# Modelling the effects of prevention and early diagnosis on HIV/AIDS infection diffusion

P. Di Giamberardino, L. Compagnucci, C. De Giorgi and D. Iacoviello *Member, IEEE,*

***Abstract*—In this paper a new model describing the HIV-AIDS epidemic spread is proposed. The improvement with respect to the known models has been driven by recent results obtained from historical data collection and the suggestions given by the World Health Organization: the characteristics of the virus diffusion, mainly by body fluids, imply the trivial fact that wise behaviours of healthy subjects and fast timely recognition of a new positive diagnosis should reduce the spread quite fast. Therefore, the set of susceptible subjects is divided into two categories: the wise people that, suitably informed, avoid dangerous behaviours, and the ones that, with irresponsible acts, could get the infection. The set of infected subjects is constituted by people who are still not aware of being infected (and therefore are responsible of the HIV spread), along with the subjects aware of being infected by HIV or AIDS. Inspired by the international guidelines suggestions, three controls are introduced, aiming both at the prevention and at the cure: an informative campaign, a test campaign, and a HIV/AIDS therapy action. Among them, the core of the control effort is a fast HIV diagnosis. The equilibrium points, their stability and the influences of the introduced inputs to the system behaviour are studied, yielding to preliminary statements for prospective works on suitable control design approaches.**

**Keywords: Analytical models, System analysis and design, Systems performance analysis and prediction, Nonlinear dynamical systems, Public health-care**

## I. Introduction

Mathematical modelling has shown its power describing models of different outbreaks like SIR, SARS, SIRC, SEIR, HIV [1], [2], [3], [4], [5], and therefore discussing possible control strategies, such as vaccination, drug medication, quarantine and so on [6], [7], [8], [9], [10], [11], [12], [13], [14], [15]. Moreover, epidemic models can be applied in different scenarios, such as biological and social networks, as in [16], where the authors formulated an SIS model with the infection transmission delay, or in [17], where again the SIS case was studied introducing a switching network for the epidemic modelling. Pioneering studies on epidemic models with switching parameters and dynamics are presented in [18]. The prediction of epidemic spread behaviour is analysed in [19] in a rather general context to propose a network architecture for resource usage prediction.

The Human Immunodeficiency Virus (HIV) is responsible for the Acquired Immuno Deficiency Syndrome (AIDS); it infects cells of the immune system, destroying or impairing their function: the immune system becomes weaker, and the person is more susceptible to infections. The AIDS is the most advanced stage of the HIV infection and it can be reached in 10-15 years from the infection. It can be transmitted only by some body fluids: blood, semen, pre-seminal fluid, rectal fluid, vaginal fluid, and breast milk; therefore, it is mainly transmitted by a subject during unprotected sex, sharing needles or syringes and, less commonly, by oral sex, blood transfusion or from mother to child during pregnancy or breastfeeding. The data from the World Health Organization (WHO) confirm the seriousness of the HIV being 1.1 million the number of people died of AIDS-related illnesses worldwide in 2016 (last update); in the same period the number of people living with HIV-AIDS is about 36.7 million. A significant aspect that contributed to the inspiration of this paper is that only 54% of people with HIV is aware of the infection. Currently no vaccine exists and the treatment consists in standard AntiRetroviral Therapy (ART) to maximally suppress the HIV virus and stop its progression; using condoms and regular blood analysis on subjects belonging to risk-categories could help in contrasting the spread of this virus. Available models of the HIV-AIDS infection may be divided into two main groups, one focused on the dynamics at cells level [20], [21], [7], [22] and one dealing with the dynamics of subjects interactions [23], [24], [25], [26], [27], [28]. Regarding the first approach, the attention is focused on the essential components of the immune system, the CD4 T-cells. An HIV patient is expected to develop AIDS; he is classified as an AIDS patient when he has less than 200 CD4 T-cells in $mm^3$ of blood [29]. A Long Term Non Progressor (LTNP) is the status of the HIV patients who still have enough CD4 T-cells to contrast the HIV and the other infections, never developing the AIDS; they represent less than 1% of the HIV infected patients, [30], [31]. In [32], the two equilibrium points, LTNP and AIDS, functions of medication, are studied in the state space, showing their asymptotic stability. The aim is to drive, with medication, the state of the patient into the LTNP region of attraction. In this framework, the medication strategy is studied in [7] and [20]; in particular, in [20] a state dependent coefficient that weights differently the control according to the number of the infected cells is introduced. These considerations inspired the works [14] and [33], where control strategies with a state dependent switching cost index are introduced, referring to a SIR and an HIV model respectively. As far as the approach concerning the relations between subjects, generally four classes are introduced: the Susceptible one, $S$, with the subjects which are not yet infected but may get the virus; the Infected one, $I$, with the individuals that still do not know their illness condition; the HIV group with the pre-AIDS patients, $P$, and the one with the AIDS patients, $A$. In [23], this four-classes model considers a constant individuals inflow in the $S$ and the $I$ classes and

P. Di Giamberardino, L. Compagnucci, C. De Giorgi and D. Iacoviello are with the Dept. of Computer, Control and Management Engineering Antonio Ruberti, Sapienza University of Rome, Via Ariosto 25, 00185, Rome, Italy emails: paolo.digiamberardino@uniroma1.it, compagnucci.luca@gmail.com, chiaradegiorgi.x@gmail.com, iacoviello@dis.uniroma1.it

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a natural death is introduced in addition to the one due to HIV/AIDS; the transitions considered are from $S$ to $I$ for the unconscious infected subjects, from $I$ to $P$ or $A$ depending on the diagnosed infection characteristics, and from $P$ to $A$. Subjects in LTNP status are not here considered. In [34], a description of a dynamic compartmental simulation model for Botswana and India considers sex behavioural compartment, high risk and low risk, to identify the best strategies for preventing the spread of the HIV/AIDS. They are focused on a behavioural intervention on female sex workers, a program for the treatment of sexually transmitted infections, for the prevention of mother-to-child transmission, for medication of the entire population, and one specific for sex workers only. In [26] the four-classes model is analysed to determine the best strategy to control the disease burden; the conclusions enhanced the importance of controlling the effective contact rate of the infected population and of isolating the susceptible part. The HIV/AIDS models present nonlinear interactions; the mathematical analysis of the global dynamics of the spread is faced in [28]. The main finding is that, whereas treatment can help in containing the HIV/AIDS spread, it can also lead to the evolution of drug resistance, which can reverse the benefits of treatment. Nevertheless, treatment is useful in reducing the infection and the mortality in infected individuals. Recently, in [25] a new model, the SICA, has been proposed, focusing on the antiretroviral therapy; four classes are introduced: the susceptible subjects (S), the infected individuals (I) with no symptoms of AIDS, the infected subjects (C) under ART treatment, the infected patients with AIDS (A). The analysis on the nonlinear SICA model showed the importance of an early ART treatment for the infected subjects, thus putting in evidence the urgency of a fast HIV/AIDS detection. In [35], the effects of people interactions in case of extreme events are studied when facing dangerous and risky actions; the uncertainties on behaviour diffusion are considered in the rather general contest of social networks in [36], where the differences with respect to virus spreading are pointed out. As it will be discussed in Section II, in this paper two classes of non-infected subjects are introduced: the subjects that are not aware of irresponsible acts and, therefore, could become infected, and the subjects representing the wise population that, suitably informed, avoid dangerous acts. The infected subjects are divided into three groups: the individuals that are still not aware of being infected, and therefore are responsible of the spreading of the HIV/AIDS, and the infected aware patients, which may be in the HIV class or in the AIDS one. The definition of the health care systems represents the first step when planning intervention. A framework in which such systems can be studied is proposed in [37] where a metamodel with different views is proposed: care of patients, resource and organization.

In the present paper, three different control actions are introduced, consistent with the three levels of intervention suggested by the World Health Organization:

- primary prevention: it is designed for healthy people to reduce the possibility of new infections; in our approach it corresponds to the information effort aiming at using wise attitudes to prevent the non-infected subjects to acquire the infection;
- secondary prevention: it is devoted to a fast identification of new infections and risk conditions to improve the percentage of subjects that become aware of their illness by regular blood tests;
- medication: it is the cure to the aware infected patients.

The costs of primary and secondary preventions represent an immediate economic effort, whereas their effects could be noted only in the future. Therefore, the preliminary study on the effects of the different strategies is useful in the cost/benefit analysis.

The main points of this paper may be summarized as follows:

- a new model of the HIV/AIDS spread is proposed: it has been inspired by the simple consideration that all the subjects in the population are susceptible and with wise behaviours the spread could stop immediately, since the HIV may be transmitted only by body fluids contact. Therefore, the important distinction between subjects with wise behaviours and the others has been modelled;
- a deep analysis of the proposed model is presented, showing its consistency with known results [23], [30]. It could represent the basis for further implementations, as it will be outlined in the Conclusions;
- interventions coherent with the suggestions of the World Health Organization are introduced and their effects on the spread are studied. Of course, the medication on the AIDS patients does not influence the spread in the proposed model, since it is assumed that, after a positive diagnosis, a patient can infect nobody. However, it is introduced for sake of completeness;
- the intuitive consideration that a fast HIV/AIDS recognition could help in reducing the spread has been confirmed by the numerical results.

The paper is organized as follows. In Section II, the proposed mathematical model is illustrated and motivated, while in Section III its stability properties are studied. In Section IV the numerical analysis of the dynamics is presented and the results obtained are discussed. Conclusions and future developments are outlined is Section V.

## II. The mathematical model

As presented in the Introduction, when dealing with the HIV infection analysis and control the most common choice for its mathematical modelling makes use of the cellular and the virus dynamics and interactions [21], [20]. An epidemiological approach is only sometimes followed, mainly to put in evidence some peculiar characteristics of its dynamics. One of the most interesting among them is proposed in [23], where some interesting aspects in the epidemic transmission and diffusion are introduced and investigated. Starting from its analysis, recalled in the next Subsection II-A, the mathematical model here proposed is derived and discussed in Subsection II-B.

### A. The reference mathematical model

In [23], the total population is denoted by $N(t)$. It contains the susceptible individuals $S(t)$, the ones that can become infected

through sexual contacts, and three kinds of infected individuals: the subjects unconscious of their illness status $I(t)$, the HIV diagnosed subjects $P(t)$, and the AIDS diagnosed ones $A(t)$. The hypothesis there adopted is that the infected individuals $I(t)$ and $P(t)$ can transmit the infection to the healthy ones, while for the $A(t)$ no risky sexual relationship is allowed. As a final step, the unconscious infected individuals, once diagnosed, transit to one of the two classes $P$ or $A$, according to a prefixed probability. Then, the dynamics there proposed, corresponding to such choices, is

$$\begin{aligned}
\dot{S}(t) &= Q_1 - dS(t) - \frac{\beta c S(t) I(t)}{N(t)} - \frac{\beta' c S(t) P(t)}{N(t)} \\
\dot{I}(t) &= Q_2 + \frac{\beta c S(t) I(t)}{N(t)} + \frac{\beta' c S(t) P(t)}{N(t)} - (\delta + d) I(t) \\
\dot{P}(t) &= \varepsilon \delta I(t) - (\alpha_1 + d) P(t) \\
\dot{A}(t) &= (1-\varepsilon)\delta I(t) + \alpha_1 P(t) - (\alpha + d) A(t) \qquad (1)
\end{aligned}$$

The parameters in (1) are: $Q_1$, the rate of the uninfected external population incoming, and $Q_2$, the same for the infected ones; $d$, the rate of natural death, not related to the infection; $\beta$ the sexual contact rate between $S(t)$ and $I(t)$ and $\beta'$ the same between $S(t)$ and $P(t)$, with $\beta \gg \beta'$; $c$, the average number of sexual partners; $\delta$, the rate of movement from infectious class due to the symptomatic effects; $\varepsilon$, the fraction of the natural evolution of the illness toward the HIV infection; $\alpha_1$, the rate of the HIV infected subjects moving to the AIDS class; $\alpha$ the constant death rate due to the AIDS infection. In this model, the external actions introduced are the prefixed constant external rates of population variations $Q_1$ and $Q_2$.

### *B. The proposed model*

The model here proposed integrates the one recalled in Subsection II-A in order to better explain particular realistic behaviours of the phenomenon and to take into account the suggestions from the World Health Organization. The first change in the model is the splitting of the healthy class $S$ into two groups of non infected persons: the first, $S_1$, containing people that are not aware of the risks of the infection; the second, $S_2$, the part of healthy population which, suitably informed, gives a great attention to the partners and to the protections. As a consequence, only the interaction between the individuals in $S_1$ and the ones in $I$ can produce new infected subjects. There are two kind of interactions between $S_1$ and $S_2$ introduced in the proposed model. The first one represents the possibility that, under a driven informative campaign, a fraction of unaware healthy persons in $S_1$ moves to the class $S_2$. The effective strength of the information campaign is represented by the external input $u_1(t)$ and can be assumed dependent on the amount of the (economic) resources invested and on the sensibility of the population with respect to the risk; therefore, a term of the form $S_1(t)u_1(t)$ is introduced. The second interaction is represented by the fact that some individuals, despite well informed, may accidentally become potentially at risk of infection. This contribution is considered proportional to the number of individuals $S_2(t)$ and its effect is modelled by the term $\gamma S_2(t)$. The flux of new individuals is here supposed to be represented by uninfected ones only (growing children, checked people and so on), without hypothesis on their awareness of the unsafe contacts, so that only $Q_1$ is present, renamed as $Q$. A further hypothesis is that the individual consciousness and the social prophylaxis lead any infected person who knows his/her status to have only safe relationships. This means that $\beta' = 0$ is assumed. Then, the following two dynamical equations model the uninfected population:

$$\begin{aligned}
\dot{S}_1(t) &= Q - dS_1(t) - \frac{\beta c S_1(t) I(t)}{N_c(t)} - S_1(t)u_1(t) + \gamma S_2(t) \\
\dot{S}_2(t) &= S_1(t)u_1(t) - (\gamma + d)\, S_2(t) \qquad (2)
\end{aligned}$$

Note that the term $N(t)$ has been changed in $N_c(t) = S_1(t) + S_2(t) + I(t)$ in order to consider the subdivision of $S(t)$ into $S_1(t)$ and $S_2(t)$ and the fact that $P(t)$ and $A(t)$ are not involved in risky sexual relationships. The second improvement concerns the transition from $I(t)$ to the diagnosed $P(t)$ and $A(t)$. A natural transition, represented by the term $\delta I(t)$, is also present in (1). Here, in addition, a second input $u_2(t)$ is introduced to represent the external contribution to a test campaign aiming at discovering the infection as soon as possible. The corresponding action considers the total amount of candidate subjects $N_c(t)$ and defines the quantity $\frac{u_2(t)}{N_c(t)}$ as the cost for each individual. The costs for $S_1(t)$, $S_2(t)$ and $I(t)$ are given by $S_1(t)\frac{u_2(t)}{N_c(t)}$, $S_2(t)\frac{u_2(t)}{N_c(t)}$ and $I(t)\frac{u_2(t)}{N_c(t)}$, respectively. The first two terms do not contribute to the dynamics, since they represent the cost to confirm that an uninfected person is actually safe. On the contrary, the third one contributes to reveal the infected status and to transfer such individuals to the diagnosed classes. Representing this effect as a proportional contribution, the term $-\psi I(t)\frac{u_2(t)}{N_c(t)}$ is introduced in the $I(t)$ dynamics to model such a transition. The decrement in $\dot{I}(t)$ must correspond to how much $\dot{P}(t)$ and $\dot{A}(t)$ increase, dividing such quantity in a fraction $\phi$ for $\dot{P}(t)$ and $(1-\phi)$ for $\dot{A}(t)$, $0 \leq \phi \leq 1$. Then, the equations added to (2), under these considerations, are

$$\begin{aligned}
\dot{I}(t) &= \frac{\beta c S_1(t) I(t)}{N_c(t)} - (d+\delta) I(t) - \psi I(t) \frac{u_2(t)}{N_c(t)} \\
\dot{P}(t) &= \varepsilon \delta I(t) - (\alpha_1 + d) P(t) + \phi \psi I(t) \frac{u_2(t)}{N_c(t)} \\
\dot{A}(t) &= (1-\varepsilon)\delta I(t) + \alpha_1 P(t) - (\alpha + d) A(t) + \\
&\quad + (1-\phi)\psi I(t) \frac{u_2(t)}{N_c(t)} \qquad (3)
\end{aligned}$$

Finally, a term taking into account the degeneration of the illness from HIV to AIDS is introduced. The natural transition is modelled, as in (1), by the proportional term depending on the parameter $\alpha_1$. In addition, a form of therapy is introduced by the action of the third input $u_3(t)$, so that the transition from HIV to AIDS becomes dependent also on such an input, proportionally to the number of individuals $P(t)$, thus reducing the natural transition given by $\alpha_1 P(t)$. Therefore, the term $P(t)u_3(t)$ is introduced.

Under all these considerations, the whole model here proposed is

$$
\begin{aligned}
\dot{S}_1(t) &= Q - dS_1(t) - \frac{c\beta S_1(t)I(t)}{N_c(t)} + \gamma S_2(t) - S_1(t)u_1(t) \\
\dot{S}_2(t) &= -(\gamma + d)S_2(t) + S_1(t)u_1(t) \\
\dot{I}(t) &= \frac{c\beta S_1(t)I(t)}{N_c(t)} - (d+\delta)I(t) - \psi\frac{I(t)}{N_c(t)}u_2(t) \\
\dot{P}(t) &= \varepsilon\delta I(t) - (\alpha_1 + d)P(t) + \phi\psi\frac{I(t)}{N_c(t)}u_2(t) + \\
&\quad + P(t)u_3(t) \\
\dot{A}(t) &= (1-\varepsilon)\delta I(t) + \alpha_1 P(t) - (\alpha + d)A(t) + \\
&\quad + (1-\phi)\psi\frac{I(t)}{N_c(t)}u_2(t) - P(t)u_3(t)
\end{aligned} \tag{4}
$$

that can be expressed in the compact form

$$
\dot{\xi} = F(\xi) + g_1(\xi)u_1 + g_2(\xi)u_2 + g_3(\xi)u_3 \tag{5}
$$

where $\xi = \begin{pmatrix} S_1 & S_2 & I & P & A \end{pmatrix}^T$ denotes the five dimensional state vector of (4), and

$$
F(\cdot) = \begin{pmatrix} f_1(\cdot) \\ f_2(\cdot) \\ f_3(\cdot) \\ f_4(\cdot) \\ f_5(\cdot) \end{pmatrix} = \begin{pmatrix} Q - dS_1 - \frac{c\beta S_1 I}{N_c} + \gamma S_2 \\ -(\gamma + d)S_2 \\ \frac{c\beta S_1 I}{N_c} - (d+\delta)I \\ \varepsilon\delta I - (\alpha_1 + d)P \\ (1-\varepsilon)\delta I + \alpha_1 P - (\alpha + d)A \end{pmatrix}
$$

$$
g_1(\cdot) = \begin{pmatrix} -S_1 \\ S_1 \\ 0 \\ 0 \\ 0 \end{pmatrix};\ g_2(\cdot) = \begin{pmatrix} 0 \\ 0 \\ -\frac{\psi I}{N_c} \\ \frac{\phi\psi I}{N_c} \\ \frac{(1-\phi)\psi I}{N_c} \end{pmatrix};\ g_3(\cdot) = \begin{pmatrix} 0 \\ 0 \\ 0 \\ P \\ -P \end{pmatrix}
$$

## III. Equilibria and stability analysis

In this Section, the existence of equilibrium conditions for the dynamics (4) and the corresponding stability properties are studied.

### A. Equilibria computation

The computation of the equilibrium points is performed by solving the equation

$$
F(\xi^e) = 0 \tag{6}
$$

It can be noted that, under the hypothesis of absence of relationships between subjects with the infection diagnosed $P(t)$ and $A(t)$, and the other ones, the uncontrolled dynamics can be decomposed into a first three dimensional system, whose state variables are $S_1(t)$, $S_2(t)$ and $I(t)$, and a two dimensional one, with state variables $P(t)$ and $A(t)$. The second system is influenced by the first one through the number of infected subjects $I(t)$. A scheme is depicted in Figure 1. Accordingly to this decomposition, the state space $\xi \in I\!R^5$ can be partitioned as $\xi = \begin{pmatrix} \xi_1^T & \xi_2^T \end{pmatrix}^T$ where $\xi_1 = \begin{pmatrix} S_1 & S_2 & I \end{pmatrix}^T \in I\!R^3$ and $\xi_2 = \begin{pmatrix} P & A \end{pmatrix}^T \in I\!R^2$ and, correspondingly,

$$
F(\xi) = \begin{pmatrix} F_1(\xi_1) \\ F_2(\xi_1, \xi_2) \end{pmatrix} = \begin{pmatrix} F_1(S_1, S_2, I) \\ F_2(I; P, A) \end{pmatrix}
$$

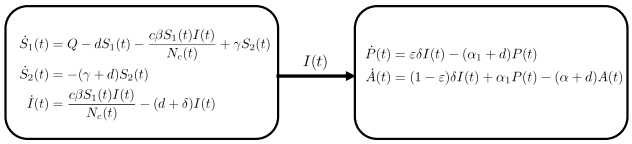


Fig. 1: Decomposition of the uncontrolled dynamics

Then, the analysis of the equilibrium points and their stability properties can be carried on referring to the two systems separately. The computation of the equilibrium points for the first block yields to the two solutions

$$
\xi^e_{1,1} = \begin{pmatrix} S_1^{e,1} \\ S_2^{e,1} \\ I^{e,1} \end{pmatrix} = \begin{pmatrix} \frac{1}{d} \\ 0 \\ 0 \end{pmatrix} Q,
$$

$$
\xi^e_{1,2} = \begin{pmatrix} S_1^{e,2} \\ S_2^{e,2} \\ I^{e,2} \end{pmatrix} = \begin{pmatrix} \frac{1}{c\beta - \delta} \\ 0 \\ \frac{c\beta - (d+\delta)}{(d+\delta)(c\beta - \delta)} \end{pmatrix} Q
$$

The existence and the positiveness of $\xi^e_{1,2}$ depend on the fulfillment of the condition

$$
c\beta - (d + \delta) > 0 \tag{7}
$$

Note that if $c\beta - (d+\delta) = 0$, then $\xi^e_{1,2} = \xi^e_{1,1}$; this fact is deeply analysed hereinafter, when the stability of such equilibria is studied. As the value of $c\beta - (d+\delta)$ increases, the second equilibrium $\xi^e_{1,2}$ becomes feasible and moves away from $\xi^e_{1,1}$. In order to study what happens when $c\beta - (d+\delta)$ becomes very large, the computation of

$$
\lim_{c\beta - (d+\delta) \to +\infty} \xi^e_{1,2} \tag{8}
$$

may be useful. Such a computation can be simplified observing that, since $d \geq 0$ and $\delta \geq 0$, the quantity $c\beta - (d+\delta)$ indefinitely increases only if $c\beta$ does. So, instead of (8), one can compute,

$$
\lim_{c\beta \to +\infty} \xi^e_{1,2} = \begin{pmatrix} 0 \\ 0 \\ \frac{1}{(d+\delta)} \end{pmatrix} Q
$$

This shows that, being $c\beta$ the coefficient regulating the infection propagation, its increment produces a higher number of infected individuals and, correspondently, a decrement of the uninfected subjects; at the limit situation, all the population is infected. As far as the second block is concerned, equation (6), restricted to the $P - A$ subspace, yields

$$
\xi^e_2 = \begin{pmatrix} P^e \\ A^e \end{pmatrix} = \begin{pmatrix} 0 \\ 0 \end{pmatrix}
$$

where the presence of the state variable $I(t)$ is assumed as an external input acting from the first subsystem to the second one and then, in this analysis, it can be set equal to zero.

*B. Stability analysis*

In order to study the stability properties of the equilibrium points, the results based on the local linear approximation are used. Due to the structure of the full dynamics, each of the two subsystems are here addressed separately. Starting with the first block, it is required the computation of the dynamical matrices of the linear approximations in a neighbourhood of each equilibrium point, $\xi^e_{1,1}$ and $\xi^e_{1,2}$. To this purpose, the Jacobian matrix $\frac{\partial F_1}{\partial \xi_1}$ must be computed and then evaluated at each equilibrium point. The classical well known structure for the Jacobian is

$$\frac{\partial F_1}{\partial \xi_1} = \begin{pmatrix} \frac{\partial f_1}{\partial S_1} & \frac{\partial f_1}{\partial S_2} & \frac{\partial f_1}{\partial I} \\ \frac{\partial f_2}{\partial S_1} & \frac{\partial f_2}{\partial S_2} & \frac{\partial f_2}{\partial I} \\ \frac{\partial f_3}{\partial S_1} & \frac{\partial f_3}{\partial S_2} & \frac{\partial f_3}{\partial I} \end{pmatrix} \tag{9}$$

The nonzero terms in (9) are

$$\begin{aligned} &\frac{\partial f_1}{\partial S_1} = -d - \frac{c\beta I}{N_c} + \frac{c\beta S_1 I}{(N_c)^2}, \quad \frac{\partial f_1}{\partial S_2} = \gamma + \frac{c\beta S_1 I}{(N_c)^2} \\ &\frac{\partial f_1}{\partial I} = -\frac{c\beta S_1}{N_c} + \frac{c\beta S_1 I}{(N_c)^2}, \quad \frac{\partial f_2}{\partial S_2} = -(\gamma + d) \\ &\frac{\partial f_3}{\partial S_1} = \frac{c\beta I}{N_c} - \frac{c\beta S_1 I}{(N_c)^2}, \quad \frac{\partial f_3}{\partial S_2} = -\frac{c\beta S_1 I}{(N_c)^2} \\ &\frac{\partial f_3}{\partial I} = -(d+\delta) + \frac{c\beta S_1}{N_c} - \frac{c\beta S_1 I}{(N_c)^2} \end{aligned}$$

Evaluating (9) at $\xi^e_{1,1}$, one gets the matrix

$$A_1^1 = \left(\begin{array}{cc|c} -d & \gamma & -c\beta \\ 0 & -(\gamma+d) & 0 \\ \hline 0 & 0 & c\beta - (\delta + d) \end{array}\right) \tag{10}$$

whose eigenvalues are $\lambda_1 = -d$, $\lambda_2 = -(\gamma + d)$, $\lambda_3 = c\beta - (d + \delta)$ and then the equilibrium point $\xi^e_{1,1}$ is locally asymptotically stable under the condition

$$c\beta - (d + \delta) < 0 \tag{11}$$

If condition (11) is not satisfied, the equilibrium point $\xi^e_{1,1}$ is unstable. However, as it can be suggested by the components of the eigenvectors $v_1$ and $v_2$ of the linear approximation,

$$v_1 = \begin{pmatrix} 1 \\ 0 \\ 0 \end{pmatrix}, v_2 = \begin{pmatrix} 0 \\ 1 \\ 0 \end{pmatrix}, v_3 = \begin{pmatrix} 1 \\ 0 \\ \frac{-\gamma - c\beta + d + \delta}{c\beta} \end{pmatrix}$$

for any initial condition in the two dimensional manifold

$$\Sigma = \left\{ \begin{pmatrix} S_1 & S_2 & I \end{pmatrix}^T \in \Re^3 \, : \, I = 0 \right\}$$

the corresponding trajectory lies in the $S_1 - S_2$ subspace and converges to the point $\xi^e_{1,1}$. In fact, from (4), the autonomous dynamic of the $I(t)$ variable, rewritten as

$$\dot{I}(t) = I(t) \left( \frac{c\beta S_1(t)}{N_c(t)} - (d + \delta) \right)$$

shows that if $I(t_0) = 0$, then $\dot{I}(t) = 0$ and $I(t) = const = I(t_0) = 0$. Under this hypothesis, the dynamics of $S_1(t)$ and $S_2(t)$ under zero inputs become

$$\begin{aligned} \dot{S}_1(t) &= Q - dS_1(t) + \gamma S_2(t) \\ \dot{S}_2(t) &= -(\gamma + d) S_2(t) \end{aligned}$$

that is, a linear structure with dynamical matrix

$$A_0 = \begin{pmatrix} -d & \gamma \\ 0 & -(\gamma - d) \end{pmatrix}$$

Its integration, starting from $S_1(0)$ and $S_2(0)$, due to the nonsingularity of $A_0$, gives

$$\begin{pmatrix} S_1(t) \\ S_2(t) \end{pmatrix} = e^{A_0 t} \left( \begin{pmatrix} S_1(0) \\ S_2(0) \end{pmatrix} + A_0^{-1} \begin{pmatrix} Q \\ 0 \end{pmatrix} \right) - A_0^{-1} \begin{pmatrix} Q \\ 0 \end{pmatrix}$$

with

$$A_0^{-1} \begin{pmatrix} Q \\ 0 \end{pmatrix} = \frac{1}{d(\gamma - d)} \begin{pmatrix} -(\gamma - d) & -\gamma \\ 0 & -d \end{pmatrix} \begin{pmatrix} Q \\ 0 \end{pmatrix} = \begin{pmatrix} -\frac{Q}{d} \\ 0 \end{pmatrix}$$

The conclusion is that if

$$\begin{pmatrix} S_1(0) \\ S_2(0) \end{pmatrix} = -A_0^{-1} \begin{pmatrix} Q \\ 0 \end{pmatrix} = \begin{pmatrix} \frac{Q}{d} \\ 0 \end{pmatrix}$$

the time evolution is

$$\begin{pmatrix} S_1(t) \\ S_2(t) \end{pmatrix} = -A_0^{-1} \begin{pmatrix} Q \\ 0 \end{pmatrix} = \begin{pmatrix} \frac{Q}{d} \\ 0 \end{pmatrix}$$

Otherwise,

$$\lim_{t \to \infty} \begin{pmatrix} S_1(t) \\ S_2(t) \end{pmatrix} = -A_0^{-1} \begin{pmatrix} Q \\ 0 \end{pmatrix} = \begin{pmatrix} \frac{Q}{d} \\ 0 \end{pmatrix}$$

which proves that the equilibrium point $\begin{pmatrix} S_1^{e,0} & S_2^{e,0} \end{pmatrix}^T = \begin{pmatrix} \frac{Q}{d} & 0 \end{pmatrix}^T$, projection of $\xi^e_{1,1}$ on the $S_1 - S_2$ subspace, is an asymptotic stable one, once the dynamics is restricted to such a subspace.

To complete the stability analysis, the second equilibrium point $\xi^e_{1,2}$ is now considered. Then, the Jacobian (9) has to be evaluated at the equilibrium point $\xi^e_{1,2}$; setting $H = \frac{d+\delta}{c\beta}$ and $K = 1 - H$, one gets the matrix

$$A_1^2 = \begin{pmatrix} -d - c\beta K^2 & \gamma + c\beta HK & -c\beta H^2 \\ 0 & -(\gamma + d) & 0 \\ c\beta K^2 & -c\beta HK & -c\beta HK \end{pmatrix} \tag{12}$$

The structure yields the eigenvalue $\lambda_1 = -(\gamma + d)$ whereas for $\lambda_2$ and $\lambda_3$ one has to find the solutions of

$$\lambda^2 + (c\beta - \delta)\lambda + \frac{(d+\delta)(c\beta - (d+\delta))(c\beta - \delta)}{c\beta} = 0 \tag{13}$$

from which the eigenvalues $\lambda_2$ and $\lambda_3$ can be numerically computed once the values of the set of parameters is known. This will be done in Section IV for the case under investigation. Here, as far as the stability of $\xi^e_{1,2}$ is concerned, the two solutions of (13) have negative real parts, and then $\xi^e_{1,2}$ is stable, if and only if (by Descartes' rule of signs)

$$\begin{aligned} c\beta - \delta &> 0 \\ (d+\delta)(c\beta - (d+\delta))(c\beta - \delta) &> 0 \end{aligned} \tag{14}$$

Since $d + \delta > 0$, (14) is equivalent to (7) and then $\xi^e_{1,2}$ is locally asymptotically stable if and only if (7) holds. It is interesting to observe that if $\xi^e_{1,1}$ is the unique equilibrium point (i.e. (7) is not satisfied), it is locally asymptotically stable, while the second equilibrium point $\xi^e_{1,2}$ exists, and is asymptotically stable, if and only if $\xi^e_{1,1}$ is unstable. If $c\beta = d + \delta$, the matrix (10) of the linear approximation in a neighbourhood of $\xi^e_{1,1}$ has one eigenvalue equal to zero. This fact suggests to study the

presence of a bifurcation once the control parameter $\bar{\beta} = c\beta$ is considered. In fact, it can be noted that the solutions $\xi^e_{1,1}$ and $\xi^e_{1,2}$ always exist and they coincide when $\bar{\beta} = d + \delta$, i.e. when there is the null eigenvalue. This means that a Stationary Bifurcation is present; in this case it is a Simple Bifurcation, since the augmented Jacobian, evaluated at $\xi^e_{1,1}$ with $c\beta = d + \delta$,

$$\left( \begin{array}{cc} \frac{\partial F_1}{\partial \xi_1} & \frac{\partial F_1}{\partial \bar{\beta}} \end{array} \right)_{\xi_1 = \xi^e_{1,1}} = \left( \begin{array}{ccc|c} -d & \gamma & -c\beta & 0 \\ 0 & -(\gamma + d) & 0 & 0 \\ 0 & 0 & 0 & 0 \end{array} \right)$$

has rank $\rho < 3$. In Figure 2 it is depicted the diagram where the Transcritical Bifurcation is reported with reference to the state variable $I$, while in Figure 3 the same is performed for the state variable $S_1$. Here, the numerical values of the parameters given in Table I are used for simplicity of interpretation.

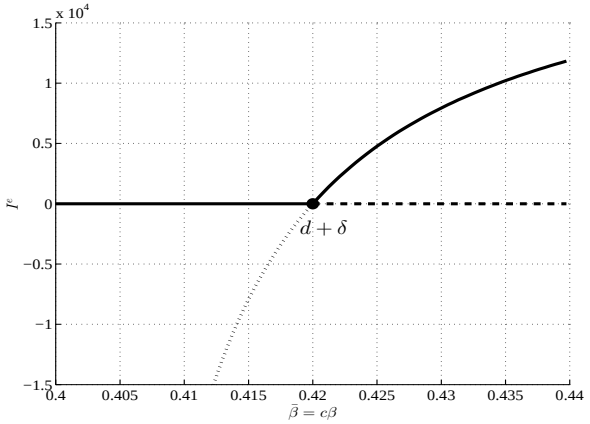


Fig. 2: Transcritical bifurcation diagram for component $I(t)$

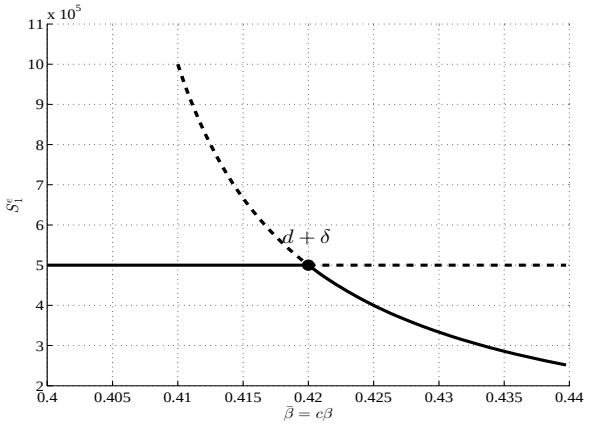


Fig. 3: Transcritical bifurcation diagram for component $S_1(t)$

Figure 2 shows with solid lines the stable equilibrium values for $I^e$ when $I^e = I^{e,1} = 0$ for $\bar{\beta} < d + \delta$ and $I^e = I^{e,2} = \frac{c\beta - (d+\delta)}{(d+\delta)(c\beta - \delta)}$ for $\bar{\beta} > d + \delta$. The dotted part of $I^{e,2}$, for $\bar{\beta} < d + \delta$, denotes the unfeasible (negative) unstable solution, while the dashed part of $I^{e,1}$ marks the unstable part of the solution. Also in Figure 3 the solid lines denote the stable equilibrium values, while the dashed ones indicate the unstable solutions. Such a behaviour depends on the parameters $c\beta$, $d$ and $\delta$. On the basis of their meanings, the existence and the stability of the equilibrium points depend on the values of the probability of the infection transmission ($c\beta$) and on the rate of reduction of the infected subjects $I(t)$ by natural death ($d$) or by infection diagnosis ($\delta$). The stability analysis of the equilibrium point for the second subsystem can be carried on computing the Jacobian

$$\frac{\partial F_2}{\partial \xi_2} = \left( \begin{array}{cc} \frac{\partial f_4}{\partial P} & \frac{\partial f_4}{\partial A} \\ \frac{\partial f_5}{\partial P} & \frac{\partial f_5}{\partial A} \end{array} \right) \tag{15}$$

Since its nonzero terms are

$$\frac{\partial f_4}{\partial P} = -(\alpha_1 + d), \quad \frac{\partial f_5}{\partial P} = \alpha_1, \quad \frac{\partial f_5}{\partial A} = -(\alpha + d)$$

matrix (15), evaluated at $\xi^e_2$, becomes

$$A_2 = \left( \begin{array}{cc} -(\alpha_1 + d) & 0 \\ \alpha_1 & -(\alpha + d) \end{array} \right) \tag{16}$$

whose eigenvalues are $\lambda_1 = -(\alpha_1 + d)$ and $\lambda_2 = -(\alpha + d)$. It is worth noting that, since $I(t)$ acts as an input for the second subsystem, when the equilibrium $I^{e,2} \neq 0$ for the first subsystem is considered, $P(t)$ and $A(t)$ assume values different from zero, corresponding to the steady state condition under constant feed $I^{e,2}$.

## IV. Numerical analysis of the dynamics

The results obtained in the previous Section III are now applied to the case study here considered, assuming the model parameters as in Table I

| Parameter | Value | | Parameter | Value |
|---|---|---|---|---|
| $d$ | 0.02 | | $c$ | 10 |
| $\beta$ | 0.15 | | $\gamma$ | 0.2 |
| $\delta$ | 0.4 | | $\psi$ | 100000 |
| $\varepsilon$ | 0.6 | | $\alpha_1$ | 0.5 |
| $\phi$ | 0.95 | | $\alpha$ | 1 |

TABLE I: Numerical values used for the model parameters

Whenever applicable, the same values for the parameters as in [23] are used. The constant value $Q = 10000$ is assumed as the flow of new individuals in equations (4). This choice does not affect the system analysis, being all the equilibrium points proportional to such a value.

### *A. Numerical values of equilibrium points and their stability analysis*

In the numerical evaluation, the structure of the system, a series of a three dimensional dynamics with a two dimensional one, should be considered. The connection is represented by the action of $I(t)$, that can be assumed as the output of the first block and the input of the second one. Being the state evolution of the second subsystem dependent on $I(t)$, for any consideration about the values assumed by the two state variables $P(t)$ and $A(t)$ it is necessary to know its behaviour. These considerations suggest to study the full system for simplifying the numerical analysis and the results interpretation. This is the approach followed in this Section, in which all the results showed and discussed refer to the full five dimensional

dynamics. Since condition (7) is fundamental for the stability characteristics, it is important to observe that in the present case $c\beta - (d+\delta) = 1.08$ and the condition (7) is satisfied. Then, according to what stated in Subsection III-A, it is possible to claim that for the first subsystem there are two equilibrium points $\xi^e_{1,1}$ and $\xi^e_{1,2}$, the first one unstable and the second one locally asymptotically stable. The numerical values for the two equilibria are $\xi^e_{1,1} = (\,500000 \quad 0 \quad 0\,)^T$ and $\xi^e_{1,2} = (\,9100 \quad 0 \quad 23400\,)^T$. The corresponding steady state values for the second subsystem are $\xi^e_{2,1} = \xi_2|_{\xi^e_{1,1}} = (\,0 \quad 0\,)^T$ and $\xi^e_{2,2} = \xi_2|_{\xi^e_{1,2}} = (\,10800 \quad 8800\,)^T$

Then, in the sequel the two full equilibrium points

$$\xi^e_1 = \begin{pmatrix} \xi^e_{1,1} \\ \xi^e_{2,1} \end{pmatrix}, \qquad \xi^e_2 = \begin{pmatrix} \xi^e_{1,2} \\ \xi^e_{2,2} \end{pmatrix} \tag{17}$$

are considered.

The influence of $c\beta$ on $\xi^e_2$, highlighted in Subsection III-A, can be numerically evidenced computing this equilibrium point for different increasing values of $c\beta$, starting from $d+\delta = 0.42$. The results are depicted in the Figure 4 for the $S^e_1$ and $S^e_2$ components and in Figure 5 for the $I^e$, $P^e$ and $A^e$ ones.

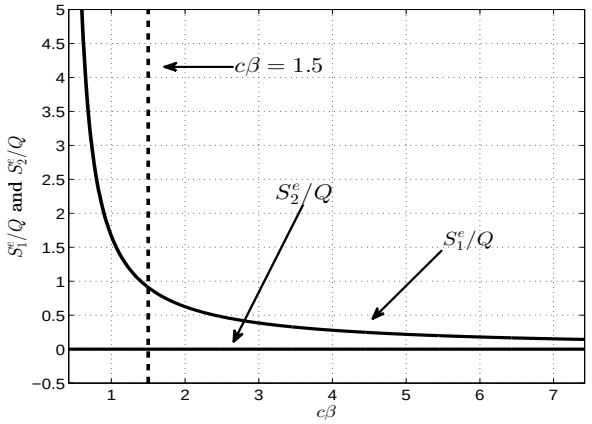


Fig. 4: Dependency of $S^e_1$ and $S^e_2$ from $c\beta$

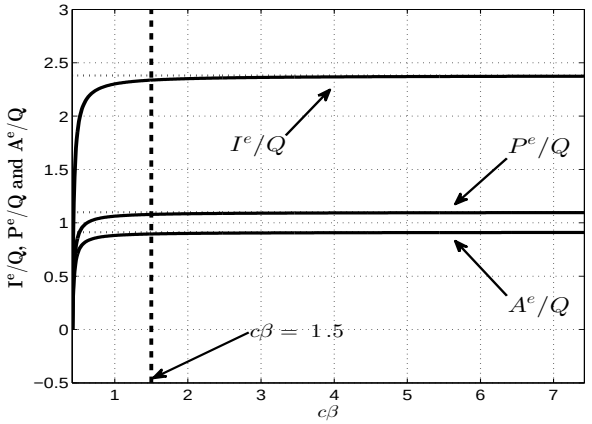


Fig. 5: Dependency of $I^e$, $P^e$ and $A^e$ from $c\beta$

The variations of the equilibrium state components $S^e_2$, $I^e$, $P^e$ and $A^e$ with respect to (17) are evident: the only equilibrium component highly affected by the value of $c\beta - (d+\delta)$ is the first one, $S^e_1$. As the product $c\beta$ increases, the second equilibrium point asymptotically tends to

$$\xi^e_2 = (\,0 \quad 0 \quad 23800 \quad 11000 \quad 9100\,)^T$$

In order to verify the unstability condition for the equilibrium point $\xi^e_1$, the numerical computation of the Jacobian evaluated in the first equilibrium point yields

$$A_1 = \left.\frac{\partial F}{\partial \xi}\right|_{\xi=\xi^e_1} = \begin{pmatrix} A^1_1 & 0 \\ * & A_2 \end{pmatrix} =$$

$$= \left(\begin{array}{ccc|cc} -0.02 & 0.2 & -1.5 & 0 & 0 \\ 0 & -0.22 & 0 & 0 & 0 \\ 0 & 0 & 1.08 & 0 & 0 \\ \hline * & * & * & -0.52 & 0 \\ * & * & * & 0.5 & -1.02 \end{array}\right)$$

with $A^1_1$ from (10) and $A_2$ from (16).

Its eigenvalues are $\lambda_1 = -0.02$, $\lambda_2 = -0.22$, $\lambda_3 = 1.08$, $\lambda_4 = -0.52$, $\lambda_5 = -1.02$, and then $\xi^e_1$ is confirmed to be unstable due to the presence of $\lambda_3 > 0$.

As far as $\xi^e_2$ is concerned, the matrix

$$A_2 = \left.\frac{\partial F}{\partial \xi}\right|_{\xi=\xi^e_2} = \begin{pmatrix} A^2_1 & 0 \\ * & A_2 \end{pmatrix} =$$

$$= \left(\begin{array}{ccc|cc} -0.80 & 0.50 & -0.12 & 0 & 0 \\ 0 & -0.22 & 0 & 0 & 0 \\ 0.78 & -0.30 & -0.30 & 0 & 0 \\ \hline * & * & * & -0.52 & 0 \\ * & * & * & 0.5 & -1.02 \end{array}\right)$$

must be considered, with $A^2_1$ from (12) and $A_2$ from (16).

Its eigenvalues are $\lambda_1 = -0.22$, $\lambda_2 = -0.55 + 0.17i$, $\lambda_3 = -0.55 - 0.17i$, $\lambda_4 = -0.52$, $\lambda_5 = -1.02$, all with negative real part, as expected, being (7) verified.

Figures from 6 to 9 show the projection to different subplanes of the state evolution in the neighbourhood of the equilibrium point $\xi^e_2$. Six initial conditions $\xi^i_0$, $i = 1, ..., 6$, reported in Table II, have been chosen:

| | $\xi^1_0$ | $\xi^2_0$ | $\xi^3_0$ | $\xi^4_0$ | $\xi^5_0$ | $\xi^6_0$ |
|---|---|---|---|---|---|---|
| $S_1$ | 92000 | 60000 | 5000 | 500 | 45000 | 90000 |
| $S_2$ | 0 | 32000 | 30000 | 10000 | 2000 | 2500 |
| $I$ | 8000 | 5000 | 50000 | 10000 | 5000 | 50000 |
| $P$ | 0 | 2000 | 5000 | 25000 | 2500 | 15000 |
| $A$ | 0 | 1000 | 14000 | 5000 | 10000 | 1000 |

TABLE II: Initial conditions used for the state trajectories analysis near the equilibrium point $\xi^e_2$

In Figure 6, where the relations between the subjects $S_1(t)$ and the infected ones $I(t)$ are depicted, it is possible to note that initially the number of the infected subjects increases, due to the term $\frac{c\beta S_1(t) I(t)}{N_c(t)}$, and then it decreases and converges to the equilibrium point. Exceptions are represented by the cases of initial conditions $\xi^3_0$ and $\xi^4_0$; in these cases, due to the high ratio between the infected $I_0$ and the susceptibles $S_{1,0}$, an initial decrement of the infected subjects is present. This behaviour can be explained considering that in these conditions, the high

value of $I_0$ implies a high flux of subjects towards the class $P$ and $A$, as confirmed by Figure 7, while the contemporary low number of susceptible subjects corresponds to an initially low interaction between the two classes and, then, a lower number of new infected individuals. This effect is more evident for the case of $\xi_0^3$ due to the particularly high number of infected $I_0$.

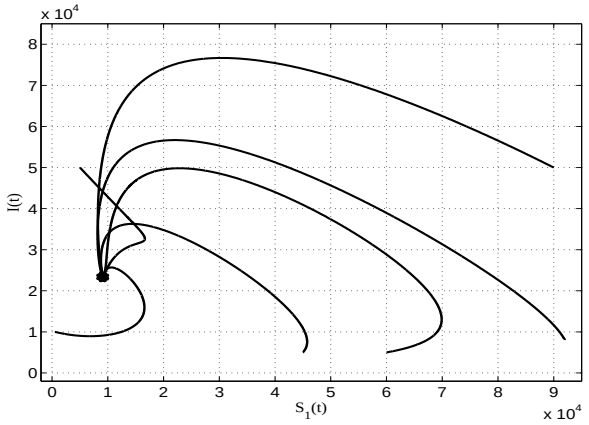


Fig. 6: Family of state trajectories in the $S_1$ – $I$ plane for initial conditions as in Table II

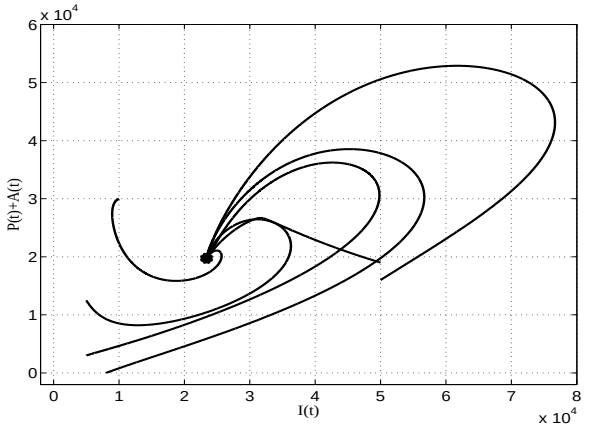


Fig. 7: Family of state trajectories in the $I$ – $(P + A)$ plane for initial conditions as in Table II

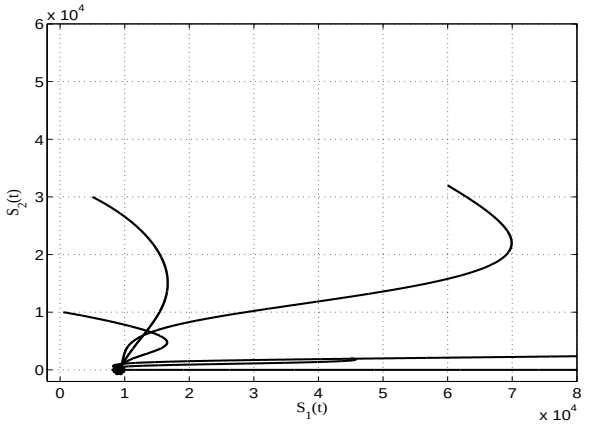


Fig. 8: Family of state trajectories in the $S_1$ – $S_2$ plane for initial conditions as in Table II

The same situation may be also analysed considering the relationship between $S_1$ and $S_2$, in Figure 8. It could be noted that $S_2(t)$ decreases while $S_1(t)$ converges to the equilibrium point showing a small oscillation.

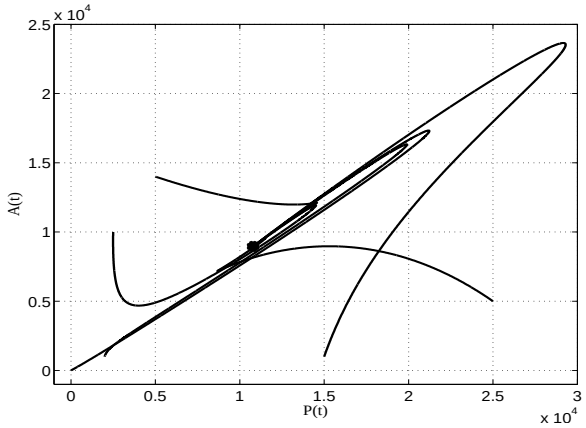


Fig. 9: Family of state trajectories in the $P$ – $A$ plane for initial conditions as in Table II

This oscillatory behaviour is well evidenced in Figure 7, where the evolution of the totality of the diagnosed patients $(P(t)+A(t))$ versus the infected subjects $I(t)$ is reported, and in Figure 9, where the projection on the $P-A$ plane of the free evolutions is depicted. There is, generally, a sort of spiral-like pattern which reaches the equilibrium point, intuitively due to the presence of the coupling terms. In fact, for example, when the infected subjects increase, their larger number increments the number of the illness classes $P$ and $A$, that, successively, decrease for the death terms proportionally to their number.

### *B. Numerical results under constant inputs*

In this Subsection it is developed a first analysis of the influences of the introduced inputs. It must be noted that, while the input $u_1(t)$ is a control of the transition between the two classes of uninfected population introduced in the present work, the input $u_2(t)$ as well as the input $u_3(t)$ contribute to modify two natural transitions. In fact, the input $u_2(t)$ has the same effect, with a different structure, of the natural transition term $\delta I(t)$, while the input $u_3(t)$ acts in the same way of the term $\alpha_1 P(t)$, but working in the opposed direction so counteracting the free trasition from $P$ to $A$. Then, it is expected that some natural behaviours of the dynamics evolutions, like the ones illustrated in Subsection IV-A, can be accentuated and some others attenuated.

In order to check if and how the previous considerations are verified, the effects of the introduced inputs $u_1$, $u_2$ and $u_3$ have been shown through some numerical simulations.

Each of the three inputs is considered separately, in order to better identify the corresponding contribution.

All the simulations have been performed making use of the initial conditions $\xi_0^2$ in Table II.

*1) Case $u_1 \neq 0$, $u_2 = u_3 = 0$:* in this case, the contribution of $u_1(t)$ to the dynamics is evaluated simulating the state evolution for different constant values of the first input $u_1$,

more precisely for $u_1 = 0, 0.1, 0.25, 0.4, 0.5$, while keeping the other two inputs equal to zero. Figures from 10 to 12 show the results of such simulations. In all of them, the dotted curve corresponds to $u_1 = 0$.

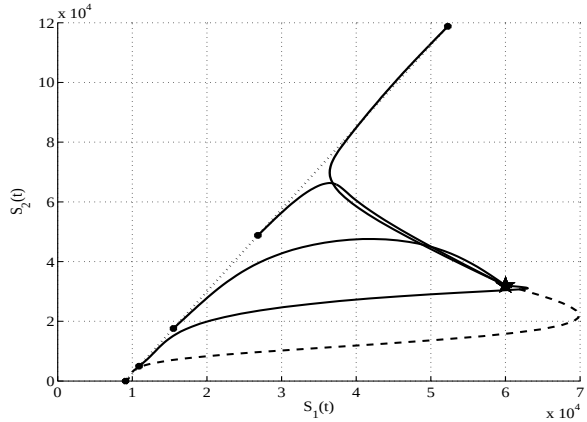


Fig. 10: State trajectories in the $S_1 - S_2$ plane and corresponding equilibrium points for different values of $u_1$

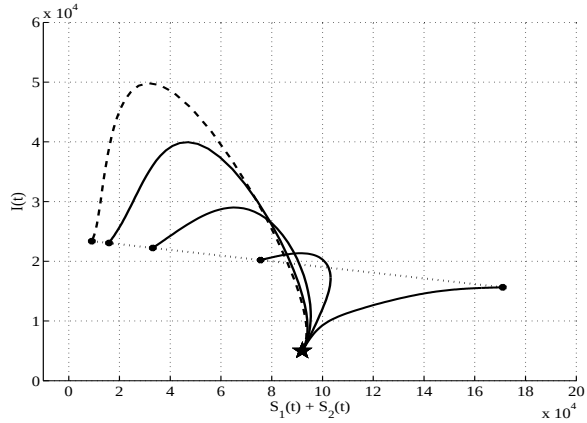


Fig. 11: State trajectories in the $(S_1 + S_2) - I$ plane and corresponding equilibrium points for different values of $u_1$

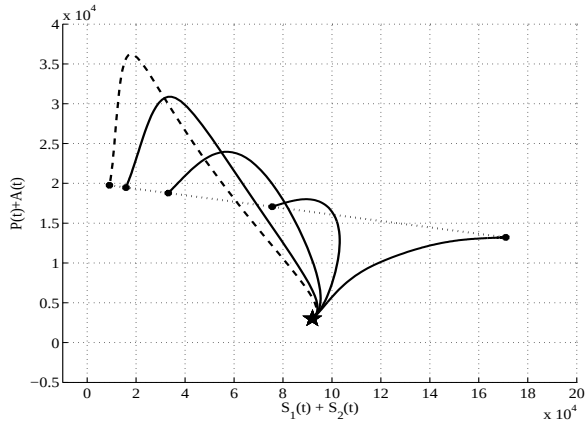


Fig. 12: State trajectories in the $(S_1 + S_2) - (P + A)$ plane and corresponding equilibrium points for different values of $u_1$

The most evident and expected result is visible in Figure 10: as $u_1$ increases, the $S_1(t)$ and $S_2(t)$ variables evolve reducing the oscillatory evolution discussed in Subsection IV-A (dotted curve), and change their steady state conditions, so that both $S_1(t)$ and $S_2(t)$ increase. The effectiveness of the primary prevention $u_1$ is evident in Figures 11 and 12, in which it can be noted both the progressive attenuation of the oscillatory evolution and the long term ($t \to +\infty$) reduction of both the infected subjects $I(t)$ and $P(t) + A(t)$ in a global context of increment of the whole population, as depicted in Figure 13.

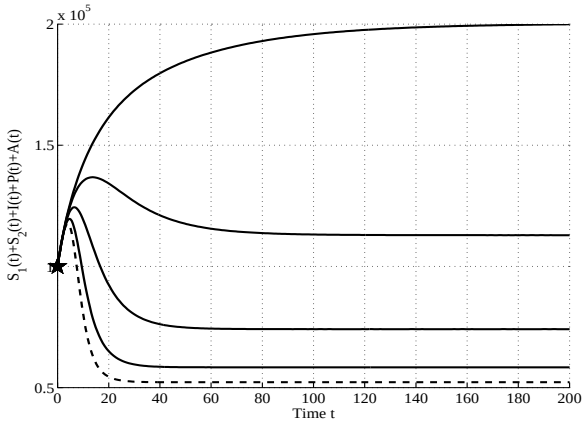


Fig. 13: Time history of the total population for different values of $u_1$

*2) Case $u_2 \neq 0$, $u_1 = u_3 = 0$:* the input $u_2$ corresponds to an action aiming at reducing, more than the autonomous effect modelled by $\delta I(t)$, the number of the infected subjects $I(t)$ responsible of the propagation of the infection. In this case simulations have been performed for different values of $u_2$ in the set $[0, 0.1, 0.25, 0.4, 0.5]$, while the other two inputs have been kept equal to zero.

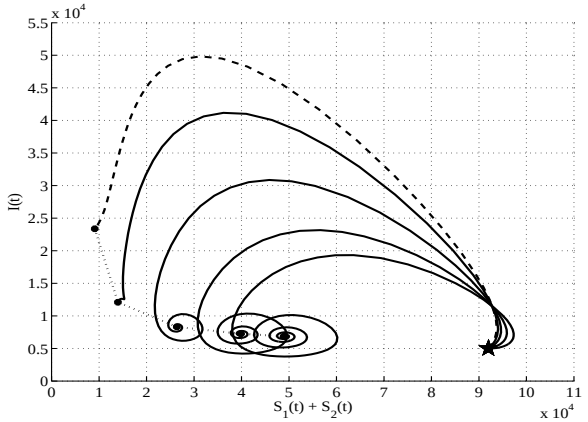


Fig. 14: State trajectories in the $(S_1 + S_2) - I$ plane, and corresponding equilibrium points, for different values of $u_2$

Figure 14 shows the evolution of $I$ versus the total unifected population $S_1 + S_2$. This figure can be more interesting once compared with Figure 11. Both the controls $u_1(t)$ and $u_2(t)$ contribute to decrement the number of the dangerous infected individuals $I$. But, tough the action of $u_2$ produces a

significant oscillatory behaviour, evident as $u_2(t)$ increases, it reduces the number of infected subjects $I(t)$ more than the control $u_1(t)$. In fact, from Figure 11, the steady state number of infected individuals $I(t)$ decreases up to about 16000 patients under different values of the $u_1$ control, while the decrement is up to 6-7000 units under the control $u_2$ of the same order as $u_1$, see Figure 14.

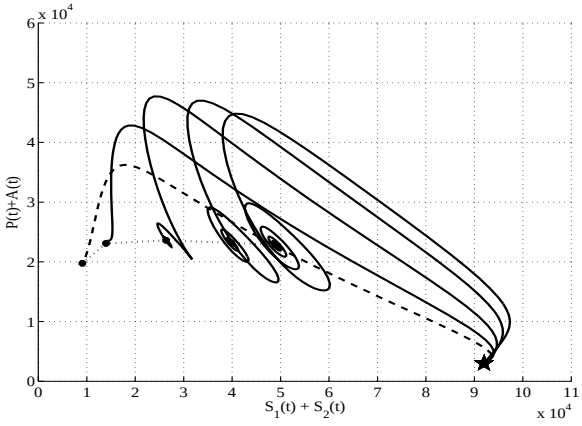


Fig. 15: State trajectories in the $(S_1 + S_2)$ – $(P + A)$ plane, and corresponding equilibrium points for different values of $u_2$

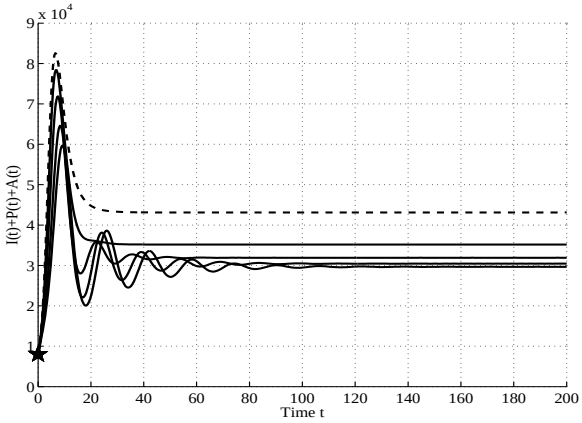


Fig. 16: Time history of the total infected population $(I(t) + P(t) + A(t))$ for different values of $u_2$

Figure 15 depicts the state evolution of the diagnosed infected patients $P(t)+A(t)$ with respect to the unifected ones $S_1(t)+S_2(t)$. In this case, it seems that $u_2$ has not a positive effect on the number of infected patients $P(t) + A(t)$: the steady state value is greater as $u_2$ increases. Actually, this reflects the positive effect of a larger number of diagnosed infected subjects, with a corresponding decrement of $I(t)$ (Figure 14). The contribution of $u_2$ to the reduction of the total number of infected individuals $I(t) + P(t) + A(t)$ is evident in Figure 16, as well as the increment of the total population (Figure 17); the advantages are also confirmed by the higher number of healthy population $S_1(t) + S_2(t)$, depicted in Figure 18.

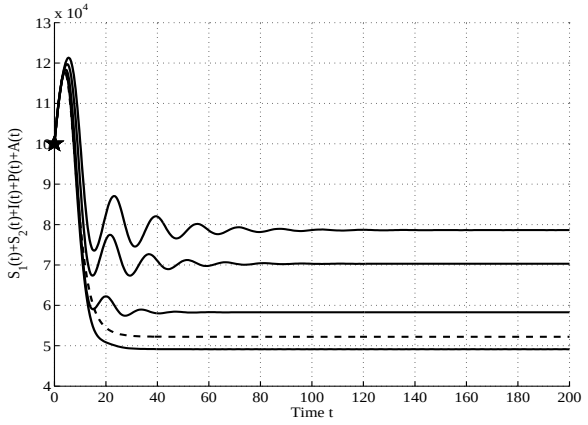


Fig. 17: Time history of the total population for different values of $u_2$

The oscillatory characteristics are present in all the evolutions and increase with the amplitude of $u_2$. Differently from $u_1$, the control $u_2$ does not modify the convergence to zero of $S_2(t)$. In Figure 19 it is shown how $S_2(t)$ goes to zero for any $u_2$ while $S_1(t)$, with an oscillatory transient, reaches a steady state value depending on $u_2$.

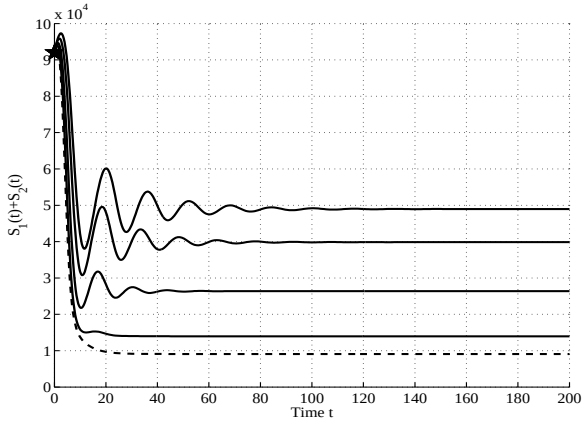


Fig. 18: Time history of the total uninfected population $(S_1(t) + S_2(t))$ for different values of $u_2$

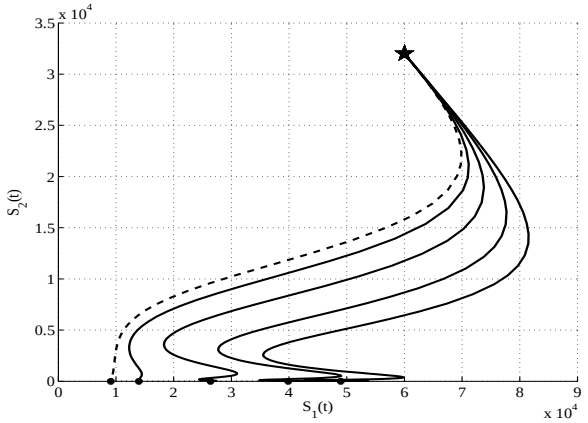


Fig. 19: State trajectories in the $S_1$ – $S_2$ plane, and corresponding equilibrium points, for different values of $u_2$

*3) Case $u_3 \neq 0$, $u_1 = u_2 = 0$:* the control $u_3$ represents the effects of the therapy on the pre-AIDS patients to prevent their transition to the AIDS status; then, it affects the dynamics of $P$ and $A$ only.

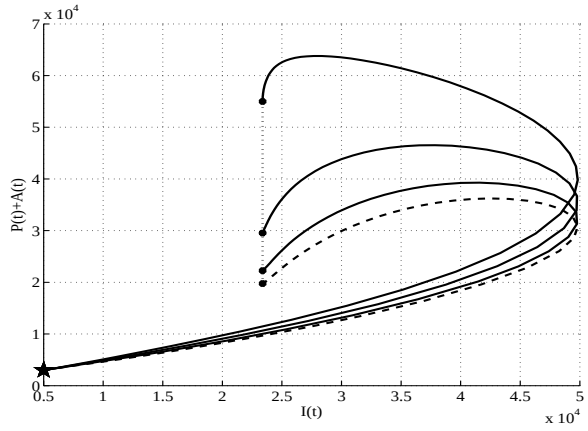


Fig. 20: State trajectory in the $I - (P + A)$ plane for the different values of $u_3$

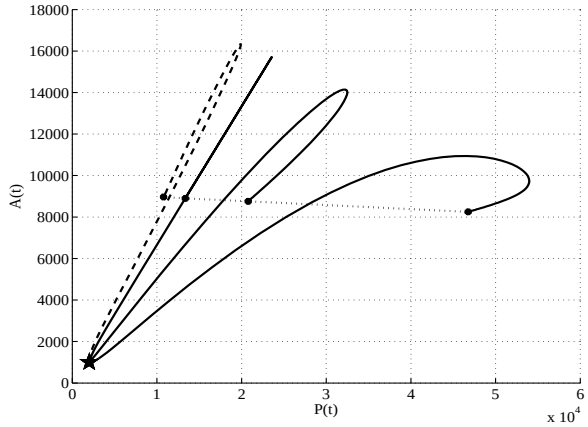


Fig. 21: State trajectory in the $P - A$ plane for the different values of $u_3$

For $S_1$ and $S_2$ it is obvious; for the part $I$ of the infected population it is possible to see that the independence from $u_3$ still holds.

For $u_3 = 0, 0.1, 0.25, 0.4$ the steady state value for $I(t)$ is always $2.34 \cdot 10^4$ while different values for $P(t) + A(t)$ are asymptotically reached, as depicted in Figure 20.

Figure 21 illustrates the main effect of the $u_3$ action. As $u_3$ increases, the steady state values and the maximum peaks for $A(t)$ decrease, the second more significantly. At the same time, $P(t)$ shows an increasing steady state value, since the effect of the therapy is to keep the pre-AIDS patients in such a state as much as possible.

## V. Conclusions

A new model describing the HIV-AIDS spread is proposed, providing an accurate description of the susceptible subjects and of the infected ones. The former are divided into two categories, the wise ones, that avoid dangerous behaviours, and the subjects that, with irresponsible acts, could become infected. The infected subjects are divided into three categories, the infected not yet aware of their status (therefore dangerous for the spread of the virus), and the subjects in the HIV and in the AIDS status. Beyond the obvious action aiming at medication but not influencing the spread, two more controls are introduced to increase the information among susceptible subjects (and therefore to avoid dangerous behaviours) and to induce the unaware infected individuals to a fast identification of the infection, thus reducing the spread of the HIV/AIDS. From the simulation results, the test campaign appears to be the core of the success of a prevention control effort: being aware of the health status as soon as possible reduces the period, up to 10-15 years, in which risky contacts are possible and induces infected subjects to start the medication. A deep analysis of the system, the equilibria points, their stability and the influences of the control actions are studied, confirming intuitive results and constituting the basis for future possible developments, as the determination of the control actions resulting from an optimization procedure with constraints on the resources. The non-availability of the full information on the state can be also taken into account: generally the only reliable information is related to the number of subjects with HIV/AIDS positive diagnosis, whereas the category of susceptible individuals should include all the subjects that have not done the test yet. Therefore, to determine a control strategy, an observer should be determined.

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**Paolo Di Giamberardino** He received the Masters (Electronic Engineering) in 1991 and the Ph.D. (Systems Engineering) degrees in 1995 from Sapienza University of Rome, Italy. He is Assistant Professor since 1996 at the Department of Computer, Control, and Management Engineering Antonio Ruberti, Sapienza University of Rome. He is coordinator of Research Projects founded by his University or by the Italian Ministry. He is member of the Scientific or Organising Committee of international Conferences and reviewer for several international journals. He is head of the "Control and Systems Laboratory" and co-head of the "E-Learning Systems and Applications Laboratory". His main research interests are in the analysis and control of nonlinear discrete time and sampled systems, in the problems of motion planning, control and coordination of mobile robots and sensor networks, in design, identification and analysis of mathematical models, in architectures and systems for e-learning.

**Luca Compagnucci** He received his Bachelor's degree in Clinical Engineering from Sapienza University of Rome, Italy, in 2015. Currently, he is completing a Masters in Biomedical Engineering at the same University. His main research interests are in biomedical instruments, physiological cybernetics, bioinformatics.

**Chiara De Giorgi** She received her Bachelor's degree in Clinical Engineering from Sapienza University of Rome, Italy, in 2015. Currently, she is completing a Masters in Biomedical Engineering at the same University. Her main research interests are in biomedical instruments, physiological cybernetics, bioinformatics.

**Daniela Iacoviello** (M'17) She received the Laurea degree in Mathematics in 1992 and the Ph.D. degree in Systems Engineering in 1998, both from Sapienza University of Rome, Italy. Since 2002 she has been assistant professor in Automatic Control at the Department of Computer, Control and Management Engineering Antonio Ruberti of Sapienza University of Rome. She is coordinator of Research Projects founded by his University or by the Italian Ministry. She is member of the Scientific or Organising Committee of international Conferences and reviewer for several international journals. Her main research interests are in signal processing and image analysis, estimation theory, system identification, physiological cybernetics and optimal control.