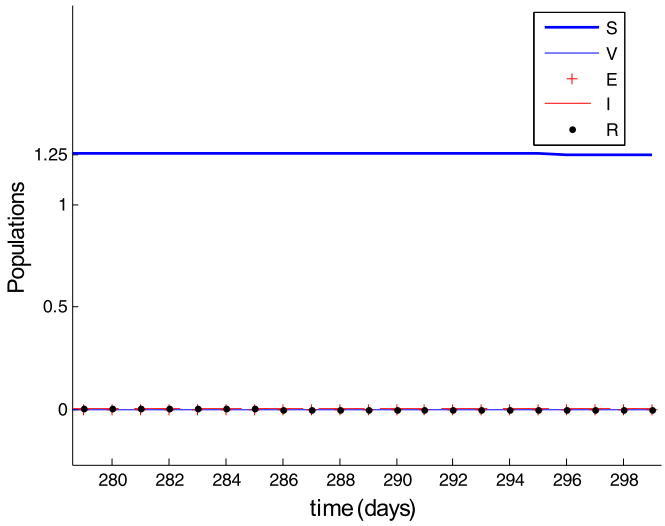

Fig. 2. Zoom on the solution trajectories for the disease-free equilibrium point for $V_c = 0$.

Note from Figs. 1–2 that the vaccinated, exposed, infected and removed-by-immunity(recovered) populations converge to zero. This situation corresponds to the case when the disease is naturally eradicated from the population. On the other hand, the susceptible presents a different dynamics, converging to a non-zero equilibrium point. Figure 2 shows that the vaccinated, exposed, infectious and recovered populations are actually zero (which is represented by the superimposed graphics) while the susceptible converges to 1.25. Furthermore, these values correspond to the ones stated in Proposition 1 for $V_c = 0$, since all the populations vanish except the susceptible which converges to $S^* = \frac{b}{b-\nu} = 1.25$. If $V_c = 1$ then the solution trajectories converge to the equilibrium point as depicted in Fig. 3.

In this case, only the exposed and infected tend to zero while the remaining populations tend to the values calculated in Proposition 1 when $V_c = 1$:

$$S^* = 1, V^* = \frac{\nu b}{(\gamma_1 + b)(b - \nu)} = 0.107,$$
$$R^* = \frac{\nu\gamma_1}{(\gamma_1 + b)(b - \nu)} = 0.14, \qquad E^* = I^* = 0.$$

5.2. *Endemic Equilibrium Point*

In order to study the endemic equilibrium point, the value of β is changed now to a new value $\beta = 0.099$ satisfying the condition $\beta \geqslant \eta e^{b\tau}(\gamma + b + \alpha)$ stated in Proposition 4(i) for $V_c = 0$ and $\nu = 0.65b$. Thus, the model trajectory solutions are depicted in Fig. 4:

A zoom on Fig. 4 will show the equilibrium point of the system as represented in Fig. 5.

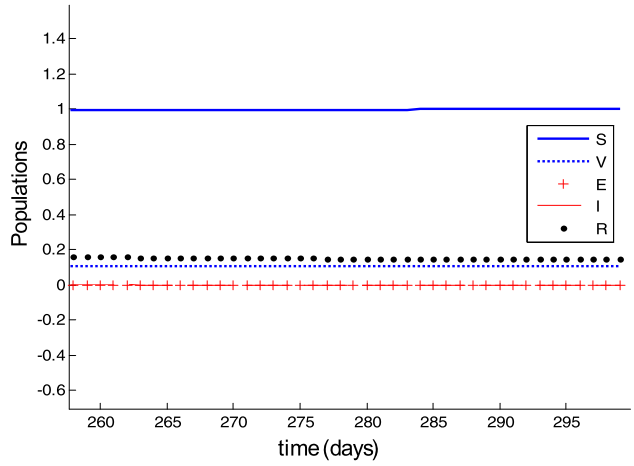

Fig. 3. Disease-free equilibrium point for $V_c = 1$.

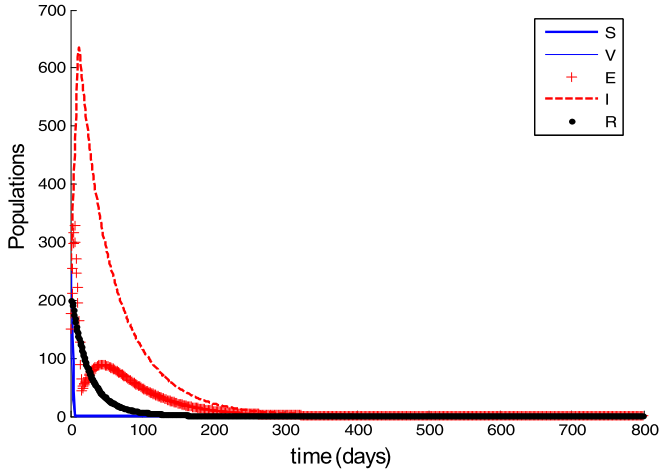

Fig. 4. Solution trajectories converging to an endemic equilibrium point.

Figure 5 shows that there is an endemic equilibrium point, associated to non-zero populations of exposed and infectious, whose coordinates in view of Proposition 4(i) satisfy the constraints:

$$E^* = \frac{\beta}{b}(1 - \mathrm{e}^{-b\omega})(\frac{S^*}{1+\eta S^*} + \frac{\delta V^*}{1+\eta V^*})I^* = 0.7,$$
$$R^* = \frac{\gamma_1 V^* + \gamma(1 - \mathrm{e}^{-b\omega})I^*}{b} = 0.043.$$

while the remaining variables, which are not given explicitly in Proposition 4(i), are $I^* = 1.1$, $V^* = 0$ and $S^* = 1.38$. Thus, the validity of the results is numerically verified.

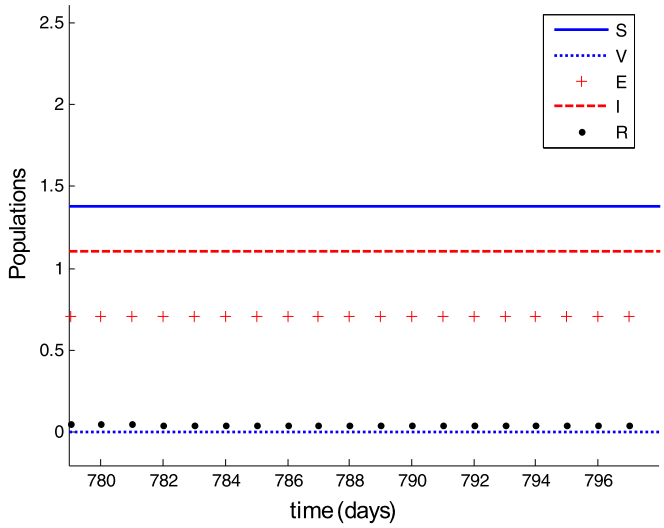

Fig. 5. Zoom of the solution trajectories showing the endemic equilibrium point.

5.3. *The Epidemic Model Versus the Evolution of Fractional Partial Populations*

It is interesting to discuss the practical use of the model with fractional or percentage of populations with respect to a total population by making the model to be more versatile. Such fractions of the partial total populations can be taken, for instance, with respect to the initial total population of the habitat under study or with respect to that of the disease-free equilibrium. Note that in time-varying models or even in time-invariant ones with external interchange of population, newborn vaccination strategy or mortality associated with the disease, it can happen that overshoots and undershoots with respect to the unit Heaviside function of some of the total population evolution through time can occur. The reason is that the total population is not necessarily constant. The percentages of the partial populations can be manipulated by using initial conditions in the model which are themselves percentages or by using absolute values of populations and then displaying the percentage evolution of the populations through time. A numerical simulation is made with initial conditions $S(0) = 25,\ V(0) = 15,\ E(0) = 15,\ I(0) = 25,\ R(0) = 20.$ The parameters of the model are $b = 0.075$ days $^{-1}$, $\nu = 0.995b < b$ and $N(0) = N^* = S^* = \frac{b}{b-\nu} = 200$. The results are depicted in Figs. 6 and 7 for $V_c = 0$ and $V_c = 1$, respectively. Figure 8 displays a zoom of the final evolution of the vaccinated and recovered populations towards the disease-free equilibrium for $V_c = 1$.

6. Concluding Remarks and Several Recommendations

This paper has investigated the disease-free and endemic equilibrium points of a modified epidemic SVEIRS model with the five typical populations of susceptible, vaccinated,

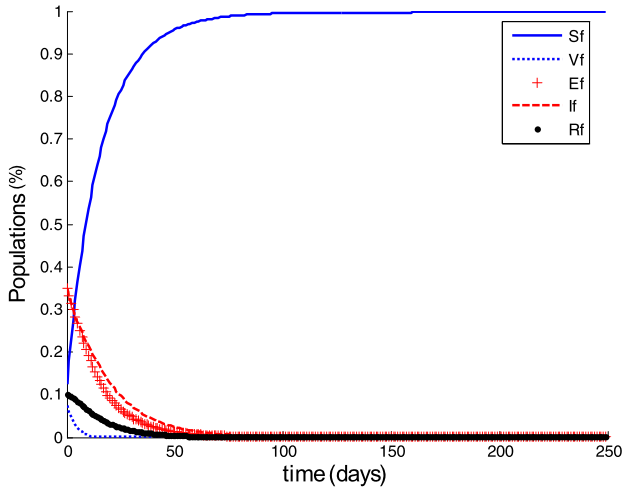

Fig. 6. Percentages of populations evolving to the disease-free equilibrium point for $V_c = 0$.

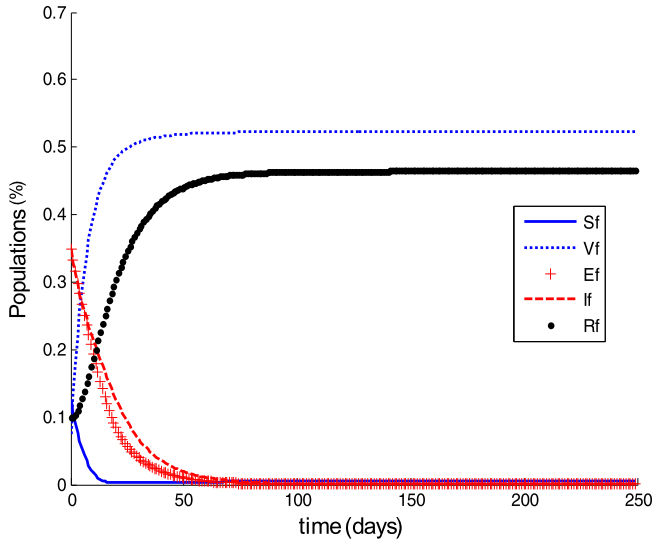

Fig. 7. Percentages of populations evolving to the disease-free equilibrium point for $V_c = 1$.

exposed, infected and recovered populations. The model is of true-mass action type and takes into account the loss of immunity of newborns. It contains potential latent and immune periods, which are internal delays in the model, and the total population is not considered constant, in general. A constant regular vaccination forcing term is incorporated to interchange numbers of susceptible and vaccinated populations. The incorporation of such a term is one of the main novelties of the proposed SVEIRS model since SVEIRS models do not incorporate usually such a vaccination action being common in SEIRS models for interchanging of susceptible and immune populations. The existence

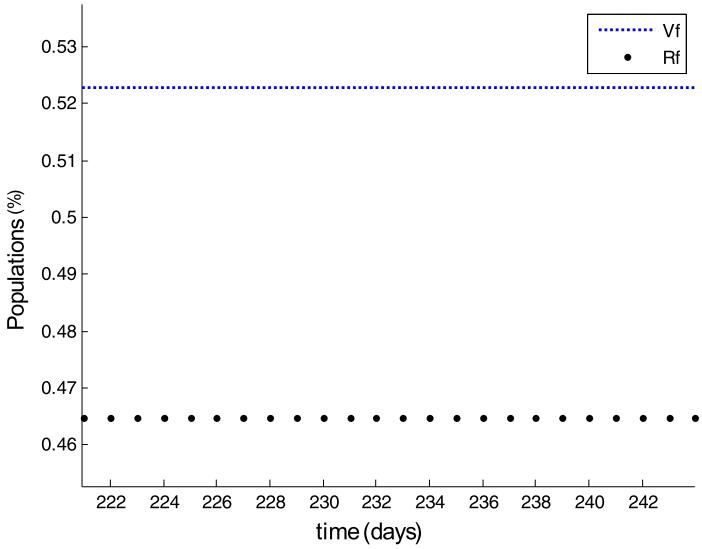

Fig. 8. Zoom of the final evolution of the fractional vaccinated and recovered populations towards the disease-free equilibrium point for $V_c = 1$.

and uniqueness of a disease-free equilibrium point as well as that of an endemic equilibrium point have been proven, and also, conditions of positivity and stability have been formulated and proven for reasonable constraints on the parameterization. A reproduction number threshold has been computed to elucidate the maintenance of the local asymptotic stability of the disease-free equilibrium for sufficiently small delays in the model. Roughly speaking, the disease-free equilibrium stability margin increases with the value of the constant vaccination while it decreases as the disease transmission constant increases.

The main vaccination recommendation is to increase the constant vaccination effort as much as possible to a threshold value being compatible with the stability of the disease-free equilibrium point given by the reproduction number. This strategy has a triple joint objective, namely, (a) to increase the recovered population at a stable disease-free equilibrium point while jointly decreasing the susceptible one, (b) to increase the effective value of the disease transmission constant being compatible with the stability of such an equilibrium, and (c) to avoid the convergence of the trajectory solutions to the endemic equilibrium point for larger values of the transmission constant compared to the case of absence of vaccination.

Another recommendation on a practical way of proceeding with the model use, provided that either the disease-free equilibrium point is known or the various formulas defining such an equilibrium are known, is as follows: (1) Firstly, consider that the susceptible, vaccinated and recovered disease-free equilibrium populations are multiplicative coefficients of a given standard testing total population while, obviously, the exposed and infected populations are zero at a stable disease-free equilibrium. This is a logic strategy to evaluate the partial populations evolving through time versus the disease-free equilib-

rium since their numbers are not ensured to be integer numbers without incorporating a discrete quantization model; (2) Secondly, perform an initialization at zero time of interest of the model time with values of the initial susceptible, vaccinated and recovered populations being close to their corresponding above mentioned equilibrium susceptible and recovered equilibrium coefficients while the exposed and infected populations are initially close to zero, but nonzero (otherwise, the infection would never appear and propagate); (3) Run the model evolution through time. The total and the various partial real populations are calculated at any time instant with the various multiplicative coefficients applied to the standard testing population. This set up would be a logic scenario to model common infectious diseases since, in these situations, the exposed and infected populations remain for all time within small deviations with respect to the whole population under study.

A relevant extension of this formalism could be devoted to the incorporation of the hybrid modeling by a simultaneous consideration of both discrete-time modeling (for instance, for the system's dynamics) and continuous time-modeling (for instance, for the vaccination effort). Hybrid systems are of greate interest in different problems of Control Theory because of their ability to a combined treatment of the formal accommodation and use of models which have continous-time and discrete-time (or eventually digital) coupled dynamics or for the use of discrete-controllers operating on continuous time systems. See, for instance, De la Sen (2006), Marchenko and Zaczkiewicz (2009a), Marchenko and Loiseau (2009b).

On the other hand, work is in progress concerning the design strategies of non-constant regular vaccination strategies by taking into account the measurable total and partial populations through time and the design of impulsive vaccination rules to remove the convergence to the endemic equilibrium point of the state trajectory solutions for larger values of the disease transmission constant.

Acknowledgements. The authors thank the Spanish Ministry of Education by its support through Grant DPI2009-07197. They are also grateful to the Basque Government by its support through Grants IT378-10, SAIOTEK S-PE08UN15, and SAIOTEK SPE09UN12. The authors are also grateful to the referees for their useful suggestions for the improvement of the first and the various revised versions of this manuscript.

References

Barreiro, A., Banos, A. (2010). Delay-dependent stability of reset systems. *Automatica*, 46(1), 216–221.

Boichuk, A., Langerov'a, M., Skorkov'a, J. (2010). Solutions of linear impulsive differential systems bounded on the entire real axis. *Advances in Difference Equations*, Article number 494379.

Bougeard, S., Qannari, E.M., Lupo, C., Hanafi, M. (2011). From multiblock partial least squares to multiblock redundancy analysis. A continuum approach. *Informatica*, 22(1), 11–26.

Daley, D.J., Gani, J. (2005). Epidemic Modelling: An Introduction. *Studies in Mathematical Biology*, Vol. 15, Cambridge University Press, New York.

D'Amico, G., Di Biase, G., Janssen, J., Manca, R. (2011). Hiv evolution: a quantification of the effects due to age and medical progress. *Informatica*, 22(1), 27–42.

De la Sen, M. (1998). A method for general design of positive real functions. *IEEE Transactions on Circuits and Systems I – Fundamental Theory and Applications*, 45(7), 764–769.

De la Sen, M. (2003). Absolute stability of single-input single-output systems with constant internal point delays. *Informatica*, 15(3), 357–374.

De la Sen, M. (2006). A note about the total stability of a class of hybrid systems. *Informatica*, 17(4), 565–576.

De la Sen, M. (2007a). Quadratic stability and stabilization of switched dynamic systems with uncommensurate internal point delays. *Applied Mathematics and Computation*, 185(1), 508–526.

De la Sen, M. (2007b). On positivity of singular regular linear time-delay time-invariant systems subject to multiple internal and external incommensurate point delays. *Applied Mathematics and Computation*, 190(12), 382–401.

De la Sen, M. (2008a). About the properties of a modified generalized Beverton–Holt equation in ecology models. *Discrete Dynamics in Nature and Society*, Article number 592950.

De la Sen, M. (2008b). The generalized Beverton–Holt equation and the control of populations. *Applied Mathematical Modelling*, 32(11), 2312–2328.

De la Sen, M., Alonso-Quesada, S. (2008b). A control theory point of view on Beverton–Holt equation in population dynamics and some of its generalizations. *Applied Mathematics and Computation*, 199(2), 464–481.

De la Sen, M., Alonso-Quesada, S. (2008c). Model-matching-based control of the Beverton–Holt equation in ecology. *Discrete Dynamics in Nature and Society*, Article number 793512.

De la Sen, M., Alonso-Quesada, S. (2009). Control issues for the Beverton–Holt equation in population in ecology by locally monitoring the environment carrying capacity: non-adaptive and adaptive cases. *Applied Mathematics and Computation*, 215(7), 2616–2633.

De la Sen, M., Alonso-Quesada, S. (2010a). On vaccination control tools for a general SEIR-epidemic model. In: *Proceedings of the 18th IEEE Mediterranean Conference on Control and Automation*, Vol. 1, pp. 1322–1328.

De la Sen, M., Luo, N.S. (2004). On the uniform exponential stability of a wide class of linear time-delay systems, *Journal of Mathematical Analysis and Applications*, 289(2), 456–476.

De la Sen, M., Agarwal, R.P., Ibeas, I., Alonso-Quesada, S. (2010b). On a generalized time-varying SEIR epidemic model with mixed point and distributed time-varying delays and combined regular and impulsive vaccination controls. *Advances in Difference Equations*, Article ID 281612. doi:10.1155/2010/281612.

Dzemyda, G., Sakalauskas, L. (2011). Large-scale analysis using heuristic methods. *Informatica*, 22(1), 1–10.

Erturk, V.S., Momani, S. (2008). Solutions to the problem of prey and predator and the epidemic model via differential transform method. *Kybernetes*, 37(8), 1180–1188.

Gao, S., Teng, Z., Xie, D. (2008). The effects of pulse vaccination on SEIR model with two time delays. *Applied Mathematics and Computation*, 201(1–2), 282–292.

Ibeas, A., De la Sen, M. (2004), Representations of multi-model based controllers by using artificial intelligence tools. *Informatica*, 15(3), 337–362.

Jiang, Y., Huiming, Y.W., Song, X., Mei, L. Su, G., Qiu, S. (2009). Global attractivity and permanence of a delayed SVEIR epidemic model with pulse vaccination and saturation incidence. *Applied Mathematics and Computation*, 213(2), 312–321.

Keeling, M.J., Rohani, P. (2008). *Modeling Infectious Diseases in Humans and Animals*, Princeton University Press, Princeton and Oxford.

Khan, Q.J.A., Krishnan, E.V. (2003). An epidemic model with a time-delay transmission. *Applications of Mathematics*, 48(3), 193–203.

Khan, H., Mohapatra, R.N., Varajvelu K., Liao, S.J. (2009). The explicit series solution of SIR and SIS epidemic models. *Applied Mathematics and Computation*, 215(2), 653–669.

Luo, N.S., Delasen, M., Rodellar, J. (1997). Robust stabilization of a class of uncertain time delay systems in sliding mode. *International Journal of Robust and Nonlinear Control*, 7(1), 59–74.

Maciulis, A., Paunksnis, A., Barzdziukas, V., Kriauciuniene L., Buteikiene, D., Piuziene, V. (2009). Digital model of blood circulation analysis system. *Informatica*, 20(4), 539–554.

Marchenko, V.M., Zaczkiewicz, Z. (2009a). Representation of solutions of control hybrid differential-difference impulse systems. *Differential Equations*, 45(12), 1811–1822.

Marchenko, V.M., Loiseau, J.J. (2009b). On the stability of hybrid difference-differential systems, *Differential Equations*, 45(5), 743–756.

Mollison, D. (Ed.) (2003). *Epidemic Models: Their Structure and Relation to Data*. Publications of the Newton Institute, Cambridge University Press, Cambridge, 1995, Denis Mollison Editor (transferred to digital printing 2003).

Mukhopadhyay, B., Bhattacharyya, R. (2007). Existence of epidemic waves in a disease transmission model with two-habitat population. *International Journal of Systems Science*, 38(9) 699–707.

Nieto, J.J., Tisdell, C.C. (2010). On exact controllability of first-order impulsive differential equations. *Advances in Difference Equations*, Article number 136504.

Ortega, N., Barros, L.C., Massad, E. (2003). Fuzzy gradual rules in epidemiology. *Kybernetes*, 32(3–4), 460–477.

Piccardi, C., Lazzaris, S. (1998). Vaccination policies for chaos reduction in childhood epidemics. *IEEE Transactions on Biomedical Engineering*, 45(5), 591–595.

Pupeikis, R. (2010). On a time-varying parameter adaptive self-organizing system in the presence of large outliers in observations. *Informatica*, 21(1), 79–94.

Song, X., Jiang, Y., Wei, H. (2009). Analysis of a saturation incidence SVEIRS epidemic model with pulse and two time delays. *Applied Mathematics and Computation*, 214(2), 381–390.

Yildirim, A., Cherruault, Y. (2009). Analytical approximate solution of a SIR epidemic model with constant vaccination strategy by homotopy perturbation method. *Kybernetes*, 38(9), 1566–1575.

Yu, H.G., Zhong, S.M., Agarwal, R.P., Xiong, L.L. (2010). Species permanence and dynamical behaviour analysis of an impulsively controlled ecological system with distributed time delay. *Computers & Mathematics with Applications*, 59(2), 3824–3835.

Zhang, T.L., Teng, Z.D. (2008). Dynamic behavior of a delayed impulsive SEIRS model in epidemiology. *Rocky Mountain Journal of Mathematics*, 38(5), 1841–1862.

Zhang, T.L., Liu, J.L., Teng, Z.D. (2009). Dynamic behaviour for a nonautonomous SIRS epidemic model with distributed delays. *Applied Mathematics and Computation*, 214(2), 624–631.

Zutautaite-Seputiene, I., Augutis, J., Telksnys, T. (2010). Parameters estimation in modelling of gas-gap in RBMK type reactor using Bayesian approach. *Informatica*, 21(2), 295–306.

Appendix A: Solution Trajectory of the SVEIRS Model

The solution trajectories of the SVEIRS differential model (1.1)–(1.5) are given below. Equation (1.1) yields:

$$S(t) = e^{-\int_0^t (b+\beta \frac{I(\xi)}{1+\eta S(\xi)})\,\mathrm{d}\xi} S(0) + \int_0^t e^{-\int_\xi^t (b+\beta \frac{I(\sigma)}{1+\eta S(\sigma)})\,\mathrm{d}\sigma} \big(\gamma I(\xi-\omega)e^{-b\omega} + \nu(1-V_c)N(\xi) + b\big)\mathrm{d}\xi. \quad \text{(A.1)}$$

Equation (1.2) yields:

$$V(t) = e^{-\int_0^t (\gamma_1+b+\frac{\delta\beta I(\xi)}{1+\eta V(\xi)})\,\mathrm{d}\xi} V(0) + \nu V_c \int_0^t e^{-\int_\xi^t (\gamma_1+b+\frac{\delta\beta I(\sigma)}{1+\eta V(\sigma)})\,\mathrm{d}\sigma} N(\xi)\,\mathrm{d}\xi. \quad \text{(A.2)}$$

Equation (1.3) is already in integral form. Equation (1.4) yields:

$$I(t) = e^{-(\gamma+b+\alpha)t}[I(0) + \beta e^{-b\tau} \int_0^t e^{(\gamma+b+\alpha)\xi} \left(\frac{S(\xi-\tau)I(\xi-\tau)}{1+\eta S(\xi-\tau)} + \frac{\delta V(\xi-\tau)I(\xi-\tau)}{1+\eta V(\xi-\tau)} \right) \mathrm{d}\xi]. \quad \text{(A.3)}$$

Equation (1.5) yields:

$$R(t) = e^{-bt}\bigg[R(0) + \int_0^t e^{b\xi}\big(\gamma_1 V(\xi) + \gamma(I(\xi) - I(\xi-\omega)\mathrm{e}^{-b\omega})\big)\,\mathrm{d}\xi\bigg]. \tag{A.4}$$

Appendix B: Disease-Free Equilibrium Stability for Sufficiently Small Delays with Quotient Parameterization

The following alternative result to Proposition 2 is based on an existence of the first sufficiently small destabilizing delay size of the linearized system about the equilibrium provided that the linearized zero-delay model is asymptotically stable about the disease-free equilibrium point.

Proposition B.1. Assume that $b > \nu$ and

$$R_{p0} = \beta\bigg(\frac{b-\nu V_c}{b-\nu+\eta(b-\nu V_c)} + \frac{\delta b\nu V_c}{(b+\gamma_1)(b-\nu)+\eta b\nu V_c}\bigg)\frac{1}{b+\alpha+\gamma} < 1.$$

Then, the SVEIRS epidemic model is locally asymptotically stable about the disease-free equilibrium point for $\tau = \lambda\omega$; $\forall\omega \in [0,\omega^*)$, any prefixed $\lambda \in \mathbf{R}_+$ and some $\omega^* \in \mathbf{R}_+$ if the so-called reproduction number satisfies the following constraint:

$$\begin{aligned} R_p(\lambda,\omega^*) := {} & \beta\mathrm{e}^{-b\lambda\omega^*}\bigg(\frac{b-\nu V_c}{b-\nu+\eta(b-\nu V_c)} + \frac{\delta b\nu V_c}{(b+\gamma_1)(b-\nu)+\eta b\nu V_c}\bigg) \\ & \times \frac{1}{b+\alpha+\gamma} < 1. \end{aligned}$$

For any prefixed, $(\lambda,\omega) \in \mathbf{R}_+^2$, the above property holds for sufficiently small disease transmission constant that satisfies:

$$\beta < \mathrm{e}^{b\lambda\omega^*}(b+\alpha+\gamma)\bigg(\frac{b-\nu V_c}{b-\nu+\eta(b-\nu V_c)} + \frac{\delta b\nu V_c}{(b+\gamma_1)(b-\nu)+\eta b\nu V_c}\bigg)^{-1}.$$

Proof. Consider the linearized system about the disease-free equilibrium point of state vector $\tilde{x}(t) := (\tilde{S}(t), \tilde{V}(t), \tilde{E}(t), \tilde{I}(t), \tilde{R}(t))^T$ characterized in Proposition 1 which satisfies the differential system (2.15) which becomes for $x^*(t) = x^*(t-\tau) = x^*(t-\omega)$:

$$\dot{\tilde{x}}(t) = A^*(\tau,\omega)\tilde{x}(t) = (A_0^* + A_\tau^* + A_\omega^*)\tilde{x}(t); \quad \tilde{x}(0) = \tilde{x}_0. \tag{B.1}$$

where $A^*(\tau,\omega) = .\frac{\partial\dot{\tilde{x}}}{\partial\tilde{x}^T}]_{(S^*,V^*,0,0,R^*)^T}$ is the Jacobian matrix of (1.1)–(1.5) at the disease-free equilibrium point. Define the delay quotient $\lambda = \tau/\omega$ for $\omega \neq 0$, resulting in $\lambda = \infty$ if $\omega = 0$ and $\tau \neq 0$, with such a definition modified as $\lambda = 0$ if $\tau = \omega = 0$.

Then, there is a bijective mapping from the Jacobian matrix $A^*(\tau,\omega)$ to $\bar{A}^*(\lambda,\omega)$, for a such a definition of λ, for any triple $(\tau,\omega,\lambda)\in\mathbf{R}^3_{0+}$ where:

$$\bar{A}^*(\lambda,\omega) := \begin{pmatrix} -b+(1-V_c)\nu & (1-V_c)\nu & (1-V_c)\nu & \mathrm{e}^{-b\omega}\gamma - \frac{\beta S^*}{1+\eta S^*} + (1-V_c)\nu & (1-V_c)\nu \\ \nu V_c & -b-\gamma_1+\nu V_c & \nu V_c & -\frac{\delta\beta V^*}{1+\eta V^*} + \nu V_c & \nu V_c \\ 0 & 0 & -b & \beta(1-\mathrm{e}^{-b\omega})(\frac{S^*}{1+\eta S^*}+\frac{\delta V^*}{1+\eta V^*}) & 0 \\ 0 & 0 & 0 & -b-\alpha-\gamma+\beta\mathrm{e}^{-b\lambda\omega}(\frac{S^*}{1+\eta S^*}+\frac{\delta V^*}{1+\eta V^*}) & 0 \\ 0 & \gamma_1 & 0 & (1-\mathrm{e}^{-b\omega})\gamma & -b \end{pmatrix}.$$

The eigenvalues of $\bar{A}^*(\lambda,\omega)$ are:

$$\Bigg(-b,-b,-b-\gamma_1,-(b+\alpha+\gamma) + \beta\mathrm{e}^{-b\lambda\omega}\left(\frac{b-\nu V_c}{b-\nu+\eta(b-\nu V_c)}+\frac{\delta b\nu V_c}{(b+\gamma_1)(b-\nu)+\eta b\nu V_c}\right), -b+\nu\Bigg).$$

Assume that $\bar{A}^*(0,0)$ is a stability matrix so that the above matrix has eigenvalues of negative real parts, i.e., $b>\nu$, and

$$R_p(\lambda,\omega) := \beta\mathrm{e}^{-b\lambda\omega}\left(\frac{b-\nu V_c}{b-\nu+\eta(b-\nu V_c)}+\frac{\delta b\nu V_c}{(b+\gamma_1)(b-\nu)+\eta b\nu V_c}\right) \times \frac{1}{b+\alpha+\gamma} < 1.$$

Thus the linearized system about the disease-free equilibrium is asymptotically stable and the nonlinear one is locally asymptotically stable for zero delays $\omega=0,\ \tau=\lambda\omega=0\ (\lambda=0)$. By continuity arguments of the eigenvalues with respect to the parameters, for any prefixed $\lambda\in\mathbf{R}^+$, there exist $\omega^*\in\mathbf{R}^+$ and $\tau^*=\lambda\omega^*\in\mathbf{R}^+$ such that the linearized system about the disease-free equilibrium is asymptotically stable and also the nonlinear one is locally asymptotically stable for $\tau=\lambda\omega;\ \forall\omega\in[0,\omega^*)$, that is if $b>\nu$ and the reproduction number

$$R_p\big(\lambda,\omega^*\big) := \beta\mathrm{e}^{-b\lambda\omega^*}\left(\frac{b-\nu V_c}{b-\nu+\eta(b-\nu V_c)}+\frac{\delta b\nu V_c}{(b+\gamma_1)(b-\nu)+\eta b\nu V_c}\right) \times \frac{1}{b+\alpha+\gamma} < 1.$$

If $R_p(\lambda,\omega^*)\geqslant 1$ then the linearized system is either critically stable or unstable.

Remark B.1. Note that for small model delays, the disease-free equilibrium stability margin decreases as the transmission constant increases for a given vaccination term. However, the modification of the value of the vaccination effort to a new appropriate value can compensate a certain increase of the transmission constant to still keep the disease-free equilibrium point stability.

Appendix C: Allocation of a Unique Endemic Equilibrium Point for the Vaccination-Free Case $V_c = 0$

This appendix contains the location of the endemic equilibrium points in the special case corresponding to $V_c = 0$ so that (2.2) implies $V^* = 0$. Furthermore, (2.4) yields for the endemic point:

$$\frac{\beta \mathrm{e}^{-b\tau}}{(\gamma + b + \alpha)} \left(\frac{S^*}{1 + \eta S^*} \right) = 1. \tag{C.1}$$

whence the value of S^* can be obtained:

$$S^* = \frac{\gamma + b + \alpha}{\beta \mathrm{e}^{-b\tau} - (\gamma + b + \alpha)\eta}. \tag{C.2}$$

In order to obtain a positive value for S^* the constraint $\beta > \eta \mathrm{e}^{b\tau}(\gamma + b + \alpha)$ must be satisfied which is the one required in Proposition 4(i) for the presence of an endemic equilibrium point. The remaining variables can be deduced from Equations (2.1), (2.3) and (2.5) by using the value of the total population in the equilibrium is the sum of all partial populations at such an equilibrium point. Hence, the total population in the equilibrium is obtained by zeroing the left-hand side of (4.2), i.e.:

$$0 = (\nu - b)N^* + b - \alpha I^* + \beta \frac{S^* I^*}{1 + \eta S^*} \big(\mathrm{e}^{-b\tau} - \mathrm{e}^{-b\omega} \big). \tag{C.3}$$

Thus,

$$\begin{aligned} N^* &= \frac{b}{(b - \nu)} + \frac{1}{(b - \nu)} \left[\beta \frac{S^*}{1 + \eta S^*} \big(\mathrm{e}^{-b\tau} - \mathrm{e}^{-b\omega} \big) - \alpha \right] I^* \\ &= \frac{b}{(b - \nu)} + \frac{1}{(b - \nu)} \big[(\gamma + b + \alpha)(1 - \mathrm{e}^{b(\tau - \omega)}) - \alpha \big] I^*. \end{aligned} \tag{C.4}$$

On the other hand, (2.3) together with (C.2) implies that:

$$E^* = \frac{(\gamma + b + \alpha)}{b} (\mathrm{e}^{b\tau} - \mathrm{e}^{b(\tau - \omega)}) I^*. \tag{C.5}$$

and (2.5) becomes

$$R^* = \frac{\gamma}{b} \big(1 - \mathrm{e}^{-b\omega} \big) I^*, \tag{C.6}$$

for $V_c = 0$. The total population is then given using (C.1)–(C.6) by:

$$\begin{aligned} N* &= \frac{b}{(b - \nu)} + \frac{1}{(b - \nu)} \big[(\gamma + b + \alpha) \big(1 - \mathrm{e}^{b(\tau - \omega)} \big) - \alpha \big] I^* \\ &= \frac{\gamma + b + \alpha}{\beta \mathrm{e}^{-b\tau} - (\gamma + b + \alpha)\eta} + 0 + \frac{(\gamma + b + \alpha)}{b} \big(\mathrm{e}^{b\tau} - \mathrm{e}^{b(\tau - \omega)} \big) I^* \\ &\quad + I^* + \frac{\gamma}{b} \big(1 - \mathrm{e}^{-b\omega} \big) I^*. \end{aligned} \tag{C.7}$$

and the value of I^* is given by:

$$I^* = \frac{(\gamma+b+\alpha)b(b-\nu)-b^2(\beta \mathrm{e}^{-b\tau}-(\gamma+b+\alpha)\eta)}{(\beta \mathrm{e}^{-b\tau}-(\gamma+b+\alpha)\eta)} \times \frac{1}{[(-b+\nu(1-\mathrm{e}^{-b\omega}))(\gamma+b+\alpha)\mathrm{e}^{b\tau}+\nu(b+\gamma)+\gamma(b-\nu)\mathrm{e}^{-b\omega}]}. \quad \text{(C.8)}$$

Thus, the remaining components of the endemic equilibrium point, which is seen to be unique, are given by (C.6) and (C.7) by using (C.8):

$$\begin{aligned} E^* &= (\gamma+b+\alpha)\big(\mathrm{e}^{b\tau}-\mathrm{e}^{b(\tau-\omega)}\big) \\ &\quad \times \frac{(\gamma+b+\alpha)(b-\nu)-b(\beta \mathrm{e}^{-b\tau}-(\gamma+b+\alpha)\eta)}{(\beta \mathrm{e}^{-b\tau}-(\gamma+b+\alpha)\eta)} \\ &\quad \times \frac{1}{[(-b+\nu(1-\mathrm{e}^{-b\omega}))(\gamma+b+\alpha)\mathrm{e}^{b\tau}+\nu(b+\gamma)+\gamma(b-\nu)\mathrm{e}^{-b\omega}]}, \\ R^* &= \gamma\big(1-\mathrm{e}^{-b\omega}\big)\frac{(\gamma+b+\alpha)(b-\nu)-b(\beta \mathrm{e}^{-b\tau}-(\gamma+b+\alpha)\eta)}{(\beta \mathrm{e}^{-b\tau}-(\gamma+b+\alpha)\eta)} \\ &\quad \times \frac{1}{[(-b+\nu(1-\mathrm{e}^{-b\omega}))(\gamma+b+\alpha)\mathrm{e}^{b\tau}+\nu(b+\gamma)+\gamma(b-\nu)\mathrm{e}^{-b\omega}]}. \end{aligned} \quad \text{(C.9)}$$

M. De la Sen was born in Arrigorriaga, Bizkaia, Basque Country, Spain. He received the MSc degree (honors) in applied physics (electronics and automation) and the PhD degree (high honors and special mention) in applied physics from the University of Basque Country, Bilbao, Spain in 1975 and 1979, respectively, and the degree of docteur – d'Etat es – sciences physiques (specialite automatique et traitement du signal, high honors) from the University of Grenoble, France, in 1987. He is currently professor of systems engineering and automatic control at the University of Basque Country where he serves as the head of the Institute of Research and Development of Processes. He has co-authored about seven hundred papers in scientific journals and proceedings of conferences.

Professor De la Sen was a visiting professor at the universities of Grenoble (France), Newcastle (Australia) and Australian National University (Canberra, Australia) and he was formerly a member of the Editorial Board of the Electrosoft Journal (CML Mechanical and Computational Engineering Publications). He is currently associate editor of the following journals: Applied Mathematical Sciences; Nonlinear Analysis, Modelling and Control; Mathematics in Engineering, Science and Aerospace – the Transdisciplinary International Journal; Discrete Dynamics in Nature and Society; Nonlinear Studies – the International Journal; and Fixed Point Theory and Applications.

His main interest research areas are: discrete and sampled-data control systems, non-periodic and adaptive sampling, adaptive control, fixed point theory, positive systems, stability, models for ecology, epidemic models, time-delay systems, artificial intelligence tools for dynamic systems and ordinary differential equations.

A. Ibeas was born in Bilbao, Spain, on July 7, 1977. He received the MSc degree in applied physics and the PhD degree in automatic control from University of the Basque Country, Spain, in 2000 and 2006, respectively. He is currently working as lecturer of electrical engineering at Autonomous University of Barcelona, Spain. His research interests include mathematical systems theory, time-delayed systems, robust adaptive control and applications of artificial intelligence to control systems design.

S. Alonso-Quesada was born in Bilbao, Bizkaia, in the Spanish Basque Country. He obtains the MSc degree in automatic and electronics physics from the Faculty of Sciences of the Basque Country University in 1992 and the PhD degree in physics from the Department of Electricity and Electronics of the same University in 2001. He has had several teaching and research positions in the Basque Country University where he is currently doctor aggregate professor in the area of engineering system and automatic in the Department of Electricity and Electronics. He has been co-author of some papers in the fields of adaptive control systems and control applications in biological systems. His research interest is in the adaptive control, control theory and control applications in epidemics models. He acts and has acted as reviewer for some international journals and conferences of control theory and control applications.

R. Nistal graduated in physics at the Science Faculty of University of the Basque Country in 2008. He received a MS degree in biophysics from University of Barcelona in 2010 where he worked on mathematical models for organ differentiations and gene networks. He is currently a PhD student at the University of Basque Country working on disease propagation models and the efficiency of possible responses.

Apie SVEIRS epideminio modelio pusiausvyros taškus, apribojimus bei privalumus esant reguliariai ir apribotai vakcinacijai

Manuel De la SEN, Asier IBEAS, Santiago ALONSO-QUESADA, Raul NISTAL

Straipsnyje nagrinėjami ligos plitimo SVEIRS modelio pusiausvyros taškai, modelio privalumai bei apribojimai esant pastoviai ir reguliariai vakcinacijai. Į modelį įtrauktos prielaidos apie natūralų populiacijos augimą, išmirimą ir naujagimių imuniteto praradimą, priklausantį nuo ligos išplitimo. Daroma prielaida, kad yra du riboti vėlavimai, kurie veikia įtariamos, stebimos, pagydytos ir infekuotos populiacijos dinamiką.