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An Epidemic on a Weighted Network

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Abstract

We introduce a weighted configuration model graph, where *edge weights* correspond to the probability of infection in an epidemic on the graph, focusing on two different weights. We study the *basic reproduction number* R_0, the *probability of a large outbreak* and the *relative final size of a large outbreak*, using discrete time and Markovian continuous time settings. Results are compared with those for a calibrated unweighted graph. The degree distributions are based both on empirical network data and on theoretical constructs. Using copulas to model the dependence between the degrees of the different edge types allows for modeling the correlation over a wide range. The weighted model produces much richer results than the unweighted model. Also, while R_0 always increases with increasing correlation between the two degrees, this is not necessarily true for the probability of an epidemic nor for the relative final size of it. The copula model can produce results that are similar to those of the empirical degree distributions, indicating that it is a viable alternative to using the full empirical data.

Keywords - epidemics, basic reproduction number, weighted graph, configuration model, final size, large outbreak, copula.

1 Introduction

The configuration model is a well known graph model where each vertex is assigned a number of half-edges which are then connected uniformly at random (Molloy & Reed, 1995; Bollobás, 2001). This model is often used when a specific degree distribution or degree sequence is desired. The development of epidemics can then be studied on these graphs. Three important quantities derived from such epidemics are the *basic reproduction number* R_0, the *probability of a large outbreak* and the *relative final size of a large outbreak*, see e.g. (Britton, 2010). Often all edges are treated as identical and for instance the transmission risk (of the infection) is assumed to be the same for all edges, which is not true for many real world networks.

In some recent models edges have been divided into different types with different epidemic properties for each edge type. E.g. in (Britton et al., 2011) a degree is assigned to each vertex and for each half-edge a weight is assigned independently from a weight distribution that is only allowed to depend on the degree of the vertex. Half-edges of identical weight are then connected to each other as in the configuration model. In (Kamp et al., 2013) vertices are assigned a degree and a number of *interactions* from a simultaneous distribution. Each interaction is then distributed independently and uniformly among all edges of the vertex. The number of interactions constitute the weight of the edge. Half-edges having the same (or similar) weight are then connected according to the configuration model. Both of these models place restrictions on the allowed degree distributions.

One possible generalization is a model with an arbitrary number of edge types, each with its own weight, allowing for an arbitrary dependence between the degrees of the different edge types. Each vertex is assigned a multivariate degree which specifies how many half-edges of each type it has. The degree can be assigned from a given degree sequence (such as from an empirical graph) or from a given degree distribution. Half-edges *of the same type* are then paired just as in the normal configuration model to create a multilayer configuration model, where two layers are only connected at vertices which have edges of both types. In this paper we study this a model, but for simplicity restrict it to two types of edges and thus two weights, and the development of SIR epidemics (explained in *Section 2.4*) on it. Even then it is possible to study some interesting configurations - e.g. we can assume that each person has two different types of contacts with other people and that the probability of infection differs on these types. Examples of such situations are family relations vs job relations, or casual vs more permanent sexual relationships. The theoretical foundations for the model, including the graph model and the epidemic model, are presented in *Section 2*.

Important questions are if the quantities of interest (described above) differ on the weighted model versus a calibrated unweighted model and also how these quantities are affected by the level of correlation between the degrees of the different types of edges. We construct the unweighted model from the weighted model by ignoring edge type and create a single configuration model network from all edges, but without doing any other changes to the degree distribution. We calibrate the weighted and unweighted models such that both have the same *mean infectious activity* (see *Section 2.5*). We then compare the development of SIR epidemics on the weighted and the unweighted models. For the weighted model we also vary the correlation between the two degrees of an individual and study the effect on the quantities of interest. We see that the weighted model produces much richer results, in that all the studied parameters typically show much more variation over the allowed range of the parameters that we can vary, compared with the unweighted model. Results also indicate that a model, where the dependence structure between the two degrees has been defined through a standard normal copula, often works equally well as a model where the dependence structure is taken from the empirical degree distribution. The copula model allows for varying the correlation through a wide range that is only limited by the marginal degree distributions (for the different edge types). Some theoretical results are given in *Section 3* while numerical results are shown in *Section 4*. A discussion can be found in *Section 5*.

2 The Model

In this section we briefly discuss graphs in *Section 2.1* and then present the unweighted and the weighted graph models in *Section 2.2* and *Section 2.3*. Specific examples are discussed in *Section 2.6*. The use of copulas to model the correlation between the degrees of the two edge types is mainly discussed in *Appendix B*. The modeling of SIR epidemics on such graphs in discrete and continuous time is discussed in *Section 2.4*.

2.1 Graphs

In this paper we use the words *graph* and *network* interchangeably. The number of vertices n is given and (typically) the case $n \to \infty$ is studied, although we do not always mention this explicitly. In the context of epidemics on graphs, vertices represent people and edges represent some type of relationship making transmission of an infection possible. We work with undirected graphs, so if two vertices are connected by an edge they can both

infect each other. Edges can be of different type and with different properties. We use ξ as an index to indicate the edge type, whenever appropriate. In this paper we limit the analysis to two edge types, so $\xi \in \{1, 2\}$.

The degrees of all vertices in the graph is called a *degree sequence*. Graphs and degree sequences can e.g. be obtained from empirical data or from more theoretical constructs. Graphs that are created by random processes are called random graphs. One such random graph model is the configuration model which is discussed in *Section 2.2* and *Section 2.3*.

2.2 Unweighted Configuration Model

The configuration model has already been thoroughly investigated (see e.g (Molloy & Reed, 1995) or (Britton et al., 2006)) and here we just briefly recapitulate how a configuration model graph is created and some properties of it.

A configuration model graph is always finite, having n vertices, but asymptotic results are obtained by letting $n \to \infty$. Initially each vertex is assigned a number of yet unconnected half-edges that can e.g. be drawn from some given degree distribution, D. Then half-edges are paired uniformly at random. Parallel edges (several edges going between the same vertices) and self loops (edges with both ends going to the same vertex) can occur, but with suitable restrictions on the degree sequence or the degree distribution the number of such edges is small compared with the total number of edges in the graph and thus (asymptotically) do not affect the properties of the graph. A finite first moment is needed in order for the configuration model to converge in distribution as $n \to \infty$ and a finite second moment is needed in order to obtain a finite first moment for the *size-biased* distribution (see below).

The degree distribution is defined by

$$p_i = \mathbb{P}(D{=}i).$$

This is the degree of a vertex that is chosen uniformly at random from the graph. The important properties of the graph that we return to later in this paper are the mean and the variance:

$$\begin{aligned} \mu &= \mathrm{E}(D), \\ \sigma^2 &= \mathrm{Var}(D). \end{aligned}$$

If a vertex is instead chosen by first selecting an edge and then selecting one of the vertices connected to the edge with the same probability we obtain

the *size-biased* distribution $\widetilde{D}$. The size-biased distribution has the following (asymptotic) properties:

$$\widetilde{p}_i = \mathbb{P}(\widetilde{D}{=}i) = \frac{ip_i}{\mu}, \tag{1}$$

$$\widetilde{\mu} = \mathrm{E}(\widetilde{D}) = \frac{\mathrm{E}(D^2)}{\mu} = \mu + \frac{\sigma^2}{\mu}. \tag{2}$$

2.3 Weighted Configuration Model

In the weighted model we have two types of edges (labeled 1 and 2 in this paper). Starting with a given number of vertices, each vertex is assigned a number of half-edges drawn independently for each vertex from a given degree distribution $\mathbf{D} = (D_1, D_2)$. Half-edges *of the same type* are then connected uniformly at random, effectively creating two configuration model graphs that are connected only at vertices that have both types of edges.

The properties of this graph are given by the degree distribution $\mathbf{D} = (D_1, D_2)$. The distribution is defined by the probabilities

$$p_{ij} \quad = \quad \mathbb{P}(D_1{=}i, D_2{=}j).$$

We only place a minimum set of requirements on this distribution. First we require that at least one $p_{ij} > 0$ for some $i, j > 0$. Otherwise we effectively have two different vertex types, one with only type 1 edges and another with only type 2 edges and these never interact - creating two separate configuration models. Secondly we require that the first and second moments are finite, so that $\mathrm{E}(D_\xi) < \infty$ and $\mathrm{Var}(D_\xi) < \infty$, where $\xi \in \{1, 2\}$ indicates the edge type. This ensures that the parallel edges and self loops can be ignored in the resulting configuration model graphs (Britton et al., 2006) and that the first moment of the size-biased distribution are finite (just as for the unweighted configuration model).

When studying this distribution the following definitions are useful:

$$\begin{aligned} \mu_\xi &= \mathrm{E}(D_\xi), \\ \sigma_\xi^2 &= \mathrm{Var}(D_\xi), \\ \sigma_{12} &= \mathrm{Cov}(D_1, D_2), \\ \rho &= \frac{\sigma_{12}}{\sigma_1\sigma_2}, \text{ if } \sigma_1, \sigma_2 > 0 \end{aligned}$$

where the last one is the correlation coefficient between D_1 and D_2.

The degree of a vertex selected uniformly at random from the graph is distributed according to $\mathbf{D}$. If we instead select a vertex by following an

edge of specified type, selected uniformly at random, the resulting degree distribution is different and also depends on the type of the edge that we follow. Given that we follow a uniformly selected edge of type $\xi \in \{1, 2\}$ the *size-biased* degree distribution is $\widetilde{\mathbf{D}}_\xi = (\widetilde{D}_{1|\xi}, \widetilde{D}_{2|\xi})$. The tilde above the symbols indicates quantities obtained from the size-biased distribution. The probability mass function $\widetilde{p}_\xi(i, j) = \mathbb{P}(\widetilde{D}_{1|\xi} = i, \widetilde{D}_{2|\xi} = j)$ distribution is then

$$\widetilde{p}_1(i, j) = \frac{ip_{ij}}{\mu_1}, \tag{3}$$

$$\widetilde{p}_2(i, j) = \frac{jp_{ij}}{\mu_2}, \tag{4}$$

when following an edge of type 1 and 2, respectively. *Eq. (3)* can be understood intuitively by realizing that when following an edge selected uniformly at random the probability of connecting to a vertex with degree i is proportional to i (thus the name *size-biased* distribution). This probability must also be proportional to the relative occurrence of vertices with this degree (quantified by p_{ij}). Finally the $1/\mu_1$ is a norming constant needed to make $\widetilde{p}_1(i, j)$ a proper probability mass function. *Eq. (4)* can be in the same way.

When following an edge of type 1 we now obtain (using *Eq. 3 and 4*)

$$\widetilde{\mu}_{1|1} = \mathrm{E}(\widetilde{D}_{1|1}) = \frac{\mathrm{E}(D_1^2)}{\mu_1} = \mu_1 + \frac{\sigma_1^2}{\mu_1},$$
$$\widetilde{\mu}_{2|1} = \mathrm{E}(\widetilde{D}_{2|1}) = \frac{\mathrm{E}(D_1 D_2)}{\mu_1} = \mu_2 + \frac{\sigma_{12}}{\mu_1}.$$

The corresponding equations are valid when starting with an edge of type 2 - just switch 1 and 2 in the equations. We return to these equations in *Section 2.4*.

In later sections we compare epidemics on the weighted model with epidemics on the corresponding unweighted model where we simply neglect the weights so $D = D_1 + D_2$. Expressions for the mean, the variance and the probability mass function in the unweighted model are

$$\mu = \mu_1 + \mu_2,$$
$$\sigma^2 = \sigma_1^2 + \sigma_2^2 + 2\sigma_{12},$$
$$p_i = \mathbb{P}(D{=}i) = \sum_{k=0}^{i} p_{k,i-k}.$$

These quantities can be used directly in the results for the *size-biased* distribution for the unweighted configuration model in *Section 2.2*.

2.4 SIR Epidemics

We work with the SIR (Susceptible - Infectious - Recovered) model in discrete and in continuous time, see e.g. (Lefèvre, 1990). In this model vertices represent people and edges represent paths by which people can infect each other. Initially only one vertex (the *index case*) is infected. An infected vertex is infectious (can infect susceptible neighbors) until it has recovered. After recovering the vertex is immune forever and cannot ever infect any other vertex. The epidemic stops when there are no more infected vertices. At this time typically some portion of all vertices are recovered and some are still susceptible. The *proportion* of recovered vertices we call the *relative final size* of the epidemic. The expected number of vertices that a typical infected vertex infects early on in the epidemic, when the population is almost completely susceptible, is called the *basic reproduction number* (denoted R_0). We are also interested in the probability of a large outbreak. All quantities are derived in the limit $n \to \infty$, although this is not always mentioned explicitly and the derivations are not formal.

In discrete time in each time step each infected vertex tries to infect its susceptible neighbors after which the vertex recovers. Thus in the next time step only newly infected vertices continue to spread the infection. We assume that for each edge (among the susceptible neighbors) infection occurs independently with probability π_ξ that is allowed to depend only on the edge type $\xi \in \{1, 2\}$. This is called the Reed-Frost model (see e.g. (Bailey et al., 1975), Section 8).

In continuous time we restrict the analysis to Markovian models where an infected vertex has an infectious period that is exponentially distributed with recovery rate γ. An infectious vertex has infectious contacts with each susceptible neighbor independently according to a Poisson process with intensity β_ξ that depends only on the edge type. The probability of an arbitrary edge of an infected vertex passing on the infection to a susceptible edge before the end of the infectious period is then

$$\pi_\xi = \frac{\beta_\xi}{\beta_\xi + \gamma}.$$

When comparing the discrete time and the continuous time models we choose β_ξ such that π_ξ are the same in both models. We must, however, keep in mind that the infectious period affects all neighbors of a vertex and thus in the Markovian case, the events that an infection is propagated along different edges of the same vertex are no longer independent. This must be taken into account when determining the probability of a large outbreak.

In the next section we analyze both the weighted and the unweighted

models in discrete and in real time, to obtain explicit expressions or algorithms for calculating R_0, the probability of a large outbreak and the relative final size of the epidemic. First we discuss the choice of π_ξ.

2.5 The Mean Infectious Activity

We *calibrate* the weighted and unweighted epidemics by setting

$$\pi_1\mu_1 + \pi_2\mu_2 = C = \pi\mu \tag{5}$$

in the weighted (left side) and the unweighted model (right side). C is a measure of the *the mean infectious activity* in the graph, as it is the expected number of secondary infections caused by a single infected vertex, *selected uniformly*, when all other vertices are susceptible. If we increase the mean infectious activity (without changing anything else) the epidemic spreads more easily, resulting in a larger R_0, an increased relative final size and an increased probability of a large outbreak. Remembering that in the unweighted model $D = D_1 + D_2$, we thus have

$$\pi = \frac{\pi_1\mu_1 + \pi_2\mu_2}{\mu_1 + \mu_1},$$

since $\mu = \mu_1 + \mu_2$.

If we divide *Eq. (5)* by C we obtain

$$r_1 + r_2 = 1,$$

where $r_\xi = \frac{\pi_\xi\mu_\xi}{C}$ determines how the mean infectious activity is distributed between the different edge types - we call it the *relative infectious activity*. We use the mean infectious activity together with the relative infectious activity in the theoretical results (*Section 3*) and in the numerical results (*Section 4*) where they allow for a consistent way of plotting the figures.

2.6 Theoretical and Empirical Distributions

To illustrate the weighted configuration model we numerically analyze some theoretical and empirical degree distributions. Two empirical bivariate degree distributions are modeled using the weighted configuration model, assigning different weights to the two edge types. To further increase the distributions that we can analyze we use copulas (see *Appendix B*) to map one dimensional (marginal) degree distributions into bivariate degree distributions. This allows us to vary the correlation between the degrees of the two edge types, given only two marginal degree distributions. We also compare

the copula model with a model based on the original empirical data. Below we briefly describe the different theoretical distributions and the empirical datasets.

2.6.1 Theoretical Distributions

- *A bivariate binomial distribution* where the marginal distributions are both binomial regardless of the correlation between D_1 and D_2 was defined in (Biswas & Hwang, 2002). For a more complete description see *Appendix A*. Here we only mention that it has five parameters, n_1, p_1, n_2, p_2 and ρ (the correlation). The mean and the variance of the marginal distributions are

$$\begin{aligned} \mathrm{E}(D_\xi) &= n_\xi p_\xi, & (6) \\ \mathrm{Var}(D_\xi) &= n_\xi p_\xi \left(1 - p_\xi\right), & (7) \end{aligned}$$

  for $\xi \in \{1, 2\}$. These can thus be varied separately, although $\mathrm{Var}(D_\xi)$ is restricted to the range $[0, \mathrm{E}(D_\xi)]$. The correlation can be varied within the full range $[-1, 1]$ only when $n_1 = n_2$ and $p_1 = p_2$. This distribution can also be used to approximate a bivariate Poisson distribution if $\{n_\xi\}$ are chosen to be large and $\{p_\xi\}$ are chosen to be small.

- *A heavy tailed distribution* that is based on typical empirical distributions, e.g. distributions that appear for various *preferential attachment* models. Such distributions often have a tail that goes as $k^{-\alpha}$, where α is a parameter that is often in the range $2 - 4$ for empirical networks. For low degrees the empirical distributions often do not decay as rapidly. We choose a distribution that approximates the described properties. Let

$$p_k = \frac{1}{c}(k + k_0)^{-\alpha},\ k = 0, 1, ...$$

  where c is a norming constant (the Hurwitz zeta function) and k_0 essentially determines the shape of the probability mass function for lower degrees.

2.6.2 Empirical Distributions

- *Sexual relationships* for a heterosexual population[1]. People have stated how many casual and how many stable sexual relationships they have

[1]Data kindly supplied by Veronika Fridlund, Department of Sociology, Stockholm University

had during the last year, see (Hansson et al., 2018) for details. The dataset analyzed here consists of 645 individuals. This information is treated as a bivariate edge distribution where the transmission probability of the two types of edges is allowed to differ. It is important to note that this is not a valid model for the actual sexual interactions within this group of people since the two sexes interact (mainly) with someone of the other sex, while in our model we treat all individuals as identical (apart from the bivariate degree). Also, this is not a random subset of the general population. Here we use the dataset only as an example of an empirical dataset. Letting 1 represent casual relationships and 2 represent stable relationships we have $\mu_1 \approx 1.42, \mu_2 \approx 1.70, \sigma_1 \approx 0.99, \sigma_2 \approx 1.73$ and $\rho \approx -0.0652$.

- *The Swedish population*[2] is a large network that is based on data containing only the workplace and family affiliation of people in Sweden, see (Holm et al., 2006) for details. Edges to family members and within workplaces are treated as different types. Some workplaces are very large and to reduce computational workload and make the model somewhat more realistic people are randomly assigned to work groups within each workplace. Letting 1 represent family edges and 2 represent company edges we have $\mu_1 \approx 2.00, \mu_2 \approx 20.98, \sigma_1 \approx 1.44, \sigma_2 \approx 27.8$ and $\rho \approx -0.241$.

In both cases people completely without edges have been excluded. This needs to be remembered when comparing different models, since in some models vertices without edges are included.

2.6.3 Network Models using Copulas

Given two marginal distributions a bivariate distribution can be obtained by using a copula, see e.g. (Nelsen, 2007). The property of the copula is to maintain the marginal distributions, while varying how the variables depend on each other. Within this constraint, different copulas are possible and these will result in different properties for the resulting bivariate distribution. For this paper we choose to use a copula based on the bivariate standard normal distribution. It is simple to simulate and it allows for modeling the correlation between the degrees of the two edge types through a large range. We obtain the bivariate distribution function

$$F(i,j) \quad = \quad C_\rho(F_1(i), F_2(j)),$$

[2]Data kindly supplied by Fredrik Liljeros, Department of Sociology, Stockholm University

where $C_\rho(\cdot,\cdot)$ denotes the bivariate standard normal copula with correlation ρ and $F_\xi(\cdot)$ represents the marginal distribution function for edge type ξ. The marginal distributions for each edge type can be taken from empirical networks or from theoretical distributions. In this paper we do both. Note that the correlation derived from $F(i,j)$ will depend not only on the copula, but also on the marginal distributions and will thus typically be different from the ρ that is used in the equation above. More information on copulas can be found in *Appendix B*.

3 Theoretical Results

3.1 Basic Reproduction Number

In the configuration model the expected degree of a vertex that is reached by following an edge from an infected vertex is determined by the size-biased distribution (see *Section 2.3* and *Eq. (2)*). When looking at the number of edges that can spread an infection in a mostly susceptible population we must deduct one edge, since the infection cannot spread back along the infecting edge, and must also multiply by the probability that an edge infects a susceptible vertex.

3.1.1 Unweighted Model

In the unweighted model we obtain

$$R_0 = \pi\left(\widetilde{\mu}-1\right) = \pi\left(\mu+\frac{\sigma^2}{\mu}-1\right) = C\left(1+\left(\frac{\sigma}{\mu}\right)^2-\frac{1}{\mu}\right),$$

remembering that the mean infectious activity $C = \pi\mu$ in the final step. We want to compare the unweighted model with the weighted model, where simultaneous degree distribution $\mathbf{D} = (D_1, D_2)$ is given, and set $D = D_1+D_2$, counting only the total number of edges, regardless of type. Using results from *Section 2.5* we obtain:

$$R_0 \;=\; C\left(1+\frac{\sigma_1^2+\sigma_2^2+2\rho\sigma_1\sigma_2}{(\mu_1+\mu_2)^2}-\frac{1}{\mu_1+\mu_2}\right). \tag{8}$$

This is strictly increasing in ρ (all other parameters being fixed) and obtains its minimum and maximum values when ρ obtains its minimum and maximum values, respectively. The allowed range of ρ is obtained from the weighted model, see below.

A special case is when $\mu_\xi = \sigma_\xi^2$ (such as for the Poisson distribution) and then

$$R_0 \;=\; C\left(1 + 2\rho\frac{\sigma_1\sigma_2}{\sigma_1^2+\sigma_2^2}\right).$$

If, in addition, $\rho = 0$ we obtain $R_0 = C$.

3.1.2 Weighted Model

In the weighted model we need to take into account which type of edge that infected the vertex since the size-biased distribution depends on this. We must remove one edge *of the type that infected the vertex*, since a vertex cannot reinfect its infector. This results in the *next generation matrix*

$$\mathbf{K} \;=\; \begin{pmatrix} \pi_1\left(\widetilde{\mu}_{1|1}-1\right) & \pi_2\,\widetilde{\mu}_{2|1} \\ \pi_1\,\widetilde{\mu}_{1|2} & \pi_2\left(\widetilde{\mu}_{2|2}-1\right) \end{pmatrix}$$

(see also *Section 2.3* for definitions). This matrix consists of the expected number of infecting edges of each type (column 1 and column 2) in the next generation when a vertex is infected through an edge of type 1 (row 1) and type 2 (row 2). R_0 is given by the largest eigenvalue of this matrix. The eigenvalue λ is obtained as a solution to the characteristic equation

$$\det(\mathbf{K}-\lambda\mathbf{I}) \;=\; 0.$$

The solution can be written in relatively compact form using some definitions from *Section 2.5* and some additional definitions, including the coefficient of variation $\mathcal{CV}_\xi$:

$$\begin{aligned} C &= \pi_1\mu_1 + \pi_2\mu_2, \\ r_\xi &= \frac{\pi_\xi\mu_\xi}{C}, \\ \mathcal{CV}_\xi &= \frac{\sigma_\xi}{\mu_\xi}, \\ \nu_\xi &= \mathcal{CV}_\xi{}^2 - \frac{1}{\mu_\xi} \end{aligned}$$

remembering that $\xi \in \{1,2\}$ indicates the edge type. The full solution is

$$\begin{aligned} R_0 \;=\; \frac{C}{2}\Bigg(&1+r_1\nu_1+r_2\nu_2 + \\ &\sqrt{\left(1+r_1\nu_1+r_2\nu_2\right)^2 + 4r_1r_2\left(\left(\rho\,\mathcal{CV}_1\,\mathcal{CV}_2+1\right)^2 - \left(\nu_1+1\right)\left(\nu_2+1\right)\right)}\Bigg). \end{aligned} \tag{9}$$

The allowed range of ρ depends on the marginal distributions of the degrees of the two edge types as well as on the correlation structure between them. An important observation is that R_0 increases when ρ increases. Thus the minimum and maximum must be obtained for the minimum and maximum value of ρ, respectively.

The full solution simplifies in some cases, e.g. if $\mu_\xi = \sigma_\xi^2$ (such as for the Poisson distribution). Then $\nu_\xi = 0$ and we have

$$R_0 = \frac{C}{2}\left(1 + \sqrt{1 + 4r_1 r_2\left((\rho\, \mathcal{CV}_1\, \mathcal{CV}_2 + 1)^2 - 1\right)},\right)$$

If we, in addition, require that $\rho = 0$ this gives that $R_0 = C$, just as for the unweighted model. An *example* is when $\{D_\xi\}$ are *independent* Poisson distributed variables.

3.2 Probability of a Large Outbreak

In the configuration model (in the limit of an infinite population) the probability of a large outbreak can be calculated as the probability of survival of a Galton-Watson branching process where the offspring is the number of new infected vertices in each generation, see e.g. (Britton et al., 2007) and also *Appendix C*. In our application infection starts with the index case and then spreads through one type of edge (the unweighted configuration model) or two types of edges (the weighted configuration model). In the weighted model the degree distribution of a newly infected vertex depends on the type of edge through which the vertex was infected. The distribution of the number of vertices that are actually infected is different in discrete time and in continuous time (both in the unweighted and the weighted models). In discrete time each edge from an infected vertex *independently* infects a new vertex with probability π or π_ξ (depending on model), while in the continuous time Markovian model edges are *dependent* since the infectious period of a vertex applies to all edges simultaneously. We thus need to treat discrete and continuous time slightly differently. The difference, in some sense, is subtle since it only affects a mapping function that we introduce. Other models than the discrete time and continuous models that we treat here can be dealt with by just changing the mapping function.

3.2.1 Unweighted Model

Let $\{p_i^*\}$ define the distribution of the number of *infecting* edges (edges that do spread the infection) of the index case. Let $\{\widetilde{p}_i^*\}$ define the distribution

of the number of infecting edges of a vertex infected after the index case (corresponding to following an edge in the configuration model, see *Section 2.2*). Define the probability generating functions

$$\begin{aligned} f^*(s) &= \sum_{i=0}^{\infty} s^i p_i^* \\ \widetilde{f}^*(s) &= \sum_{i=0}^{\infty} s^i \widetilde{p}_i^* \end{aligned}$$

The probability q that the branching process dies out, given that we start with an infecting *edge*, is the solution to the fixed-point equation

$$q = \widetilde{f}^*(q),$$

as discussed in *Appendix C*. We then apply this solution to each edge of the index case, giving the probability τ of a large outbreak (the probability that the process does not die out)

$$\tau = 1 - f^*(q).$$

What remains is to obtain expressions for the probability mass functions p_i^* and $\widetilde{p}_i^*$. These can be derived from the distributions p_i and $\widetilde{p}_i$ (see *Section 2.2* and *Section 2.3*). Study a given infected vertex that has exactly k edges that could spread the infection. Then the actual number of edges that *do* spread the infection is between 0 and k. Let $\phi(i \mid k)$ denote the probability that exactly i edges out of the k available spread the infection. Clearly $\phi(i \mid k)$ depends on the model for spreading the infection - e.g. we can expect it to be different in discrete time and in continuous time. However, if $\phi(i \mid k)$ is known we can easily obtain p_i^* and $\widetilde{p}_i^*$ through

$$p_i^* = \sum_{k=i}^{\infty} \phi(i \mid k) p_k \text{ and} \tag{10}$$

$$\widetilde{p}_i^* = \sum_{k=i}^{\infty} \phi(i \mid k) \widetilde{p}_{k+1}, \tag{11}$$

remembering that $k+1$ is required for the *size-biased* distribution since the infection cannot spread back on the edge that infected the current vertex.

In discrete time infecting edges are independent and, given the probability π that an edge spreads an infection, the number of edges that spread the infection is distributed as $\mathrm{Bin}(k, \pi)$ so that

$$\phi(i \mid k) = \binom{k}{i} \pi^i (1-\pi)^{k-i}.$$

When this result is inserted into *Eq. (10)* and *Eq. (11)* typically further simplification is not possible except in some special cases, like when D is Poisson or binomially distributed.

In continuous time the derivation of $\widetilde{p}_i^*$ is slightly more complicated. If we fix the duration of the infectious period $T{=}t$, the edges become independent and each edge infects another vertex with probability $\pi(t) = 1{-}e^{-\beta t}$, independently of the other edges. Let D^* denote the number of edges that pass on the infection. Then (when $k \geq i$)

$$\begin{aligned}
\mathbb{P}\left(D^*{=}i \mid k, T=t\right) &= \binom{k}{i}\pi(t)^i(1{-}\pi(t))^{k-i}, \text{ and so} \\
\phi(i \mid k) &= \int_0^\infty \binom{k}{i}\pi(t)^i(1{-}\pi(t))^{k-i} f_T(t)\mathrm{d}t \\
&= \int_0^\infty \binom{k}{i}(1{-}e^{-\beta t})^i(e^{-\beta t})^{k-i}\gamma e^{-\gamma t}\mathrm{d}t
\end{aligned}$$

When this result is inserted into *Eq. (10)* and *Eq. (11)* typically further simplification is not possible.

3.2.2 Weighted Model

Results for the weighted model follow the same method as for the unweighted model, taking into account the two different edge types. Let $p^*(i,j)$ define the distribution of *infecting* edges of the index case. Let $\widetilde{p}_\xi^*(i,j)$ define the distribution of the number of infecting edges of a vertex that were infected after the index case (through a type $\xi \in \{1,2\}$ edge, see *Section 2.3*). Let $\mathbf{s} = (s_1, s_2)$ and define the probability generating functions

$$\begin{aligned}
f^*(\mathbf{s}) &= \sum_{i=0}^\infty \sum_{j=0}^\infty s_1^i s_2^j p^*(i,j) \\
\widetilde{f}_\xi^*(\mathbf{s}) &= \sum_{i=0}^\infty \sum_{j=0}^\infty s_1^i s_2^j \widetilde{p}_\xi^*(i,j)
\end{aligned}$$

The probabilities $\{q_\xi\}$ that the branching process dies out, *given that we start with an infecting edge* of type $\xi \in \{1,2\}$, are given by the solution to the fixed-point equation

$$\mathbf{q} = (\widetilde{f}_1^*(\mathbf{q}), \widetilde{f}_2^*(\mathbf{q})),$$

where $\mathbf{q} = (q_1, q_2)$, as discussed in *Appendix C*. Just as in the unweighted case, we then apply this solution to each edge of the index case, giving the

probability τ of a large outbreak (the probability that the process does not die out)

$$\tau \;=\; 1 - f^*(\mathbf{q}).$$

The probability mass functions $p^*(i,j)$ and $\widetilde{p}^*_\xi(i,j)$ can be derived from the distributions p_{ij} and $\widetilde{p}_{ij}$ (see *Section 2.2* and *Section 2.3*). Study a given infected vertex that has k edges of type 1 and l edges of type 2 that could spread the infection. Let $\phi(i,j \mid k,l)$ denote the probability that (i,j) edges out of the (k,l) available spread the infection. Then

$$p^*(i,j) \;=\; \sum_{k=i}^{\infty}\sum_{l=j}^{\infty} \phi(i,j \mid k,l) p_{k,l}, \tag{12}$$

$$\widetilde{p}^*_1(i,j) \;=\; \sum_{k=i}^{\infty}\sum_{l=j}^{\infty} \phi(i,j \mid k,l) \widetilde{p}_{k+1,l} \text{ and} \tag{13}$$

$$\widetilde{p}^*_2(i,j) \;=\; \sum_{k=i}^{\infty}\sum_{l=j}^{\infty} \phi(i,j \mid k,l) \widetilde{p}_{k,l+1}, \tag{14}$$

again remembering that the infection cannot spread back on the edge that infected the current vertex.

In discrete time edges are independent and if the probability than an edge spreads an infection is π_ξ then the number of edges that spread the infection are distributed independently as $\mathrm{Bin}(k,\pi_1)$ and $\mathrm{Bin}(l,\pi_2)$ so that

$$\phi(i,j \mid k,l) \;=\; \binom{k}{i}\pi_1^i(1-\pi_1)^{k-i}\binom{l}{j}\pi_2^j(1-\pi_2)^{l-j}.$$

When this result is inserted into *Eq. (12)*, *Eq. (13)* and *Eq. (14)* then typically further simplification is not possible except in some special cases, e.g. when D_ξ are independently Poisson distributed.

In continuous time, given that the duration of the infectious period $T = t$, each edge infects another vertex with probability $\pi_\xi(t) = 1 - e^{-\beta_\xi t}$, independently of the other edges. Let D^*_ξ denote the number of edges of type ξ that pass the infection on. Then (when $k \geq i$ and $l \geq j$)

$$\mathbb{P}\left(D^*_1{=}i, D^*_2{=}j \mid k,l,T{=}t\right) = \binom{k}{i}\pi_1(t)^i(1{-}\pi_1(t))^{k-i}\binom{l}{j}\pi_2(t)^j(1{-}\pi_2(t))^{l-j},$$

and so

$$\begin{aligned}\phi(i,j \mid k,l) \;&=\; \int_0^\infty \binom{k}{i}\pi_1(t)^i(1{-}\pi_1(t))^{k-i}\binom{l}{j}\pi_2(t)^j(1{-}\pi_2(t))^{l-j} f_T(t)\mathrm{d}t \\ &=\; \int_0^\infty \binom{k}{i}\left(1{-}e^{-\beta_1 t}\right)^i\left(e^{-\beta_1 t}\right)^{k-i}\binom{l}{j}\left(1{-}e^{-\beta_2 t}\right)^j\left(e^{-\beta_2 t}\right)^{l-j}\gamma e^{-\gamma t}\mathrm{d}t\end{aligned}$$

When this is inserted into *Eq. (12)*, *Eq. (13)* and *Eq. (14)* typically further simplification is not possible.

3.3 Final Size of a Large Outbreak

In discrete time (when edges are undirected and independent) the relative final size of the epidemic is equal to the probability of a large outbreak, see (Britton et al., 2007). This has already been calculated in *Section 3.2*.

In continuous time edges are dependent and so further analysis is needed. Instead of studying how the epidemic develops forward in time (starting with the index case), we instead select a vertex uniformly at random and study which vertices that would infect it if they were infected. We continue this process (in the limit of an infinite population) by following edges backwards in time to create the *susceptibility set*, see (Ball & Neal, 2008). In the configuration model this once again corresponds to a Galton-Watson branching process and edges (by which a vertex may have been infected) are independent since they are attached to different vertices which are independent. The probability that the branching process (that creates the susceptibility set) *survives* is equal to the relative final size of the epidemic. The probability of infection is the same as when calculating the probability of a large outbreak in the discrete case in *Section 3.2* and so the size of the outbreak in continuous time is equal to the probability of a large outbreak in discrete time! Note that, in continuous time, the *relative final size of the epidemic* and the *probability of a large outbreak* are in general not equal.

4 Numerical Results

4.1 Sexual Network

Figure 1 shows R_0 and the relative final size of the epidemic (for fixed C) for the sexual relationship network modeled using the bivariate standard normal copula so that ρ can be varied. In the figure the correlation is plotted on the x-axis and r_2 is plotted on the y-axis. Low or high values of r_2 indicate that the infection is spread mainly by type 1 or type 2 edges, respectively. The horizontal red line indicates when the probability of infection is the same for type 1 and type 2 edges. From the plot we see that both R_0 and the final size depend greatly on the correlation between the degrees *and* on the balance between the probability of infection on type 1 and type 2 types.

While R_0 must always increase with ρ (as can be seen from *Eq. (8)* and *Eq. (9)*) this is not the case for the relative final size. When $C = 1.6$ we see

that the relative final size achieves a maximum *inside* the plotted region both for the weighted and for the unweighted model. To the right of the maximum an increase of the correlation results in a decrease in the relative final size. From the plot we note that changing the balance between type 1 and type 2 edges clearly affects the epidemics that develop on the graph. When the balance is strongly shifted towards type 1 edges, large outbreaks tend to be small (or not occur at all). When the balance is strongly shifted towards type 2 edges epidemics are typically large. Results for the probability of a large outbreak are similar (not shown here). Such plots thus give information on which type of edge (which type of relationship) that should be targeted in order to best best reduce the probability and the size of large outbreaks. Clearly, the unweighted model does not provide this information.

We also compare the probability mass function generated using the standard normal copula with the empirical probability mass function. We do this for $C = 1$ and fixed correlation, and vary only the balance between the degrees of type 1 and type 2 edges. Results in *Figure 2* show a very close correspondence between empirical results and copula generated results. Note that in the left figure, depicting R_0, the curves match perfectly, which they must. Somewhat more interesting is that the figures for the relative final size (center) and the probability of a large outbreak in continuous time (right) also match very well.

4.2 Sweden Network

Figure 3 shows R_0 and the relative final size of the epidemic for the Swedish population network modeled using the bivariate standard normal copula so that ρ can be varied. The result depends greatly on both the correlation and on the balance between the degrees of type 1 and type 2 edges. We do not see the same variation when looking at the unweighted plots. As mentioned before, R_0 always increases with increasing ρ (see *Section 3.1*). However, from the plot where $C = 1.3$ we observe that as ρ increases the relative final size decreases in the weighted model for some values of the balance parameter (e.g. follow the 0.5 balance line towards the right side of the figure). The unweighted model shows a small, but steady decrease through the range of ρ.

We again compare results for the copula generated probability mass function with the empirical probability mass function. We do this for $C = 1$ and fixed correlation, and vary only the balance between the degrees of type 1 and type 2 edges. Results in *Figure 4* show a good correspondence between empirical results and copula generated results, just as was the case for the sexual contact network (*Figure 1*). We also expect that the difference may be

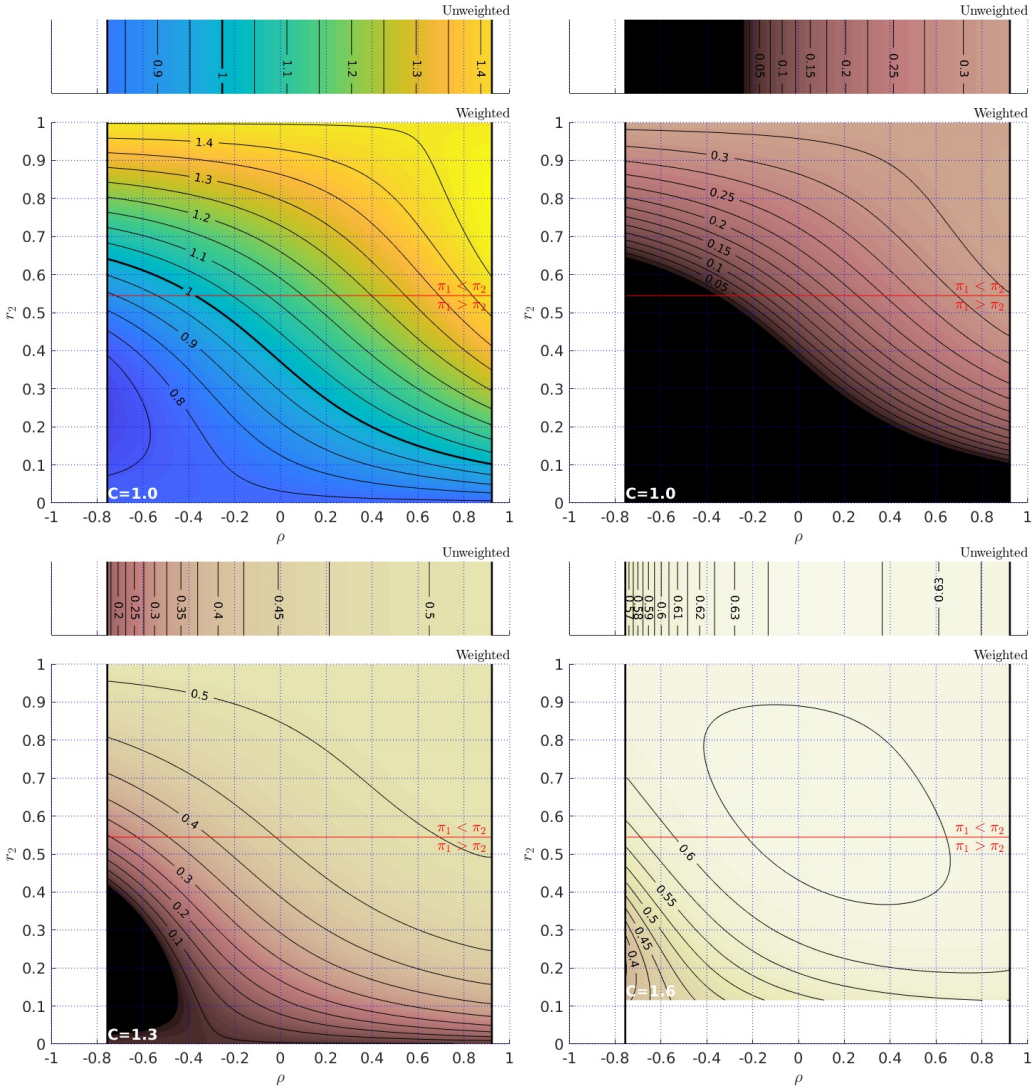

Figure 1: The figure shows contour plots of R_0 (upper left) and the relative final size (the remaining three) for different values of C for the sexual relationship network modeled using the bivariate standard normal copula. Each plot consists of a larger (lower) area corresponding to the weighted model and a smaller (upper) area corresponding to the unweighted model. The horizontal axis represents the correlation and the vertical axis represents the balance between the degrees of type 1 and type 2 edges (only in the weighted model). Note that R_0-results for other values of C can be obtained simply by multiplying the values in the R_0-plot with C, since this plot was based on $C = 1$ and R_0 is directly proportional to C.

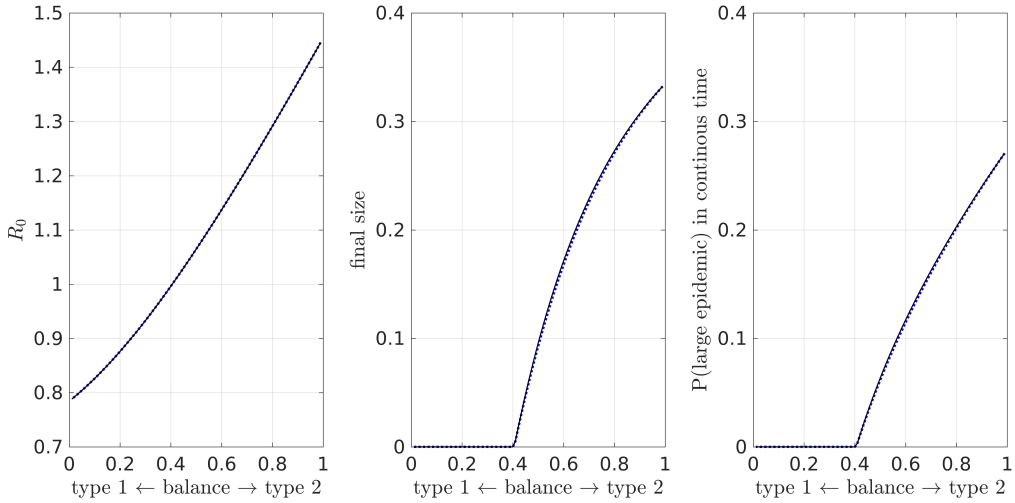

Figure 2: For the sexual network the figure shows a comparison between results from the empirical probability mass function (blue dotted lines) and results from the standard normal copula applied to the marginal distributions (solid black lines). All curves use the same correlation coefficient $\rho \approx -0.0652$ (from the empirical distribution) and $C = 1$. Note that all three curves depict two curves, but because they are so similar they appear almost as one curve. In the left figure the curves do coincide exactly.

larger if C is higher, since then more of the original probability mass function is preserved. We test this by setting $C = 1.3$. Results in *Figure 5* indeed show a slight difference in the relative final size and in the probability of a large outbreak.

4.3 Heavy Tail Network

Figure 6 shows R_0 and the relative final size of the epidemic for a heavy-tailed degree distribution (see *Section 2.6*) modeled using the bivariate standard normal copula so that ρ can be varied. For type 1 edges the parameters $\alpha = 10$ and $k_0 = 20$ were used and the distribution was truncated at 25 edges (allowing only degrees between 0 and 25 to have positive probability). For type 2 edges the parameters $\alpha = 4$ and $k_0 = 20$ were used and the distribution was truncated at 200 edges (allowing only degrees between 0 and 200 to have positive probability). Truncating the distributions is necessary to be able to perform the simulation within reasonable time.

4.4 Binomial Network

Figure 7 shows R_0, the relative final size and the probability of a large outbreak for a bivariate binomial distribution as described in *Section 2.6* and also

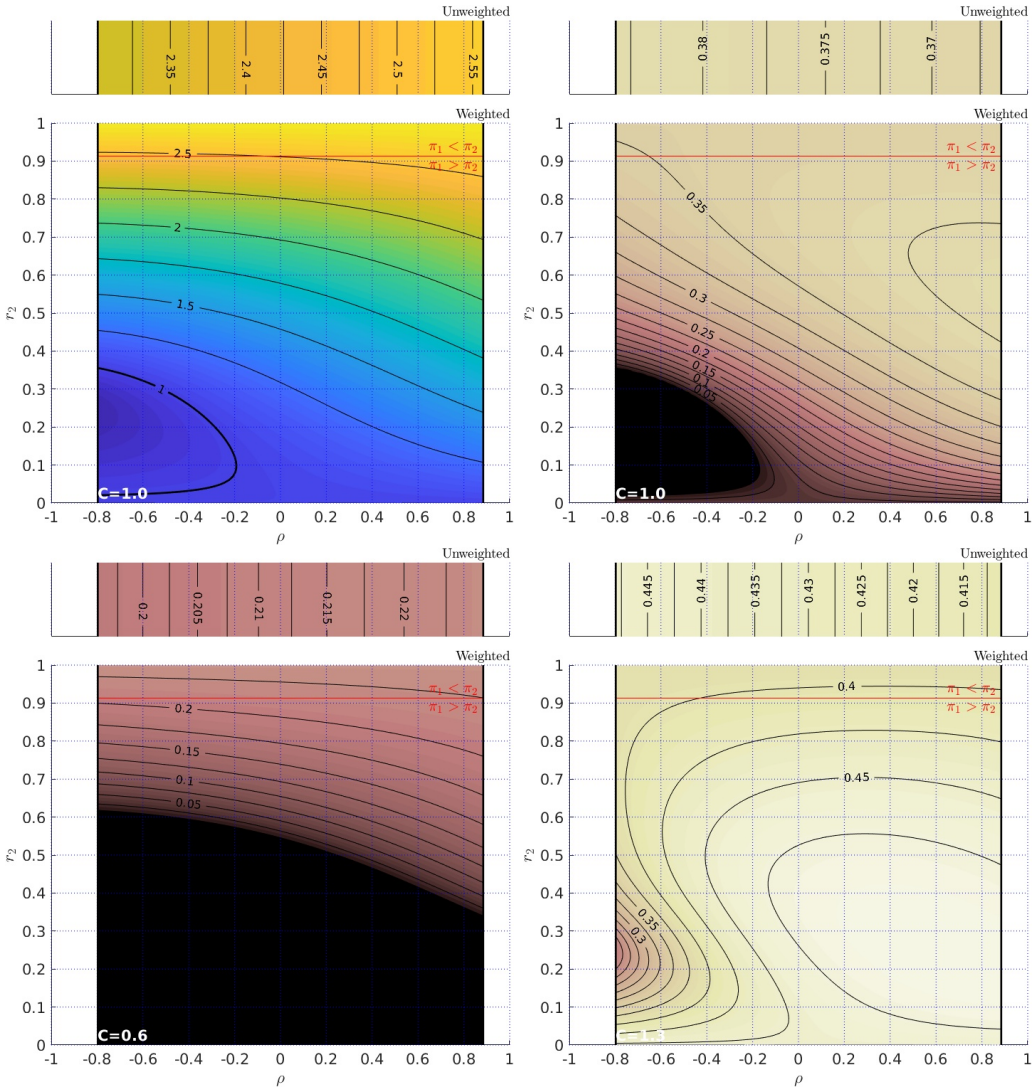

Figure 3: The figure shows contour plots of R_0 (upper left) and the relative final size (the remaining three) for different values of C for the Swedish population network modeled using the bivariate standard normal copula. For additional information see *Figure 1*.

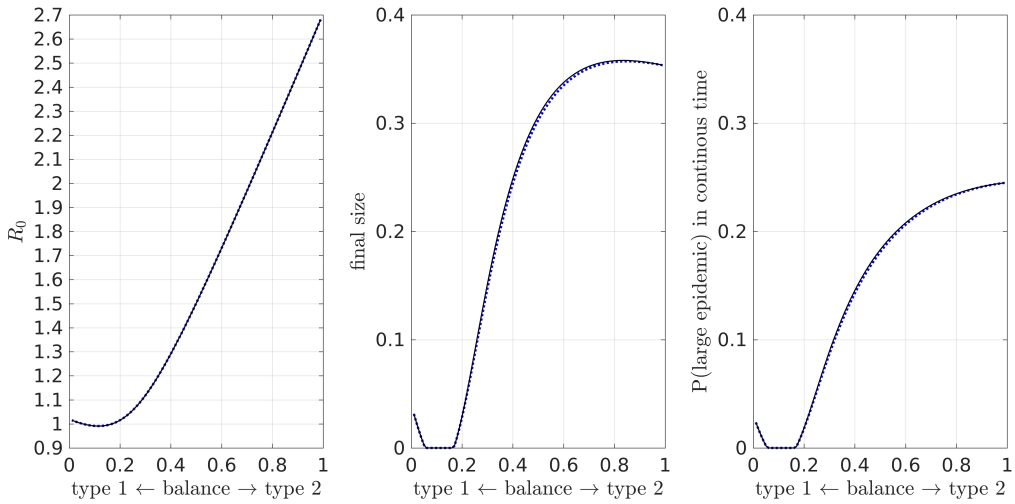

Figure 4: For the Swedish population network the figure shows a comparison between results from the empirical probability mass function (blue dotted lines) and results from the standard normal copula applied to the marginal distributions (solid black lines). All curves use the same correlation coefficient $\rho \approx -0.241$ (from the empirical distribution) and $C = 1$.

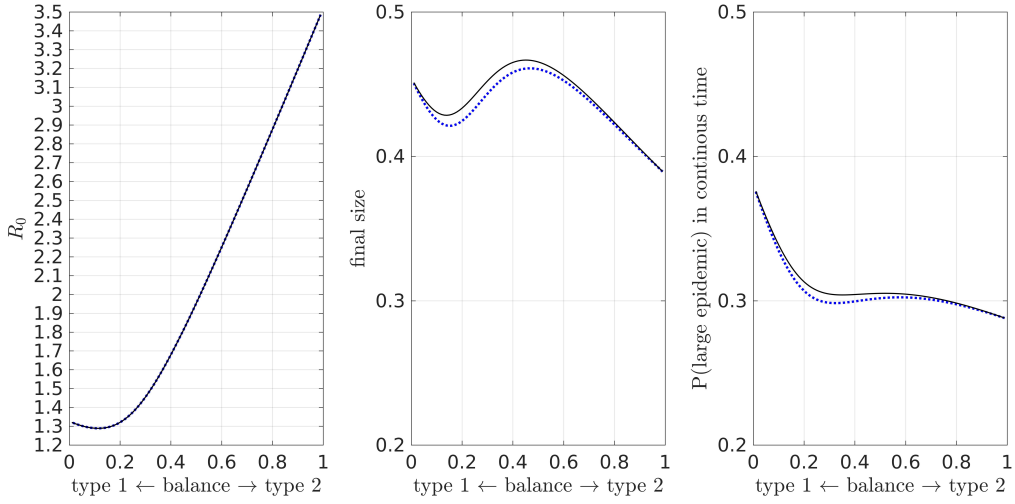

Figure 5: The same plot as in *Figure 4*, except for setting $C = 1.3$. Some differences can be seen for this higher value of C.

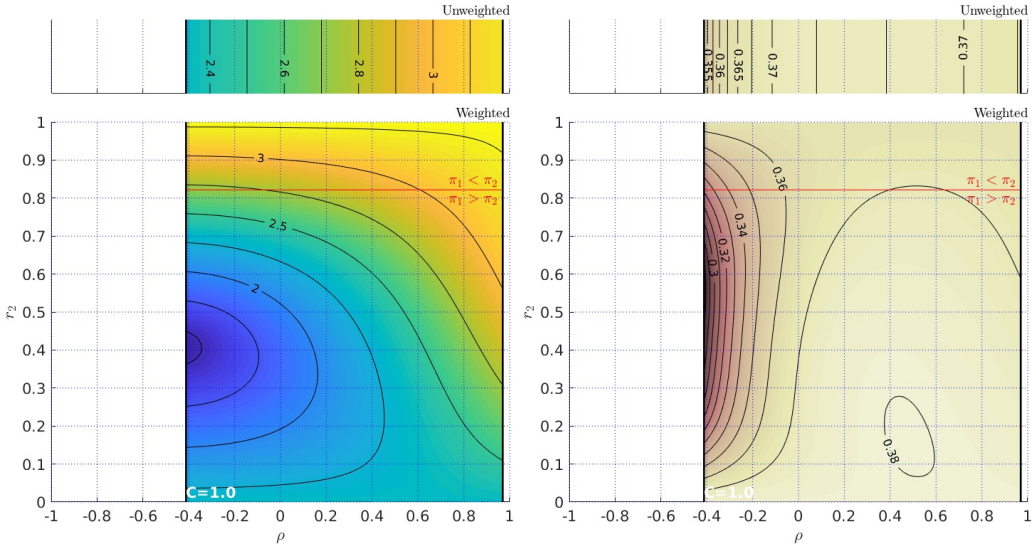

Figure 6: The figure shows contour plots of R_0 (upper left) and relative final size for the heavy tailed network modeled using the bivariate standard normal copula.

compared with a copula model. The parameters were $n_1 = 5$ and $p_1 = 0.5$ and $n_1 = 20$ and $p_1 = 0.5$ for edge type 1 and 2, respectively. The copula model uses the two marginal distributions and models the correlation through the bivariate normal copula. The three left figures show the bivariate binomial model and the three right figures show the copula model. We note that the models produce almost identical results in the region where both are defined, but that the copula model allows for a wider range of ρ.

5 Discussion

We have modeled weighted and unweighted configuration model networks based on different empirical and theoretical distributions, and have studied epidemics taking place on these networks. We show that the weighted network model produces much richer results in terms of the variation of R_0, the probability of a large outbreak and the relative final size of an epidemic as functions of ρ and the balance between the edge types.

We have used a parametrization that separates

(a) the mean infectious activity in the network,

(b) how the activity is distributed between the different edge types and

(c) the correlation between the degrees of the edge types.

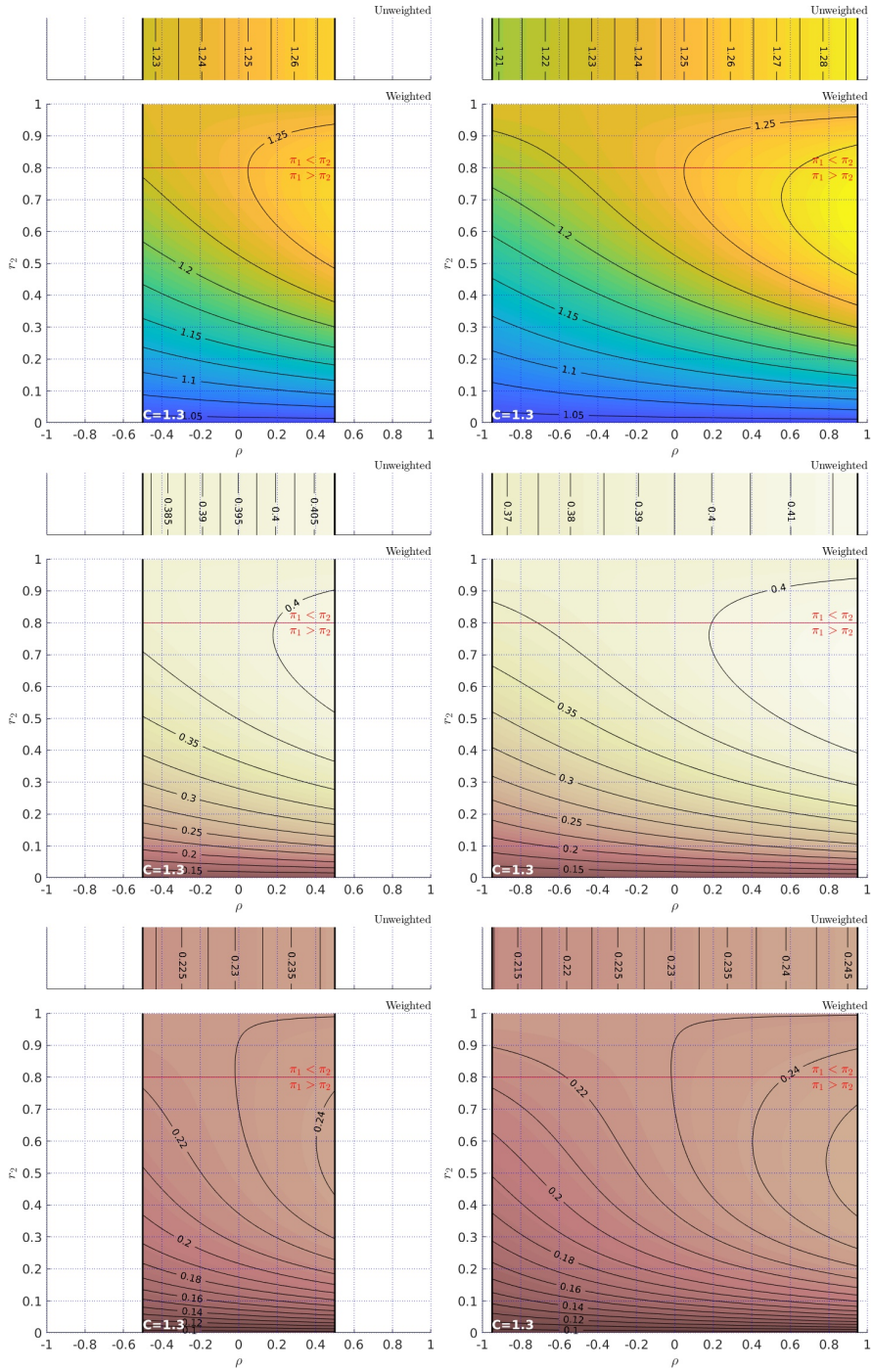

Figure 7: The figure shows contour plots of R_0 (upper), the relative final size (middle) and the probability of a large outbreak in continuous time (lower) for the bivariate binomial model (left) and for the same marginal distributions modeled through the bivariate normal copula (right).

This parametrization simplifies visualization of results. This is especially evident for R_0 plotted for $C = 1$ as R_0 can be obtained for other values of C simply by multiplying the values in the plot by C. The plot gives immediate information as to which combination of parameters that can produce large outbreaks. The data needed can be obtained by only studying the so called egocentric degree distribution of individuals sampled from the population together with estimates of the activity on different types of edges. Modeling of the network is then done through the configuration model.

The introduction of copulas allows for modeling situations outside the range of the empirical data. As an example we can use the sexual network analyzed in *Section 4.1* for which we have a single sample with fixed correlation. By applying a bivariate standard normal copula to the marginal edge distributions we are able to model the network for other values of the correlation ρ. Results indicate that the copula model can produce results that are almost identical to those generated by the original network data model (when setting the same correlation in both models). Thus, if the correlation and the marginal distributions are known, the exact dependence between the degrees of the different edge types may not always be so important. The copula approach also gives some insight to what is possible for a specific set of marginal distributions. For instance, the minimum and the maximum copulas can be used to calculate which minimum and maximum correlation that is possible for the given marginal distributions.

The disadvantage with the described modeling approach is that it is fairly computer intensive, requiring some care when writing the software used to calculate parameters from the model. Expanding the model to more than two edge types may require some simplifications of the model. Such simplifications may indeed be possible when the infection probabilities π_ξ are low so that the exact dependence structure in the empirical data is less important.

Future work could include an investigation of what simplifications of the model that are possible, without affecting results significantly, and also how well the model matches real world networks. E.g. if the relative final size of a large outbreak on a *finite* empirical network is comparable to the *asymptotic* relative final size in the configuration model.

Acknowledgments

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A The Bivariate Binomial Distribution

This bivariate binomial distribution was defined in (Biswas & Hwang, 2002). The distribution has five parameters, where the first four (n_1, p_1, n_2 and p_2) define the two marginal binomial distributions and the last parameter α is directly related to the correlation coefficient ρ.

The distribution is defined so that $D_\xi = \sum_{i=1}^{n_\xi} X_{\xi,i}$, for $\xi \in \{1, 2\}$. $X_{1,i}$ are independent Bernoulli distributed variables with parameter p_1. $X_{2,i}$ are independent Bernoulli distributed variables, each with parameter

$$p_{2,i} = \begin{cases} \frac{p_2+\alpha(p_2-p_1)+\alpha X_{1,i}}{1+\alpha}, & \text{if } i \leq n_1, \\ p_2 & \text{if } i > n_1 \end{cases}$$

Thus, D_2 depends on D_1, but because of the specific choice of parameters, D_2 is still distributed as $\text{Bin}(n_2, p_2)$. For the analytical form of the simultaneous probability mass function we refer the reader to (Biswas & Hwang, 2002). Because the marginals are Binomially distributed

$$\begin{aligned} \mu_\xi &= \text{E}(D_\xi) = n_\xi p_\xi, \\ \sigma_\xi^2 &= \text{Var}(D_\xi) = n_\xi p_\xi (1 - p_\xi), \end{aligned}$$

The correlation ρ can be both positive and negative and α is closely related to it through

$$\rho = \sqrt{\frac{\min(n_1, n_2)}{\max(n_1, n_2)}} \left(\frac{\alpha}{1+\alpha}\right) \sqrt{\frac{p_1(1-p_1)}{p_2(1-p_2)}}.$$

The allowed range for the correlation coefficient is a function of the first four parameters. This range is not correctly given in (Biswas & Hwang, 2002) so we give it here (there are several cases). We assume that $p_\xi > 0$ and $n_\xi > 0$ for $\xi \in \{1, 2\}$ to avoid a degenerate distribution.
The lower limit:

$$p_1 + p_2 < 1 \implies \begin{cases} \alpha \geq -\frac{p_2}{1+p_2-p1} \\ \rho \geq -\sqrt{\frac{\min(n_1,n_2)}{\max(n_1,n_2)}} \sqrt{\frac{p_1}{1-p_1} \cdot \frac{p_2}{1-p_2}} \end{cases},$$

$$p_1 + p_2 \geq 1 \implies \begin{cases} \alpha \geq -\frac{1-p_2}{1+p_1-p_2} \\ \rho \geq -\sqrt{\frac{\min(n_1,n_2)}{\max(n_1,n_2)}} \sqrt{\frac{1-p_1}{p_1} \cdot \frac{1-p_2}{p_2}} \end{cases}.$$

The upper limit:

$$
\begin{aligned}
p_1 < p_2 \quad &\Longrightarrow \quad \begin{cases} \alpha \leq \frac{1-p_2}{p_2-p_1} \\ \rho \leq \sqrt{\frac{\min(n_1,n_2)}{\max(n_1,n_2)}} \sqrt{\frac{p_1}{1-p_1} \cdot \frac{1-p_2}{p_2}} \end{cases} , \\
p_1 > p_2 \quad &\Longrightarrow \quad \begin{cases} \alpha \leq \frac{p_2}{p_1-p_2} \\ \rho \leq \sqrt{\frac{\min(n_1,n_2)}{\max(n_1,n_2)}} \sqrt{\frac{1-p_1}{p_1} \cdot \frac{p_2}{1-p_2}} \end{cases} , \\
p_1 = p_2 \quad &\Longrightarrow \quad \begin{cases} \alpha \text{ no upper limit} \\ \rho \leq \sqrt{\frac{\min(n_1,n_2)}{\max(n_1,n_2)}} \end{cases} .
\end{aligned}
$$

Only when $n_1{=}n_2$ and $p_1{=}p_2$ can ρ take on any value in the range $[-1, 1]$.

B Modeling Distributions through Copulas

Copulas define the correlation structure between random variables with given marginal distributions. They can be defined for any number of variables, but we limit the scope to bivariate distributions. Let X and Y be two random variables with given (marginal) distributions $F_X(x)$ and $F_Y(y)$. We define the simultaneous distribution $F_{X,Y}(x, y) = \mathbb{P}(X \leq x, Y \leq y)$ through a copula $C(u, v)$ as

$$F_{X,Y}(x, y) \quad = \quad C(F_X(x), F_Y(y)).$$

The function $C(u, v)$ can be viewed as the simultaneous distribution function for two uniformly distributed random variables on [0,1]. The *Fréchet-Hoeffding Bounds* (see (Nelsen, 2007), page 9) are

$$M(u, v) \leq C(u, v) \leq W(u, v) \; \forall \, u, v, \tag{15}$$

where

$$
\begin{aligned}
M(u, v) \quad &= \quad \min(u, v), \\
W(u, v) \quad &= \quad \max(u{+}v{-}1, 0).
\end{aligned}
$$

In our application we are mainly interested in using the copula to vary the correlation between the variables, while maintaining the marginal distributions. We thus limit our study to copulas that are uniquely defined by the correlation ρ and indicate this by writing $C_\rho(u, v)$.

Noting that

$$\rho \quad = \quad \frac{\mathrm{Cov}(X,Y)}{\sqrt{\mathrm{Var}(X)\mathrm{Var}(Y)}},$$

using *Hoeffding's Identity* (e.g. see (Nelsen, 2007), page 154)

$$\mathrm{Cov}(X,Y) \quad = \quad \int_{-\infty}^{+\infty}\int_{-\infty}^{+\infty} (F_{X,Y}(x,y) - F_X(x)F_Y(y))\ \mathrm{d}x\ \mathrm{d}y$$

and using *Eq. (15)* we see that $\mathrm{Cov}(X,Y)$ has bounds that can be found by applying $W(u,v)$ (for the lower bound) and $M(u,v)$ (for the upper bound). We use these bounds when calculating and presenting results in *Section 4*. At the bounds the copulas are unique and results obtained by using the copulas are also unique. However, between the two extremes the copula that results in a specific correlation coefficient is not unique and thus results obtained from the simultaneous distribution function is also not necessarily unique.

In our application we want ρ to span as large a range as possible. Not all copulas are able to span the maximum range, but one that is is the bivariate normal copula. It is defined as

$$C_\rho(u,v) \quad = \quad N_\rho(\Phi^{-1}(u), \Phi^{-1}(v)),$$

where $\Phi(x)$ and $N_\rho(x,y)$ are the univariate and the bivariate standard normal distributions, respectively.

In our application we work with empirical bivariate degree distributions. From these we can extract all parameters for the distribution, such as means, variances and the correlation, but we are not able to vary the correlation coefficient. However, taking the marginal distributions and applying a copula, such as the standard normal copula, enables us to vary the correlation coefficient.

As is mentioned above the choice of copula will affect the results (except at the boundaries of the maximum allowed range for ρ), so care is needed when drawing conclusions based on results from the use of (arbitrary) copulas.

C Branching Processes

Here we only give a brief overview of the part of the theory that we need for this paper. For a more thorough treatment of the theory of branching processes subject we refer the reader to (Harris, 2002).

Early on in the epidemic the growth of the number of infected vertices can be modeled through a branching process. In our application the branching process starts with a single infected individual, the index case, and we study how the number of new infected vertices develops in each generation. Unfortunately, the degree distribution is different for the index case and for the subsequent generations. In this section we deal with the most simple form where we assume that the same degree distribution is valid through the entire branching process. This is still applicable in our model for all epidemic generations after the index case. Given that we understand how the branching process develops for all future generations it is then an easy task to include the index case in the model. In our application we only need to obtain the probability that the branching process goes extinct. This same quantity can be used to calculate both the asymptotic probability of a large outbreak and the relative final size of it.

Below we will talk of the *offspring* of an individual and in our application this corresponds to the number of people that the individual infects. We start with the unweighted model where there is only one edge type. Then we continue with the weighted model where we have two different types of edges that the epidemic can spread through. Then we need to take into account through which type of edge an individual was infected and also through how many edges of each type the epidemic continues.

We begin with a model with only one type of individual (corresponding to the unweighted configuration model. We study the number of individuals Z_i in each generation $i = 0, 1, ...$ We start with a single individual so $Z_0 = 1$. Each new generation then consists of the offspring of the individuals in the previous generation so that $Z_i = \sum_{j=1}^{Z_{i-1}} X_{i,j}$, where $X_{i,j}$ (the offspring of individual j in generation i) are all independent and identically distributed $X_{i,j} \sim X$. The offspring distribution is defined by $p_k = \mathbb{P}(X{=}k)$. In the following we will assume that $\mathrm{E}(X) < \infty$ and that $p_k < 1$ for $k = 1, 2$. One important property of a branching process is the probability that it dies out, i.e. that $Z_i = 0$ for some i. Through the probability generating function

$$f(s) \;=\; \sum_{i=0}^{\infty} s^i p_k.$$

we obtain the probability of extinction q as the smallest non-negative solution to the fixed point equation

$$q \;=\; f(q).$$

Note that there can be at most 1 solution $q \in [0, 1)$ and that there is always a solution $q = 1$.

We now continue with a multi-type model which corresponds to the weighted configuration model where there are different types of edges. We restrict the analysis to two types of edges and in effect study how the number of infections via each type of *edge* develops. Let $p_\xi(i,j)$ be the probability that an individual of type $\xi \in \{1,2\}$ has offspring i individuals of type 1 and j individuals of type 2. Some assumptions are needed and these correspond to the requirements for the single type branching process described above. We assume that the expected number of offspring is finite. In addition we assume that the process is not *singular* and that it is *positively regular*, for definitions see (Harris, 2002). In our application positively regular means that, regardless of which edge type we start with, at some time in the future the process is able to produce the other edge type. In turn this means that at least one $p_\xi(i,j) > 0$ for some $i, j > 0$. If this is not true then the graph can be separated into subgraphs, each one consisting only of vertices connected by a single edge type. Such separate configuration model graphs are not within the scope of this paper.

Further, let $\mathbf{s} = (s_1, s_2)$ and define the probability generating functions

$$f_\xi(\mathbf{s}) \quad = \quad \sum_{i=0}^{\infty}\sum_{j=0}^{\infty} s_1^i s_2^j p_\xi(i,j)$$

The probability q_ξ that the branching process dies out, *given that we start with an individual of type* ξ, is a solution to the fixed-point equation

$$\mathbf{q} \quad = \quad (f_1(\mathbf{q}), f_2(\mathbf{q})), \tag{16}$$

where $\mathbf{q} = (q_1, q_2)$. If there exists a solution $\mathbf{q} \in [0,1)^2$ (and there can be at most one such solution), then this is the correct solution. Otherwise the solution is $\mathbf{q} = (1,1)$. As above, *Eq. (16)* can be solved iteratively

1. Let $\mathbf{q}^{(0)} = (0,0)$.
2. Let $\mathbf{q}^{(k)} = (f_1(\mathbf{q}^{(k-1)}), f_2(\mathbf{q}^{(k-1)}))$, for $k = 1, 2, ...$
3. The probability that the process dies out is $\mathbf{q} = \lim_{k\to\infty} \mathbf{q}^{(k)}$.

The quantities q and q_ξ are used in *Section 3.3* and *Section 3.2*.