Annales Univ. Sci. Budapest., Sect. Comp. 52 (2021) 293–312

STOCHASTIC PROCESSES ON RANDOM GRAPHS WITH MULTIPLE TYPE EDGES

Bence Rozner (Budapest, Hungary)

Communicated by Imre Kátai

(Received May 31, 2021; accepted July 3, 2021)

Abstract. In this paper we examine the spread of an infectious disease on several random graph models with multiple type edges. The introduction of the types of the edges allows us to use more adequate models, because the probabilities of the propagations may depend on the variety of the connections in the graph. At first, we generalize the SIR-process for graphs with multi-type edges. Then, we further generalize the process by introducing latency (i.e. infected individuals do not show symptoms for a random period of time) and quarantine (i.e. infected individuals who show symptoms are temporarily separated from the population). Finally, the empirical results of some stochastic simulations related to the different processes and underlying structures are presented.

1. Introduction

Stochastic processes on (random) graphs have been widely used to model infectious diseases on large networks (see e.g. [8, 12, 16]). A social network can

Key words and phrases: Epidemic spread, random graph, stochastic simulation.
2010 Mathematics Subject Classification: 92D30.
The Project is supported by the "Integrált kutatói utánpótlás-képzési program az informatika és számítástudomány diszciplináris területein (Integrated program for training new generation of researchers in the disciplinary fields of computer science) ", No. EFOP-3.6.3-VEKOP-16-2017-00002. The project has been supported by the European Union and co-funded by the European Social Fund.

The work was made under the supervision of Ágnes Backhausz. I would like to express my gratitude for her aid, comments and suggestions which contributed to the quality of the article.

be modelled by a graph, where vertices represent the individuals in the population, and two vertices are connected if there is a relationship between the two corresponding entities. In order to understand the spread of an infectious disease on the graph, we assign different states to the vertices (e.g. *susceptible*, *infectious*, *recovered*, *carrier*, *exposed* and so on). Then, a discrete or a continuous time stochastic process is defined on the phase space of the states of vertices, where the evolution of the process depends on the structure of the underlying graph.

In many applications, the structure of the graph can be extended by various features, i.e. we can assign some kind of characteristics to the vertices or to the edges. In a social network, infectious diseases are spread through human contact. Since the relationships between the individuals can be different in nature, the probability of spreading is also different among different people. In this study, we examine various types of epidemics on random graphs with multiple type edges. More precisely, we assign a type to the edges of the graph, which is chosen from a finite set of possibilities by certain random dynamics. Then, the infectious disease spreads among the vertices of the graph, so that the probability of infection is different on different types of edges. By using stochastic simulations, we examine the behaviour of the spread of epidemics, when there is also a connection between the types of the edges and the parameters of the process.

In some applications, we can control the spread of the disease, up to a certain level, by separating infected individuals in order to slow down the contagion. We can also assign a state to the edges of the graph, i.e. *active* or *inactive*. We assume that the virus cannot spread on *inactive* edges. At this point, it is clear that if all the edges of the graph are inactive, then the epidemic cannot spread further and all the infected individuals will recover in time, but in practice our goal is to slow down the spread of the infection by eliminating as few connections as possible. Again, by stochastic simulations, we examine the effect of separation (or quarantine), which can be considered as a graph with two types of edges, dynamically changing over time.

We will show that the spread of the epidemic depends on the structure of the underlying graph model, and the introduction of the types of the edges (with the different propagation probabilities) or the quarantine can lead to different results.

The underlying graph models that we have used in this study are the multitype versions of the preferential attachment graph, the model of independent edges ([5, 6]), and a generalized version of a random graph model with *duplication* and *deletion* ([4]).

Related works. There are several articles on models describing the spread of epidemics that include quarantine. One possible direction of the modelling of the spread of infectious diseases is the use of so-called *compartmental models*.

In this approach, we use differential equations to define the dynamics of the change in the number (or in the proportion) of individuals of a given state. In [13], [17], [15] the state of *quarantine* is also introduced in order to enhance the SIS-, SIR- and $SEIR$-processes.

There are some multi-type preferential attachment graph models that have been investigated, see [3, 5, 6, 18]. These models can be used as the underlying structure of spread of epidemic processes. In [2], they investigate the effects of individual decisions on social distancing and isolation in graphs with multi-type vertices. In [1], they include groups of age and risk in the SIR-model and find the optimal strategies for quarantine.

Outline. In Section 2, we define the multi-type random graph models that we use in the following sections. The spread of epidemic process is defined in Section 3. In Section 4, the results of various stochastic simulations are examined. Finally, in Section 5, we mention some possibilities to enhance the models that we have investigated in order to obtain more realistic models.

2. Models

In this section, we define the graphs that are used as the underlying structures in the modelling of the spread of epidemics. These are the (multi-type) *preferential attachment graph*, the *model of independent edges* ([6]) and a generalized version of the *duplication and deletion model* ([4]). In terms of their definitions, these graphs are either *static* or *dynamic*. This means that the structure of the graph of a given size is either defined by a specified rule, or a sequence of graphs is defined where the set of vertices or edges are modified by a given dynamics.

First, let us introduce some notations for the *dynamic* graph models. Let $(G_n)_{n=0}^{\infty}$ be a sequence of finite random graphs. The vertex set and the edge set of G_n are denoted by V_n and E_n, respectively. In the sequel, the number of different types of edges, which is denoted by N, will be fixed. For every $k \in [N] = \{1, 2, \dots, N\}$ let $E_n^{(k)}$ denote the set of edges of type k in G_n. We assume that the different types form a partition of the edges (where we allow empty sets in the partition), i.e. for every n we have $E_n = \bigcup_{k=1}^{N} E_n^{(k)}$ and for every k, l we have $E_n^{(k)} \cap E_n^{(l)} = \emptyset$ whenever $k \neq l$. We assume that the initial configuration G_0 is a finite deterministic graph, moreover for every $k \in [N]$ we have $\left|E_0^{(k)}\right| > 0$. Finally, for every n let $\mathcal{F}_n$ denote the σ-algebra generated by the first n multi-type graphs. We can choose $\mathcal{F}_0$ to be the trivial σ-algebra, since G_0 is deterministic. Notice that $\mathcal{F} = (\mathcal{F}_n)_{n=0}^{\infty}$ is a filtration.

For the *static* graph models, we simply omit the notation indicating the size of the graph from the indices of the vertex and edge sets.

2.1. Preferential attachment graph

In this section, we are going to define the *multi-type preferential attachment graph* model. The *single-type preferential attachment graph* is defined in [10]. First, we have a look at the definition of the *single-type* version. Let $\beta > 0$ be a fixed parameter. Let $V = \{v_1, v_2, \dots, v_n\}$ be the set of vertices. In order to define the set of edges we are going to create a sequence $(v_i^*)_{i=1}^{2m}$ from the elements of V. We start with the empty sequence. If the current length of the sequence equals to k, then we choose the next element v_{k+1}^* to be equal to $v \in V$ with probability $\frac{d(v)+\beta}{k+n\beta}$, where $d(v)$ is the multiplicity of v in the sequence $v_1^*, \dots, v_k^*$. We define the edge set as $E = \big\{\{v_{2i-1}^*, v_{2i}^*\}, i = 1, 2, \dots, m\big\}$. The *single-type preferential attachment graph* with n vertices, m edges and parameter β is denoted by $\mathrm{PAG}_\beta(n, m)$.

Remark 2.1. We are going to choose $m = n$, so we use the sparse preferential attachment graph, because the simulation processes are much faster and more reliable.

Remark 2.2. The *single-type preferential attachment graph* is not the same as, however it is motivated by the (sparse) Barabási–Albert graph model in [7], specified in [9].

In order to obtain a *multi-type preferential attachment graph* denoted by

$$N\text{-}\mathrm{PAG}_\beta(n, m_1, m_2, \dots, m_N),$$

we define N independent *single-type preferential attachment graphs* on the vertex set V. Let us denote these independent graphs by

$$\mathrm{PAG}_\beta^{(1)}(n, m_1),\ \mathrm{PAG}_\beta^{(2)}(n, m_2), \dots,\ \mathrm{PAG}_\beta^{(N)}(n, m_N).$$

Then, the different edges which belong to $\mathrm{PAG}_\beta^{(k)}(n, m_k)$ form the set of edges of type k, where $k \in [N]$. Notice that the number of edges of the N-type preferential attachment graph $N\text{-}\mathrm{PAG}_\beta(n, m_1, m_2, \dots, m_k)$ equals to $N \cdot \sum_{k=1}^{N} m_k$.

2.2. Model of independent edges

The *model of independent edges* is a *dynamic* graph model. There are two different versions.

Version I. This model is a modification and a multi-type version of the models in [11] and [14]. Let $\lambda > 0$ be a fixed parameter. In the n^{th} step, we have the following dynamics:

(i) a new vertex v_n is born, thus $V_n = V_0 \cup \{v_1, \ldots, v_n\}$;

(ii) every existing vertex $v \in V_{n-1}$, independently of each other, is connected to v_n with an edge of type k with probability equal to $\frac{\deg_{n-1}^{(k)}(v)}{2|E_{n-1}|}$, where $\deg_{n-1}^{(k)}(v)$ denotes the number of edges of type k connected to v in G_n. The choices for the edges of different types are also independent of each other.

Version II. Another version of the model was defined in [5]. In the original definition, we have a sequence $(\lambda_n)_{n=1}^{\infty}$ which meets certain conditions. In this article, we assume that $\lambda_n = \lambda$ for every n, where $\lambda > 0$ is fixed. In the n^{th}, step we have the following dynamics:

(i) a new vertex v_n is born, thus $V_n = V_0 \cup \{v_1, \ldots, v_n\}$;

(ii) every existing vertex $v \in V_{n-1}$, independently of each other, is connected to v_n with $\Delta_n^{(k)}(v)$ edges of type k, where $\Delta_n^{(k)}(v) \sim \text{Poi}\left(\lambda \frac{\deg_{n-1}^{(k)}(v)}{2|E_{n-1}|}\right)$, where $\text{Poi}(\mu)$ denotes the Poisson distribution with parameter $\mu > 0$. The number of edges of different types are also independent of each other.

The existence and some properties of the *asymptotic degree distribution* have been proven in [5]. The *degree distribution* of a graph is defined as the proportion of the vertices with a given degree. For graphs with multi-type edges, the *(generalized) degree distribution* is the proportion of vertices which are adjacent to a specific number of edges of each type. The *(generalized) asymptotic degree distribution* is the almost sure limit of these proportions for all possible configurations. For the *model of independent edges*, the *degree distribution* is a family of random variables. If the number of vertices tends to infinity, these proportions stabilize, but they are functions of the asymptotic proportion of the edges of different types in the graph, which is a random variable, i.e. the *asymptotic degree distribution* is a family of mixed random variables. In the spread of epidemic processes, the probabilities of propagation depends on the types of the edges, thus the spreading also depends on the structure of the graph, even if the number of vertices is large.

2.3. Duplication model

For every vertex $v \in V$ we denote by $\mathcal{N}_n(v)$ the set of neighbours of v in G_{n-1}.

In the n^{th} step

(i) a new vertex v_n is born, thus $V_n = V_0 \cup \{v_1, \ldots, v_n\}$.

(ii) We choose a vertex v from V_{n-1} uniformly at random and we connect v_n to every vertex in $\mathcal{N}_{n-1}(v)$. The type of the new edges will be exactly the same as the type of the edges connected to v. (*Duplication.*)

(iii) We choose a vertex w from V_{n-1} uniformly at random and we delete all the edges which are incident to w in G_{n-1}. Notice that the vertices v and w are not necessarily different. (*Deletion.*)

3. Epidemic spread

In this chapter, we introduce the processes that may be suitable for modelling the spread of infectious diseases. These processes can be categorized according to the possible states of vertices of the underlying graph.

For every spread of infection processes, we have an underlying graph to model the structure of the individuals of the population. Let us have a finite random graph on n vertices with multi-type edges denoted by G. The set of vertices and the set of edges of type k are denoted by V and $E^{(k)}$, where $k \in [N]$, respectively.

We will use the following notations:

$$\Pi^k = \left\{ \pi = (\pi_1, \pi_2, \ldots, \pi_k) \in \mathcal{P}^k(V) : \bigcup_{i=1}^{k} \pi_i = V \text{ and } \pi_i \cap \pi_j = \emptyset \text{ for every } i \neq j \right\}$$

$$\Sigma^k = \left\{ \sigma = (\sigma_1, \sigma_2, \ldots, \sigma_k) \in (\mathbb{N}_0)^k : \sum_{i=1}^{k} \sigma_i = n \right\},$$

where $\mathcal{P}^k(V)$ is the set of k-dimensional vectors of subsets of V and $(\mathbb{N}_0)^k$ is the set of k-dimensional vectors of non-negative integers.

3.1. *SIR*-process

There are three different states for the vertices: *susceptible*, *infectious* and *recovered*. Susceptible vertices represent individuals who are healthy, but can be infected. Infectious vertices play the role of entities who are infected and infectious, i.e. they can spread the infection to susceptible vertices. Finally, recovered vertices represent the individuals who are not infectious any longer,

and immune, i.e. cannot be infected again. The following flow diagram shows the transitions between the different states.

Let us fix $J \in \mathbb{N}^+$, i.e. the total number of steps. For every $j \in [J]$, in the j^{th} step the set of vertices of state *susceptible*, *infectious* and *recovered* are denoted by $\mathcal{S}_j$, $\mathcal{I}_j$ and $\mathcal{R}_j$, respectively. We will also use the notations $S_j = |\mathcal{S}_j|$, $I_j = |\mathcal{I}_j|$ and $R_j = |\mathcal{R}_j|$. Since the structure of the underlying graph does not change during the spread of the epidemics, for every $j \in [J] \cup \{0\}$, we have $\mathcal{S}_j \cup \mathcal{I}_j \cup \mathcal{R}_j = V$, thus $S_j + I_j + R_j = n$.

We define the following (discrete-time) stochastic processes:

$$\mathbf{X} : \{0\} \cup [J] \to \Pi^3, \qquad \mathbf{X} = (X_j)_{j=0}^J = (\mathcal{S}_j, \mathcal{I}_j, \mathcal{R}_j)_{j=0}^J$$
$$\mathbf{Y} : \{0\} \cup [J] \to \Sigma^3, \qquad \mathbf{Y} = (Y_j)_{j=0}^J = (S_j, I_j, R_j)_{j=0}^J .$$

Remark 3.1. Notice that we have $Y_j \sim \sigma(X_j)$ for every $j \in [J]$, i.e. Y_j is measurable to X_j, thus the value of Y_j can be calculated from X_j.

As in the previous section, the σ-algebra generated by the underlying random multi-type graph G is denoted by $\mathcal{F}_n$. Let us define $\mathcal{G}_j = \sigma\left(\mathcal{F}_n, (X_i)_{i=0}^j\right)$, i.e. the σ-algebra generated by G and the first j steps of the process $\mathbf{X}$.

We assume that the initial sets of the vertices of different states $\mathcal{S}_0$, $\mathcal{I}_0$ and $\mathcal{R}_0$ are given. For every $j \in [J]$, in the j^{th} step we have the following dynamics:

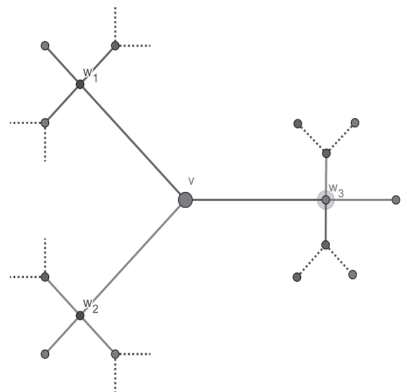
(i) every *susceptible* vertex $v \in \mathcal{S}_{j-1}$ becomes *infectious* with probability

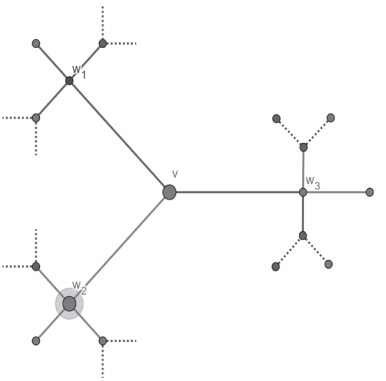
$$\mathbf{P}\left(v \in \mathcal{I}_j \middle| \mathcal{G}_{j-1}\right) = 1 - \prod_{k=1}^{N} (1 - p_k)^{i_{j-1}^{(k)}(v)},$$

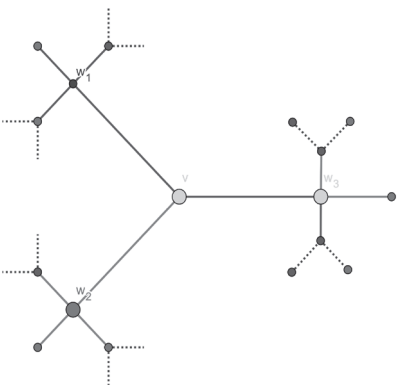
where $i_{j-1}^{(k)}(v)$ is the number of edges of type k which connect v to an *infectious* vertex in step $j-1$. Notice that the types of edges remain unchanged, but the states of the vertices may be different over the steps.

(ii) Every *infectious* vertex $v \in \mathcal{I}_{j-1}$ becomes *recovered* with probability $\mathbf{P}\left(v \in \mathcal{R}_j \middle| \mathcal{G}_{j-1}\right) = q$.

An illustration of the dynamics of the SIR-process can be seen in the be seen in the figures below.







Top left: Vertex v may infect w_1 and w_2 with different probabilities, because they are connected to v with different types of edges. Here, vertex w_3 cannot be infected, because it has already been infected.
Top right: Let us assume that vertex w_2 has been infected by v.
Bottom: After a while infectious vertices become recovered and they can no longer become infectious again. Here, vertices v an w_3 became recovered.

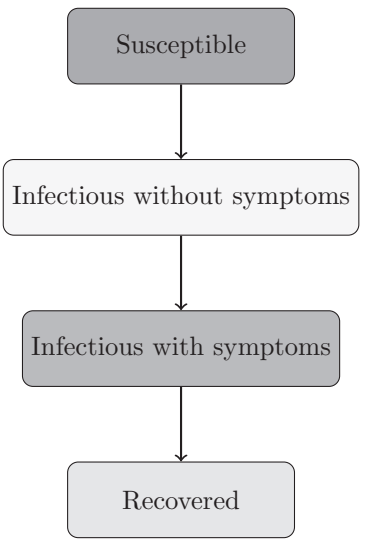
In the SIR-process, the parameters are the probabilities of the spread of infection on the different types of edges $(p_1, p_2, \ldots, p_N)$, the probability of recovery (q) (which is the same for all vertices), the finite time horizon (T) and the underlying graph.

3.2. Dynamical SI_1I_2R-process

Many infectious diseases are known in which the infected patient does not initially produce symptoms but is nevertheless contagious. Epidemics caused by such diseases are particularly difficult to control. It is advisable to separate any infectious patient from other people as soon as possible, but in this case the fact of the infection is initially unknown. In this section, we present a modification of the previously discussed model that can be used to model the spread of infectious diseases that have a latency period.

This is a modified version of the SIR-process. There are four different states for the vertices: *susceptible*, *infectious without symptoms* (I_1), *infectious with symptoms* (I_2) and *recovered*.

The following flow diagram shows the transitions between the different states.



In this model we assume that the edges of the underlying graph model are either *open* or *closed*. *Closed* edges indicate the separation of the corresponding points, i.e. the disease cannot spread through *closed* edges.

Let us fix $J \in \mathbb{N}^+$, i.e. the total number of steps. For every $j \in [J]$, in the j^{th} step the set of vertices of state *susceptible*, *infectious without symptoms*, *infectious with symptoms* and *recovered* are denoted by $\mathcal{S}_j$, $\mathcal{I}_j^{(1)}$, $\mathcal{I}_j^{(2)}$ and $\mathcal{R}_j$, respectively. We will also use the following notations for the cardinality of the sets of vertices of different states:

$$S_j = |\mathcal{S}_j|, I_j^{(1)} = |\mathcal{I}_j^{(1)}|, I_j^{(2)} = |\mathcal{I}_j^{(2)}| \text{and} R_j = |\mathcal{R}_j|.$$

Since the structure of the underlying graph does not change during the spread of the epidemics, for every $j \in [J] \cup \{0\}$, we have $\mathcal{S}_j \cup \mathcal{I}_j^{(1)} \cup \mathcal{I}_j^{(2)} \cup \mathcal{R}_j = V$, thus $S_j + I_j^{(1)} + I_j^{(2)} + R_j = n$.

We define the following (discrete-time) stochastic processes:

$$\mathbf{X} : \{0\} \cup [J] \to \Pi^4, \qquad \mathbf{X} = (X_j)_{j=0}^J = \left(\mathcal{S}_j, \mathcal{I}_j^{(1)}, \mathcal{I}_j^{(2)}, \mathcal{R}_j\right)_{j=0}^J$$

$$\mathbf{Y} : \{0\} \cup [J] \to \Sigma^4, \qquad \mathbf{Y} = (Y_j)_{j=0}^J = \left(S_j, I_j^{(1)}, I_j^{(2)} R_j\right)_{j=0}^J.$$

As in the previous section, we have $\mathcal{G}_j = \sigma\left(\mathcal{F}_n, (X_i)_{i=0}^{j}\right)$, i.e. the σ-algebra generated by G and the first j steps of the process $\mathbf{X}$.

We assume that the initial sets of the vertices of different states $\mathcal{S}_0$, $\mathcal{I}_0^{(1)}$, $\mathcal{I}_0^{(2)}$ and $\mathcal{R}_0$ are given. For every $j \in [J]$, in the j^{th} step we have the following dynamics:

(i) every *susceptible* vertex $v \in \mathcal{S}_{j-1}$ becomes *infectious without symptoms* with probability

$$\mathbf{P}\left(v \in \mathcal{I}_j^{(1)} \middle| \mathcal{G}_{j-1}\right) = 1 - \prod_{k=1}^{N} (1-p_k)^{i_{j-1}^{(k)}(v)},$$

where $i_{j-1}^{(k)}(v)$ is the number of *open* edges of type k which connect v with an *infectious with symptoms* vertex in the step $j-1$. Notice that the types of edges remain unchanged, but the states of the vertices may be different over the steps.

(ii) Every *infectious without symptoms* vertex $v \in \mathcal{I}_{j-1}^{(1)}$ becomes *infectious with symptoms* with probability

$$\mathbf{P}\left(v \in \mathcal{I}_j^{(2)} \middle| \mathcal{G}_{j-1}\right) = r.$$

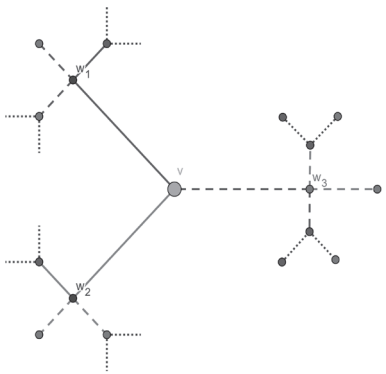
Then, every edge becomes *closed* which are incident to the vertex v.

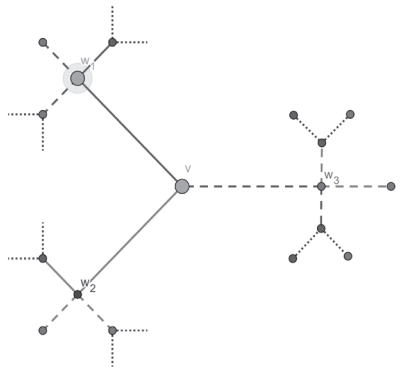
(iii) Every *infectious with symptoms* vertex $v \in \mathcal{I}_{j-1}^{(2)}$ becomes *recovered* with probability

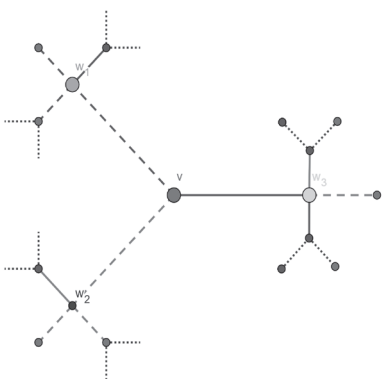
$$\mathbf{P}\left(v \in \mathcal{R}_j \middle| \mathcal{G}_{j-1}\right) = q.$$

Then, every edge becomes *open* which are incident to v, except those which are incident to an *infectious with symptoms* vertices.

As in the previous section, an illustration of the dynamics of the SI_1I_2R-process can be seen in the figures below.







Top left: Vertex v may infect w_1 and w_2 with different probabilities, because they are connected to v with different types of edges. Here, vertex w_3 has already been infected, thus it is temporarily removed from the rest of the graph like all other I_2-vertices.
Top right: Let us assume that vertex w_1 has been infected by v.
Bottom: After a while I_1-vertices become I_2-vertices and they show symptoms of the disease. Then, they are separated from the rest of the graph, like vertex v in this graph. The I_2-vertices become recovered after a random period of time. Then, they are reconnected to the rest of the graph, except to their neighbours which are I_2-vertices. Here, vertex w_3 became recovered.

In the SI_1I_2R-process, the parameters are the probabilities of the spread of infection on the different types of edges $(p_1, p_2, \ldots, p_N)$, the probability of appearance of symptoms (r), the probability of recovery (q) (which are the same for all vertices), the finite time horizon (T) and the underlying graph.

4. Sensitivity analysis of the parameters

In this section, we have a look at the results of some stochastic simulations. We generated random graphs with two types of edges according to the models in Section 2, then we simulated the spread of epidemic processes on these graphs.

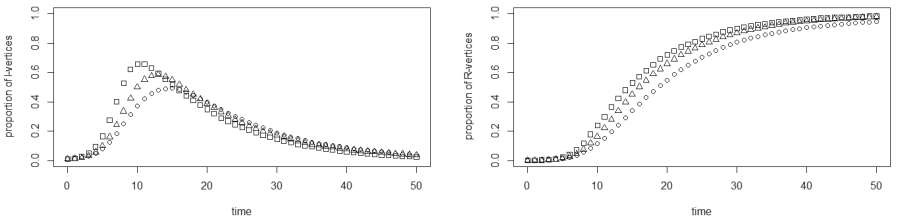
For each graph model, we examine the SIR-process at first. As a reminder, in this variant, the symptoms of the infectious disease become apparent immediately after infection. After recovery, the individuals become immune to the disease so that they can no longer become infected. In the following sections, we study the change of the proportion of vertices with different states over time, and we compare the results of different parametrizations. Then, we have a look at the SI_1I_2R-process. In this variant, it takes some random number of steps until the symptoms of the infectious disease become apparent after infection. Similarly to the SIR-process, the individuals become immune to the disease after recovery so that they can no longer become infected.

4.1. Preferential attachment graph

In this section, we use the multi-type *preferential attachment* model as the underlying graph of the process. For a given parametrization, we generated $N = 10$ random graphs on $n = 1000$ vertices with two types of edges. We had $m_1 = 2000$ edges of the first type and $m_2 = 1000$ edges of the second one. We examined three different parametrizations of the SIR-process:

$p_1 = 0.1$	$p_2 = 0.1$	$q = 0.1$	$T = 50$
$p_1 = 0.05$	$p_2 = 0.1$	$q = 0.1$	$T = 50$
$p_1 = 0.1$	$p_2 = 0.05$	$q = 0.1$	$T = 50$

For the SIR-processes, 10% of the vertices are *infectious* and all the other vertices are *susceptible* at the beginning, and for the SI_1I_2R-processes, 10% of the vertices are *infectious without symptoms* and the rest of the vertices are *susceptible*. The trajectories of the average of the N scenarios for the three parametrizations can be seen on the following images. The trajectories, marked with $\circ$, $\square$ and $\triangle$, represent the 1^{st}, the 2^{nd} and the 3^{rd} parametrization, respectively.

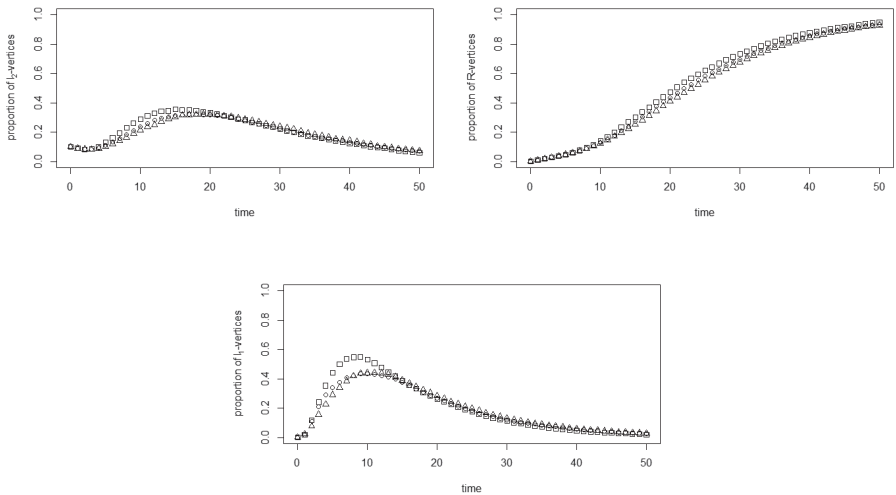


SIR-process in the preferential attachment model

The ∘-trajectory represents the "single-type case", because $p_1 = p_2$. We can see the constant decrease of the proportion of *susceptible* vertices and the constant increase of the *recovered* vertices. However, as for the *infectious* vertices, we observe the rapid increase and then the slow decrease.

Since the number of the edges of the 1^{st} and the 2^{nd} types are different, decreasing the probability of propagation on the edges of the 1^{st} type has a more severe impact than the same decrease on the edges of the 2^{nd} type.

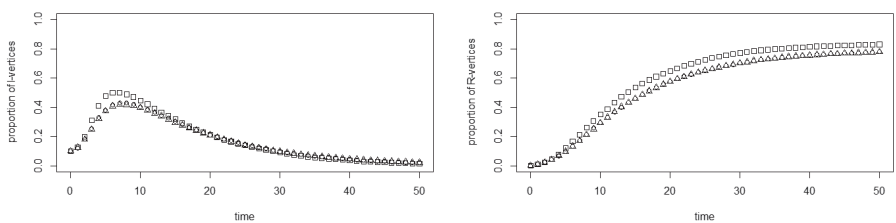
We use the same parametrization for the SI_1I_2R-process with an additional parameter, which is $r = 0.1$ (the probability that symptoms of an infected individual become detectable). As a result of the isolation of patients, who are *infectious with symptoms*, the epidemic curve flattens. Even though there is a latency period, if we isolate people when the symptoms appear, it already improves a lot. This holds in general, regardless of the type of edges. We can see that the introduction of quarantine makes the model less sensitive to changes in the parameters. However, the slowing effect of the quarantine is much severe for the 2^{nd} and 3^{rd} than it is in the single-type case.



SI_1I_2R-process in the preferential attachment model

4.2. Model of independent edges - version I

In this section, we use the first version of the *model of independent edges* as the underlying graph of the process. Again, for a given parametrization, we generated $N = 10$ random graphs on $n = 1000$ vertices with two types of edges. The parametrization of the SIR-process is the same as in the previous section.

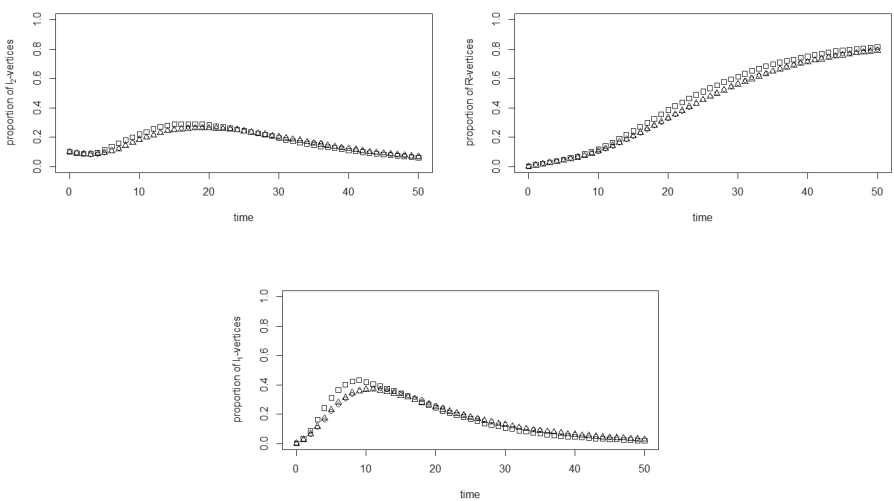


SIR-process in the model of independent edges - I

The structure of both versions of the *model of independent edges* depend on the finite configuration which is the initial graph of the growing sequence. In

the simulations, the initial configuration is a graph with two vertices which are connected with 2 edges of the 1^{st} and 1 edge of the 2^{nd} type. Similarly to the *preferential attachment* model, the underlying graph contains more edges of the 1^{st} type than the 2^{nd} type, but the results are less sensitive to changes in the probabilities of propagation. We can also see that the spread of the infection is slower even in the single-type case compared to the *preferential attachment* model. This is due to the fact that edges of the same type are more likely grouped in the *model of independent edges*.

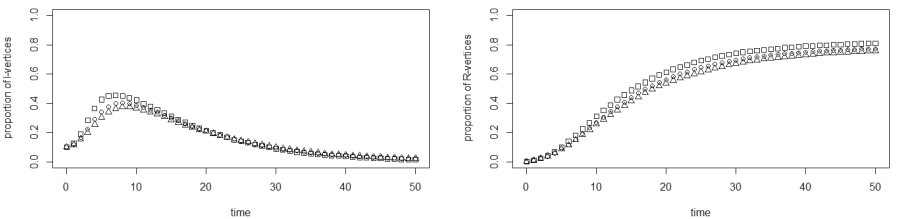
For the SI_1I_2R-process, we use the same parametrization as in the previous section. In this case, the impact of having two different types for the edges is less severe. The observable data is almost the same as it is in the single-type case.



SI_1I_2R-process in the model of independent edges - I

4.3. Model of independent edges - version II

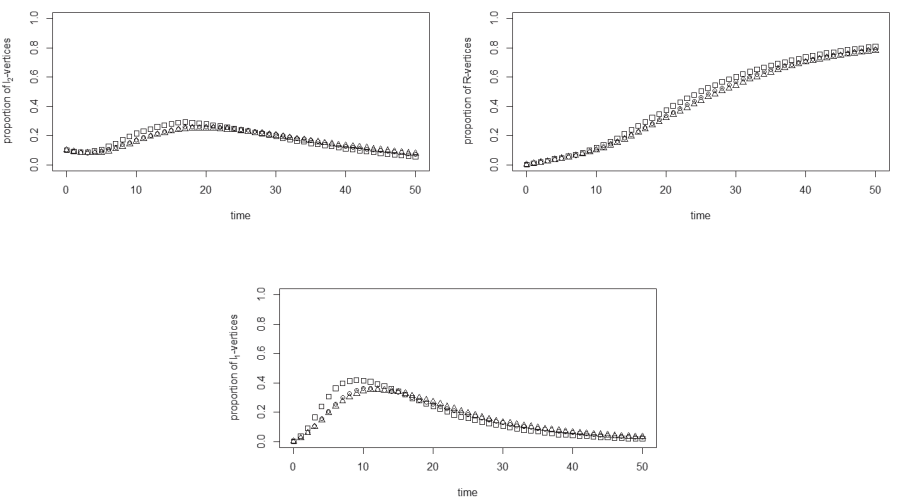
In this section, we use the second version of the *model of independent edges* as the underlying graph of the process. Again, for a given parametrization, we generated $N = 10$ random graphs on $n = 1000$ vertices with two types of edges. We have chosen $\lambda = 1$, i.e. the fixed parameter of the graph. The parametrization of the SIR-process is still the same as in the previous section.



SIR-process in the model of
independent edges - II

Although the construction of the underlying graph is different, the results are very similar to the previous version of the *model of independent edges*. In the first version of the model, the (random) degrees of the vertices are binomially distributed, while they follow Poisson distribution in the second version of the model. In this case when the number of vertices is sufficiently large, these distributions have almost the same behaviour.

Again, for the SI_1I_2R-process, we use the same parametrization as in the previous section.

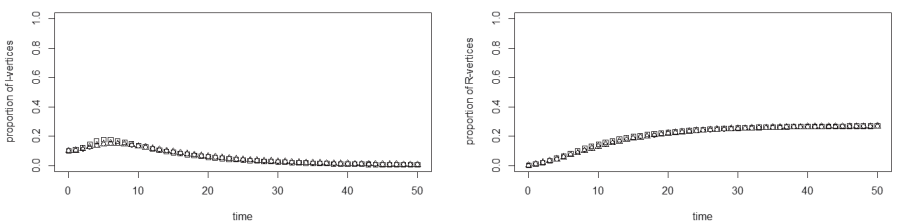


SI_1I_2R-process in the model of
independent edges - II

4.4. Duplication model

In this section, we use the *duplication model* as the underlying graph of the process. Again, for a given parametrization, we generated $N = 10$ random graphs on $n = 1000$ vertices with two types of edges. Similarly to the *model of independent edges*, the structure of the *duplication model* depends on the initial finite configuration. In our simulations, the initial configuration contains two independent Erdős–Rényi graphs on 900 vertices, and the probability that a pair of vertices is connected by an edge equals to 0.1. Because of the choice of parameters, both graphs contain a single giant connected component with high probability. Then, we apply 100 duplication and deletion steps. The edges of these graphs define the set of edges of different types in the multi-type configuration. The resulting graph is highly *clustered*, i.e. it mainly consists of independent cliques. This corresponds to a model where we segregate well-isolated groups through restrictive measures, such as the individuals only meet with those who are living in the same household, or the schools are partially open but the classes are isolated. If there are many independent components and only a negligible part of the vertices are *infectious*, then the results depend on the structure of the typical components and not on the global structure of the graph.

The parametrization of the SIR-process is still the same as in the previous section.

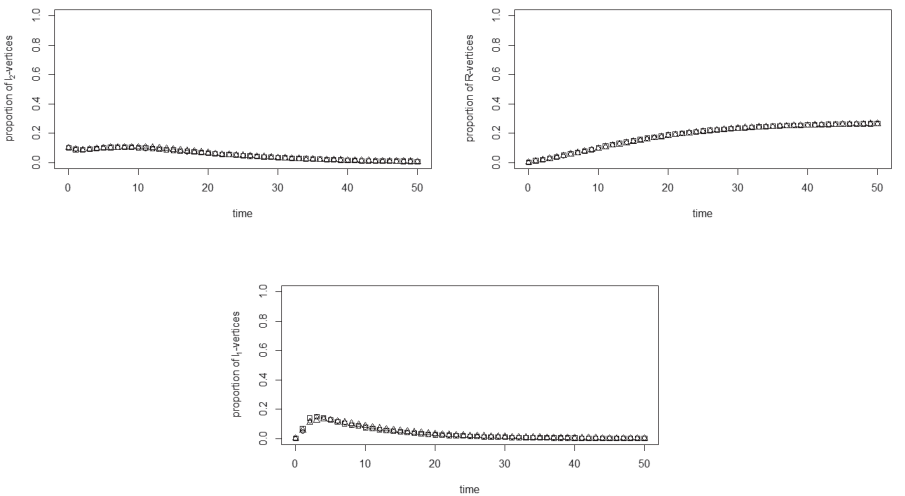


SIR-process in the duplication model

Because of the clustering properties of the underlying graph model, the results are not sensitive to the changes in probabilities of propagation. Initially, 10% of the vertices is *infectious* (for the SIR-process) or *infectious without symptoms* (for the SI_1I_2R-process). Within the dense connected components which contain some *infectious* vertices at the beginning, the epidemic will spread, no matter how small the infection probabilities are.

Again, for the SI_1I_2R-process, we use the same parametrization as in the previous section. Because of the clustering properties of the model, the effect

of quarantine is less significant. In the connected components, the epidemic will spread among the individuals before we can detect the symptoms and remove the edges between some of the vertices. It also means that if we create isolated bubbles with the help of restrictive measures (e.g. school classes are well-separated), then no more quarantine is necessary, and it is not a problem if there are edges with higher propagation probabilities, the epidemic will spread too a much smaller extent.



SI_1I_2R-process in the duplication model

5. Further research possibilities

We have seen that the introduction of different types of edges and the presence of segregated groups can have a severe impact on the spread of the epidemic.

Due to the diversity of processes describing the spread of the epidemic, there are many opportunities for further research. One possibility is to introduce a model in which a group of vertices of the graph represent the medical employees. In some applications, the infected individuals require some kind of medical treatment. In this case, the medical employees (doctors, nurses, etc.) are assigned to the infected individuals. We may assume that the medical em-

ployees can also be infected, and then, they also require medical treatment. One can examine how much capacity is required in the healthcare for the infected patients to receive appropriate treatment under different parameters of the infection.

Another possibility is to study continuous-time models, i.e. models in which the events describing the infections and recoveries occur on a continuous time scale. For example, we can use exponentially distributed random times that determine when these events occur. Continuous-time models are typically more complex than the discrete-time versions, but they often describe epidemics in reality in a more natural way.

References

[1] **Acemoglu, D., V. Chernozhukov, I. Werning and M.D. Whinston,** A multi-risk SIR model with optimally targeted lockdown. Technical report, National Bureau of Economic Research (2020).

[2] **Amini, H. and A. Minca,** Epidemic Spreading and Equilibrium Social Distancing in Heterogeneous Networks, arXiv preprint (2020) `https://arxiv.org/pdf/2007.04210v2.pdf`

[3] **Antunović, T., E. Mossel and M. Rácz,** Coexistence in preferential attachment networks, *Combinatorics, Probability and Computing*, **25(6)** (2016), 797–822.

[4] **Backhausz, Á. and T. F. Móri,** Further properties of a random graph with duplications and deletions, *Stochastic Models*, **32(1)** (2016), 99–120.

[5] **Backhausz, Á. and B. Rozner,** Asymptotic degree distribution in preferential attachment graph models with multiple type edges, *Stochastic Models*, **35(4)** (2019), 496–522.

[6] **Backhausz, Á. and B. Rozner,** Barabási–Albert random graph with multiple type edges with perturbation, *Acta Mathematica Hungarica*, **161(1)** (2020), 212–229.

[7] **Barabási, A-L., R. Albert,** Emergence of scaling in random networks, *Science*, **286** (1999), 509–512.

[8] **Berger, N., C. Borgs, J. Chayes and A. Saberi,** On the spread of viruses on the internet, *Proceedings of the 16th ACM-SIAM Symposium on Discrete Algorithm* (*SODA*), 2005.

[9] **Bollobás, B., O. Riordan, J. Spencer and G. Tusnády,** The degree sequence of a scale-free random graph process, *Random Structures Algorithms*, **18(3)** (2001), 279–290.

[10] **Borgs, C., J. Chayes, L. Lovász, V. T. Sós and K. Vesztergombi,** Limits of randomly grown graph sequences, *Eur. J. Combin.*, **32(7)** (2011), 985–999.

[11] **Dereich, S. and P. Mörters,** Random networks with sublinear preferential attachment: degree evolutions, *Electronic Journal of Probability*, **14** (2009), 1222–1267.

[12] **Durrett, R.,** *Random Graph Dynamics*, Cambridge University Press, Cambridge, 2006.

[13] **Hamelin, F., A. Iggidr, A. Rapaport, S. Sallet and M.O. Souza,** Identifiability and observability of the SIR model with quarantine, arXiv preprint (2021)
https://arxiv.org/pdf/2103.04614v1.pdf

[14] **Katona, Z. and T.F. Móri,** A new class of scale free random graphs, *Statistics & Probability Letters*, **76(15)** (2006), 1587–1593.

[15] **Kiouach, D., Y. Sabbar and S. El-idrissi,** New results on the asymptotic behavior of an SIS epidemiological model with quarantine strategy, stochastic transmission, and Lévy disturbance, arXiv preprint (2020)
https://arxiv.org/pdf/2012.00875v1.pdf

[16] **Kiss, I.Z., J. Miller and P. Simon,** *Mathematics of Epidemics on Networks*, Springer (2017).

[17] **Kumar, M. and S. Abbas,** Analysis of steady state solutions to an age structured SEQIR model with optimal vaccination, arXiv preprint (2020)
https://arxiv.org/pdf/2012.10068v1.pdf

[18] **Rosengren, S.,** A multi-type preferential attachment tree, *Internet Mathematics*, **1(1)** (2018). https://doi.org/10.24166/im.05.2018

B. Rozner
Department of Probability Theory and Statistics
Faculty of Science
ELTE Eötvös Loránd University
Budapest
Hungary
bence.rozner@ttk.elte.hu