Abstract. We study the spread of an SIRS-type epidemics with vaccination on network. Starting from an exact Markov description of the model we investigate the mean epidemic lifetime by providing a sufficient condition for the fast extinction, depending on the topology of the network.

Then, we pass to consider a first-order mean-field approximation of the exact model. At this point, we dwell on the stability properties of the system by relying on the graph-theoretical notion of equitable partition. In the case of graphs possessing this kind of partition, we find a positively invariant set which contain the endemic equilibrium, that can be computed by using a lower-dimensional dynamical system. Finally, in the special case of regular graphs, we show that when the recovery rate is higher than the vaccination rate, the aforementioned invariant set is contained in the domain of attraction of the endemic equilibrium.

Keywords: Susceptible-infective-removed-susceptible model, Networks, Time to extinction, Equitable partition, Stability.

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1. Introduction

The spread and persistence of infectious diseases are a result of the complex interactions between individual units (e.g. people, city, county, etc), disease characteristics and possible control policies. Consequently, the aim of many mathematical models is to gain insight into how diseases transmit and to identify the most effective strategies for their prevention and control. Vaccination is considered to be the most effective intervention policy as well as a cost-effective strategy to reduce both the morbidity and mortality of individuals.

Over the past few decades a high variety of compartmental models, where the population is divided into different classes (compartments), depending on the stage of the disease, have been formulated. A relevant amount of these models, including those that incorporate a vaccination strategy, assumes a homogeneous mixing approximation \[20, 11, 15, 32, 8\]. Basically, individuals in the population interact with each other completely at random (with no preferential interaction), each generating the same number of contacts. Although the simplicity of the model allows to include more specific characteristics, such as birth and deaths, vaccination by age etc., the homogeneous mixing assumption ignores details such as geographical location, presence of community structures, or the specific role of each individual in the contagion spreading, while the underlying contact structure of the population plays a crucial role in the spreading of the epidemics \[5, 27, 19\].
Epidemic models have been used also to describe a wide range of other phenomena, like social behaviors, diffusion of information, computer viruses etc., indeed the dynamical behavior of these phenomena can be described by the same type of equations, although their basic mechanisms may differ [28]. For example, networks through which agents communicate with one another are frequently used to propagate electronic viruses. Thus, epidemiological modeling method can help to understand how such viruses spread on a network for building proper effective strategies to stem the viral prevalence, e.g. to implement antivirus techniques [35, 3]. For a review on epidemics models on networks see, e.g., [27, 12].

In our model, we classify each individual in the population according to her viral state: susceptible, infected or recovered. An individual in the susceptible state can be infected if she is in contact with any infected individual (equivalently, they are adjacent nodes in the network). After the infection is over, the individual enters in the recovery state, and while in the recovery state, she cannot undergo to a new infection. However, in this work we analyze a model where the recovery state is not permanent, hence the individual returns, after an exponentially distributed time, to the susceptible state.

Moreover, a further mechanism exists that change the state of an individual, that is vaccination. Vaccination takes place for susceptible individuals who are moved directly to the recovery state. We do not add a compartment for the vaccinated individuals, not distinguishing the vaccine-induced immunity from the natural one acquired after the virus contraction. In several examples in applications, actually, vaccination does not confer a long-life immunity (in the field of infectious disease, think, e.g., to influenza, diptheria, pertussis and pneumococcal vaccine).

Overall, the model we consider can be classified as a SIRS susceptible-infected-removed-susceptible model with vaccination, on networks, that we shall refer to with SIRS\(_v\). Moreover, we adopt an individual (node)-based approach , see also [35], as opposite to a large part of the literature where the structure of the network is simplified by using a degree-based mean-field (DBMF) approach, where all nodes with the same degree are assumed to be statistically equivalent, see, e.g., [11, 22, 29, 37].

1.1. Outline and main results. In Sec\[2\] we start to consider the exact stochastic SIRS model with vaccination. As stated before, we have a population of \(N\) individuals where each of them can be classified in one of three states, \(S\), \(I\) or \(R\). Therefore, the process describing the spreading of the epidemics among the population counts \(3^N\) possible states. Our system evolves as a continuous-time Markov model: all the involved processes, vaccination, infection, recovery and loss of immunity, are thought as independent Poisson processes each with its own rate (that allows to jump from a state to another). This approach describes the global change in the state probabilities of the network exactly.

In this context, we investigate the mean time in which the epidemic is active (at least one node is infected), trying to understand in which way the network topology, and the parameters of the model, are responsible for a quick epidemic extinction. We also provide some numerical investigations to assess the role of the loss-immunity parameter in the extinction mean time.

The exponential growth of the state space with \(N\) makes the search for solution neither analytically nor computationally tractable, except for very small networks. Hence, it is necessary to derive an approximation of the original model. A direct
approach for deriving an approximate model is to start from a node-level description of the underlying exact stochastic process (Sec. 2.1), as proposed in [30], and then, through a first-order mean-field approximation, obtain a set of $3N$ nonlinear differential equations, specifying the state probabilities of each node (Sec. 3). Basically, we consider an extension of the $N$-intertwined mean-field approximation (NIMFA), provided for the SIS and SIR models in [33] and [36] respectively, to a SIRS model (with vaccination).

In Sec. 4 based on the stability results in [35], we provide the critical threshold which separates an extinction region from an endemic one in terms of the parameters of the model and the network topology. From the stability analysis provided in [35], we have a sufficient condition ensuring the global attractivity of the positive equilibrium, above the threshold. However, this condition is quite restrictive, we show, e.g., that it is never satisfied in the case of regular graphs. Thus, in Sec. 4.1 we focus on the domain of attraction of the positive equilibrium for these specific graphs. For this purpose, we use the notion of equitable partitions [17, 6]. Thus, first we prove the existence of a positively invariant set for the system when a graph posses an equitable partition, then we show that when the initial conditions belong to this set the whole epidemic dynamics can be expressed by a reduced system of $3n$ equations, where $n < N$. Moreover, this invariant set contains the endemic equilibrium (besides the disease-free equilibrium) that can be computed by means of the reduced system. Since a regular graph is a special case of graph with equitable partition, we show that, when the recovery rate is higher than the vaccination rate, the aforementioned invariant set is contained in the domain of attraction for the endemic equilibrium. Finally, in Sec. 5 we provide some numerical investigations.

2. The Exact Model

We consider a continuous-time Markovian susceptible-infective-removed-susceptible (SIRS) model with vaccination, on networks. Specifically, the epidemics spreads over an undirected connected graph $G = (V, E)$, where the node set $V$ represents the individuals in the population and the links between nodes are specified by the edge set $E$. The connectivity of $G$ is conveniently expressed by the symmetric $N \times N$ adjacency matrix $A$.

Each node can be, at time $t$, in one of the three states $S, I,$ or $R$ with a certain probability. The viral state of a node $i$, at time $t$, will be denoted by the random variable $X_i(t)$. We assume that the infection process is a per link Poisson process where the infection rate between a susceptible and an infected node is $\beta$. The recovery process of an infected node is poissonian too, with rate $\delta$, and once cured the individual pass from the state $I$ to $R$. We denote by $\tau = \beta/\delta$ the so-called effective infection rate. In a SIRS model the immunity acquired after receiving the infection is temporary (unlike the most studied SIR model). A recovered individual stays in the state $R$ for an exponentially distributed time with mean $1/\gamma$, before returning to the susceptible state. In addition, we include the possibility of vaccination for a healthy individual. We assume that each susceptible can receive vaccination at a constant rate $\sigma$ (again we have a Poisson process for vaccination), and that the vaccine is totally effective in preventing infection, although it does not provide a long-life immunity. We do not distinguish the vaccine-induced immunity from the natural one acquired after the contraction of the disease. Namely, we do not consider a vaccination state into the basic model, but the vaccinated individual
pass to the state $R$. Thus, each individual loses the immunity either given by the vaccine or by recovering with the same rate $\gamma$. All the involved Poisson processes are independent.

The state of the network $Y(t)$ at time $t$ is defined by all possible combinations of viral states in which the $N$ nodes can be at time $t$. Let us denote the $3^N$ possible configurations that the state $Y(t)$ can assume by

$$Y_k = (X_N, \ldots, X_1),$$

where $X_i \in \{S, I, R\}$ represents the state of node $i$, and $k = 0, \ldots, 3^N - 1$. We label the state in this way: by setting $S = 0$, $I = 1$, and $R = 2$, we can consider the vector state $Y_k$ as the ternary representation of $k$, that is $k = \sum_{i=1}^N X_i 3^{i-1}$.

The only transition for the process that can take place from a given configuration $Y_k$ are those differing form it in a single component $X_i$. The epidemics process can be described by a continuous-time Markov chain with $3^N$ states specified by the infinitesimal generator $Q$ with elements

$$q_{ij} = \begin{cases} 
\delta, & \text{if } z = j - 3^{m-1} \land X_m = 1; \ m = 1, \ldots, N \\
\beta \sum_{i=1}^N a_{mi} 1_{\{X_i = 1\}}, & \text{if } z = j - 3^{m-1} \land X_m = 0; \ m = 1, \ldots, N \\
\gamma, & \text{if } z = j + 2 \cdot 3^{m-1} \land X_m = 2; \ m = 1, \ldots, N \\
\sigma, & \text{if } z = j - 2 \cdot 3^{m-1} \land X_m = 0; \ m = 1, \ldots, N \\
- \sum_{i=0; i \neq z}^{3^N-1} q_{zi}, & \text{if } z = j \\
0, & \text{otherwise}
\end{cases}$$

(1)

where, $X_m$ is the state of node $m$ in the network state $z$.

Let us note that the set of all states with no infected individuals, that is those states $Y_k$, where $X_i \in \{0, 2\}$, for all $i = 1, \ldots, N$, forms a final class. This differentiates the SIRS$_v$ model from the standard SIRS one, where there is only one absorbing state, that is $Y_0 = (X_N = S, X_{N-1} = S, \ldots, X_2 = S, X_1 = S)$.

Conversely, the set of states where $X_i = 1$, for some $i$, forms a transient class. Standard results in Markov theory implies that the process will enter the final class in finite time, $P$-a.s., which is equivalent to say that the epidemic reaches the extinction (no more infected nodes) almost surely.

Let us define the probability state vector

$$v(t) = (v_0(t), \ldots, v_{3^N-1}(t)), \quad (2)$$

with components

$$v_k(t) = \mathbb{P}(Y(t) = Y_k).$$

The rate of change of every network state is given by the following differential equation:

$$\frac{dv^T(t)}{dt} = Qv^T(t), \quad (3)$$

whose solution is

$$v^T(t) = e^{Qt}v^T(0).$$
The exact system (3) fully describes the Markov process, however the number of equations increases exponentially with the number of nodes; this poses several limitations in order to determine the solutions even for small networks. Hence, often, it is necessary to formalize models that are an approximation of the original one, but allows a better analytical and numerical analysis. A direct approach for deriving an approximate model is to start from a node level description of the underlying stochastic process, that we report in the next section. Then, through a mean-field type approximation (see Sec. 3), it is possible to obtain a reduced set of $3N$ nonlinear differential equations describing the time-change of the state probabilities of each node.

2.1. Node-level Markov description of the SIRS process with vaccination.

Alternatively to the approach adopted in the previous section, we can describe the spreading process by a node-level approach, i.e., by specifying the probability for each node $i$ to move from a state to the others, conditioned on the network state $Y(t)$ \[3\]. Given a node $i$, we shall denote in the sequel $Y_{-i}(t)$ the state of all the other nodes $j \neq i$ in the network.

We can consider the representation for finite state Markov processes by means of all the involved Poisson processes in the model \[29,7\]. For a susceptible individual, the process of being infected by one infected neighbor, during the interval time $(t, t+dt]$ is independent of the process of receiving infection from another neighbor. Indeed, all the infected neighbors compete with each other and the susceptible node become infected when one of the neighbors succeeds in transmitting the infection.

Now, let us define $\mathbb{1}_{\{E\}}$ the indicator random variable (which equals one if the condition $E$ is true, else it is zero). Since for the Poisson processes the probability that $q$ events occur in a time interval $dt$ is of order $(dt)^q$, we can write the probability of having an infection for the node $i$, during the time interval $(t, t+dt]$, as

$$\mathbb{P}(X_i(t+dt) = I | X_i(t) = S, Y_{-i}(t)) = \beta \sum_{j=1}^{N} a_{ij} \mathbb{1}_{\{X_j(t) = I\}} dt + o(dt), \quad (4)$$

since the sum of independent Poisson processes (i.e., the infection processes) is again a Poisson process with rate equals to the sum of the individual rates. The probability of not having a transition from the infected state to the removed state, during $(t, t + dt]$, is:

$$\mathbb{P}(X_i(t+dt) = I | X_i(t) = I, Y_{-i}(t)) = 1 - \delta dt + o(dt). \quad (5)$$

Then from (4) and (5), we have

$$\mathbb{P}(X_i(t + dt) = I | Y(t)) = \mathbb{1}_{\{X_i(t) = S\}} \beta \sum_{j=1}^{N} a_{ij} \mathbb{1}_{\{X_j(t) = I\}} dt + \mathbb{1}_{\{X_i(t) = I\}} (1 - \delta dt) + o(dt). \quad (6)$$

By noticing that

$$\mathbb{P}(X_i(t + dt) = I | Y(t)) = \mathbb{E}[\mathbb{1}_{\{X_i(t + dt) = I\}} | Y(t)],$$
Then, if we compute the expected value of each side of (6), by the law of iterated expectation, we get

\[
E[\mathbb{1}_{\{X_i(t+dt)=I\}}] = E\left[\mathbb{1}_{\{X_i(t)=S\}}\sum_{j=1}^{N} a_{ij} \mathbb{1}_{\{X_j(t)=I\}}\right] dt \\
+ E\left[\mathbb{1}_{\{X_i(t)=I\}}\right] (1 - \delta dt) + o(dt).
\]

After dividing both members by \(dt\) and letting \(dt \to 0\), we have, by exploiting again the properties of the indicator random variable

\[
\frac{d}{dt} P(X_j(t) = I) = \beta \sum_{j=1}^{N} a_{ij} P(X_i(t) = S, X_j(t) = I) - \delta P(X_i(t) = I). \tag{7}
\]

The probability to be recovered, for node \(i\), during the interval time \((t, t + dt]\) is

\[
P(X_i(t + dt) = R | X_i(t) = I, Y_{-i}(t)) = \delta dt + o(dt).
\]

The probability to get vaccinated during \((t, t + dt]\) is

\[
P(X_i(t + dt) = R | X_i(t) = S, Y_{-i}(t)) = \sigma dt + o(dt),
\]

and, finally, the probability that no transition from the removed state happens (that is no loss of immunity occurs) during \((t, t + dt]\), is

\[
P(X_i(t + dt) = R | X_i(t) = R, Y_{-i}(t)) = 1 - \gamma dt + o(dt).
\]

Thus, proceeding as above, we have

\[
\frac{dP(X_i(t) = R)}{dt} = \delta P(X_i(t) = I) + \sigma P(X_i(t) = S) - \gamma P(X_i(t) = R). \tag{8}
\]

With the same arguments as before, we can also discuss the variation of the probability to be in the susceptible state, to get

\[
\frac{dP(X_i(t) = S)}{dt} = -\beta \sum_{j=1}^{N} a_{ij} P(X_i(t) = S, X_j(t) = I) \\
+ \gamma P(X_i(t) = R) - \sigma P(X_i(t) = S). \tag{9}
\]

It seems that we have described the dynamic of the system by means of \(3N\) equations in the unknowns \(P(X_i(t) = x), i = 1, \ldots, N, x = S, I, R\). Unfortunately, equations (7) and (9) are not closed since they contain the joint probabilities \(P(X_i(t) = S, X_j(t) = I)\). We can show that it is possible to derive a system of differential equations for each two-pair probabilities, but even those are not closed, since they involve higher order joint probabilities. In the end, again, a system of \(3^N\) linear equations appears and, as for (3), for large values of \(N\) the system is neither analytically nor computationally tractable. Instead, to reduce the \(3^N\) state-space size, in Sec. 3, we adopt a closure approximation technique to obtain a system of \(3N\) differential equations.
2.2. Time to extinction for the SIRS model. In this section we use the dynamic described in equations (7)-(8)-(9) and we discuss the average lifetime of the epidemics before its extinction (which occurs with probability 1, since the class $Y^0 = \{X_i \neq I, \ i = 1, \ldots, N\}$ is final). Our aim is to find conditions for a quick extinction in order to avoid a long-term epidemic persistence.

First, let us investigate the average time the epidemic is active, that is, at least one node is infected. We focus on the SIRS model with $\sigma = 0$, (although the sufficient condition (13) for fast extinction holds also when $\sigma > 0$), and consider $Y_0$, the set of the states with no infected nodes, which we refer to as the final set.

The next proposition gives us an upper bound on $\mathbb{P}\left(\sum_{i=1}^{N} \mathbb{1}_{\{X_i(t) = I\}} > 0\right)$, that is the probability of not being in the final set $Y_0$, at time $t$.

**Proposition 1.** Let $A$ be the adjacency matrix of the graph $G$, and $\lambda_1(A)$ its spectral radius. Then, for any initial condition $X_0 = (X_1(0), \ldots, X_N(0))$, and all $t \geq 0$, it holds:

$$\mathbb{P}\left(\sum_{i=1}^{N} \mathbb{1}_{\{X_i(t) = I\}} > 0\right) \leq \sqrt{N} \sum_{i=1}^{N} \mathbb{1}_{\{X_i(0) = I\}} \exp((\beta \lambda_1(A) - \delta)t).$$

**Proof.** Let us consider the SIRS governing equation (7), by invoking the law of total probability, it can be rewritten as (11)

$$\frac{d}{dt} \mathbb{P}[X_i(t) = I] = \beta \sum_{j=1}^{N} a_{ij} \mathbb{P}(X_j(t) = I) - \delta \mathbb{P}(X_i(t) = I)$$

$$- \beta \sum_{j=1}^{N} a_{ij} \mathbb{P}(X_i(t) = I, X_j(t) = I) - \beta \sum_{j=1}^{N} a_{ij} \mathbb{P}(X_i(t) = R, X_j(t) = I),$$

for $i = 1, \ldots, N$. Consequently,

$$\frac{d}{dt} \mathbb{P}[X_i(t) = I] \leq \beta \sum_{j=1}^{N} a_{ij} \mathbb{P}(X_j(t) = I) - \delta \mathbb{P}(X_i(t) = I), \quad (12)$$

that written in matrix form is

$$\frac{d}{dt} P(t) \leq (\beta A - \delta I_N) P(t),$$

where $P(t) = [\mathbb{P}(X_1(t) = I), \ldots, \mathbb{P}(X_N(t) = I)]^T$ and $I_N$ is the identity matrix with dimension $N$. The solution of the linear differential inequality above for the vector of infection probabilities is

$$P(t) \leq \exp(t(\beta A - \delta I_N)) P(0),$$

where, $P(0)$ is determined by means of the initial condition $X_0$. In the sequel we let $u$ be the all-one row vector. We notice that, for any $i = 1, \ldots, N$,

$$\mathbb{P}(X_i(t) = I) = \sum_{k: Y_k(i) = 1} \mathbb{P}(Y(t) = Y_k);$$
further,  
\[ P(Y(t) \not\in Y^0) = \sum_{k: Y_k \not\in Y^0} P(Y(t) = Y_k) \leq \sum_{i=1}^{N} \sum_{k: Y_k \not\in Y^0} P(Y(t) = Y_k) \]
\[ = \sum_{i=1}^{N} P(X_i(t) = I) \leq \sum_{i=1}^{N} \left( \exp(t(\beta A - \delta I_N))P(0) \right)_i \]
\[ = u \cdot \exp(t(\beta A - \delta I_N))P(0). \]

By invoking the Cauchy-Schwarz inequality and considering that the matrix \(A\) is symmetric, we obtain (see [14, Thm 8.2])

\[ P \left( \sum_{i=1}^{N} \mathbb{1}_{\{X_i(t) = I\}} > 0 \right) \leq ||u||_2 \exp((\beta \lambda_1(A) - \delta) t) ||P(0)||_2 \]
\[ = \sqrt{N} \sum_{i=1}^{N} \mathbb{1}_{\{X_i(0) = I\}} \exp((\beta \lambda_1(A) - \delta) t) \]

as claimed. \( \square \)

**Corollary 2.** Let \( \tau^{FS} \) denote the hitting time to the final set \( Y^0 \). Then, under the condition
\[ \frac{\beta}{\delta} < \frac{1}{\lambda_1(A)} \]  
(13)

it holds that
\[ \mathbb{E}(\tau^{FS}) \leq \frac{\log(N) + 1}{\delta - \beta \lambda_1(A)}. \]  
(14)

**Proof.** Following the proof of [14] Cor. 8.6 we have
\[ \mathbb{E}(\tau^{FS}) = \int_0^{\infty} P(\tau^{FS} > t) dt = \int_0^{\infty} P \left( \sum_{i=1}^{N} \mathbb{1}_{\{X_i(t) = I\}} > 0 \right) dt \]
\[ \leq \int_0^{\infty} \min \{1, N \exp(-(\delta - \beta \lambda_1(A)) t)\} dt. \]  
(15)

Since \( N \exp(-(\delta - \beta \lambda_1(A)) t) < 1 \) when \( t > \log(N)/(\delta - \beta \lambda_1(A)) \) we can split the intervals of integration in \([0, t^*] \) and \([t^*, \infty) \) obtaining that
\[ \mathbb{E}(\tau^{FS}) \leq t^* + \frac{N}{\delta - \beta \lambda_1(A)} \exp(-(\delta - \beta \lambda_1(A)) t^*) = \frac{\log(N) + 1}{\delta - \beta \lambda_1(A)}. \]

\( \square \)

The above result states that if we consider a sequence of graphs \( G_N \) on \( N \) nodes, for instance regular graphs with fixed degree \( k \) (notice that they share the same spectral radius \( \lambda_1(A_N) = k \)) then the condition \( \delta - \beta \lambda_1(A_N) \geq c > 0 \), for some constant \( c \), implies that the expected time to the infection eradication grows at most logarithmically in \( N \). In this setting, for large \( N \), by using Markov’s inequality we have that the time to eradication is of order \( (\log(N))^\alpha \) with high probability, for any \( \alpha > 1 \).

The result in Proposition 1 implies that the condition (13) is sufficient for fast extinction. This coincides with what is known for the SIS model in [14] Thm 8.2.
where the bound is over the probability that at time $t$ the process has not yet reached the absorbing state (the overall-healthy state).

**Time to absorbing state.** In the previous section we have considered the probability of the persistence of the epidemics (meaning that at least one infected node remains in the network) and the mean time to hit the final set, where there are no more infectious nodes. Now, instead we want to consider the probability of no absorption for the SIRS model ($\sigma = 0$) (that is the probability that the process is not in the zero state, where all nodes are susceptible).

**Proposition 3.** Under the same hypothesis of the Prop. 1 and assuming that $-\gamma$ does not belong to the spectrum of $\beta A - \delta I_N$, it holds that

$$\mathbb{P}\left(\sum_{i=1}^N X_i(t) > 0\right) \leq C \sqrt{N \sum_{i=1}^N 1_{\{X_i(0) = I\lor R\}}} \exp(\max(\beta \lambda_1(A) - \delta, -\gamma) t), \quad (16)$$

where $C$ is a positive constant that depends on the adjacency matrix $A$, and on the parameters $\beta, \delta, \gamma$.

**Proof.** By considering equations (12) and (8), we can write

$$\frac{d\mathcal{P}(t)}{dt} \leq \mathcal{A} \mathcal{P}(t),$$

where $\mathcal{P}(t) = [P_I(t), P_R(t)]^T$, with $P_I(t) = [\mathbb{P}(X_1(t) = I), \ldots, \mathbb{P}(X_N(t) = I)]^T$ and $P_R(t) = [\mathbb{P}(X_1(t) = R), \ldots, \mathbb{P}(X_N(t) = R)]^T$, and

$$\mathcal{A} = \begin{bmatrix} \beta A - \delta I_N & 0 \\ \delta I_N & -\gamma I_N \end{bmatrix}.$$

Thus,

$$\mathcal{P}(t) \leq \exp(t\mathcal{A})\mathcal{P}(0),$$

with $\mathcal{P}(0) = [P_I(0), P_R(0)]^T$, which is determined by the initial condition $X_0$. Consequently

$$\mathbb{P}\left(\sum_{i=1}^N X_i(t) > 0\right) \leq u \exp(t\mathcal{A})\mathcal{P}(0).$$

By invoking Cauchy-Schwarz inequality we arrive at

$$\mathbb{P}\left(\sum_{i=1}^N X_i(t) > 0\right) \leq ||u||_2 ||\exp(t\mathcal{A})||_2 ||\mathcal{P}(0)||_2.$$

The matrix $\mathcal{A}$ is diagonalizable if $-\gamma$ does not belong to the spectrum of $\beta A - \delta I_N$. Indeed, it easy to see that under this hypothesis a basis of eigenvectors of $\mathcal{A}$ can be found. Thus, we have that $||\exp(t\mathcal{A})||_2 = ||M \exp(Dt)M^{-1}||_2$, where $D$ is the diagonal matrix containing the eigenvalues of $\mathcal{A}$ and $M$ the matrix containing the corresponding eigenvectors. Finally, we have

$$\mathbb{P}\left(\sum_{i=1}^N X_i(t) > 0\right) \leq C \sqrt{N \sum_{i=1}^N 1_{\{X_i(0) = I\lor R\}}} \exp(\lambda_1(D) t) \quad (17)$$
Figure 1. Stochastic simulations of the SIRS and SIRS with vaccination average fraction of infected nodes as function of time and $\gamma$, for a complete graph with $N = 50$. a) SIRS model, $\beta = 0.25$, $\delta = 0.4$. b) SIRS$_v$ model, $\beta = 1$, $\delta = 0.4$, $\sigma = 0.45$. At time 0 there is one infected node.

where

$$\lambda_1(D) = \max\{\beta \lambda_1(A) - \delta, -\gamma\}$$

is the maximum eigenvalue of the matrix $\mathcal{A}$, and $C = ||M||_2||M^{-1}||_2$.

Numerical investigations. We investigate numerically the role of the loss-immunity parameter $\gamma$ in the exact models. We consider the averaged $10^3$ sample paths resulting from a discrete event simulation of the exact models. The discrete event simulation is based on the generation of independent Poisson processes for the infection of healthy nodes, the recovery of infected, and for the loss of immunity of the removed, and for the vaccination of susceptible in the SIRS$_v$, i.e., when $\sigma > 0$.

We can see that $\gamma$ influences the dynamics of the average fraction of infected nodes (the prevalence). Specifically, in Fig. 1 a), we show the behavior of the prevalence for the exact SIRS as function of time and $\gamma$. We consider the complete graph with $N = 50$ and fixed values of $\beta$ and $\delta$ for which the condition (13) does not hold. We observe that for some low values of $\gamma$ the average fraction of infected nodes decays towards zero in a quite short time window. As $\gamma$ grows the time to extinction tends to increase, and after a certain critical value of $\gamma$ the prevalence tends to stabilize around a positive quantity for long time (resembling the behavior of the mean-field model that above the threshold reaches the positive equilibrium point (Sec. 3)). The same behavior can be observed for the exact SIRS$_v$ in b). Thus, we are led to assert that the value of $\gamma$ influences the time to extinction of the exact models.

3. Mean-field approximation

Let us come back to the nodal level description for the Markov model (Sec. 2.1). As we pointed out the equations (7) and (9) are not closed since they contain the joint probabilities $P(X_i(t) = S, X_j(t) = I)$. We can "close" the equations providing an approximation for the joint probabilities in terms of the marginal probabilities, assuming the independence between the dynamic states of two neighbors, the so-called first-order mean-field type approximation [30]. Thus, let $(i,j) \in E$, we assume
\[ P(X_i(t) = S, X_j(t) = I) = P(X_i(t) = S)P(X_j(t) = I). \] (18)

Let us define the state probabilities of individual \( i \), at time \( t \), as

\[ S_i(t) = P[X_i(t) = S], \quad I_i(t) = P[X_i(t) = I], \quad R_i(t) = P[X_i(t) = R]. \]

Then, by means of the assumption (18), we have the following mean-field equations for the SIRS model

\[
\begin{align*}
\frac{dS_i(t)}{dt} &= -S_i(t) \sum_{j=1}^{N} \beta a_{ij} I_j(t) + \gamma R_i(t) - \sigma S_i(t) \\
\frac{dI_i(t)}{dt} &= S_i(t) \sum_{j=1}^{N} \beta a_{ij} I_j(t) - \delta I_i(t) \\
\frac{dR_i(t)}{dt} &= \delta I_i(t) - \gamma R_i(t) + \sigma S_i(t),
\end{align*}
\] (19)

for \( i = 1, \ldots, N \), with initial conditions

\[ ((S_1(0), \ldots, S_N(0), I_1(0), \ldots, I_N(0), R_1(0), \ldots, R_N(0)) \in \hat{\Gamma},\]

\[ \hat{\Gamma} = \{(S_1, \ldots, S_N, I_1, \ldots, I_N, R_1, \ldots, R_N) \in \mathbb{R}_+^{3N} | S_i + I_i + R_i = 1, i = 1, 2, \ldots, N \} \]

where, \( \mathbb{R}_+^{3N} \) is the non-negative orthant of \( \mathbb{R}^{3N} \). Since \( S_i(t) + I_i(t) + R_i(t) = 1 \), we can omit the equation for the probability of being in the susceptible state and obtain

\[
\begin{align*}
\frac{dI_i(t)}{dt} &= (1 - I_i(t) - R_i(t)) \sum_{j=1}^{N} \beta a_{ij} I_j(t) - \delta I_i(t), \\
\frac{dR_i(t)}{dt} &= (\delta - \sigma)I_i(t) - (\gamma + \sigma)R_i(t) + \sigma,
\end{align*}
\] (20)

for \( i = 1, \ldots, n \), with initial conditions

\[ ((I_1(0), \ldots, I_N(0), R_1(0), \ldots, R_N(0)) \in \Gamma,\]

where

\[ \Gamma = \{(I_1, \ldots, I_N, R_1, \ldots, R_N, \ldots) \in \mathbb{R}_+^{2N} | I_i + R_i \leq 1, i = 1, 2, \ldots, N \}. \]

The region \( \hat{\Gamma} \) and \( \Gamma \) are positively invariant for the system (19) and (20), respectively (see [35]).

As discussed for the SIS and SIR model in literature, we conjecture that also for the SIRS model the following inequality holds

\[ P(X_i(t) = S, X_j(t) = I) \leq P(X_i(t) = S)P(X_j(t) = I), \] (21)

that is
\[ P(X_i(t) = S | X_j(t) = I) \leq P(X_i(t) = S). \]

The intuitive idea behind this is that an infected neighbor does not increase the probability of an individual to remain susceptible [36]. A first rigorous proof of the positive correlation between infection states was provided in [13] for the SIS Markov model and for a general (non-Markov) SIR model, and much later again proved for the Markovian SIS in [9] (see also the discussion in [10]).

If (21) holds for our SIRS model, we would have that the derivative of the infection probability in (19) is always overestimated as a consequence of the independence assumed in (18). Thus, the probability of infection for each node in the approximated model would provide an upper bound of the exact infection probability in the Markov model. This seems also to be confirmed by the simulation reported in Section 5, where we compare the exact model with the approximated one. Hence, from a practical point of view, to prevent epidemics in a network, the mean-field model would put us always on the safe side, as provided for other epidemic models. [33, 36, 10].

4. Stability Analysis

The disease free equilibrium (DFE) of the system (20) is given by the vector \( P_0 = (I_0^1, \ldots, I_0^N, R_0^1, \ldots, R_0^N) \), where

\[ I_i^0 = 0, \quad \text{and} \quad R_i^0 = \frac{\sigma}{\gamma + \sigma}, \quad i = 1, \ldots, N. \]

(22)

Let us note that for the SIRS model without vaccination, i.e. \( \sigma = 0 \), \( R_i^0 = 0 \) and \( I_i^0 = 0 \), for \( i = 1, \ldots, N \).

The positive constant solution, i.e., the endemic equilibrium \( P^* \), for the SIRS model (20) has the following components

\[ I_i^* = \frac{1}{\delta} \frac{\gamma \sum_{j=1}^N \beta a_{ij} I_j^*}{\sum_{j=1}^N \beta a_{ij} I_j^*(1 + \gamma / \delta) + (\gamma + \sigma)}, \]

\[ R_i^* = 1 - I_i^* - \frac{\gamma}{\sum_{j=1}^N \beta a_{ij} I_j^*(1 + \gamma / \delta) + (\gamma + \sigma)}, \]

for \( i = 1, \ldots, N \). Summing \( I_i^* \) over all nodes, and divided by \( N \), we obtain the average fraction of infected nodes in the steady state, \( \bar{I}^* \).

Theorem 4. Let us consider the system (20) and let \( D = \frac{\gamma}{\gamma + \sigma} \beta A - \delta I_N \times N \), whose maximum eigenvalue is

\[ \lambda_1(D) = \frac{\gamma}{\gamma + \sigma} \beta \lambda_{\max}(A) - \delta. \]

The following statements hold

a) If \( \tau \leq \frac{\gamma}{\gamma + \sigma} \frac{1}{\lambda_{\max}(A)} \) the disease free equilibrium \( P_0 \) is globally asymptotically stable in \( \Gamma \). \( P_0 \) is the unique equilibrium of the system (20) on the boundary of \( \Gamma \).
b) If \( \tau > \frac{\gamma + \sigma}{\gamma} \cdot \frac{1}{\lambda_1(A)} \), \( P_0 \) is a saddle point, the system (20) is uniformly persistent and it has a unique positive constant solution \( P^* \) in \( \bar{\Gamma} \). Moreover if \( \delta \geq \sigma \), \( P^* \) is asymptotically stable.

Proof. See [35]. □

Thus, for the SIRS\(_v\) model, the critical threshold separating the region of extinction from the persistent one is

\[
\tau^{(1)}_{c,SIRS_v} = \frac{\gamma + \sigma}{\gamma} \cdot \frac{1}{\lambda_1(A)}. \tag{23}
\]

In [35], the authors give also sufficient conditions for the global stability of the endemic equilibrium in \( \bar{\Gamma} \). Precisely, in the homogeneous setting, we would have:

**Theorem 5.** Let \( \tau > \frac{\gamma + \sigma}{\gamma} \cdot \frac{1}{\lambda_1(A)} \). Then, \( P^* \) is globally asymptotically stable in \( \bar{\Gamma} \), if one of the following two conditions hold:

a) \( \delta > \sigma \), and \( \lambda_1(A) < \frac{1}{\beta} \cdot \min \left\{ \frac{S_i^*}{(S_i^*)^2} \right\} \cdot \min \left\{ \frac{S_i^*}{1-S_i^*} \right\} \),

b) \( \delta = \sigma \).

Proof. See [35]. □

Let us note that the condition in a), regarding the maximum eigenvalue of \( A \), might not hold in some circumstances. For example, in the Remark 2, we shall prove that for the case of regular graphs condition a) is never satisfied, hence it cannot be used for verifying the global attractivity of the endemic equilibrium. Thus, in the next section, we shall investigate the attractivity of the endemic equilibrium in this specific case. Specifically, for dynamics over a regular graph, we find an invariant subset of \( \bar{\Gamma} \) (and, consequently of \( \Gamma \)), and we prove that, above the threshold and under the condition \( \delta > \sigma \), this subset is included in the domain of attraction of the endemic equilibrium. To prove this we pass through the theory of equitable partitions and we shall see how in this particular case the equilibrium points can be computed by a reduced system.

4.1. **Attractivity of the the endemic equilibrium: regular graphs.** In this section we dwell on the graph-theoretical notion of equitable partition [31, 17, 24]. A network with an equitable partition of its node set posses certain structural regularity of the graph connectivity. Based on this, we shall analyse the domain of attraction of the endemic equilibrium in the case of regular graphs that can be seen as a graph with an equitable partition.

4.1.1. **Equitable partitions.** In the following, we report the definition of equitable partition [31].

**Definition 6.** Let \( G = (V, E) \) be an undirected graph. The partition \( \pi = \{V_1, \ldots, V_n\} \) of the node set \( V \) is called equitable if for all \( i, j \in \{1, \ldots, n\} \), there is an integer \( d_{ij} \) such that

\[
d_{ij} = \deg(v, V_j) := \# \{ e \in E : e = \{v, w\}, w \in V_j \}. \tag{24}
\]

independently of \( v \in V_i \).
An equitable partition generates the quotient graph $G/\pi$, which is a multigraph with the cells $V_1, \ldots, V_n$ as nodes and $d_{ij}$ edges between $V_i$ and $V_j$. For simplicity, one can identify $G/\pi$ in a (simple) graph having the same node set, and where an edge exists between $V_i$ and $V_j$ if at least one exists in the original multigraph [6].

This partition of the node set can be adopted for representing a population divided in communities, a framework that captures some of the most salient structural inhomogeneities in contact patterns in many applied contexts [2, 26]. For an overview of the use of equitable partitions, from a theoretical and practical point of view, see e.g., [6, 26, 25]. One can identify the set of all nodes in $V_i$ as the $i$-th community of the whole population. In particular, each $V_i$ induces a subgraph, $G_i$, of $G$ that is necessarily regular. Hereafter, as in [6], we consider two infection rates: the intra-community infection rate $\beta$ for infecting individuals in the same community and the inter-community infection rate $\epsilon\beta$ i.e., the rate at which individuals among different communities get infected. We assume $0 < \epsilon < 1$, the customary physical interpretation is that infection across communities occur at a much smaller rate. Clearly the model can be extended to the case $\epsilon \geq 1$.

In the case of two different infection rates, we replace the unweighted adjacency matrix in the system (19) with its weighted version, incorporating the parameter $\epsilon$ (see [6, Example 3.1]). Interestingly, the spectral radius of the smaller quotient graph (that is of the quotient matrix related to the quotient graph (see [6])) is equal to the spectral radius of the matrix $A$ (see [6, Prop 3.3]).

In [6], the authors show that it is possible to reduce the number of equations representing the time-change of infection probabilities when all nodes belonging to the same cell have the same initial conditions. After proving the existence of a positively invariant set for the original system of $N$ differential equations, they show that the endemic equilibrium belongs to this invariant set and that it can be computed by the reduced system of $n$ equations. In the following, we want to prove the same for the case of the SIRS model (with vaccination).

Let us consider the average value of the state probabilities at time $t$ of nodes in $G_h$,

$$ S_h(t) = \frac{1}{k_h} \sum_{i \in G_h} S_i(t), \quad T_h(t) = \frac{1}{k_h} \sum_{i \in G_h} I_i(t), \quad R_h(t) = \frac{1}{k_h} \sum_{i \in G_h} R_i(t), $$

where $k_h$ is the cardinality of $G_h$, $h = 1, \ldots, n$. Then, it holds

**Theorem 7.** Let $G = (V, E)$ an undirected graph and $\pi = \{V_h, h = 1, \ldots, n\}$ an equitable partition of the node set $V$. Let $G_h$ be the subgraph of $G = (V, E)$ induced by the cell $V_j$. Let $Y = (S_1, \ldots, S_N, I_1, \ldots, I_N, R_1, \ldots, R_N) \in \tilde{\Gamma}$. Then, the subset of $\tilde{\Gamma}$

$$ \tilde{\Omega} = \{ Y \in \tilde{\Gamma} | S_i = S_h, I_i = T_h, R_i = R_h, \forall i \in G_h, h = 1, \ldots, n, \} $$

is positively invariant for the system (19).

**Proof.** From (19), we have for all $i \in G_h$, $h = 1, \ldots, n$
that (29) is equal to zero. Finally, from (25) and (28), we have
\[
\forall i, r \in G_h, m \in \{1, \ldots, n\}, \sum_{z \in G_m} (a_{iz} - a_{rz}) = 0,
\]
we have
\[
\sum_{z \in G_m} (a_{iz} - a_{rz}) \delta_h = \sum_{z \in G_m} (a_{iz} - a_{rz}) \delta_h,
\]
which implies
\[
\frac{d(R_i - \overline{R_h})}{dt} = \delta(I_i - \overline{I_h}) - \gamma(R_i - \overline{R_h}) + \sigma(S_i - \overline{S_h}),
\]
(27)

Now, let us focus on the nonlinear part in (25) (and in (26)). We have
\[
\frac{d(S_i - \overline{S_h})}{dt} = -\beta \left[ S_i \sum_{z=1}^{N} a_{iz} I_z - \frac{1}{k_h} \sum_{r \in G_h} \sum_{z=1}^{N} S_r a_{rz} I_z \right] + \gamma(R_i - \overline{R_h}) \tag{25}
\]
\[
- \sigma(S_i - \overline{S_h})
\]
\[
\frac{d(I_i - \overline{I_h})}{dt} = \beta \left[ S_i \sum_{z=1}^{N} a_{iz} I_z - \frac{1}{k_h} \sum_{r \in G_h} \sum_{z=1}^{N} S_r a_{rz} I_z \right] - \delta(I_i - \overline{I_h}), \tag{26}
\]
\[
\frac{d(R_i - \overline{R_h})}{dt} = \delta(I_i - \overline{I_h}) - \gamma(R_i - \overline{R_h}) + \sigma(S_i - \overline{S_h}), \tag{27}
\]
Now, from the last equation in (28)
\[
\frac{1}{k_h} \sum_{r \in G_h} \sum_{m=1}^{n} (a_{iz} - a_{rz}) = 0, \tag{29}
\]
Then, since \( \forall i, r \in G_h \) and \( \forall m \in \{1, \ldots, n\} \), the sum
\[
\sum_{z \in G_m} (a_{iz} - a_{rz}) = 0,
\]
we have that (29) is equal to zero. Finally, from (26) and (28), we come to have
\[ \frac{d(S_i - \overline{S}_h)}{dt} = -\beta \frac{1}{k_h} \left[ \sum_{r \in G_h} \sum_{m=1}^{n} \sum_{z \in G_m} (a_{iz}(S_i - \overline{S}_h) - a_{rz}(S_r - \overline{S}_h))(I_z - \overline{T}_m) \right. \]
\[ \left. + \sum_{r \in G_h} \sum_{m=1}^{n} \sum_{z \in G_m} (a_{iz}(S_i - \overline{S}_h) - a_{rz}(S_r - \overline{S}_h))I_m \right] - \sigma(S_i - \overline{S}_h) + \gamma(R_i - \overline{R}_h), \]
\( \forall i \in G_h, h = 1, \ldots, n. \)

Similarly,
\[ \frac{d(I_i - \overline{T}_h)}{dt} = \beta \frac{1}{k_h} \left[ \sum_{r \in G_h} \sum_{m=1}^{n} \sum_{z \in G_m} (a_{iz}(S_i - \overline{S}_h) - a_{rz}(S_r - \overline{S}_h))(I_z - \overline{T}_m) \right. \]
\[ \left. + \sum_{r \in G_h} \sum_{m=1}^{n} \sum_{z \in G_m} (a_{iz}(S_i - \overline{S}_h) - a_{rz}(S_r - \overline{S}_h))I_m \right] - \delta(I_i - \overline{T}_h), \]
\( \forall i \in G_h, h = 1, \ldots, n. \)

Now, let us denote by \( g(t) \) the solution of the system \( \mathcal{G} \), with equations (25), (26), (27), where \( g : \mathbb{R} \rightarrow \mathbb{R}^{3N} \) and consider the case where
\[ S_i(0) - \overline{S}_h(0) = 0, \quad I_i(0) - \overline{T}_h(0) = 0, \quad R_i(0) - \overline{R}_h(0) = 0 \quad \forall i \in G_h \] (32)

that means, \( S_i(0) = S_r(0), I_i(0) = I_r(0), R_i(0) = R_r(0) \), for all \( i, r \in G_h, h = 1, \ldots, n \). Then, from (30), (31), (27) we can easily see that the identically zero function \( g \equiv 0 \) is the unique solution of the system \( \mathcal{G} \), with initial conditions (32). Indeed, \( g \equiv 0 \), means that for all \( t \geq 0, \)
\[ S_i(t) = S_r(t), \quad I_i(t) = I_r(t), \quad R_i(t) = R_r(t), \quad \forall i, r \in G_h, \] (33)

\( h = 1, \ldots, n. \) Moreover, the vector with components as in (33) is a solution of (19) and it is unique in \( \tilde{\Gamma} \), with respect to the initial conditions (32), hence \( g \equiv 0 \) is a unique solution of \( \mathcal{G} \). Thus, we have that \( \tilde{\Omega} \) is positively invariant for the system (19).

\[ \square \]

Thus, under the hypothesis in Thm. 7, considering initial conditions in \( \tilde{\Omega} \), we can reduce the original system (19) of \( 3N \) differential equations and describe the time-change of the state probabilities by a system of \( 3n \) equations. The same argument can be applied to the system (20). Specifically, we have
\[
\frac{dS_h}{dt} = -\beta S_h \sum_{m=1; m \neq h}^{n} \varepsilon d_{hm} I_m - \beta S_h d_h I_h + \gamma R_h - \sigma S_h, \tag{34}
\]

\[
\frac{dI_h}{dt} = \beta S_h \sum_{m=1; m \neq h}^{n} \varepsilon d_{hm} I_m + \beta S_h d_h I_h - \delta I_h,
\]

\[
\frac{dR_h}{dt} = \delta I_h - \gamma R_h + \sigma S_h,
\]

where \(d_h\) is the internal degree of \(G_h\).

**Remark 1.** From the uniqueness argument in Thm 4 b), it is immediate to deduce that when \(G\) is a graph with equitable partitions the endemic equilibrium of the system (19) must belong to \(\hat{\Omega} \cap \hat{\Gamma}\). Thus, it can be computed by means of the reduced system (34).

### 4.1.2. Regular Graphs

In this section we consider regular graphs. We can consider these graphs, where all nodes have the same degree, as having an equitable partition with a single cell. It holds

**Theorem 8.** Let \(G = (V, E)\) be an undirected regular graph with degree \(d_G\), and \(\tau > \frac{\gamma + \sigma}{\lambda_1(A)}\).

Then, if \(\delta > \sigma\), the endemic equilibrium \(Y^* = (S_1^*, \ldots, S_N^*, I_1^*, \ldots, I_N^*, R_1^*, \ldots, R_N^*)\) is asymptotically stable in \(\hat{\Gamma}\) and \(\hat{\Omega} \cap \hat{\Gamma}\) is a subset of the domain of attraction of \(Y^*\).

**Proof.** The asymptotic stability is provided in Thm 4 b). Thus, we have to prove that under the condition \(\delta > \sigma\) we can identify a subset of the domain of attraction of the endemic equilibrium in the case of regular graphs.

We can apply the Thm 7 in the case of \(G\) regular graph. From (34), and since \(S + I + R = 1\), we obtain

\[
\frac{dI}{dt} = \beta d_G(1 - I - R)I - \delta I, \tag{35}
\]

\[
\frac{dR}{dt} = \delta I - \gamma R + \sigma(1 - I - R),
\]

with initial conditions \((I(0), R(0)) \in \hat{\Gamma}'\), with \(\Gamma' = \{(I, R) \in \mathbb{R}_+^2 | I + R \leq 1\}\). By Thm 7, \(S_i(t) = S(t), I_i(t) = I(t), R_i(t) = R(t), i = 1, \ldots, N,\) for all \(t \geq 0\), since all nodes have the same trajectories when starting with the same initial conditions.

Now, let us consider the Volterra-type function, \(U = I - I^* - I^* \ln(I/I^*)\), used by many authors [18, 16, 4, 21] and the common quadratic function \(Z = \frac{1}{2}(R - R^*)^2\). Since from the equilibrium equations \(\beta(1 - I^* - R^*)dI^* - \delta I^* = 0\) and \(\delta I^* - \gamma R^* + \sigma(1 - I^* - R^*) = 0\), after some manipulations, we obtain

\[
U' = -\beta d_G(I - I^*)^2 - \beta d_G(I - I^*)(R - R^*),
\]

\[
Z' = (\delta - \sigma)(R - R^*)(I - I^*) - (\gamma + \sigma)(R - R^*).\]
Let us define $V = cU + Z$, where $c = \frac{(\delta - \sigma)}{\beta d}$, $\beta > 0$. Then,

$$V' = - (\delta - \sigma) (I - I^*)^2 - (\gamma + \sigma)(R - R^*)^2,$$

so that $V' \leq 0$ and $V' = 0$ if and only if $I = I^*$ and $R = R^*$. Thus, $V$ is a Lyapunov function for the system (35) and by a classical theorem of Lyapunov we have the global attractivity (and the local stability) of the endemic equilibrium $(I^*, R^*)$ in $\tilde{\Gamma}$.

Consequently, for a dynamics over a regular graphs, when $Y(0) \in \tilde{\Omega} \cap \tilde{\Gamma}$, the trajectories of the original system (19) coincide with the solution obtained from the reduced system (35) (recalling that $S = 1 - I - R$). Hence, $\lim_{t \to \infty} Y(t) = Y^*$, where $Y^* \in \tilde{\Omega} \cap \tilde{\Gamma}$.

Remark 2. Let us note that in the case of regular graphs, above the threshold (23), the condition in Thm. 4

$$\lambda_1(A) < \frac{1}{\beta} \cdot \min \left\{ \frac{\delta I^*_i}{(S^*_i)^2} \right\} \cdot \min \left\{ \frac{S^*_i}{1 - S^*_i} \right\},$$

is never satisfied. Indeed, from Remark 1, we have that $S^*_i = S^*_j$ and $I^*_i = I^*_j$ for all $i, j = 1, \ldots, N$, and we can use the reduced system (35) for computing the steady state vector. Thus, from the equilibrium equation $(\beta S^*d_G - \delta)I^* = 0$, when $I^* \neq 0$, we have $S^* = \frac{\delta}{\beta d_G}$. Since $I^* < 1 - S^*$, we obtain

$$\frac{1}{\beta} \cdot \frac{\delta I^*}{S^*} < \frac{\delta}{\beta S^*} = d_G = \lambda_1(A).$$

4.2. Notes on the basic SIRS epidemic model. From Thm 4 we can see that for a basic SIRS model, i.e., by setting $\sigma = 0$, it holds

$$\tau^{(1)}_{c: SIRS} = \frac{1}{\lambda_1(A)}. \quad (36)$$

Moreover, from Thm. 4b), above $\tau^{(1)}_{c: SIRS}$ the asymptotic stability of the endemic equilibrium is always ensured, without further conditions.

Let us note that

$$\tau^{(1)}_{c: SIRS} = \tau^{(1)}_{c: SIS} = \tau^{(1)}_{c: SIR}, \quad (37)$$

see, indeed, for the SIS and SIR threshold, e.g., [33, 6, 34]. Comparing (36) with (23), it is clear how the introduction of vaccination extends the region of extinction, that is values of $\delta$ and $\beta$ for which the epidemics would persist without vaccination can be instead sufficient to drop the epidemics if the vaccination is introduced in the population. The mean-field threshold for the SIRS model is not able to capture the role of $\gamma$ in the extinction and persistence of epidemics. However, the value of $\gamma$ in the mean-field model explicitely influences the average fraction of infected nodes in the steady state, indeed for the SIRS model the positive equilibrium point has the following components:

$$I^*_i = \frac{1}{\frac{1}{1 + \delta/\gamma} \left( 1 - \frac{1}{\frac{1}{1 + \tau(1 + \delta/\gamma) \sum_{j=1}^{N} a_{ij} I_j} } \right) }, \quad (38)$$
\[ R_i^* = \frac{\delta}{\gamma} I_i^*, \quad S_i^* = 1 - \left(\frac{\gamma + \delta}{\gamma}\right) I_i^*, \]  

(39)

for \( i = 1, \ldots, N \). We can see that for fixed values of \( \beta \) and \( \delta \), as \( \gamma \) increases the steady state solution \( I_i^* \) approaches that of the SIS model (see [33]). This is easy to understand since, as \( \gamma \) increases, the average immune period tends to decrease (a removed individual quickly return to the susceptible state) and the behaviour of the SIRS model approaches that of the SIS model. Conversely, if the value of \( \gamma \) goes down (the return to the susceptible state is protracted) the probability of being infectious tends to decreases, detaching from the SIS meta-stable solution.

5. Numerical investigations

In Fig. 2, we consider the average fraction of infected nodes of the SIRS \(_v\) models, as function of time and \( \sigma \), for a complete graph with \( N = 50 \), by fixing \( \beta = 0.25 \), \( \delta = 0.4 \) and \( \gamma = 0.2 \). We can see how increasing the value of the rate of vaccination \( \sigma \), the steady-state average fraction of infected nodes decreases, thus passing from a region of persistence to a region of extinction. Thus, once known the other parameters, we can calibrate the value of \( \sigma \) to guide the epidemic towards the extinction.

In Fig. 3 we report the steady-state average fraction of infected nodes, \( \bar{I}^* \), as function of \( \gamma \), for different values of \( \sigma \), by considering a complete graph with \( N = 50 \), \( \beta = 0.25 \) and \( \delta = 0.9 \). We can see that, by fixing the value of \( \sigma \), the value of \( \bar{I}^* \) increases as \( \gamma \) increases, thus a shorter immunity period leads to a more aggressive epidemic. Viceversa, by fixing \( \gamma \), the value of the prevalence in the steady-state clearly decreases as \( \sigma \) increases. Thus, the less time each individual remains unvaccinated, the more the entire population will benefit in terms of percentage of infected individuals in the long-run.

![Figure 2. SIRS \(_v\) average fraction of infected nodes as function of time and \( \sigma \), for a complete graph with \( N = 50 \), \( \beta = 0.25 \), \( \delta = 0.4 \), \( \gamma = 0.2 \). At time 0 there is one infected node.](image-url)
Fig. 3. SIRS\(_v\) steady-state average fraction of infected nodes, \(\bar{I}^*\), as function of \(\gamma\), for different values of \(\sigma\), for a complete graph with \(N = 50\), \(\beta = 0.25\), \(\delta = 0.9\).

Fig. 4 depicts the solutions of the system (20) for two nodes of a regular graph with \(N = 50\) and \(d_G = 10\), starting with different initial conditions. These solutions are compared with the one computed using the reduced system obtained from (34), with initial conditions equal to \(I_h(0)\), and \(R_h(0)\). We can see that trajectories starting outside the invariant set \(\Omega\) tend to approach the one starting in \(\Omega\) as time elapses. It can be seen that, as pointed out in the Remark, the positive equilibrium belongs to \(\Omega \cap \Gamma\), and can be computed with the reduced system. Thus, from the numerical investigation, we can note that even when the initial conditions of the nodes are different, the trajectories are attracted by the endemic equilibrium.

In Fig. 5 we consider a complete graph with \(N = 50\) and provide a comparison between the dynamics of the prevalence, obtained from the solution of the ODE system (20), and the averaged \(2 \cdot 10^4\) sample paths resulting from the discrete event simulation of the exact SIRS\(_v\) process. In Fig. 5a) we consider values of the parameters such that \(\tau < \tau_{c_{SIRS}}^{(1)}\), while in b) and c) values for which \(\tau > \tau_{c_{SIRS}}^{(1)}\). We can see that in a) only in the early phase the approximated model is slightly above the exact dynamics. In b) for the chosen parameters values, i.e. \(\beta = 1\), \(\delta = 0.45\), \(\gamma = 0.2\), \(\sigma = 0.4\), there is a quite perfect match. Interestingly, in c) when we consider the same values for \(\beta\), \(\delta\) and \(\sigma\), but \(\gamma = 0.06\) we have a different qualitative behavior between the exact and the approximated model after a certain point in time. Indeed, we can see that the exact prevalence, after reaching the peak, starts to decrease towards the state with no infected quite early, while in the approximate model, the infection remains persistent.

In Fig. 6 we report the same type of comparison done in Fig. 5 but for a regular graph with \(N = 50\) and \(d_G = 10\). We can see that, for the chosen parameters, the solution of the approximated model stays slightly above that of the exact model, thus providing an upper bound for the exact dynamics. However we can note that in a), as well as in b) for the time window considered, the qualitative behavior
is the same between the two models. For the chosen values of the parameters in b), the stochastic dynamics seems to stand on a positive value for long time before reaching the absorption, resembling the behavior of the mean-field model that above the threshold reaches the positive equilibrium. However, in c), as for the complete graph case, when we have the same \( \beta, \delta \) and \( \sigma \), but a lower \( \gamma \) than in b), the qualitative behavior between the dynamics of the two models is different and after the peak the exact dynamics reaches the extinction quite early. Thus, let us rewrite the condition for the extinction \( \tau \leq \tau_{c;SIRS_v}^{(1)} \) in the following way

\[
\rho \leq \frac{1}{\lambda_1(A)} = \tau_c, \quad \text{where} \quad \rho = \frac{\beta \gamma}{\delta(\gamma + \sigma)}.
\]

Then, we can assert that from Fig. 5 and Fig. 6, in some \( \rho \)-region around \( \tau_c \), we can observe deviations between the mean-field and the exact model. Thus we could expect that, in general, deviations between the two models are expected for intermediated value of \( \beta \gamma / (\delta(\gamma + \sigma)) \). This behavior can also be observed in the SIS model in a \( \tau \)-region around \( \tau_c \) [33].

![Figure 4](image-url)
Figure 5. Stochastic simulation versus numerical solution obtained from (20), for the SIRS model with vaccination. Complete graph with $N = 50$, $\sigma = 0.4$. a) $\beta = 0.1$, $\delta = 0.9$, $\gamma = 0.1$, $\tau < \tau^{(1)}_{c,SIRS_v}$. b), $\beta = 1$, $\delta = 0.45$, $\gamma = 0.2$, $\tau > \tau^{(1)}_{c,SIRS_v}$. c) $\beta = 1$, $\delta = 0.45$, $\gamma = 0.06$, $\tau > \tau^{(1)}_{c,SIRS_v}$. At time 0 there is one infected node.

6. Conclusion

In this work, we started by considering the exact stochastic Markov description of a SIRS model, with vaccination, on networks. In this context, we investigated the mean time of the epidemic, through the topological properties of the graph. We found conditions for a quick extinction, when there are no more infected, to avoid a long-term persistence.

Starting from a node-level description of the exact Markov process, that becomes neither analytically nor computationally tractable with increasing number of nodes $N$, we derived an approximation of it by means of a first-order meanfield technique. We obtained a set of $3N$ nonlinear differential equations, specifying the state probabilities of each node. Based on the stability results in [35], we provided the critical threshold, which separates an extinction region from an endemic one, in terms of the parameters and the network topology. In this way it is made explicit to what extent the threshold and the steady-state solutions are influenced by the value of the loss-immunity parameter, and by the introduction of the vaccination, comparing the results with the basic SIRS model.

Since, as we showed, a sufficient condition provided in [35], ensuring the global attractivity of the positive equilibrium is never satisfied in the case of regular graphs,
we analyzed the domain of attraction of the positive equilibrium, for these specific
graphs. For this purpose we used the notion of equitable partitions. We proved the
existence of a positively invariant set for the system when a graph posses an equi-
table partition, and we showed that, when the initial conditions belong to this set,
the whole epidemic dynamics can be expressed by a reduced system. This reduced
system can be used for the computation of the endemic equilibrium that belongs
to the invariant set. Since a regular graph is a special case of graph with equitable
partition, we showed that, when the recovery rate is higher than the vaccination
rate, the aforementioned invariant set is contained in the domain of attraction of
the endemic equilibrium. Finally, we provided numerical investigations, comparing
also the exact stochastic model with the approximated one.
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