Epidemic spreading model of COVID-19

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We study Susceptible-Exposed-Asymptomatic-Infectious-Recovered (SEAIR) epidemic spreading model inspired by two characteristics of the infectiousness of COVID-19: delayed start and its appearance before onset of symptoms, or even with total absence of them. The model is theoretically analyzed in continuous-time compartmental version and discrete-time version on random regular graphs and complex networks. We show analytically that there are relationships between the epidemic thresholds and the equations for the susceptible populations at the endemic equilibrium in all three versions, which hold when the epidemic is weak. We provide theoretical arguments that eigenvector centrality of a node approximately determines its risk to become infected.

I. INTRODUCTION

Understanding epidemic spreading of contagious diseases and effectiveness of various countermeasures is of high interest for public health and the society in general, with important contributions provided by epidemiologists, mathematicians and physicists as well. Although earliest theoretical work in mathematical epidemiology dates back to Daniel Bernoulli [1], the development of the modern approach started in the beginning of the past century [2,3]. In the last two decades, since the emergence of the complex networks theory, epidemic modeling has gained novel insights. By modeling the contacts between the individuals with complex networks, association was found between the epidemic threshold with the network properties like the degree distribution or eigenvalue of the adjacency matrix [4-10]. Moreover, the epidemic spreading has grown as a concept that extends its original design for modeling diffusion of infectious disease to sharing ideas, rumors, or computer viruses [11].

In the classical approach, the individuals are conveniently grouped in compartments or classes. The mathematical models for epidemic spreading are systems of differential equations for the evolution of the size of those compartments. In such models, there is assumption of homogeneous mixing, which means that the pathogen can spread between each pair of individuals with equal probability. In a more realistic modeling each individual is considered as node in certain network of contacts, where infection can spread only among neighbors in that network. This is particularly relevant, because real world networks besides being random, among others they possess properties like small-world phenomenon [11], or scale-free distribution of the node degrees [12]. One approach for studying the disease spreading on networks is the heterogeneous mean-field [6,13] in which all nodes with the same degree are assumed to be statistically equivalent. The quenched mean-field technique is applied in even more realistic scenario, where each node is treated separately [14,15]. Among major contributions in the field of epidemic spreading on networks are also the studies of spreading on multilayer networks [16], disease localization [17] and the assumption of non-exponential distribution of periods between consecutive events [18,19].

Various infections are characterized with different stages in the course of development of the disease in the host, starting from contracting the pathogen to the healing. Depending on the disease under study, several compartments, or classes are defined in order to differentiate between the stages. The most frequently used compartments are the Susceptible (S), Exposed (E), Infectious (I) and Recovered (R) [4,20]. The meaning of the compartmental symbols usually is: S – healthy individuals subjected to infection, E – infected which do not transmit the disease yet, I – infected and infectious and R – cured which cannot become infected again. The most popular models are the SIS and SIR, which are sufficiently simple to provide mathematical tractability, and powerful enough to capture the features of epidemics of many contagious diseases [20].

The ongoing COVID-19 is the largest pandemic in modern history. The understanding of the virus and its influence on the infected individuals are still in progress. However, it was uncovered that it features a slow progress that might take a long period from inception to cure and some infected individuals can be unnoticed because they might not have any symptoms while spreading the disease [21]. Some studies even report that the infectiousness is strongest before and at the onset of appearance of symptoms [22]. In order to properly describe such contagion one needs a model which incorporates delay effects and also has different stages with possibly different virulence. Since its emergence, numerous mathematical models of the COVID-19 with different complexity have appeared (a short and not exhaustive list of references is [23-26]). In this theoretical work we con-

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sider the known Susceptible-Exposed-Infected-Recovered (SEIR) model augmented with another state, the asymptomatic state (A), which precedes the infectious. The chosen SEAIR model has a built-in delay which means that the infected person do not start to spread the disease immediately, allows to address the different contagiousness of the disease in different phases and captures the presence of undetected spreaders. Also, in our opinion it is simple enough to allow for theoretical study of different properties. We study the SEAIR model with two approaches: the classical, which uses differential equations, and the statistical physics-based, by considering discrete-time evolution model on complex networks. We study the latter model on random regular graph and on complex network separately. For the three versions we obtain the epidemic threshold, equation for the fraction of susceptible individuals at the end of epidemic and study linear stability of the disease-free and endemic equilibria. The results for the random regular graph hold when the contagiousness is weak, while for the complex network it is also needed that epidemic is weak in a sense that small fraction of population is affected by the epidemic. We furthermore study the roles of the leading eigenvalue and the principal eigenvector of the adjacency matrix in the epidemic spreading. It is already known that leading eigenvalue determines the epidemic threshold and thus determines the stability [14, 27]. We show that the principal eigenvector and its associated eigenvector centrality have important role in estimation of the risk of infection. Finally, the techniques which we apply for analysis of the Jacobian matrix of the evolution might be relevant in studies of linear stability of coupled multidimensional dynamical systems.

The paper is organized as follows. In Section II we introduce and analyze the SEAIR compartmental model. In the following Section III is studied the discrete-time version, while in Section IV we present some results of numerical experiments. The paper ends with the Conclusion.

II. SEAIR COMPARTMENTAL MODEL

Let the variables $S$, $E$, $A$, $I$ and $R$ denote the fraction of individuals which are respectively susceptible, exposed, asymptomatic, infectious and recovered. We assume that the exposed stage corresponds to the incubation period when host has the pathogen, but he or she cannot infect the others. In the asymptomatic stage the individual spreads the disease, possibly with higher virulence without being aware about having the virus. The person can even recover without ever noticing that he, or she had the disease. Certain percentage of carriers of the virus will show symptoms and we classify them as infectious. Let the infecting rate of the asymptomatic persons is $\alpha$, while of infectious ones is $\beta$. The rate at which exposed individuals become asymptomatic is $\gamma$. The growth of the fraction of infectious hosts, which have symptoms is determined with rate $\sigma$. For simplicity, we assume that healing of both the asymptomatic and infectious persons is modeled with same rate $\mu$. We emphasize that $\mu$ does not exactly correspond to the time of complete healing, but the period in which a person can infect the others. With these assumptions one has the following SEAIR compartmental model

\[
\begin{align*}
S &= -\alpha AS - \beta IS, \\
E &= \alpha AS + \beta IS - \gamma E, \\
A &= \gamma E - \sigma A - \mu A, \\
I &= \sigma A - \mu I, \\
R &= \mu A + \mu I.
\end{align*}
\]  

(1)

We have neglected the births and deaths in the population and one can easily verify that the total number of persons in all states is constant $S(t) + E(t) + A(t) + I(t) + R(t) = 1$.

One trivial solution of the system (1) is the disease-free state $S = 1$, when the pathogen is absent. If some virus is introduced, an epidemic can occur. Then there is an endemic equilibrium which corresponds to the situation when the fraction of susceptibles is not sufficient for further spread of the disease. When epidemic occurs, the number of unaffected people can be obtained by standard technique [28]. If we sum the top four equations in (1) the following relationship will hold

\[
\frac{d(S + E + A + I)}{dt} = -\mu(A + I).
\]  

(2)

In situations when the epidemic starts with negligibly small number of virus bearers, by taking that at the finish of the epidemic the fractions of individuals with the pathogen is zero, after integration of the last equation, one obtains

\[
S(0) - S(\infty) = \mu \int_0^\infty (A(t) + I(t)) \, dt.
\]  

(3)

The first equation in (1) can be rewritten as

\[
\frac{dS}{S} = -(\alpha A + \beta I)dt,
\]  

(4)

which by integration will result in another relationship between the initial and final fractions of susceptibles

\[
\ln \frac{S(0)}{S(\infty)} = \alpha \int_0^\infty A(t)dt + \beta \int_0^\infty I(t)dt.
\]  

(5)

One can also integrate the fourth equation in (1) on both sides to obtain

\[
I_\infty - I_0 = \sigma \int_0^\infty A(t)dt - \mu \int_0^\infty I(t)dt \approx 0.
\]  

(6)

The last result provides a relationship between asymptomatic and infectious fractions in the course of the whole epidemic

\[
\sigma \int_0^\infty A(t)dt = \mu \int_0^\infty I(t)dt.
\]  

(7)
By combining the relationships (3), (5) and (7), the following equation for the fraction of unaffected individuals is obtained

$$ S(0) - S(\infty) = \frac{\mu(\mu + \sigma)}{\beta\sigma + \alpha\mu} [\ln S(0) - \ln S(\infty)]. \hspace{1cm} (8) $$

Using the fact that \( f(x) = \ln x \) is steeper than \( g(x) = x \) for \( x < 1 \), one can verify that

$$ S(0) - S(\infty) < \ln S(0) - \ln S(\infty). \hspace{1cm} (9) $$

This implies that the transcendental equation (8) has a solution only if

$$ \mu(\mu + \sigma) < \beta\sigma + \alpha\mu. \hspace{1cm} (10) $$

The last inequality is the condition of existence of endemic equilibrium \( S = S(\infty); E = A = I = 0; R = S(0) - S(\infty) \) of the system (4).

To study the linear stability of the equilibrium states, one should linearize the system (4). The respective Jacobian matrix \( J = \left[ \frac{\partial B}{\partial \partial C} \right]; B, C \in \{ S, E, A, I, R \} \), reads

$$ J = \begin{bmatrix}
-\alpha A - \beta I & 0 & -\alpha S & -\beta S & 0 \\
\alpha A + \beta I & -\gamma & \alpha S & \beta S & 0 \\
0 & \gamma & -\sigma - \mu & 0 & 0 \\
0 & 0 & \sigma & -\mu & 0 \\
0 & 0 & \mu & \mu & 0
\end{bmatrix}. \hspace{1cm} (11) $$

At the disease-free state for which \( S = 1 \) and \( E = A = I = R = 0 \), the Jacobian has rather simple form

$$ J_{DF} = \begin{bmatrix}
0 & 0 & -\alpha & -\beta & 0 \\
0 & -\gamma & \alpha & \beta & 0 \\
0 & \gamma & -\sigma - \mu & 0 & 0 \\
0 & 0 & \sigma & -\mu & 0 \\
0 & 0 & \mu & \mu & 0
\end{bmatrix}. \hspace{1cm} (12) $$

Because the first and last columns are zero, this Jacobian has two trivial eigenvalues zero, while the other three are the roots of the polynomial

$$ R(\lambda) = (-\mu - \lambda)[(-\gamma - \lambda)(-\sigma - \mu - \lambda) - \alpha\gamma] + \beta\gamma\sigma. $$

We show in Appendix A that the three eigenvalues of the Jacobian have negative real part, if the following relationship holds

$$ \mu(\mu + \sigma) > \alpha\mu + \beta\sigma. \hspace{1cm} (13) $$

The obtained inequality is opposite of the condition of the existence of endemic equilibrium, as one can expect. If (13) holds, (11) does not, the disease-free state is stable and epidemic will not occur. In the opposite, if (13) is not satisfied, the equilibrium \((1, 0, 0, 0, 0)\) is unstable, the epidemic will ensue, and the size of unaffected population can be obtained from (8). Thus the threshold at which epidemic can emerge is the following relationship

$$ \mu(\mu + \sigma) = \alpha\mu + \beta\sigma. \hspace{1cm} (14) $$

The linear stability analysis of the endemic equilibrium can be applied by the same procedure as for the disease-free one. In this case in the Jacobian (11) one should take \( S = S(\infty) \) and \( E = A = I = 0 \). By applying the same procedure which is explained in Appendix A it will be obtained that the only difference from the disease-free case is that instead of \( \alpha \) and \( \beta \) it should be used \( S(\infty)\alpha \) and \( S(\infty)\beta \) respectively. This would simply change the condition for stability of the endemic equilibrium to

$$ \mu(\mu + \sigma) > S(\infty)(\alpha\mu + \beta\sigma). \hspace{1cm} (15) $$

From (8) one has the following relationship for the fraction containing the parameters

$$ \frac{\mu(\mu + \sigma)}{\beta\sigma + \alpha\mu} = \frac{S(0) - S(\infty)}{\ln S(0) - \ln S(\infty)}. \hspace{1cm} (16) $$

Plugging the last relationship in (15), it will be obtained that the endemic equilibrium is stable once the following holds

$$ \frac{S(0) - S(\infty)}{\ln S(0) - \ln S(\infty)} > S(\infty). \hspace{1cm} (17) $$

The last inequality can be rearranged as

$$ \frac{S(0)}{S(\infty)} > 1 + \ln \frac{S(0)}{S(\infty)}, \hspace{1cm} (18) $$

which holds always since \( S(0) > S(\infty) \).

### III. DISCRETE-TIME SEAIR MODEL

Subsequent investigation of a disease spreading model when one needs to account for the contacts between the individuals, is to use epidemic spreading on complex networks framework. We consider discrete-time evolution version of the proposed SEAIR model with finite population of \( N \) individuals. The network of contacts is conveniently modeled with fixed undirected graph, in which the vertices are the individuals, while the links exist between those persons which have contact to each other. This means that the disease can be transmitted only between neighbors in the graph. The dynamical variables of interest in this model are the set of probabilities for each node being in certain state, \( S, E, A, I, \) or \( R \) at given moment \( n \). We will also denote the parameters with the same Greek letters as in the compartmental model. They correspond to the same transitions and have a meaning of probabilities instead of rates. To be more precise, \( \alpha \) is the probability that asymptomatic person will infect a susceptible neighbor at one time step, while \( \beta \) is the
respective probability in the case of contact between infectious and susceptible individual. Once becoming exposed, the person can proceed into asymptomatic phase with probability $\gamma$ at one time step, or remain in the same state with probability $1 - \gamma$. The respective probability to show symptoms by asymptomatic individual is $\sigma$. Again, as in the compartmental model, we assume identical probability $\mu$ to become recovered, for both the asymptomatic and infectious state.

The dynamics of the discrete-time version of the SEAIR model is build upon the model considered in \cite{14}. Denote the probabilities that the individual $i$ at the discrete moment $n$ is in respective state with $p_{S,i}(n)$, $p_{E,i}(n)$, $p_{A,i}(n)$, $p_{I,i}(n)$ and $p_{R,i}(n)$. Our model assumes a reactive process of epidemic spreading, which means that in each time step every individual has a contact with every neighbor \cite{20}. Then, certain susceptible person $i$ at the moment $n + 1$ will not receive the infection from any of its neighbors with probability \cite{14,20}

$$p_{S,i}(n + 1) = p_{S,i}(n)P_{i}(n),$$ \hspace{1cm} (19)

where $N_i$ is the set of neighbors of $i$. The individual will remain susceptible at moment $n + 1$, if he, or she, did not receive the contagion, which means that

$$p_{S,i}(n + 1) = p_{S,i}(n)P_{i}(n).$$ \hspace{1cm} (20)

Otherwise, an individual can become exposed if he or she has been susceptible before and received the contagion, or continue to be exposed at the next moment if the incubation has not finished, with probability

$$p_{E,i}(n + 1) = p_{S,i}[1 - P_{i}(n)] + (1 - \gamma)p_{E,i}(n).$$ \hspace{1cm} (21)

The probability of being asymptomatic at the next moment is

$$p_{A,i}(n + 1) = \gamma p_{E,i} + (1 - \sigma - \mu)p_{A,i}(n),$$ \hspace{1cm} (22)

where the last term accounts for the situation that neither the symptoms will appear, nor healing will happen in one time step. The node $i$ will be in state I at moment $n + 1$ with probability

$$p_{I,i}(n + 1) = \sigma p_{A,i}(n) + (1 - \mu)p_{I,i}(n),$$ \hspace{1cm} (23)

where the former term describes the probability to show symptoms, if in the previous moment the node was asymptomatic, while the last one corresponds to recovering. Finally, the probability of being recovered at some moment is

$$p_{R,i}(n + 1) = p_{R,i}(n) + \mu[p_{A,i}(n) + p_{I,i}(n)].$$ \hspace{1cm} (24)

A. Epidemic spreading on random regular graphs

We will pursue our analysis of spreading processes on random regular graphs where each node has the same degree $k$. For infinitely large random regular graphs, the probabilities of the states are equal for all nodes and one can drop the index of the node. The probability to avoid infection \cite{19} will be simplified to

$$P(n) = [1 - \alpha p_{A}(n) - \beta p_{I}(n)]^k.$$ \hspace{1cm} (25)

Then the system of equations for discrete-time epidemic spreading on random regular graph reads

$$p_{S}(n + 1) = p_{S}(n)\left[1 - \alpha p_{A}(n) - \beta p_{I}(n)\right]^k,$$

$$p_{E}(n + 1) = p_{S}(n)\left[1 - \alpha p_{A}(n) - \beta p_{I}(n)\right]^k + (1 - \gamma)p_{E}(n),$$

$$p_{A}(n + 1) = \gamma p_{E}(n) + (1 - \sigma - \mu)p_{A}(n),$$

$$p_{I}(n + 1) = \sigma p_{A}(n) + (1 - \mu)p_{I}(n),$$

$$p_{R}(n + 1) = p_{R}(n) + \mu[p_{A}(n) + p_{I}(n)].$$ \hspace{1cm} (26)

Here we have also two equilibrium points: one where all individuals are susceptible $p = (1, 0, 0, 0, 0)$ and the other where the fraction of susceptible individuals is such that it prevents further spread of the disease $p = (p_S^\ast, 0, 0, 1 - p_S^\ast)$. In the Appendix \cite{B} we show how application of similar reasoning as for the compartmental model can allow to find a closed form equation for the number of susceptible individuals at the end of epidemic. It can be applied when the contagion probabilities $\alpha$ and $\beta$ are very small. As is shown in the Appendix \cite{B} the equation for determination of the probability of susceptible state at the end of epidemic is very similar to the respective one for the compartmental case

$$p_{S}(0) - p_{S}(\infty) = \frac{\mu(\mu + \sigma)}{k(\alpha \mu + \beta \sigma)} \ln \frac{p_{S}(0)}{p_{S}(\infty)}.$$ \hspace{1cm} (27)

The last result extends the one for all-to-all coupling considered in compartmental models, where effectively each individual can get the disease from any one in the population. Here, it is obtained that appropriately modified relationship holds for restricted number of contacts. By repeating the same analysis as for the compartmental model, one can also obtain that the condition for existence of endemic equilibrium is

$$\mu(\mu + \sigma) < k(\beta \sigma + \alpha \mu).$$ \hspace{1cm} (28)

We can further make linear stability analysis of equilibria by linearizing the evolution equations. The respective Jacobian matrix at the fixed points for which $p_{E} = p_{A} = p_{I} = 0$ and $p_{S} = p_{S}^\ast$ is

$$J = \begin{bmatrix} 1 & 0 & -k\alpha & -k\beta & 0 \\ 0 & 1 & -\gamma & k\alpha & k\beta \\ 0 & \gamma & 1 - \sigma - \mu & 0 & 0 \\ 0 & 0 & \sigma & 1 - \mu & 0 \\ 0 & 0 & \mu & \mu & 1 \end{bmatrix}.$$ \hspace{1cm} (29)
Solving the characteristic equation \( Q(\lambda) = \det(J - \lambda I) = 0 \) of the Jacobian \([20]\), for \( p_S^2 = 1 \) will result in two trivial eigenvalues equal to one and other three. In vicinity of the epidemic start, the nontrivial eigenvalues \( \lambda \) are the roots of the polynomial in \( \lambda \),

\[
\mathcal{R}(\lambda) = (1 - \mu - \lambda) [(1 - \gamma - \lambda)(1 - \sigma - \mu - \lambda) - \alpha \gamma k] + \beta \gamma \sigma k. \tag{30}
\]

One can introduce new variable \( \nu = \lambda - 1 \) and obtain

\[
\mathcal{R}(\nu) = (\nu + \gamma) [(-\nu)\gamma - \alpha \beta \sigma k] + \beta \gamma \sigma k. \tag{31}
\]

The characteristic function is very similar to that which corresponds to the stability of the compartmental model \([13]\). The only difference is that here one should take \( k\alpha \) and \( k\beta \) instead of \( \alpha \) and \( \beta \) respectively, and that the independent variable is \( \nu \). Thus, one can apply the same reasoning as in that case, which is explained in Appendix\( A \) The conclusion would be that all roots \( \nu \) would have negative real part only if

\[
\mu(\mu + \sigma) > k(\alpha \mu + \beta \sigma). \tag{32}
\]

We remind that because \( \nu = \lambda - 1 \), when \( \nu \) has negative real part, \( \lambda < 1 \), and thus the same condition determines when the disease-free state is linearly stable.

The linear stability of the endemic equilibrium is established from the leading eigenvalue of the same Jacobian matrix \([20]\) as the disease-free one, but using \( p_S^2 = p_S(\infty) \) obtained from \([27]\). Again, the procedure is nearly the same as for the compartmental version and the only difference is that instead of \( k \) one should use \( kp_S(\infty) \) in all analysis. Then the endemic equilibrium will be stable, if the condition similar to \([32]\) is satisfied

\[
\mu(\mu + \sigma) > kp_S(\infty)(\alpha \mu + \beta \sigma). \tag{33}
\]

Without showing the details, we will just mention that once the endemic equilibrium in this discrete-time disease spreading model exists, it is linearly stable.

**B. Epidemic spreading on complex networks**

Let us now consider the general case when the contacts between individuals are described with complex network. In studies of interacting units coupled in a network it is typical to define the state by stacking the state vectors of each node one on top of another. In this case another ordering is more appropriate \([27]\). First, create vector of the probabilities of susceptible states of all nodes \( p_S = [p_{S,1}, p_{S,2}, \ldots, p_{S,N}]^T \), then those of the exposed states \( p_E = [p_{E,1}, p_{E,2}, \ldots, p_{E,N}]^T \), and likewise for the remaining three \( p_A, p_I \) and \( p_R \). Also, denote with A the adjacency matrix of the network of contacts between the individuals with elements \( A_{i,j} = 1 \) only if nodes \( i \) and \( j \) are neighbors and \( A_{i,j} = 0 \) otherwise. Under general circumstances, determination of the probabilities at the end of epidemic in case it happens, is very complicated, if not impossible. However, when the contagiousness is weak, which means that \( \alpha \ll 1 \) and \( \beta \ll 1 \), one can obtain similar expressions which relate initial and final probabilities of susceptible state as for the former two models. As it is explained in details in the Appendix\( C \) when \( \alpha \ll 1 \) and \( \beta \ll 1 \), the susceptibility probability vector can be calculated from the following self-consistent system

\[
p_S(0) - p_S(\infty) = \mu(\frac{1}{\mu} - \sigma)\sum_{n=0}^{\infty} p_A(n), \tag{34}
\]

\[
\ln p_S(0) - \ln p_S(\infty) = (\alpha + \beta \frac{\sigma}{\mu}) A \sum_{n=0}^{\infty} p_A(n). \tag{35}
\]

The solution of the system of transcendental equations equations \([34]\) consists of the susceptibility vector at the endemic equilibrium \( p_S(\infty) \) and the vector of sums of probabilities of asymptomatic states

\[
p_A = \sum_{n=0}^{\infty} p_A(n). \tag{36}
\]

Such transcendental system should be solved numerically, and for large networks might be impossible task. However, one can at least obtain how the solution will look like, when the epidemic is weak in a sense that only small fraction of the population is infected during its course. In such case the probability of susceptibility will not change significantly \( p_S(0) \approx p_S(\infty) \). This situation might be present for example when the contagiousness of the pathogens is slightly over the threshold. Then one can keep only the leading terms in the expansion of the logarithm and obtain

\[
\ln p_{S,i}(0) - \ln p_{S,i}(\infty) \approx p_{S,i}(0) - p_{S,i}(\infty). \tag{37}
\]

The last approximation means that effectively the left hand sides of relationships \([34]\) are equal. Then, after some algebra, by using \([35]\), from those relationships one can obtain

\[
p_A = \frac{\alpha \mu + \beta \sigma}{\mu(\mu + \sigma)} A p_A. \tag{38}
\]

The last relationship is eigenvalue equation of a matrix which is the adjacency matrix multiplied by the scalar \( (\alpha \mu + \beta \sigma)/[\mu(\mu + \sigma)] \), which corresponds to eigenvalue equal to one. Thus, the vector of sums of probabilities of asymptomatic state \([35]\) represents eigenvector of the adjacency matrix that corresponds to the eigenvalue \( \Lambda \) such that

\[
1 = \Lambda \frac{\alpha \mu + \beta \sigma}{\mu(\mu + \sigma)}. \tag{39}
\]

To determine which is the eigenvalue \( \Lambda \), observe that we can apply similar inequality as \([9]\), which means that for each node \( i \) one has

\[
p_{S,i}(0) - p_{S,i}(\infty) < \ln p_{S,i}(0) - \ln p_{S,i}(\infty). \tag{40}
\]
This implies that one has the following vector inequality

\[ \mathbf{p}_A < \frac{\alpha \mu + \beta \sigma}{\mu (\mu + \sigma)} \mathbf{A} \mathbf{p}_A, \]  

which is obtained from (34) with simple algebra. When the epidemic parameters are such that

\[ \Lambda_{\text{max}} \frac{\alpha \mu + \beta \sigma}{\mu (\mu + \sigma)} < 1, \]  

where \( \Lambda_{\text{max}} \) is the largest eigenvalue of \( \mathbf{A} \), then (38) cannot be satisfied for no one eigenvalue. That is the condition when the endemic equilibrium does not exist. To determine when it will emerge, one should increase the value of the fraction in (41), by modifying the epidemic parameters. Then, the first eigenvalue that can satisfy inequality as (38) will be exactly the largest eigenvalue \( \Lambda_{\text{max}} \). Thus the condition for existence of endemic equilibrium is

\[ \mu (\mu + \sigma) < \Lambda_{\text{max}} (\alpha \mu + \beta \sigma). \]  

The last result is generalization of the case of random regular graph for which \( \Lambda_{\text{max}} = k \).

Because the leading eigenvalue of the adjacency matrix \( \mathbf{A} \) determines the equation for \( \mathbf{p}_A \), that vector is determined from the respective eigenvector, or the principal eigenvector of \( \mathbf{A} \). However, the eigenvalue equation (37) just determines the relative magnitudes of the components of \( \mathbf{p}_A \). If it is used in the system (34) it will be obtained that for each node the change in the probability of being susceptible is the same which counters the fact that it corresponds to the respective component of the principal eigenvector. We further note that from (34) the relative magnitudes of the changes of the probabilities of susceptible state \( \mathbf{p}_{S,i}(0) - \mathbf{p}_{S,i}(\infty) \), and the resulting probabilities of recovered state \( \mathbf{p}_{R,i}(\infty) \), as colinear to \( \mathbf{p}_A \), are also proportional to the principal eigenvector of the adjacency matrix. This is in accordance to the reasoning that the individuals with highest risk of infection are those with many contacts, and particularly those which have many high-degree neighbors. Thus the eigenvector centrality of the node [31], which is the respective component in the principal eigenvector, determines the risk of infection of that node. We note that, by applying this procedure one can also show that the same conclusions about the role of the leading eigenvalue and principal eigenvector in epidemic spreading on complex networks hold for the simpler SEIR and SIR models.

We now proceed with the study of the stability of the disease-free equilibrium and determine the epidemic threshold. The associated Jacobian matrix is obtained by taking the respective derivatives in the equations (20) to (24). Also, we remind that after making differentiation, at the epidemic inception in the Jacobian it should be taken \( p_{E,i} = 0, p_{A,i} = 0, p_{I,i} = 0 \) and \( p_{S,i} = 1 \). It can be verified that the Jacobian will have the following matrix form

\[
\mathbf{J} = \begin{bmatrix}
\mathbf{I} & 0 & -\alpha \mathbf{A} & -\beta \mathbf{A} & 0 \\
0 & (1 - \gamma) \mathbf{I} & \alpha \mathbf{A} & \beta \mathbf{A} & 0 \\
0 & \gamma \mathbf{I} & (1 - \sigma - \mu) \mathbf{I} & 0 & 0 \\
0 & 0 & \sigma \mathbf{I} & (1 - \mu) \mathbf{I} & 0 \\
0 & 0 & \mu \mathbf{I} & \mu \mathbf{I} & \mathbf{I}
\end{bmatrix},
\]

where \( \mathbf{I} \) is identity matrix of the same size \( N \) as the adjacency matrix \( \mathbf{A} \) – the number of nodes in the network. One can note the similarity in the structure between the last matrix and that in (29). The eigenvalues of the last Jacobian are obtained from the characteristic equation \( \mathcal{R}(\lambda) = \det(\mathbf{J} - \lambda \mathbf{I}_N) = 0 \), where we emphasize that the involved identity matrix has size \( 5N \times 5N \). One could use the approach given in [27] to determine the dependence of the epidemic threshold on the largest eigenvalue of the adjacency matrix. We have chosen alternative approach here, based on Schur’s determinant identity

\[
\det \begin{bmatrix} \mathbf{Q} & \mathbf{R} \\ \mathbf{S} & \mathbf{T} \end{bmatrix} = \det(\mathbf{T}) \cdot \det(\mathbf{Q} - \mathbf{R} \mathbf{T}^{-1} \mathbf{S}),
\]

which is more general. Clearly, when a matrix has many zero submatrices, its application provides simpler results. By repetitive use of it, which is elaborated in the Appendix D it can be shown that the nontrivial eigenvalues can be obtained from the polynomial corresponding to the following determinant

\[
\mathcal{T}(\lambda) = \det \left[ \frac{(1 - \gamma - \lambda)(1 - \sigma - \mu - \lambda)(1 - \mu - \lambda)}{\gamma [\alpha (1 - \mu - \lambda) - \beta \sigma]} \mathbf{I} - \mathbf{A} \right].
\]

We note that the same determinant can be obtained by the procedure given in the Appendix D which even delivers the eigenvectors of the Jacobian. Currently, we cannot state whether the approach in Appendix D is just an alternative, or it might have potential to provide results when the latter is not useful. To continue with the analysis, one can substitute the multiplier of the identity matrix in the last equation as

\[
\Lambda = \frac{(1 - \gamma - \lambda)(1 - \sigma - \mu - \lambda)(1 - \mu - \lambda)}{\gamma [\alpha (1 - \mu - \lambda) - \beta \sigma]},
\]

and will obtain the characteristic function for determination of the eigenvalues of the adjacency matrix \( \det(\mathbf{A} - \mathbf{J}) \). From the relationship (46) it can be seen that for each eigenvalue of the adjacency matrix \( \Lambda \) correspond three eigenvalues of the Jacobian \( \lambda \), which are obtained from the polynomial

\[
\mathcal{R}(\lambda) = (1 - \mu - \lambda) [(1 - \gamma - \lambda)(1 - \sigma - \mu - \lambda) - \alpha \gamma \Lambda] + \beta \gamma \sigma \Lambda.
\]
where \(\Lambda_{i,j}\) is the \(i,j\)-th element of the adjacency matrix. The remaining partial derivatives in the Jacobian matrix are the same as for the disease-free state and are conveniently captured in the respective submatrices of (43). The form of the partial derivatives (51) is such that for each \(p_{S,i}\) they have identical form and likewise for the \(p_{E,i}\). To obtain a more compact form for expressing such relationship, one can introduce a diagonal matrix \(\Sigma\) which contains the endemic equilibrium probabilities \(p_{S,i}(\infty)\) along the diagonal \(\Sigma_{i,i} = p_{S,i}(\infty)\). Then, one can obtain that the partial derivatives between the susceptibility and exposed vectors with respect to the asymptomatic and infectious vectors can be compactly written as

\[
\begin{align*}
\frac{\partial p_S}{\partial p_A} &= -\alpha \Sigma A, & \frac{\partial p_S}{\partial p_I} &= -\beta \Sigma A, \\
\frac{\partial p_E}{\partial p_A} &= \alpha \Sigma A, & \frac{\partial p_E}{\partial p_I} &= \beta \Sigma A.
\end{align*}
\] (52)

Now, the Jacobian of the endemic equilibrium differs from that for the disease-free one, only in that it contains the matrix product \(\Sigma A\) instead of \(A\). Respectively, the stability of the endemic equilibrium will depend on the leading eigenvalue \(\Lambda_{\text{max}}\) of the matrix product \(\Sigma A\).

Thus, the stability condition is similar to that for the disease-free state (50)

\[
\mu(\mu + \sigma) > \Lambda_{\text{max}}(\alpha \mu + \beta \sigma).
\] (53)

In the Appendix E it is shown that in case of small contagiousness \(\sigma \ll 1\) and \(\beta \ll 1\) and when epidemic affects small population during its course, the endemic equilibrium is linearly stable.

Let us finally examine the behavior of the disease spreading in the early phase of epidemic. The one-step evolution of the probabilities of different states in disease spreading on complex networks is given by equations (20) to (23). One can combine all probabilities in single column vector as \(p = [p_S^T, p_E^T, p_A^T, p_I^T]^T\) and the right hand sides of the probability evolution equations in a vector \(F\). Then, one-step evolution of the probabilities can be compactly written in vector notation as

\[
p(n+1) = F[p(n)].
\] (54)

Consider early stages of the epidemics, when the states \(p(n)\) are sufficiently close to the disease-free equilibrium \(p_{DF} = F[p_{DF}]\). Denote with \(\delta p(n) = p(n) - p_{DF}\) the deviation from the disease-free state. Then from (54) one has

\[
\delta p(n+1) = F[p(n)] - p_{DF} = F[p(n) - p_{DF}] = p_{DF} + J_{DF}\delta p(n).
\] (55)

The linear approximation of the nonlinear function \(F\) in vicinity of the disease-free state is

\[
F[p(n)] \approx F[p_{DF}] + J_{DF}[p(n) - p_{DF}] = p_{DF} + J_{DF}\delta p(n).
\] (56)
It means that consecutive perturbations satisfy simple relationship
\[ \delta p(n+1) = J_{DF} \delta p(n). \] (57)

Thus, at the early phase of an epidemic, the perturbation at given moment \( n \) is approximately given as
\[ \delta p(n) \approx J_{DF}^{n} \delta p(0). \] (58)

Denote with \( z_{i} \) the eigenvector of the Jacobian that corresponds to the eigenvalue \( \lambda_{i} \). Also, let the vectors \( z_{i} \) are properly chosen to make orthonormal basis. Then the perturbation \( \delta p(0) \) can be expressed in terms of the Jacobian basis vectors as
\[ \delta p(0) = \sum_{i=1}^{5N} p_{i} z_{i}. \] (59)

Then, after \( n \) time steps the perturbation will approximately evolve to
\[ \delta p(n) = J_{DF}^{n} \delta p(0) = \sum_{i=1}^{5N} p_{i} \lambda_{i}^{n} z_{i}. \] (60)

It is clear that as the number of steps \( n \) increases, the projection along the principal eigenvector will dominate the others. It means that one can use the approximation
\[ \delta p(n) \approx p_{max} \lambda_{max}^{n} z_{max}, \] (61)

where \( p_{max} \) is the projection of the initial perturbation along the principal eigenvector \( z_{max} \). In the Appendix it is explained that when the epidemic starts the leading eigenvalue of the Jacobian \( \lambda_{max} \) depends on that of the adjacency matrix \( \lambda_{max} \) and that the principal eigenvector of the Jacobian in that case is determined with the principal eigenvector of the adjacency matrix. Thus, the latter determines the evolution of the epidemics at the early stages. We emphasize that this is an approximation since as \( n \) grows, the Jacobian which is used, less accurately represents the nonlinear evolution, because the state of the system goes away from the disease-free one. Although an approximation, the last result provides an estimate of the risk of being infected of the nodes in a network, by the respective eigenvector centrality. This observation needs more theoretical work for establishing the conditions when the principal eigenvector is really useful in estimation of the risk of infection.

IV. NUMERICAL EXPERIMENTS AND DISCUSSION

The focus of numerical experiments in this work is put on validation of the theoretical results for the discrete-time SEAIR model. The theoretical analysis of the compartmental model was classical and did not bring any significant novelty, which is not known for the other compartmental models. Thus, the potential of the compartmental model should be tested on real data, which is left for future study.

We have made simulations of disease spreading on random regular graphs by numerical solution of the evolution equations [26]. The aim of these numerical experiments was to check the validity of the epidemic threshold relationships, as well as the equation for the fraction of the susceptible individuals at the end of the epidemic [27]. For computational reasons, for this and the other numerical experiments in this work, we have considered networks with 1000 nodes. All versions of the SEAIR epidemic spreading model considered here, have five parameters \( \alpha, \beta, \sigma, \mu \) and \( \gamma \). However, theoretical analysis in previous sections has shown that only the first four of them are relevant for determination of the epidemic threshold and the susceptible fraction at the end of the epidemic. The contagiousness parameters’ values, \( \alpha = 0.0025 \) and \( \beta = 0.002 < \alpha \) were chosen arbitrarily, by caring to be small to ensure that the approximations made in the theoretical analysis are justified and using the observation that for COVID-19 contagiousness is bigger before the onset of symptoms. We have taken \( \gamma = 0.5 \) which should correspond to two days mean period of incubation, while the value of \( \sigma = 0.2 \) was chosen arbitrarily. The critical parameter \( \mu_{0} \) was calculated from the following quadratic equation
\[ k \alpha \mu_{0} + \beta \sigma \mu_{0} + \mu_{0} (\mu_{0} + \sigma) = 1, \] (62)

which is obtained from the condition for emergence of endemic state for the random regular graphs [28]. The value of the parameter \( \mu \) was varied in vicinity \( \mu_{0} \). All simulations were repeated for ten different networks and for each network ten different initializations were made by putting random node in exposed state (patient zero), while leaving the remaining ones as susceptible. The pathogen was considered as extinct at the moment when the total fraction of exposed, asymptomatic and infectious individuals is smaller than \( 10^{-8} \) of the population. In the figure show the average number of the susceptible individuals at the end of the epidemic. The number of susceptible individuals for each particular simulation is simply sum of the probabilities of the susceptible state over all nodes. Averaging is performed for all networks from the same type and for all initial conditions. In the blue diamonds are given the results for the random regular graph with node degree \( k = 50 \), while with red circles is the curve for the random graph with constant degree distribution in the interval \([30, 70]\). We emphasize that in this figure the threshold value \( \mu_{0} \) is obtained for the random regular graph and the same value is used for the other one. Both considered graphs have same average degree, and thus show similar results, particularly when one is far enough from the threshold \( \mu = \mu_{0} \). In vicinity of \( \mu_{0} \), as was theoretically shown for general complex networks, the epidemic threshold depends on...
Disease spreading on random regular graph and random graph with constant degree distribution. The curves represent the dependence of the fraction of susceptible individuals at the end of the epidemic on the parameter $\mu$. The meaning of the symbols is the following: orange stars – theoretical values from eq. 27 for infinite-size random regular graph with node degree 50; blue diamonds – random regular graph with the same degree and 1000 nodes; red circles – random graph with uniform degree distribution in [30,70] and 1000 nodes.

The leading eigenvalue 42, which for the graph with distributed node degree is greater than the average degree $\Lambda_{\text{max}} > \langle k \rangle$ as the Perron-Frobenius theorem claims 31. Thus, for same $\mu$ one expects more infected individuals for the graphs with distributed degree. The results from the simulations are further compared with theoretical values obtained from 27 for random regular graph with $k = 50$ nodes, which holds for network with infinite number of nodes. It can be seen a noticeable difference between the theoretical curve and the simulations. One reason for such discrepancy could be attributed in the fact that the theoretical results hold for infinitely large networks. The other factor is the initialization of the epidemics, which even in case of stable disease-free state, $\mu > \mu_0$, produces at least a small fraction of infected individuals – at least the neighbors of the patient zero.

Next, we have considered disease spreading on Erdős-Rényi (ER) 32 and Barabási-Albert (BA) 12 models of complex networks. Within the ER model, we have considered probability of existence of link between each pair of nodes $\rho_{\text{ER}} \in \{0.01, 0.03, 0.05\}$ and generated ten different networks for each case. For the BA complex networks we have taken four different values of the seed $m \in \{5, 10, 15, 20\}$. As for the random regular graphs, for each ER and BA network ten different initial conditions were considered. The parameter values for $\alpha$, $\beta$, $\gamma$ and $\sigma$ were taken identical as for the random regular graphs. In the figure 2 are shown the average number of susceptible individuals and the correlation coefficient between the recovered probability vector and the principal eigenvector of the adjacency matrix at the end of epidemic. Here, the threshold value $\mu_0$ was calculated for each network separately from the equation

$$\Lambda_{\text{max}} \frac{\alpha \mu_0 + \beta \sigma}{\mu_0(\mu_0 + \sigma)} = 1,$$

where $\Lambda_{\text{max}}$ is the leading eigenvalue of the network which had to be calculated previously.

In the figure 2 can be seen that as the parameter $\mu$ increases towards the critical value $\mu_0$, the number of susceptible at the endemic equilibrium approaches the total number of individuals, as it is expected. We remark that it is not equal to the total population even when the conditions of epidemic are not satisfied, because there is certain probability that the patient zero will infect some neighboring nodes. However, this is finite size effect, and in infinitely large network the fraction of infected individuals will be infinitesimally small. We also remark that the results about the ER network look that it is more prone to epidemic. This deception appears because the horizontal axis is in the units of the threshold value $\mu_0$ and not the absolute terms. As it is known, BA networks are more prone to epidemics and for infinite size networks the epidemic threshold is vanishing 6.

The rather high value of the correlation coefficient $\rho$, when disease is spreading suggests that indeed the principal eigenvector predicts the pattern of infection. When the epidemic is not possible, $\mu > \mu_0$, the correlation does not drop sharply, due to the finite size effects. Near the epidemic threshold there is nonzero probability of infecting the neighborhood by the initially exposed node, and particularly those with higher eigenvector centrality.

We have finally studied the behaviour of the epidemics at the onset in order to verify which nodes bear the highest risk of contracting the disease. As common wisdom suggests, highly connected nodes, and particularly those with well connected neighbors are most risky ones – just as eigenvector centrality ranks the nodes. For that reason we have calculated the evolution of correlation coefficient between the principal eigenvector of the adjacency matrix and the probability vector of recovered state as the epidemic unfolds. In the figure 3 is shown the correlation coefficient as function of time. For the BA network shown at right the parameters have the same values, while for the ER network (at left panel) $\alpha = 0.05$ and $\beta = 0.04$, while $\gamma$ and $\sigma$ are the same as in the other simulations.

One can note that generally in the early stages of the disease outbreak very high correlation is achieved, which confirms that the eigenvector centrality predicts rather well the riskiness of contraction of the pathogen. As the epidemics fades out the correlation might drop because in certain parameter combinations majority of population has high chance of becoming infected and this infection pattern can differ significantly from the predictions by the principal eigenvector of the adjacency matrix. However, the lowest curve for the ER network model shows that this is not always happening. In such situation, when epidemic is barely possible, only small fraction of population can be affected, particularly those which are close to the patient zero. This observation needs further
FIG. 2: Discrete-time epidemic model on complex networks at the end of epidemic. In all panels ten different complex networks with 1000 nodes are considered. The seed of generating the BA networks, \( m \), and the link probability for ER networks \( p_{ER} \) is given in the inset. In the top panels are shown the average number of susceptible individuals \( \langle S \rangle \), while at bottom are average correlation coefficients \( \langle \rho \rangle \) between the number of recovered individuals and the principal eigenvector of the respective adjacency matrix. The horizontal axis is given in units of the critical value of the parameter \( \mu_0 \) at the epidemic threshold which is calculated for each network separately.

investigation of the pattern of risk in case of such small outbreaks.

V. CONCLUSIONS

We have studied SEAIR epidemic spreading model inspired by the contagiousness features of COVID-19. Theoretical analysis were made for the compartmental version as well as for discrete-time epidemic spreading on random regular and complex networks. For the compartmental model the epidemic threshold was found and it was shown that it also determines the emergence of endemic equilibrium as well. When the contagiousness is weak, we have shown that for random regular graph and complex network the epidemic threshold obtained from stability analysis of the disease-free state depends in a similar way on the model parameters. As is known for many other disease spreading models, the epidemic threshold was obtained to depend on the leading eigenvalue of the adjacency matrix. We have also demonstrated that the endemic equilibrium when exists is linearly stable in the three considered models. Theoretical analysis in this work has shown that the risk to be infected certain node is dependent on its eigenvector centrality. In early stages of epidemics, the eigenvector centrality points which nodes are most likely to be first to contract the disease, while in case of mild epidemic on complex network, it shows which nodes have more risk to contract the disease during the whole course of the epidemic.

The analysis of the linear stability was based on two approaches. By appropriately organizing the probabilities of various states as dynamical variables it was obtained Jacobian matrix of the equilibria which has form that allows analytical treatment. The first approach was based on applying Schur’s determinant identity which lead to result that the nontrivial eigenvalues of the Jacobian are related to those of the adjacency matrix. In the second approach we have furthermore shown that eigenvectors corresponding to the nontrivial eigenvalues of the Jacobian are combinations of scaled eigenvectors...
FIG. 3: Evolution of correlation coefficient between the principal eigenvector of the adjacency matrix and the vector of probability of recovered state in ER (left panel) and BA (right panel) complex networks. The considered networks have 1000 nodes. Each curve is obtained by averaging ten networks with the same parameters and ten randomly chosen initially infected nodes for each network.

of the adjacency matrix. We believe that these two techniques can be applied in a range of studies where multidimensional dynamical systems interact through complex topology of contacts.

Although the motivation of studying the SEAIR epidemic spreading model was the COVID-19 pandemic, we did not make any testing about its relevance on real data. Naturally, it is the first study which should follow this one. One of the key issues would be inference of the fraction of the population which has contracted the disease, but has not shown symptoms at all. This could help in estimating the likelihood of reappearance of the epidemic and its possible size, once it weakens. Since, in general, contagiousness parameters change during epidemic, testing the validity of the relationships for the fraction of susceptible individuals at the end of epidemic might not be easy task. However, at the early phase of an epidemic these parameters could be considered as constant. Then, by using real data, it could be verified how well the eigenvector centrality anticipates which individuals bear highest risk of infection. If it proves to be useful predictor, then a follow up is investigation of its relevance to planning of vaccination and other protective measures.

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Since \( a_2 > 0 \), the condition \( a_0 > 0 \) is equivalent to

\[
\mu(\mu + \sigma) > \alpha \mu + \beta \sigma. \tag{A5}
\]

We should also verify that also \( a_2 a_1 > a_0 \) is satisfied which after multiplication of the respective values in \([A1]\) will result in

\[
\gamma \mu \sigma + \gamma^2 \sigma + \gamma \mu^2 + 2 \gamma^2 \mu - \sigma \gamma^2 \\
+ \mu \sigma^2 + \gamma \sigma^2 + \mu^2 \sigma + 2 \gamma \mu \sigma - \sigma \gamma \sigma \\
+ 2 \mu^2 \sigma + 2 \gamma \mu \sigma + 2 \mu^3 + 4 \gamma \mu^2 - 2 \gamma \alpha \mu \\
> \gamma \mu^2 + \gamma \mu \sigma - \gamma \alpha \mu - \gamma \beta \sigma. \tag{A6}
\]

By algebraic manipulation and rearrangement of the last inequality becomes

\[
4 \gamma \mu \sigma + \gamma^2 \sigma + 4 \gamma \mu^2 + 2 \gamma^2 \mu \\
- \alpha \gamma^2 + \mu \sigma^2 + \gamma \sigma^2 + 3 \mu^2 \sigma \\
- \sigma \gamma \sigma + 2 \mu^3 - \gamma \alpha \mu + \gamma \beta \sigma > 0. \tag{A7}
\]

Now, from the condition \([A5]\) one has the inequality

\[
\mu + \sigma > \alpha, \tag{A8}
\]

which implies the following relationships for the negative sign terms in \([A7]\)

\[
\gamma^2 (\mu + \sigma) > \gamma^2 \alpha, \\
\gamma \sigma (\mu + \sigma) > \gamma \sigma \alpha, \\
\gamma \mu (\mu + \sigma) > \gamma \mu \alpha. \tag{A9}
\]

By using the last three inequalities in \([A7]\), one will obtain that only positive terms will remain at the left hand side, which means that it is satisfied. Thus by the Routh-Hurwitz criterion the nontrivial eigenvalues of the Jacobian have negative real parts if and only if \([A5]\) holds.

### Appendix B: Endemic equilibrium for random regular graph

For determination of the population remaining unaffected by the epidemic in disease spreading on random regular graph, we follow the same approach as in the compartmental model. To proceed in that spirit, first sum up the first four equations in the system \([26]\) and obtain

\[
p_S(n+1) + p_E(n+1) + p_A(n+1) + p_I(n+1) \\
= p_S(n) + p_E(n) + (1 - \mu) [p_A(n) + p_I(n)]. \tag{B1}
\]

We can sum the last relationship over all moments \( n \), from the onset to finish of the epidemic, and assume negligibly small initial probabilities of infected individuals. Then, due to cancellation of the respective terms it will be obtained

\[
p_S(0) - p_S(\infty) = \mu \sum_{n=0}^{\infty} [p_A(n) + p_I(n)], \tag{B2}
\]
which corresponds to the equation (3) of the compartmental model.

Next, the fourth equation in (20) is rewritten as

$$p_I(n + 1) - p_I(n) = \alpha p_A(n) - \mu p_I(n). \tag{B3}$$

Summation of the infinite ladder of equations (B3) and using $p_I(0) = 0 = p_I(\infty)$ leads to the same relationship between the probabilities of asymptomatic and infectious states as in the case of the compartmental model (2),

$$\sigma \sum_{n=0}^{\infty} p_A(n) = \mu \sum_{n=0}^{\infty} p_I(n). \tag{B4}$$

The first equation in (20) can be rearranged as

$$\left[ \frac{p_S(n+1)}{p_S(n)} \right]^{1/k} = 1 - \beta p_I(n) - \alpha p_A(n). \tag{B5}$$

If we take logarithm of the last equation, and use approximation $\alpha p_A(n) \ll 1$ and $\beta p_I(n) \ll 1$, that holds for weak spreading $\alpha \ll 1$ and $\beta \ll 1$, it will be obtained

$$\frac{1}{k} \ln p_S(n+1) - \ln p_S(n) = -\beta p_I(n) - \alpha p_A(n). \tag{B6}$$

Summing the last relationship for all moments, after cancellations results in

$$\ln p_S(0) - \ln p_S(\infty) = k a \sum_{n=0}^{\infty} p_A(n) + k \beta \sum_{n=0}^{\infty} p_I(n). \tag{B7}$$

Using the relationships (B2), (B4) and (B7), one can obtain an estimate of the number of unaffected individuals in epidemic spreading on random regular graphs from

$$p_S(0) - p_S(\infty) = \frac{\mu(\mu + \sigma)}{\beta(\sigma + \alpha \mu)} \ln \frac{p_S(0)}{p_S(\infty)}. \tag{B8}$$

Appendix C: Endemic equilibrium for complex network

For small contagious parameters $\alpha \ll 1$ and $\beta \ll 1$, one can approximate the probability that susceptible individual will not receive the virus as

$$\prod_{j \in N_i} [1 - \alpha p_{A,j}(n) - \beta p_{I,j}(n)] 
\approx 1 - \alpha \sum_{j \in N_i} p_{A,j}(n) - \beta \sum_{j \in N_i} p_{I,j}(n). \tag{C1}$$

Then the evolution of all probabilities can be compactly written as

$$p_S(n+1) = p_S(n) \left[ 1 - \alpha A p_A(n) - \beta A p_I(n) \right],$$
$$p_E(n+1) = p_S(n) \left[ \alpha A p_A(n) + \beta A p_I(n) \right] + (1 - \gamma) p_E(n),$$
$$p_A(n+1) = \gamma p_E(n) + (1 - \sigma - \mu) p_A(n),$$
$$p_I(n+1) = \sigma p_A(n) + (1 - \mu) p_I(n),$$
$$p_R(n+1) = \mu p_I(n) + p_I(n) + p_I(n). \tag{C2}$$

We will follow the same technique as for the previous two scenarios. Summing up the first four equations in the last system will result in

$$p_S(n+1) + p_E(n+1) + p_A(n+1) + p_I(n+1) = p_S(n) + p_E(n) + (1 - \mu) p_A(n) + p_I(n). \tag{C3}$$

Now, let's sum over all moments and use the fact that the probabilities of infected states at the beginning and ending of epidemic are vanishing. Then from the last relationship will be obtained

$$p_S(0) - p_S(\infty) = \mu \sum_{n=0}^{\infty} [p_A(n) + p_I(n)]. \tag{C4}$$

Rearrangement of the fourth equation in (C2) and summing over all moments will lead to result that generalizes (B4)

$$\sum_{n=0}^{\infty} p_A(n) = \mu \sum_{n=0}^{\infty} p_I(n). \tag{C5}$$

One can write the evolution equation of probability of susceptible state for each node $i$ as

$$\frac{p_{S,i}(n+1)}{p_{S,i}(n)} = \prod_{j \in N_i} [1 - \alpha p_{A,j}(n) - \beta p_{A,j}(n)]. \tag{C6}$$

Further, take logarithm on both sides of the last equation and keep only leading terms in $\alpha$ and $\beta$ in the expansion of the logarithm of the multipliers to obtain

$$\ln \frac{p_{S,i}(n+1)}{p_{S,i}(n)} = -\alpha \sum_{j \in N_i} p_{A,j}(n) - \beta \sum_{j \in N_i} p_{I,j}(n). \tag{C7}$$

Summing (C7) over all moments will result in

$$\ln p_{S,i}(0) - \ln p_{S,i}(\infty) = \alpha \sum_{j \in N_i} \sum_{n=0}^{\infty} p_{A,j}(n) + \beta \sum_{j \in N_i} \sum_{n=0}^{\infty} p_{I,j}(n). \tag{C8}$$

Denote with $\ln p_S(n)$ the vector which components are the logarithms of probabilities of susceptible states $p_{S,i}(n)$. Then, the relationship (C8) for all nodes can be compactly written as

$$\ln p_S(0) - \ln p_S(\infty) = \alpha A \sum_{n=0}^{\infty} p_A(n) + \beta A \sum_{n=0}^{\infty} p_I(n). \tag{C9}$$

From one side, using (C5) in (C4) will result in

$$p_S(0) - p_S(\infty) = \mu (1 + \frac{\sigma}{\mu}) \sum_{n=0}^{\infty} p_A(n). \tag{C10}$$

From another side, applying (C5) in (C9) will lead to

$$\ln p_S(0) - \ln p_S(\infty) = \left( \alpha + \beta \frac{\sigma}{\mu} \right) A \sum_{n=0}^{\infty} p_A(n). \tag{C11}$$

The last two relationships are system of equations for determination of vectors of the probabilities of the susceptible and the infinite sum of the vectors of asymptomatic states.
Appendix D: Characteristic polynomial for the eigenvalues of the Jacobian of the discrete-time model

To obtain more compact expression for determination of the eigenvalues of the Jacobian, we will extensively use the Schur’s determinant identity

\[
\det \begin{bmatrix} Q & R \\ S & T \end{bmatrix} = \det(T) \cdot \det(Q - RT^{-1}S). \quad (D1)
\]

One should note that the identity does not need the matrices to be square and if at least one of the matrices \(R\) or \(S\) is zero, then one has simpler relationship

\[
\det \begin{bmatrix} Q & R \\ S & T \end{bmatrix} = \det(Q) \cdot \det(T). \quad (D2)
\]

First we can assign the role of the bottom-right submatrix \(T\) in \((D1)\) to the bottom-right identity matrix in \((D3)\). Then one can note that to the respective submatrix \(R\) corresponds zero matrix and use \((D2)\) instead to obtain

\[
\mathcal{R}(\lambda) = \det[(1 - \lambda)I] \cdot \det \begin{bmatrix} (1 - \lambda)I & 0 & -\alpha A & -\beta A \\ 0 & (1 - \gamma - \lambda)I & \alpha A & \beta A \\ \gamma I & (1 - \sigma - \mu - \lambda)I & 0 \\ 0 & \sigma I & (1 - \mu - \lambda)I \end{bmatrix}. \quad (D3)
\]

By repeating the same procedure one more time with taking top-right submatrix \((1 - \lambda)I\) as the submatrix \(Q\) in the Schur’s determinant identity, and observing that now the submatrix \(S\) is zero, one can obtain that

\[
\mathcal{R}(\lambda) = \{\det[(1 - \lambda)I]\}^2 \cdot \det \begin{bmatrix} (1 - \gamma - \lambda)I & \alpha A & \beta A \\ \gamma I & (1 - \sigma - \mu - \lambda)I & 0 \\ 0 & \sigma I & (1 - \mu - \lambda)I \end{bmatrix}. \quad (D4)
\]

To simplify notation one could first stop repetitive writing of the part which contains the trivial eigenvalue \(\lambda = 1\) which has multiplicity \(2N\), and focus on the remaining. Take the submatrix \(T = (1 - \mu - \lambda)I\) which determinant contains trivial eigenvalues \(\lambda = 1 - \mu\) and respectively the remaining submatrices \(Q, R\) and \(S\). We note that \(1 - \mu\) are not eigenvalues of the Jacobian, since in expanding the determinants as polynomial, the terms corresponding to \(1 - \mu - \lambda\) that appear in \(T\) will cancel with the same terms which will appear in the denominator in the remaining determinant as will be seen below. From the last determinant let us first consider the submatrix that corresponds to the product \(RT^{-1}S\) in the Schur’s identity \((D1)\). By using the properties of the inverse matrix one can obtain first

\[
[(1 - \mu - \lambda)I]^{-1} \cdot [0 \ \sigma I] = [0 \ \frac{\sigma}{1 - \mu - \lambda}I]. \quad (D5)
\]

Then it follows that

\[
[\beta A \ 0] \cdot [0 \ \frac{\sigma}{1 - \mu - \lambda}I] = [0 \ \frac{\beta \sigma}{1 - \mu - \lambda}I]. \quad (D6)
\]

Now, the part of the characteristic polynomial which contains the nontrivial eigenvalues is

\[
\mathcal{S}(\lambda) = \det \left\{ \begin{bmatrix} (1 - \gamma - \lambda)I & \alpha A \\ \gamma I & (1 - \sigma - \mu - \lambda)I \end{bmatrix} - \begin{bmatrix} 0 & \frac{\beta \sigma}{1 - \mu - \lambda}A \\ 0 & 0 \end{bmatrix} \right\} = \det \begin{bmatrix} (1 - \gamma - \lambda)I & (\alpha - \frac{\beta \sigma}{1 - \mu - \lambda})A \\ \gamma I & (1 - \sigma - \mu - \lambda)I \end{bmatrix}. \quad (D7)
\]

We can apply the Schur’s identity again. First observe the matrix product that corresponds to the \(RT^{-1}S\) term in \((D1)\)

\[
\left( \alpha - \frac{\beta \sigma}{1 - \mu - \lambda} \right)A \cdot [(1 - \mu - \sigma - \lambda)I]^{-1} \cdot \gamma I
\]

\[
= \frac{\gamma}{1 - \sigma - \mu - \lambda} \left( \alpha - \frac{\beta \sigma}{1 - \mu - \lambda} \right)A. \quad (D8)
\]

After simplification of the scalar at the right-hand side of the last relationship and subtract the respective matrices in the form \(Q - RT^{-1}S\) from \((D1)\) one will obtain the following characteristic polynomial of the eigenvalues
Again, the first determinant has trivial eigenvalues \( \lambda = 1 - \sigma - \mu \) with multiplicity \( N \) as well and the nontrivial ones are contained in the second determinant. By observing the second determinant in (D9), one can note that in the denominator multiplying the adjacency matrix appear terms \( 1 - \mu - \lambda \) and \( 1 - \sigma - \mu - \lambda \). Expansion of the determinants as polynomials will result in cancellation of those terms in the denominators with the respective ones in the determinants \( \det[(1 - \mu - \lambda)I] \) and \( \det[(1 - \sigma - \mu - \lambda)I] \). Finally, the characteristic polynomial resulting from the last nontrivial determinant will not change if one multiplies it with a constant. So, a more convenient form of the last determinant, and the respective characteristic polynomial is

\[
T(\lambda) = \det \left[ \frac{(1 - \gamma - \lambda)(1 - \sigma - \mu - \lambda)(1 - \mu - \lambda)}{\gamma(a(1 - \mu - \lambda) - \beta \sigma)} I - \Lambda \right].
\]  

(D10)

### Appendix E: Stability of the endemic equilibrium in disease spreading on complex networks

Since the Jacobian of the endemic and disease-free equilibrium differ only in the presence of the matrix \( \Sigma \), the characteristic equation will have the same form for both cases. However, it was previously obtained that the leading eigenvalue of the Jacobian of the disease-free equilibrium \( \lambda_{\text{max}} \) depends on the leading one of the adjacency matrix \( \Lambda_{\text{max}} \). Accordingly, for the endemic state the dependence will be on the leading eigenvalue \( L_{\text{max}} \) of the matrix product \( \Sigma \Lambda \). We will verify that this eigenvalue is related with that of the adjacency matrix as \( L_{\text{max}} < p_{S,\text{max}} \Lambda_{\text{max}} \), where \( p_{S,\text{max}} = \max p_{S,i}(\infty) \), is the maximum of the probabilities of susceptible states at the end of the epidemic. To prove that, denote with \( x \) the unit eigenvector of \( \Sigma \Lambda \), corresponding to \( L_{\text{max}} \), or \( \Sigma \Lambda x = L_{\text{max}} x \). Let \( \Lambda_i \) and \( u_i \) are the eigenvalues and the respective orthogonal basis vectors corresponding to the adjacency matrix. The vector \( x \) in the basis \( u_i \) is given as

\[
x = \sum_{i=1}^{N} a_i u_i, \tag{E1}
\]

where \( \sum a_i^2 = 1 \) because \( x \) is unit vector. Then, multiplying the matrix \( \Lambda \) with \( x \) will result in some vector

\[
y = \Lambda x = \sum_{i=1}^{N} a_i \Lambda_i u_i, \tag{E2}
\]

Due to the orthonormality of the basis \( u_i^T u_j = \delta_{i,j} \), the squared magnitude of \( y \) reads

\[
y^T y = \sum_{i=1}^{N} a_i^2 \Lambda_i^2, \tag{E3}
\]

which can be bounded as

\[
y^T y \leq \Lambda_{\text{max}}^2 \sum_{i=1}^{N} a_i^2 = \Lambda_{\text{max}}^2. \tag{E4}
\]

This means that the vector \( y \) has length not bigger than \( \Lambda_{\text{max}} \). In connected network each node will be infected with nonzero probability \( p_{S,i}(\infty) < 1 \). Thus the matrix \( \Sigma \) is symmetric positive semi-definite, and all its eigenvalues are strictly less than one. Let us now express the vector \( y \) in the orthonormal basis \( v_i \) of the matrix \( \Sigma \)

\[
y = \sum_{i=1}^{N} b_i v_i. \tag{E5}
\]

Then the vector \( \Sigma y \) can be expressed as

\[
\Sigma y = \sum_{i=1}^{N} b_i p_{S,i}(\infty) v_i, \tag{E6}
\]

since \( \Sigma \) is diagonal matrix with eigenvalues \( p_{S,i}(\infty) \). The squared magnitude of \( \Sigma y \) is bounded as

\[
y^T \Sigma^T \Sigma y = \sum_{i=1}^{N} b_i^2 p_{S,i}(\infty)^2 < p_{S,\text{max}}^2 \sum_{i=1}^{N} b_i^2 = p_{S,\text{max}}^2 |y|^2. \tag{E7}
\]

Now, combining (E2), (E4) and (E7) will result in

\[
L_{\text{max}}^2 x^T x = x^T \Lambda^T \Sigma \Lambda x < p_{S,\text{max}}^2 \Lambda_{\text{max}}^2. \tag{E8}
\]

Thus, we have just bounded the leading eigenvalue of the matrix \( \Sigma \Lambda \) as

\[
L_{\text{max}} < p_{S,\text{max}} \Lambda_{\text{max}}. \tag{E9}
\]

Recall that in the stability analysis of the endemic equilibrium one has the matrix product \( \Sigma \Lambda \) instead of \( \Lambda \) which is used in the disease-free state. So, the stability of the endemic equilibrium depends on \( L_{\text{max}} \) as the other case depends on \( \Lambda_{\text{max}} \). Correspondingly, the endemic equilibrium will be linearly stable, once the following inequality holds [refer to the respective condition (50)]

\[
\mu(\mu + \sigma) > L_{\text{max}}(\alpha \mu + \beta \sigma). \tag{E10}
\]

Now, consider the system of transcendental equations (54) and use the fact that \( p_A \) is principal eigenvector
of the adjacency matrix $A$, or $AP_A = \Lambda_{\text{max}}P_A$. By algebraic manipulations, from the system (E4) it can be shown that for each component of the susceptible probability vector holds relationship similar to (E3)

$$p_{S,i}(0) - p_{S,i}(\infty) = \frac{\mu(\mu + \sigma)}{\Lambda_{\text{max}}(\beta\sigma + \alpha\mu)} [\ln p_{S,i}(0) - \ln p_{S,i}(\infty)], \quad (E11)$$

from which one has

$$\frac{\mu(\mu + \sigma)}{\beta\sigma + \alpha\mu} = \Lambda_{\text{max}} \frac{p_{S,i}(0) - p_{S,i}(\infty)}{\ln p_{S,i}(0) - \ln p_{S,i}(\infty)}. \quad (E12)$$

Combining the endemic equilibrium stability condition (E10) with the last relationship (E12) will result in

$$\Lambda_{\text{max}} \frac{p_{S,i}(0) - p_{S,i}(\infty)}{\ln p_{S,i}(0) - \ln p_{S,i}(\infty)} > L_{\text{max}}. \quad (E13)$$

Rearranging the terms in the last inequality will result in more convenient form

$$\Lambda_{\text{max}}p_{S,i}(\infty) \left( \frac{p_{S,i}(0)}{p_{S,i}(\infty)} - 1 \right) > L_{\text{max}}. \quad (E14)$$

From the fourth row in (E1), which corresponds to the infectious state, one can obtain that

$$\sigma w_A + (1 - \mu)w_I = \lambda w_I, \quad (F2)$$

which can be rearranged into

$$\sigma w_A = (\mu + \lambda - 1)w_I. \quad (F3)$$

The last equation relates the magnitudes of the vectors $w_A$ and $w_I$ and shows that they are collinear. In similar manner, from the last row in (E1), one can show that the vector $w_R$ is collinear with the previous ones and moreover

$$\frac{\mu(\mu + \lambda + \sigma + 1)}{\mu + \lambda + 1} w_A = (\lambda - 1)w_R. \quad (F4)$$

Likewise, from the third row in (E1), it follows that the exposed probability vector $w_E$ is also collinear to the previous ones, or more precisely

$$\gamma w_E = (\mu + \lambda + \sigma - 1)w_A. \quad (F5)$$

The last inequality is satisfied since one can use (E10) and the fraction at the left-hand side is always greater than one. Thus, when an epidemic occurs such that small fraction of the population is affected, the respective endemic equilibrium is linearly stable.

### Appendix F: Eigenvalues and eigenvectors of the Jacobian at the disease-free state for epidemic spreading on complex networks

Denote the eigenvectors of the Jacobian matrix in the disease-free equilibrium with $w = [w_S, w_E, w_A, w_I, w_R]^T$ where $w_S$, $w_E$, $w_A$, $w_I$ and $w_R$ are the parts which correspond to the states $S$, $E$, $A$, $I$ and $R$ respectively. Then, the eigenvalue equation for the Jacobian $Jw = \lambda w$ in more detailed form is

$$\begin{bmatrix}
\lambda - A & 0 & -\beta A & 0 \\
0 & (1 - \gamma)I & \alpha A & 0 \\
0 & \gamma I & (1 - \sigma - \mu)I & 0 \\
0 & 0 & \sigma I & (1 - \mu)I \\
0 & 0 & \mu I & \mu I - 1
\end{bmatrix} \begin{bmatrix}
w_S \\
w_E \\
w_A \\
w_I \\
w_R
\end{bmatrix} = \lambda \begin{bmatrix}
w_S \\
w_E \\
w_A \\
w_I \\
w_R
\end{bmatrix} \quad (F1)$$

Now consider the second row in (F1), from which one has

$$\alpha Aw_A + \beta Aw_I = (\lambda + \gamma - 1)w_E, \quad (F6)$$

which after using (F3) and (F4) will result in

$$\left(\alpha + \frac{\beta\sigma}{\mu + \lambda - 1}\right)Aw_A = (\lambda + \gamma - 1)\frac{(\mu + \lambda + \sigma - 1)}{\gamma}w_A. \quad (F7)$$

The last relationship could be rearranged further as

$$Aw_A = \frac{(\lambda + \gamma - 1)(\mu + \lambda + \sigma - 1)(\mu + \lambda - 1)}{\gamma[\alpha(\mu + \lambda - 1) + \beta\sigma]}w_A. \quad (F8)$$

We have obtained eigenvalue equation for the adjacency matrix. Thus, every vector $w_A$ must be eigenvector of the adjacency matrix $A$. Since the eigenvalues of the adjacency matrix $\Lambda$ are independent on any dynamical process evolving on the network, it means that the eigenvalues of the Jacobian $\lambda$ must satisfy the relationship

$$\lambda = \frac{(\lambda + \gamma - 1)(\mu + \lambda + \sigma - 1)(\mu + \lambda - 1)}{\gamma[\alpha(\mu + \lambda - 1) + \beta\sigma]} \quad (F9)$$
The last result relates the eigenvalues of the Jacobian with those of the adjacency matrix. By expanding the terms, one can see that it is cubic polynomial in \( \lambda \), and thus for each eigenvalue \( \Lambda \) one has three possibly different eigenvalues \( \lambda \). Thus, \( N \) eigenvalues of the adjacency matrix would generate \( 3N \) eigenvalues of the Jacobian.

We remind that as is given in the Appendix D there is one trivial eigenvalue \( \lambda = 1 \) with algebraic multiplicity \( 2N \). The eigenvectors corresponding to this eigenvalue are those that span the subspace consisting of susceptible or recovered states only and zeros at the remaining states. It can be easily verified from \((\text{F1})\), that each vector of the form

\[
\mathbf{w}_{S,R} = [w_S^*, 0, 0, w_R^*]^T
\]

is eigenvector of the Jacobian.

Finally, from the first row in \((\text{F1})\) it follows that

\[
\left( \alpha + \frac{\beta \sigma}{\mu + \lambda - 1} \right) \mathbf{A} \mathbf{w}_A = (1 - \lambda) \mathbf{w}_S,
\]

in which one can use \((\text{F8})\) to obtain

\[
\mathbf{w}_S = \frac{(\mu + \lambda + \sigma - 1)(\lambda + \gamma - 1)}{\gamma(1 - \lambda)} \mathbf{w}_A. \tag{F12}
\]

Thus, \( \mathbf{w}_S \) vector is collinear with the rest as well. This implies that besides the vectors \((\text{F10})\), the remaining eigenvectors of the Jacobian \( \mathbf{w} \) consist of scaled copies of the eigenvectors of the adjacency matrix. More precisely, by using the relationships \((\text{F3}), (\text{F4}), (\text{F5}) \) and \((\text{F12})\), the eigenvector \( \mathbf{w} \) is

\[
\mathbf{w} = \begin{pmatrix}
\mathbf{w}_S \\
\mathbf{w}_E \\
\mathbf{w}_A \\
\mathbf{w}_I \\
\mathbf{w}_R
\end{pmatrix} = \begin{pmatrix}
\frac{(\mu + \lambda + \sigma - 1)(\lambda + \gamma - 1)}{\gamma(1 - \lambda)} \\
\frac{\mu + \lambda + \sigma - 1}{\gamma} \\
\frac{\mu + \lambda - 1}{\gamma(\mu + \lambda - 1)} \\
\frac{\mu + \lambda + \sigma - 1}{\gamma(\mu + \lambda - 1)} \\
\frac{\mu + \lambda - 1}{\gamma(\mu + \lambda - 1)}
\end{pmatrix} \mathbf{w}_A. \tag{F13}
\]

Since \( \mathbf{w}_A \) is eigenvector of the adjacency matrix, there are \( 3N \) linearly independent eigenvectors of the form given in \((\text{F13})\). Together with \( 2N \) linearly independent vectors of the form \((\text{F10})\), they constitute a set of \( 5N \) linearly independent vectors. When properly normalized they can be made to form an orthonormal basis.