

## Global analysis of SAIRS-type epidemic models

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### 1. INTRODUCTION

Once an infectious disease starts circulating in a population, the main goal is to contain its spread. Several control strategies may be applied to control a disease, such as detection and isolation of infectious individuals or vaccination. However, the detection of infectious individuals is far from being an easy task: various diseases, such as influenza, cholera, shigella or Covid-19, are often spread by asymptomatic individuals. The presence of asymptomatic cases allows a wide circulation of a disease in the population, since they often remain unidentified. Hence, the contribution of the so called “silent spreaders” to the infection transmission dynamics should be considered in mathematical epidemic models, as in Robinson and Stilianakis (2013). Unlike the more famous and studied epidemic models, much less attention has been paid to the SAIR(S)-type models. Thus, we think that a deeper understanding of these kinds of models is needed, and could prove to be very useful in the epidemiological field.

In this work, we consider an SAIRS (Susceptible - Asymptomatic infected - symptomatic Infected - Recovered - Susceptible) model based on the one proposed in (Robinson and Stilianakis, 2013, Sec. 2), in which the authors provide only a local stability analysis. An SAIR-type model is studied in Ansumali et al. (2020), with application to SARS-CoV-2. The proposed global stability analysis regards only a simplified version of the model in Robinson and Stilianakis (2013): first, recovered people do not lose their immunity; moreover, the infection rates of the asymptomatic and symptomatic individuals are equal, as well as their recovery rates, while in Robinson and Stilianakis (2013) these parameters are considered to be potentially different. In Ottaviano et al. (2022b), we provide a global stability analysis of the model proposed in Robinson and Stilianakis (2013), and for some variations thereof. In addition, we include in our model the possibility of vaccination. In the investigation of global stability, we answer an open problem left in Ansumali et al. (2020). In particular, we study the global asymptotic stability (GAS) of the disease-free equilibrium (DFE) and provide results related to the global asymptotic stability of the endemic equilibrium (EE) for many variations of the model. We found the expression of the basic reproduction number  $R_0$  and prove that the DFE is globally asymptotically stable if  $R_0 < 1$  and unstable if  $R_0 > 1$ , condition under which a positive endemic equilibrium (EE) exists.

### 2. FORMULATION OF THE MODEL

In our model, the total population  $N$  is partitioned into four compartments, namely  $S$ ,  $A$ ,  $I$ ,  $R$ , which represent the fraction of Susceptible, Asymptomatic infected, symptomatic Infected and Recovered individuals, respectively, such that  $N = S + A + I + R$ . Without loss of generality, we assume  $N = 1$ . The infection can be transmitted to a susceptible through contact with either an asymptomatic infected individual, at rate  $\beta_A$ , or a symptomatic individual, at rate  $\beta_I$ . From the asymptomatic compartment, an individual can either progress to the class of symptomatic infectious  $I$ , at rate  $\alpha$ , or recover without ever developing symptoms, at rate  $\delta_A$ . An infected individual with symptoms can recover at a rate  $\delta_I$ . We assume that the recovered individuals do not obtain a long-life immunity and can return to the susceptible state after an average time  $1/\gamma$ . Furthermore, we assume that a proportion  $\nu$  of susceptible individuals receive a dose of vaccine, which grants them a temporary immunity. Moreover, we consider the vital dynamics of the entire population and, for simplicity, we assume that the rate of births and deaths are the same, equal to  $\mu$ ; we do not distinguish between natural deaths and disease related deaths. The system of ODEs that describes the model is given by:

$$\begin{aligned} \frac{dS(t)}{dt} &= \mu - \left( \beta_A A(t) + \beta_I I(t) \right) S(t) - (\mu + \nu) S(t) + \gamma R(t), \\ \frac{dA(t)}{dt} &= \left( \beta_A A(t) + \beta_I I(t) \right) S(t) - (\alpha + \delta_A + \mu) A(t), \\ \frac{dI(t)}{dt} &= \alpha A(t) - (\delta_I + \mu) I(t), \\ \frac{dR(t)}{dt} &= \delta_A A(t) + \delta_I I(t) + \nu S(t) - (\gamma + \mu) R(t), \end{aligned} \tag{1}$$

with initial condition  $(S(0), A(0), I(0), R(0))$  belonging to the set

$$\bar{\Gamma} = \{(S, A, I, R) \in \mathbb{R}_+^4 \mid S + A + I + R = 1\}, \tag{2}$$

where  $\mathbb{R}_+^4$  is the non-negative orthant of  $\mathbb{R}^4$ . Assuming initial conditions in  $\bar{\Gamma}$ ,  $S(t) + A(t) + I(t) + R(t) = 1$ , for all  $t \geq 0$ ; hence, system (1) is equivalent to the following three-dimensional dynamical system:

$$\begin{aligned} \frac{dS(t)}{dt} &= \mu - \left( \beta_A A(t) + \beta_I I(t) \right) S(t) - (\mu + \nu + \gamma) S(t) + \\ &\quad + \gamma(1 - A(t) - I(t)), \\ \frac{dA(t)}{dt} &= \left( \beta_A A(t) + \beta_I I(t) \right) S(t) - (\alpha + \delta_A + \mu) A(t), \\ \frac{dI(t)}{dt} &= \alpha A(t) - (\delta_I + \mu) I(t), \end{aligned} \tag{3}$$

with initial condition  $(S(0), A(0), I(0))$  belonging to the set

$$\Gamma = \{(S, A, I) \in \mathbb{R}_+^3 \mid S + A + I \leq 1\},$$

which is positively invariant for system (3). In the following, we denote with  $\dot{\Gamma}$  the interior of the set  $\Gamma$ .

### 3. RESULTS

System (3) always admits a disease-free equilibrium, given by

$$x_0 = (S_0, A_0, I_0) = \left( \frac{\mu + \gamma}{\mu + \nu + \gamma}, 0, 0 \right). \tag{4}$$

The behaviour of the system is related to the basic reproduction number  $\mathcal{R}_0$  of (3), given by

$$\mathcal{R}_0 = \left( \beta_A + \frac{\alpha \beta_I}{\delta_I + \mu} \right) \frac{\gamma + \mu}{(\alpha + \delta_A + \mu)(\nu + \gamma + \mu)}. \tag{5}$$

**Theorem 3.1.** *The disease-free equilibrium  $x_0$  of (3) is globally asymptotically stable in  $\Gamma$  if  $\mathcal{R}_0 < 1$ , and unstable if  $\mathcal{R}_0 > 1$ .*

**Theorem 3.2.** *There exists a unique endemic equilibrium  $x^* = (S^*, A^*, I^*)$  in  $\dot{\Gamma}$  for system (3) if and only if  $\mathcal{R}_0 > 1$ .*

In Ottaviano et al. (2022b), we analyze different variations of the model. In the case of the SAIR model (i.e.  $\gamma = 0$ ) and when asymptomatic and symptomatic individuals have the same transmission rate and recovery rate (i.e.  $\beta_A = \beta_I$  and  $\delta_A = \delta_I$ ), we prove the following result providing an appropriate Lyapunov function.

**Theorem 3.3.** *The endemic equilibrium  $x^* = (S^*, A^*, I^*)$  is globally asymptotically stable in  $\dot{\Gamma}$  for system (3) if  $\mathcal{R}_0 > 1$ .*

In the general case of the SAIRS model with different rate of transmission and recovery for the two groups of infectious individuals, we use a geometric approach for the global stability of equilibria of nonlinear autonomous differential equations proposed in Lu and Lu (2017).

**Theorem 3.4.** *Assume that  $\mathcal{R}_0 > 1$  and  $\beta_A < \delta_I$ . Then, the endemic equilibrium  $x^*$  is globally asymptotically stable in  $\dot{\Gamma}$  for system (1).*

However, as illustrated by various numerical simulations in Ottaviano et al. (2022b), we are led to think that the assumption on the parameters  $\beta_A$  and  $\delta_I$  could be relaxed.

### 4. EXTENSION TO A MULTI-GROUP MODEL

Later, we generalize the SAIRS model to a multi-group model, which takes into account different groups of individual among which an epidemic can spread.

In this framework, the total population is divided into  $n$  groups. We denote with  $S_i, A_i, I_i$  and  $R_i$  the fraction of Susceptible, Asymptomatic infected, symptomatic

Infected and Recovered individuals in the  $i$ -th group, respectively, such that  $S_i + A_i + I_i + R_i = 1$  at all times.

The disease can be transmitted by individuals  $A_i$  and  $I_i$ , within their group, to the susceptible  $S_i$ , with transmission rate  $\beta_{ii}^A$  and  $\beta_{ii}^I$ , respectively, but also between different groups: e.g., individuals  $A_j$  and  $I_j$ , belonging to the  $j$ -th group, may infect susceptible individuals  $S_i$  of group  $i$  with transmission rate  $\beta_{ij}^A$  and  $\beta_{ij}^I$ , respectively. We also assume that the multi-group network is undirected and connected. The disease-related parameters, that are the average time of the symptoms developing, denoted by  $1/\alpha$ , the recovery rates from both the infectious compartments,  $\delta_A$  and  $\delta_I$ , and the average time to return to the susceptible state,  $1/\gamma$ , do not depend on the group of origin. We assume, instead, that the proportion  $\nu_i$  of vaccinated individuals depends on the group. Moreover, we consider the vital dynamics of each group, assuming that the rate of births and deaths are the same in the  $i$ -th group, equal to  $\mu_i$ .

Even though some results on SIRS-type model in Muroya et al. (2013) and SEIRS-type model in Fan et al. (2018) have been achieved, the problem of existence and global stability of an endemic equilibrium for several multi-group models remains open, as stated in Mohapatra et al. (2015).

Our results, in Ottaviano et al. (2022a), regard a generalization of Theorems 3.1-3.3 for the multi-group type model. The problem of the global asymptotically stability of the endemic equilibrium, as in Theorem 3.4, remains open.

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