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Design of a vaccination law for an age-dependent epidemic model using state feedback

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Abstract: An age-dependent epidemic model is studied with the goal of designing a state feedback stabilizing vaccination law to eradicate a disease. This model consists of a set of three nonlinear partial-integro differential equations (PIDE). A salient feature of the dynamical analysis is the fact that, if the basic reproduction number is greater than one, then the disease-free equilibrium is unstable. In view of this, we provide a linearizing state feedback vaccination law that is deduced from the one obtained for the PIDE model discretisation with respect to the age. Conditions guaranteeing stability of the closed-loop system and positivity of the feedback control are obtained using Isidori's theory and semigroup theory. Numerical simulations complete the analysis.

Keywords: Infinite-dimensional systems, positive systems, (un)stability of equilibria, nonlinear control, partial integro-differential equations, epidemiology

1. INTRODUCTION

Understanding the evolution of an epidemic is crucial to be able to act on it and to eradicate the disease. One way to counter the disaster brought by some epidemics is to develop effective vaccines and to adopt an appropriate vaccination strategy. In this paper, the second part is achieved by studying an extended version of the SIR model developed by Kermack and Mckendrick (1991) where the individuals age is taken into account. This is motivated by the fact that vaccination strategies may depend on the age of individuals. In such models, the population is assumed to be divided in three groups: the class S of susceptible individuals who can catch the disease, the class I of infected individuals who can transmit the disease and the class R of recovered individuals who are assumed to be permanently immune to the disease.

This paper is organized as follow. First, the age-dependent model is introduced. It consists of a set of three nonlinear partial-integro differential equations. Then, in Section 3, the results of the dynamical analysis of the system are reported and a result on the stability of the equilibria is highlighted. In view of this result, a feedback vaccination law is introduced in Section 4 and the global linearizing stability analysis of the feedback is performed. This analysis is inspired by Isidori's theory (Isidori (1995)) but applied to infinite dimensional system. Moreover, it is shown that, under appropriate choices of the feedback gains, the vaccination law is non-negative, ensuring its physical feasibility. Finally, in the last two sections, the results are illustrated by numerical simulations.

2. MODEL DESCRIPTION

In this paper, an age-dependent epidemic model is studied to describe the propagation of a disease. The individuals age is considered since several epidemiological factors are age-dependent, vaccination being one of them. An adapted version of the SIR model Kermack and Mckendrick (1991) is used which is inspired by the one described in Bastin and Coron (2016). The dynamics of the disease propagation is described by a system of nonlinear partial integro-differential equations (PIDE)

$$\begin{aligned}
(\partial_t + \partial_a) S(t,a) &= -(\Theta(t,a) + \mu(a)) S(t,a) \\
&\quad - \beta(a) S(t,a) \int_0^{a_{\max}} I(t,b)\, db, \\
(\partial_t + \partial_a) I(t,a) &= -(\mu(a) + \gamma(a)) I(t,a) \\
&\quad + \beta(a) S(t,a) \int_0^{a_{\max}} I(t,b)\, db, \\
(\partial_t + \partial_a) R(t,a) &= \Theta(t,a) S(t,a) + \gamma(a) I(t,a) \\
&\quad - \mu(a) R(t,a)
\end{aligned} \tag{1}$$

under non-negative initial conditions $S(0,a) = S_0(a)$, $I(0,a) = I_0(a)$, $R(0,a) = R_0(a)$ and boundary conditions $S(t,0) = B, I(t,0) = 0, R(t,0) = 0$.

The quantities $S(t,a), I(t,a)$ and $R(t,a)$ denote the age-densities of susceptible, infected and recovered individuals at time t respectively. Remark that, in the following, the terms S-, I- and R-individuals will refer to susceptible, infected and recovered individuals, respectively. By the definition of density, the number of S-individuals between two given ages b and c is obtained by integrating $S(t,a)$ on the interval $[b,c]$. The sum of those three quantities gives

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the density of the total population denoted as $P(t,a)$. The function $\Theta(t,a)$ is the input variable and represents the vaccination rate of S-individuals. In this model, we assume that the population is closed. Therefore, a change in the total size of the population is only due to birth and mortality with respective rates B, assumed to be constant, and $\mu(a)$. Moreover, we assume that the disease is transmitted by contact between S- and I-individuals with a disease transmission rate given by $\beta(a)\int_0^{a_{\max}} I(t,b)db$. Finally the recovery rate of the disease is given by the coefficient $\gamma(a)$. Observe that all these parameters are nonnegative.

3. DYNAMICAL ANALYSIS

This section is dedicated to the well-posedness of the model and the stability analysis of its equilibria.

3.1 Well-posedness and stability analysis

Assuming that the rate of vaccination is given by a Lipschitz continuous state feedback law, the existence and uniqueness of a solution can be stated using Pazy's theorem on the existence of a mild solution. See (Pazy, 1983, Chap.6, Sect. 1). Moreover, it can be shown that this solution is non-negative, as should be expected, in view of the physical interpretation of the model. This can be proven using the method of characteristics and semigroup theory, as mentioned in Inaba (1990) and Inaba (2017) for a similar model.

The stability analysis can be performed on the limiting autonomous system, using the fact that $\lim_{t\to\infty} P(t,a) = P(a) = B\exp\left(\int_0^a \mu(\eta)d\eta\right)$. Equilibria can be found for the "normalized model" where the following change of variables is made:
$S(t,a) = P(t,a)\,s(t,a)$; $I(t,a) = P(t,a)\,i(t,a)$; $R(t,a) = P(t,a)\,r(t,a)$. In that case, if the basic reproduction number $R(0)$ defined by

$$\int_0^{a_{\max}} P(b)\,\Gamma(b)\int_0^b \frac{\beta(\sigma)}{\Gamma(\sigma)}\exp\left(-\int_0^\sigma \Theta^\star(\eta)\,d\eta\right)d\sigma db,$$

where $\Gamma(b) = \exp\left(-\int_0^b \gamma(\eta)\,d\eta\right)$, is smaller than 1, there is only one equilibrium, the disease-free one ($I(t,a) = 0$). Conversely, if $R(0)$ is strictly greater than 1, there are two equilibria, an endemic one and a disease-free one.

Then, the stability analysis can be performed on the homogeneous normalized model (obtained using the change of variables: $\hat{s}(t,a) = s(t,a) - 1$). With this system it can be shown that the principle of linearized stability can be applied. See Sonveaux and Winkin (2022). Studying the stability of the linearized homogeneous normalized model, using semigroup theory and property of operators, as developed in Inaba (1990), leads to the following result.

Theorem 1. The disease free equilibrium is locally exponentially stable when $R(0) \leq 1$. Conversely it is locally exponentially unstable if $R(0) > 1$. In that case, the endemic equilibrium is locally exponentially stable.

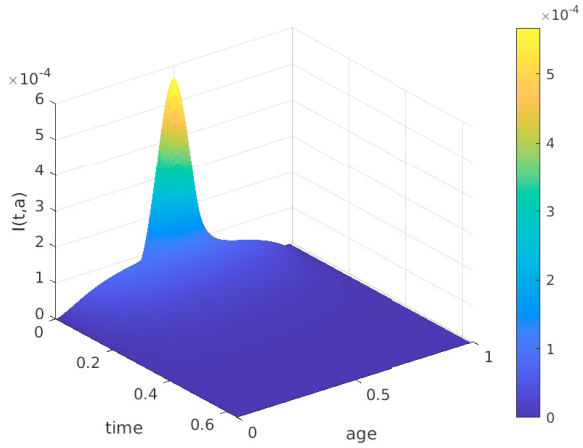

Fig. 1. Density of I-individuals for $R(0) = 0.8894$

3.2 Numerical simulations

Those results can be illustrated on numerical simulations. Note that in the simulations, the maximal age, $a_{\max}$, is fixed to 1 and the choice of parameters implies that the total population equals 1. Those parameters are the one used in Okuwa et al. (2019) where some adjustments were made for the transmission coefficient $\beta(a)$ and for the initial conditions. Here the transmission coefficient is chosen to be

$$\beta(a) = \beta_0\left(\sin(a)e^{-2a} + \frac{1}{100}\right),$$

in order to have differentiability. The choice of β_0 influences the basic reproduction number of infection and is fixed at 600 for Figure 1 and 800 for Figure 2. The change of initial conditions is given by

$$\begin{aligned} S_0(a) &= P(a) - I_0(a), \\ I_0(a) &= \begin{cases} \hat{I}_0(a) - \hat{I}_0(0) & \text{if } I_0(a) \geq 0 \\ 0 & \text{else,} \end{cases} \\ R_0(a) &= 0 \end{aligned}$$

where

$$\hat{I}_0(a) = \frac{1}{2}e^{-100\left(a-\frac{1}{2}\right)^2} \times 10^{-3} \times P(a).$$

These choices ensure consistency between initial conditions and boundary conditions. Note that $P(a)$ represents the age-density of the population. In all the simulations of this paper, a tolerance threshold is set to 10^{-8}. Therefore simulations are stopped when the trajectories reach the convergence or if the final time, fixed at 20, is reached.

Those simulations corroborate the theoretical results. Indeed, as stated in Theorem 1, when $R(0)$ is smaller than 1, the disease is eradicated. This can be observed in Figure 1 since the infected individuals converge to 0. Conversely, in Figure 2, $R(0)$ is greater than 1 and the infected individuals tend to a state where there remain infected individuals in the population.

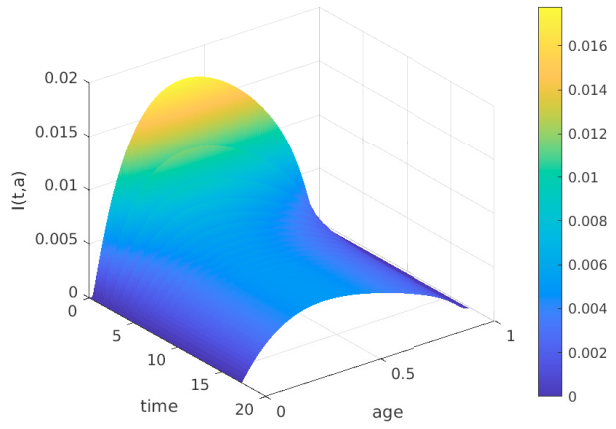

Fig. 2. Density of I-individuals for $R(0) = 1.1859$

4. FEEDBACK STABILIZATION

In this section, a state feedback law is designed to stabilize the system around the disease free equilibrium. As mentioned in Sonveaux and Winkin (2022), a feedback law can be designed, using Isidori's theory (Isidori (1995)) for a finite-dimensional model obtained by discretizing Model 1 according to the age of the individuals. Therefore, this law is linearizing for the discretized model and is stabilizing for an appropriate choice of the feedback gains. From this law, the following nonlinear continuous state feedback control law is deduced by taking a formal limit

$$\begin{aligned}\Theta(t,a) = \tilde{\alpha}_2(a) &+ \int_0^{a_{\max}} \beta(a) S(t,a)\, da - 2\mu(a) - \gamma(a) \\ &- \beta(a) \int_0^{a_{\max}} I(t,b)\, db \\ &- \frac{\int_0^{a_{\max}} (\mu(a)+\gamma(a)) I(t,a)\, da}{\int_0^{a_{\max}} I(t,b)\, db} \\ &+ \frac{I(t,a)}{\beta(a) S(t,a) \int_0^{a_{\max}} I(t,b)\, db} \Big(\tilde{\alpha}_1(a) \\ &+ (\mu(a)+\gamma(a))(\mu(a)+\gamma(a)-\tilde{\alpha}_2(a)) \Big) \end{aligned} \quad (2)$$

where $\tilde{\alpha}_1$ and $\tilde{\alpha}_2$ are the design parameters. One of the nice feature of this law is that it linearizes the closed-loop model in "normal-form": see (5) - (9). Moreover, in the following, it is shown that, for a good choice of the design parameters, this feedback law stabilizes the closed-loop system, leading to disease eradication. In addition, it is highlighted that, also for an appropriate choice of the control parameters, this law is non-negative, therefore ensuring its physical meaning with respect to the model.

4.1 Stabilizing feedback law

In this section, an approach inspired by Isidori (1995) is used with the particularity to applied on infinite dimensional system. This is motivated by the fact that the vaccination law (2) is deduced from the one obtained using Isidori's theory. First, we can observe that the closed-loop system is given by

$$\begin{aligned}(\partial_t + \partial_a) S(t,a) &= S(t,a) \Big[-\tilde{\alpha}_2 + \mu(a) + \gamma(a) \\ &\quad - \int_0^{a_{\max}} \beta(a) S(t,a) da \\ &\quad + \frac{\int_0^{a_{\max}} (\mu(a)+\gamma(a)) I(t,a) da}{\int_0^{a_{\max}} I(t,a)\, da} \Big] \\ &\quad + \frac{I(t,a)}{\beta(a) \int_0^{a_{\max}} I(t,a)\, da} [-\tilde{\alpha}_1 + \\ &\quad (\mu(a)+\gamma(a))(\tilde{\alpha}_2 - (\mu(a)+\gamma(a)))] \end{aligned} \quad (3)$$

$$\begin{aligned}(\partial_t + \partial_a) I(t,a) &= -(\mu(a)+\gamma(a)) I(t,a) \\ &\quad + \beta(a) S(t,a) \int_0^{a_{\max}} I(t,b)\, db, \\ R(t,a) &= P(a) - S(t,a) - I(t,a)\end{aligned}$$

under the same non-negative initial conditions and boundary conditions as Model 1. Observe that the R-individuals can be obtained by knowing the density of the S-individuals and the I-individuals and the age-density of the population, $P(a)$. Remark that this variable does not depend on time since we assume that the population has reached a stable age distribution. Therefore, in the following, we consider only a set of two equations. As in Isidori's theory, the following nonlinear coordinates change is applied,

$$\begin{aligned}\bar{I}(t,a) &= I(t,a), \\ \bar{S}(t,a) &= -(\gamma(a)+\mu(a)) I(t,a) \\ &\quad + \beta(a) S(t,a) \int_0^{a_{\max}} I(t,b)\, db\end{aligned} \quad (4)$$

to write the system in a so-called "normal-form". Therefore, the closed-loop system (3) rewrites

$$\begin{aligned}(\partial_t + \partial_a) \bar{I}(t,a) &= \bar{S}(t,a), \\ (\partial_t + \partial_a) \bar{S}(t,a) &= \bar{I}(t,a)[-\tilde{\alpha}_1(a) + g(a)] \\ &\quad + \bar{S}(t,a)[-\tilde{\alpha}_2(a) + h(a)]\end{aligned} \quad (5)$$

where

$$g(a) = -\beta(a) \frac{d}{da} \left(\frac{\gamma(a)+\mu(a)}{\beta(a)} \right), \quad (6)$$

$$h(a) = \frac{1}{\beta(a)} \frac{d}{da} \beta(a) \quad (7)$$

under non-homogeneous boundary conditions

$$\begin{aligned}\bar{I}(t,0) &= 0, \\ \bar{S}(t,0) &= \beta(0) B \int_0^{a_{\max}} \bar{I}(t,b)\, db\end{aligned} \quad (8)$$

and initial conditions

$$\begin{aligned}\bar{I}(0,a) &= I_0(a), \\ \bar{S}(0,a) &= \bar{S}_0(a).\end{aligned} \quad (9)$$

One can observe that the closed-loop in "normal-form" is linear. It shows that the state feedback (2) is linearizing for the model in "normal-form", as it is the case in Isidori's theory.

In order to work with homogeneous boundary conditions, the system is rewritten using Fattorini's approach on boundary control systems (see Fattorini (1968)) where the results are extended to Banach spaces. Therefore (5) - (9) is equivalent to

$$\begin{aligned} \dot{\bar{x}} &= \left(\bar{\mathcal{A}}_0 + \bar{D}\right)\bar{x} \\ \bar{x}(0) &= (0,0)^T \end{aligned} \tag{10}$$

with $\bar{x} = \left(\bar{I}, \bar{S}\right)^T$, $\bar{\mathcal{A}}_0 = \begin{pmatrix} -\frac{d\cdot}{da} & I \\ G(a)\, I & -\frac{d\cdot}{da} + H(a)\, I \end{pmatrix}$

with $\mathcal{D}\left(\bar{\mathcal{A}}_0\right) = \left\{\bar{x} \in L^1(0, a_{\max}) \times L^1(0, a_{\max}) : \bar{x}, \right.$
$\left. \frac{d\bar{x}}{da} \in AC[0, a_{\max}], \bar{x}_0 = (0,0)^T \right\}$

and $\bar{D} = \begin{pmatrix} 0 & 0 \\ \delta_0 \beta(0)\, B \int_0^{a_{\max}} \cdot \; db & 0 \end{pmatrix}$.

However, in this formulation, $\bar{D}$ is unbounded. Therefore, we use an approximation of the Dirac delta δ_0 by replacing it by $d_k(a)$, a term of a Dirac sequence, satisfying properties developed in (Hinrichsen and Pritchard, 2010, Chap. 2, Sect. 3, Lemma 2.3.4) with ∞ replaced with $a_{\max}$. The approximate system is given by

$$\begin{aligned} \dot{\bar{x}}_k &= \left(\bar{\mathcal{A}}_0 + \bar{D}_k\right)\bar{x}_k \\ \bar{x}_k(0) &= (0,0)^T \end{aligned} \tag{11}$$

with $\bar{D}_k = \begin{pmatrix} 0 & 0 \\ d_k(a)\, \beta(0)\, B \int_0^{a_{\max}} \cdot \; db & 0 \end{pmatrix}$.

Note that solutions of this approximate system are denoted by $\bar{x}_k$. In the following, it is shown that the infected individuals converge to zero. First a lemma is needed implying the convergence to zero of the new variables $\bar{I}_k$ and $\bar{S}_k$. A complete proof of this Lemma is available in Sonveaux and Winkin (2022) and uses the principle of invariance stability under system equivalence (Schumacher (1981))) and the bounded perturbation theorem ((Klaus-Jochen and Rainer, 2006, Chap. 3, Sect. 1)) to conclude.

Lemma 2. $\bar{\mathcal{A}}_0 + \bar{D}_k$ is the infinitesimal generator of an exponentially stable C_0-semigroup $\left(\tilde{T}(t)\right)_{t\geq 0}$ with growth bound

$$\omega_0(\tilde{T}) < -(c_2 + K) + (1 + K(c_1 - 1) - c_2)\,\|\bar{D}_k\| < 0$$

if c_1 and c_2 are chosen such that

$$c_1 > max\left\{1, \sup_{a\in[0,a_{\max}]} \left(\tilde{\alpha}_1(a) - g(a)\right), \frac{\beta_0 B}{K}\right\}, \tag{12}$$

$$c_2 > max\left\{0, \frac{\beta_0 B(1 + Kc_1)}{1 + \beta_0 B} - K, \sup_{a\in[0,a_{\max}]} \left(\tilde{\alpha}_2(a) - h(a)\right)\right\}, \tag{13}$$

$$c_2 \leq K(c_1 - 1). \tag{14}$$

Then, using Lemma 2 that shows that $\bar{x}_k(t,a)$ exponentially converges to zero, it follows that so does $\bar{I}_k(t,a)$ which is equal to $I_k(t,a)$ by the change of variables (4). Therefore, the eradication of the I_k-population is obtained.

Theorem 3. Let $x_{0_k} = [I_{0_k}, S_{0_k}]^T \in L^1(0, a_{\max})^+ \times L^1(0, a_{\max})^+$ where $L^1(0, a_{\max})^+$ refers to the cone of (almost everywhere) non-negative functions in $L^1(0, a_{\max})$. Assume that we choose c_1, c_2, $\tilde{\alpha}_1(a)$ and $\tilde{\alpha}_2(a)$ such that conditions (12) to (14) are satisfied. Then, the state feedback (2) implies the exponential asymptotic convergence to zero of the infected population $I_k(t,a)$ as time tends to infinity:

$$\|I_k(t,\cdot)\|_1 \to 0 \text{ as time goes to infinity.}$$

Observe that the convergence is proven for the approximate system (11). However, we are interested in the convergence of the infected individuals trajectories of system (3).
An intuition to obtain this result consists in observing the limit of the error's dynamics $\bar{E}(t,a) = \bar{x}_k(t,a) - \bar{x}(t,a)$. This dynamics tends, as k goes to infinity, to $\dot{\bar{E}} = \bar{D}\bar{E}$ whose solution is $\bar{E}$ which is identically zero. Therefore, we can assume that $\bar{x}_k(t,a)$ tends to $\bar{x}(t,a)$ as time tends to infinity. Theorem 3 would then conclude the conjecture.

Conjecture 4. The state feedback (2) implies the exponential convergence towards zero of the infected population $I(t,a)$, as time tends to infinity.

4.2 Nonnegativity of the feedback law

Since the feedback law represents the vaccination rate, it has to be non-negative to make physical sense. Therefore, the design feedback parameters need to be tuned in order to ensure non-negativity of the feedback law. Observe that the conditions (12) - (14) only affects the convergence speed of the system. Therefore, in the following, the feedback gains can be tuned appropriately to ensure the nonnegative property of the vaccination.

Theorem 5. Define

$$\nu = \sup_{a\in[0,a_{\max}]} \mu(a); \Gamma = \sup_{a\in[0,a_{\max}]} \gamma(a)$$

and N the total population. Taking

$$\tilde{\alpha}_2(a) = 3\nu + 2\Gamma + \beta(a)N \tag{15}$$

$$\tilde{\alpha}_1(a) = -\left(\mu(a) + \gamma(a)\right)\left(\mu(a) + \gamma(a) - \tilde{\alpha}_2\right) \tag{16}$$

yields the local exponential stability of the closed-loop system (3) with the non-negative vaccination law (2).

Proof. Condition (16) simplifies the vaccination law (2). Moreover, definition of ν, Γ and the estimate $\int_0^{a_{\max}} I(t,b)db \leq N$ lead to the following inequality for the vaccination law,

$$\begin{aligned} \Theta(t,a) \geq \tilde{\alpha}_2(a) + \int_0^{a_{\max}} \beta(a)\, S(t,a)\, da - 2\nu - \Gamma \\ - \beta(a)N - (\nu + \Gamma). \end{aligned}$$

Then condition (15) implies that

$$\Theta(t,a) \geq \int_0^{a_{\max}} \beta(a)\, S(t,a)\, da$$

which is always non-negative.

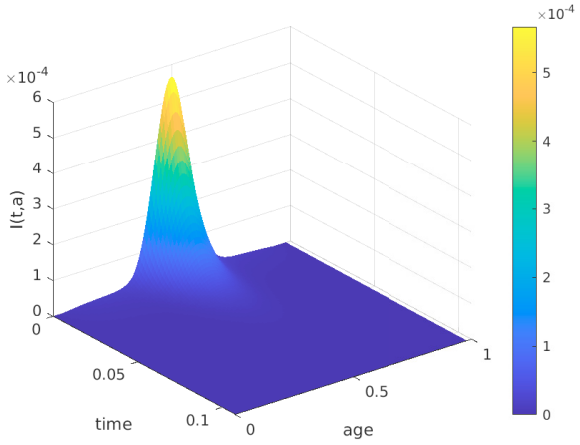

Fig. 3. Density of I-individuals for $R(0) = 1.1859$ with vaccination

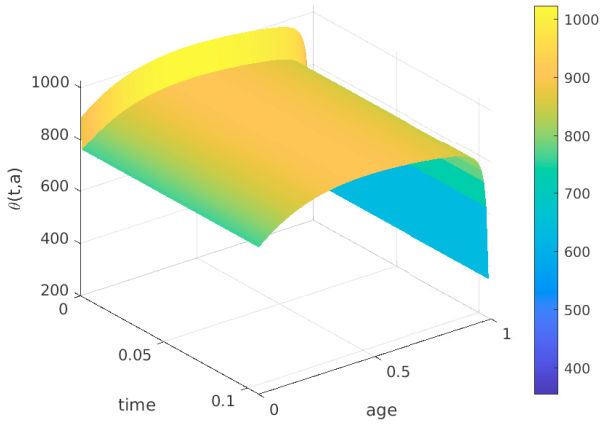

Fig. 4. Dynamics of the feedback vaccination-law

4.3 Numerical simulations

The previous results can be observed thanks to numerical simulations. The same parameters as in Section 3.2 are used. Figure 3 shows the density of I-individuals when $R(0)$ is greater than 1 and the vaccination law (2) is applied. As mentioned in Conjecture 4, we observe that the disease becomes eradicated from the population. Observe that we do not consider fatal disease in the model since only the natural death rate, described by $\mu(a)$, is taken into account. However, even for non-fatal illness, it is interesting to eradicate the disease because infected individuals can end up with serious sequels. Moreover, Figure 4 shows that the vaccination law remains non-negative all the time, ensuring its physical meaning. The strategy suggested by Figure 4 is to vaccinate less young and old people and focus the efforts on people who are in classes of age with more infected individuals initially. The proposed law features a transient phase, for each age where lots of individuals are vaccinated, that is followed by a steady-state. Therefore, it is interesting to wonder if a static vaccination law $\tilde{\Theta}(a)$ could be enough to obtain disease eradication. The last figures are obtained by applying the feedback-law $\tilde{\Theta}(a)$ (see Figure 6) corresponding to the limit of $\Theta(t, a)$, as time goes to infinity. Only numerical results were investigated in this case. Figure 5 shows that the use of a static vaccination law implies disease eradication. The convergence rate is similar as the one obtained with the state-feedback law. However, Figure 7, representing the difference between infected individuals obtained using the static law and those with the dynamical law, highlights the fact that they are less infected individuals with the dynamical law. This is a consequence of the transient phase of the dynamical law.

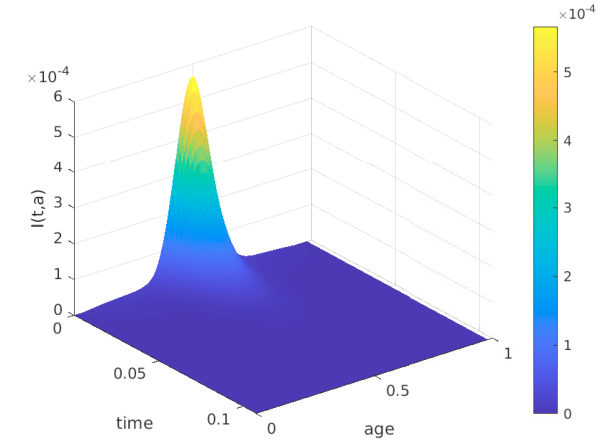

Fig. 5. Density of I-individuals for $R(0) = 1.1859$ with a static vaccination

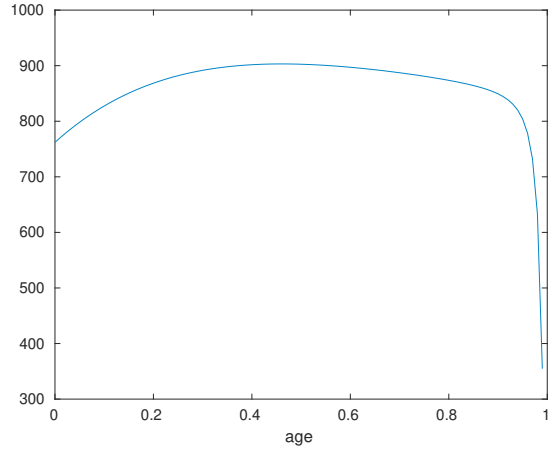

Fig. 6. Static vaccination-law profile

5. CONCLUDING REMARKS

The results of the dynamical analysis of an age-dependent SIR model were reported using the principle of linearized stability. It led to Theorem 1 where the unstability of the disease-free equilibrium has been highlighted in the case where the basic reproduction number $R(0)$ is greater than 1. Therefore, a linearizing feedback law has been proposed. It was shown that, under appropriate choices of the design parameters, it is stabilizing while being non-negative.

However, since the control law is a state feedback, the knowledge of all state variables is needed, which is not possible in practice. Therefore, the design of a state observer will be needed to reconstruct the whole state. Moreover, in view of the numerical results obtained for the static vaccination law deduced from the state-feedback

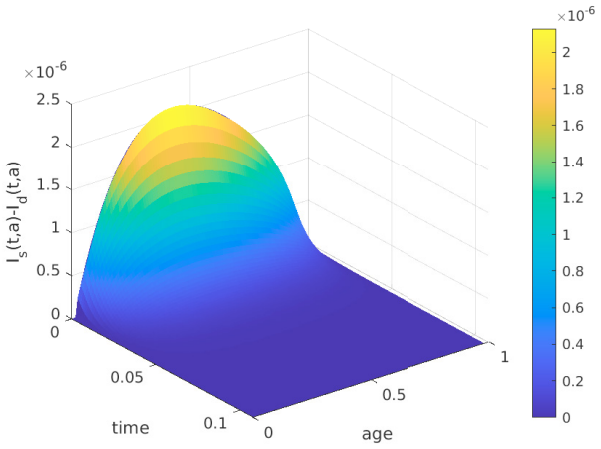

Fig. 7. Difference between I-individuals obtained with the static law (I_s) and those obtained with the dynamical law (I_d)

one, investigating the performance of this static law in the analytical framework could be interesting. It would also be of interest to apply this theory on real data, which are for instance widely available in the case of the COVID19 pandemic.

REFERENCES

Bastin, G. and Coron, J.M. (2016). *Stability and boundary stabilization of 1-D hyperbolic systems*, 40–43. coll. PNLDE Subseries in Control. Birkhauser, Switzerland.

Fattorini, H.O. (1968). Boundary control systems. *SIAM Journal on Control*, 6(3), 349–385. doi:10.1137/0306025.

Hinrichsen, D. and Pritchard, A.J. (2010). *Mathematical systems theory I modelling, state space analysis, stability and robustness*. Springer.

Inaba, H. (1990). Treshold and stability results for an age-structured epidemic model. *Journal of mathematical biology*, 411–434.

Inaba, H. (2017). *Age-Structured population dynamics in demography and epidemiology*. Springer.

Isidori, A. (1995). *Nonlinear control systems*. Springer, 3rd edition.

Kermack, W. and Mckendrick, A. (1991). Contributions to the mathematical theory of epidemics—ii. the problem of endemicity. *Bulletin of Mathematical Biology*, 53(1-2), 57–87. doi:10.1016/s0092-8240(05)80041-2.

Klaus-Jochen, E. and Rainer, N. (2006). *A Short Course on Operator Semigroups*. Springer, New York, USA.

Okuwa, K., Inaba, H., and Kuniya, T. (2019). Mathematical analysis for an age-structured sirs epidemic model. *Mathematical Biosciences and Engineering*, 16, 6071–6102.

Pazy, A. (1983). *Semigroups of linear operators and application to partial differential equations*, volume 44. Springer-Verlag New-York, USA.

Schumacher (1981). *Dynamic feedback in finite-and infinite-dimensional linear systems*. Ph.D. thesis.

Sonveaux, C. and Winkin, J. (2022). State feedback control law design for an age-dependant SIR model. Submitted.