Type reproduction number for epidemic models on heterogeneous networks

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メタデータ	言語: eng
	出版者:
	公開日: 2021-11-05
	キーワード (Ja):
	キーワード (En):
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	所属:
URL	http://hdl.handle.net/10297/00028418

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Abstract

Infection can spread easily on networks with heterogeneous degree distributions. Here, we considered targeted immunization on such networks, wherein a fraction of individuals with the highest connectivity are immunized. To quantify the effect of this targeted immunization approach on population immunity, we proposed a method using the type reproduction number. Consequently, we derived a precise and simple formula that can yield the immunization threshold, which, to the best of our knowledge, is the first such result presented in literature.

INTRODUCTION

In recent decades, advancements in the field of transportation have led to increased connectivity among people. Owing to this increased interaction, outbreaks of several new infectious diseases have occurred around the world, which are threatening the lives and health of people. In particular, these diseases spread over networks of individuals via contact between them. In a similar manner, the spread of computer viruses through the Internet has also caused significant economic damage to affected individuals and entities. Therefore, there is an urgent and important need to understand the mechanism of these spreading phenomena in networks; moreover, effective methods to control these infections are required. A key issue for effective control of infections is to determine the groups of individuals on which preventive measures such as immunization should be focused.

In epidemiology, the basic reproduction number \mathcal{R}_0 has been used to measure the transmission potential of infectious diseases[1, 2]. \mathcal{R}_0 represents the average number of secondary infections that a typical infection would directly cause in a completely susceptible population. The standard method for calculating \mathcal{R}_0 for epidemic models described by ordinary differential equations involves determining the spectral radius $\rho(A)$ of the next-generation matrix A for an infectious disease [3, 4]. When $\mathcal{R}_0 > 1$, the infection can spread in the host population; in contrast, when $\mathcal{R}_0 < 1$, the infection will not spread. Thus, \mathcal{R}_0 is a useful indicator of the effort required to eliminate an infection from the population. For unstructured models that assume well-mixed infections, if individuals in a host population are immunized at random, then the incidence of an infection will decline when the proportion of people with immunity exceeds $1 - 1/\mathcal{R}_0$, which is referred to as herd immunity fraction [1].

However, the criterion using \mathcal{R}_0 is based on the assumption that the host population is homogeneous and well mixed. If the population is divided into some types and the infection control is performed by focusing on these types, then the type reproduction number \mathcal{T} is used in the place of \mathcal{R}_0 [5–7]. The type reproduction number \mathcal{T} for a target subset of the population represents the average number of secondary cases in this subset produced by the primary cases in the same subset in a completely susceptible population. \mathcal{T} considers the secondary cases transmitted directly from the primary cases. Further, it considers the cases *indirectly* from people who are infected from the primary cases but excluded the target subset [7]. If the infection grows exclusively within the complement of the target subset, \mathcal{T} might not be well-defined. If a vaccine is only applied to this subset of the population, the required fraction of vaccine coverage in the target subset can be given by $1 - 1/\mathcal{T}$, where \mathcal{T} is the type reproduction number for the target subset. In case \mathcal{T} is not well-defined, the infection cannot be eradicated unless the entire subset population is immunized. In previous studies [8, 9], the target reproduction number has been introduced as a general extension of the type reproduction number, and a simple method for deriving the type and target reproduction number using the next-generation matrix has been proposed: if the next-generation matrix A is decomposed into the target matrix C of the terms subject to be immunized and the residual matrix A - C of the terms not subject to be immunized, then the type and target reproduction number \mathcal{T}_C is given by the spectral radius of the matrix $C(I - A + C)^{-1}$,

$$\mathcal{T}_C = \rho(C(I - A + C)^{-1}) \tag{1}$$

if A is irreducible and $\rho(A - C) < 1$ [8, 9]. In this study, the target is limited to the subset of nodes specified by the degree; thus, we use the term type reproduction number.

Considering the spread of infections in social networks, an important property of networks that should not be overlooked is its degree heterogeneity, where the degree k is defined as the number of connections each node has with other nodes [10–12]. It is well-known that the degree distribution, which is the probability distribution of this degree k over the entire network, often follows a power law for large values of k:

$$P(k) \sim k^{-\gamma}.$$
 (2)

In this case, the network is called a scale-free network [12, 13]. For example, it has been reported that the networks of human sexual contact are scale-free [14–16]. On the contrary, some other studies on the subject have rejected this notion [17, 18]. While it is still being debated how exactly real sexual networks are scale-free, it is clear that they are highly heterogeneous; this is because only a few individuals tend to have a large number of sexual partners, while most individuals only have a few sexual partners.

In the popular susceptible-infected-susceptible (SIS) model in networks [19–21], the basic reproduction number is given as follows:

$$\mathcal{R}_0 = \lambda \langle k^2 \rangle / \langle k \rangle, \tag{3}$$

where λ represents the infection rate, which is defined later. The SIS model is the simplest of the compartment model for infectious disease spread, and while there are many possible extensions, the essential properties of \mathcal{R}_0 remain the same. A similar formula for \mathcal{R}_0 has long been known in the field of epidemiology [1, 22]. If the degree distribution follows Eq. (2) and $\gamma \leq 3$, then the second moment $\langle k^2 \rangle$ diverges in the large-size limit. Thus, \mathcal{R}_0 can diverge if λ is finite. Moreover, even if λ is considerably small, the infection can become widespread. While $\langle k^2 \rangle$ must be finite for real social networks, typically, they have high $\langle k^2 \rangle$.

In this study, to develop efficient herd immunity, we considered the case wherein only a fraction of individuals in a population with the highest connectivity ($k \ge k_{\text{max}}$) are immunized; this is because it is expected that targeting individuals that act as hubs effectively reduces $\langle k^2 \rangle$. Though this case has been analyzed in previous works [21, 23], unlike those studies, herein, we quantify the effect of target immunization by using the type reproduction number. Furthermore, we also derive a new formula to calculate the immunization threshold.

RESULT

To account for the effect of heterogeneity in the degree distribution of a population, it is appropriate to consider the density $\rho_k(t)$ of infected nodes within each degree class k.

Based on the previously proposed SIS model [24, 25], the mean-field rate equation can be obtained as

$$\frac{d\rho_k(t)}{dt} = -\rho_k(t) + \lambda k [1 - \rho_k(t)] \Theta_k(t), \qquad (4)$$

where the time unit is set so that the recovery rate is equal to one. In this equation, the first term on the right-hand side represents recovery, wherein the average duration of infection is set to one, while the second term represents transmission, which is proportional to the combined product of infection rate (λ), density of susceptible nodes $(1 - \rho_k(t))$, number of neighboring vertices (k), and probability that any neighbor is infected ($\Theta_k(t)$). In particular, the probability $\Theta_k(t)$ is the average of the probabilities that a connection from a node with degree k exists to an infected node with degree k' over all degrees:

$$\Theta_k(t) = \sum_{k'=1} P(k'|k)\rho_{k'}(t),$$
(5)

where P(k'|k) represents the conditional probability that a node of degree k is connected

to a node of degree k'. Assuming that there is no degree-degree correlation [19, 20], $\Theta_k(t)$ could be considered independent of k, and thus, can be given as

$$\Theta(t) = \frac{1}{\langle k \rangle} \sum_{k=1} k P(k) \rho_k(t).$$
(6)

This is because, here

$$P(k'|k) = k'P(k')/\langle k \rangle.$$
(7)

To calculate the type reproduction number, we consider the linearized system of Eq. (4) near the disease-free solution. If the degree distribution has the maximum value k_{max} , then the next-generation matrix of eq. (4) is as follows:

$$A = \begin{pmatrix} \lambda P(1|1) & \lambda P(2|1) & \cdots & \lambda P(k_{\max}|1) \\ 2\lambda P(1|2) & 2\lambda P(2|2) & \cdots & 2\lambda P(k_{\max}|2) \\ \vdots & \vdots & \ddots & \vdots \\ k_{\max}\lambda P(1|k_{\max}) & k_{\max}\lambda P(2|k_{\max}) & \cdots & k_{\max}\lambda P(k_{\max}|k_{\max}) \end{pmatrix},$$
(8)

where A_{ij} represents the rate of infection for nodes of degree *i* due to spread of the infection from infectious nodes of degree *j*. The complete derivation of the matrix in Eq. (8) was performed using the method proposed by Diekmann et al. [4]; we decomposed the Jacobian of Eq. (4) into $T + \Sigma$, where $T_{ij} = iP(j|i)$ represents the transmission part, describing the production of new infections, and $\Sigma_{ij} = -\delta_{ij}$ is the transition part, describing changes in state, and computed $A = -T\Sigma^{-1}$. Note that A = T because the recovery rates are set equal to one for all people.

If we target nodes with k larger than k_t , the target matrix can be written as follows:

$$C = \begin{pmatrix} 0 & 0 & \cdots & 0 \\ \vdots & \vdots & & \vdots \\ 0 & 0 & \cdots & 0 \\ k_t \lambda P(1|k_t) & k_t \lambda P(2|k_t) & \cdots & k_t \lambda P(k_{\max}|k_t) \\ \vdots & \vdots & & \vdots \\ k_{\max} \lambda P(1|k_{\max}) & k_{\max} \lambda P(2|k_{\max}) & \cdots & k_{\max} \lambda P(k_{\max}|k_{\max}) \end{pmatrix}.$$
 (9)

Then, the type reproduction number $\mathcal{T}_{\geq k_t}$ is determined using Eq. (1). In the absence of degree-degree correlation (i.e., Eq. (7)), by using Eq. (1)), the type reproduction number

can be obtained as follows:

$$\mathcal{T}_{\geq k_{t}} = \frac{\frac{\lambda}{\langle k \rangle} \sum_{k=k_{t}}^{k_{\max}} k^{2} P(k)}{1 - \frac{\lambda}{\langle k \rangle} \sum_{k=1}^{k_{t}-1} k^{2} P(k)}$$

$$= 1 + \frac{\mathcal{R}_{0} - 1}{1 - \frac{\lambda}{\langle k \rangle} \sum_{k=1}^{k_{t}-1} k^{2} P(k)},$$
(10)

if the denominator is positive. The derivation of Eq. (10) is presented in the Methods section. If the denominator is negative and $\mathcal{T}_{\geq k_t}$ is not well-defined, the infection can survive even when all $k \geq k_t$ nodes have been immunized. It is obvious from Eq. (10) that when $\mathcal{R}_0 > 1$, $\mathcal{T}_{\geq k_t}$ increases monotonically with respect to k_t . Furthermore, if the entire population is targeted ($k_t=1$), the type reproduction number can be calculated as

$$\mathcal{T}_{\geq 1} = \frac{\lambda}{\langle k \rangle} \sum_{k=1}^{k_{\max}} k^2 P(k), \qquad (11)$$

which coincides with the formula for the basic reproduction number \mathcal{R}_0 given by Eq. (3). For a general case, it can be mathematically confirmed that $\mathcal{T}_{\geq k_t} > 1 \Leftrightarrow \mathcal{R}_0 > 1$ and $\mathcal{T}_{\geq k_t} < 1 \Leftrightarrow \mathcal{R}_0 < 1$ [8, 9].

We examine the characteristics of the type reproduction number $\mathcal{T}_{\geq k_t}$, using the example shown in Fig. 1, where the degree distribution $P(k) \propto k^{-3}$ for $k_{\min} \leq k \leq k_{\max}$ with $k_{\min} = 2$ and $k_{\max} = 10^4$. It should be noted that k_{\max} is an artificially introduced cutoff; however, a system with a finite size always has a similar cutoff. The value of λ is set such that $\mathcal{R}_0 = 3$; consequently, more than $1 - 1/\mathcal{R}_0 = 2/3$ of the total population would have to be randomly immunized to prevent the spread of the infection. Fig. 1(a) shows the dependency of $\mathcal{T}_{\geq k_t}$ on k_t ; in this case, because Eq. (10) is well-defined for $k_t \leq 29$, the infection cannot be eradicated by immunizing only nodes with degrees k > 29. Thus, this critical value is given by the maximum value k_t that satisfies:

$$\frac{\lambda}{\langle k \rangle} \sum_{k=1}^{k_t-1} k^2 P(k) < 1.$$
(12)

Then, the required fraction of the targeted nodes $k \ge k_t$ that need to be immunized can be obtained as follows:

$$1 - \frac{1}{\mathcal{T}_{\geq k_t}} = \frac{\mathcal{R}_0 - 1}{\frac{\lambda}{\langle k \rangle} \sum_{k=k_t}^{k_{\max}} k^2 P(k)};$$
(13)

and tends to a value of one when k_t approaches the critical value of 29 as shown in Fig. 1(b). In particular, this figure can be used to obtain the required value of k_t based on public



FIG. 1. Characteristic curves for the type reproduction number: (a) type reproduction number $\mathcal{T}_{\geq k_t}$ plotted as a function of k_t , (b) plot for required fraction of immunized nodes with degree $k \geq k_t$, and (c) plot of required amount of vaccine given by Eq. (14). Here, the degree distribution $P(k) \propto k^{-3}$ for $2 \leq k \leq 10^4$ and the infection rate is set to $\lambda = 0.22$ (such that $\mathcal{R}_0 = 3$).

health constraints. For example, if only 80% of the target population can be vaccinated, or the effective rate of vaccination is 80%, then, to eradicate the infection, k_t must be less than or equal to 7 because $1 - 1/\mathcal{T}_{\geq 7} < 0.8$ and $1 - 1/\mathcal{T}_{\geq 8} > 0.8$.

When all nodes with $k \ge k_t$ are immunized, the proportion of the population that receives immunity against the infection is $\sum_{k=k_t}^{k_{\max}} P(k)$. Because the total amount of vaccine is $\sum_{k=k_t}^{k_{\max}} P(k)$ multiplied by $1 - 1/\mathcal{T}_{\ge k_t}$, the required amount of vaccine is calculated as

$$g_c = (\mathcal{R}_0 - 1) \frac{\langle k \rangle}{\lambda} \frac{\sum_{k=k_t}^{k_{\max}} P(k)}{\sum_{k=k_t}^{k_{\max}} k^2 P(k)}.$$
(14)

It can be easily proved that g_c is a decreasing function of k_t , regardless of the degree distribution P(k) (see also Fig. 1(c)). Therefore, it was shown that the critical value of k_t obtained via Eq. (12) or using its plot (such as in Fig. 1(b)) yields the optimal value for k_t . In other words, if the immunization rate and vaccine efficiency are independent of the degree, the upper limit of k_t , where immunization measures concentrate on nodes with $k \ge k_t$, is found by Fig. 1(b), and the total proportion of vaccine is minimal at this value of k_t .

DISCUSSION

In summary, we formulated an optimal immunization strategy, which is given by Eq. (12), based on the degree and using the type reproduction number. Here, optimal immunization refers to when the amount of vaccine required to achieve herd immunity is minimal. To achieve herd immunity, it is necessary to increase the sum in the denominator in the right side of Eq. (13). As this is the sum of the square of the degree over the target nodes, it is apparent that the strategy targeting nodes in a descending order with respect to the degree is optimal. Another immunization strategy has been investigated by Pastor-Satorras and Vespignani [21, 23]. Their targeted immunization scheme was developed to *progressively* immunize the most highly connected nodes [23]. In constract, our proposed method determines the target population subset to be immunized based on the degree before immunization. Thus, their reported method for calculating the immunization threshold differs from that proposed in this study. They focused on the number of links that disappear when the high-degree nodes were removed, where the fraction of the disappearing links is given as follows:

$$p = \frac{\sum_{k=k_t}^{k_{\max}} kP(k)}{\sum_{k=1}^{k_{\max}} kP(k)}.$$
(15)

Additionally they derived the immunization threshold as follows:

$$\frac{\langle k^2 \rangle_{g_c}}{\langle k \rangle_{g_c}} = \frac{\sum_{k=1}^{k_t - 1} k^2 P(k)}{\sum_{k=1}^{k_t - 1} k P(k)} (1 - p) + p < \frac{1}{\lambda},\tag{16}$$

where $\langle \cdot \rangle_{g_c}$ represents the average of residual degrees after the links disappears. In contrast, Eq. (12) can be rewritten as

$$\frac{\sum_{k=1}^{k_t-1} k^2 P(k)}{\sum_{k=1}^{k_t-1} k P(k)} (1-p) < \frac{1}{\lambda}.$$
(17)

Thus, the critical value of k_t derived from (16) is smaller than that deduced from (17). It should be noted that the critical value of k_t in [23] does not represent the degree of the original network; instead it refers to the degree of the network with the immunized nodes removed. The immunization method assumed in this study is comparatively simple and more realistic, because it is based on the information of the static network, and the calculated threshold value is more accurate.

Furthermore, while we considered the SIS model in our study, it is easy to extend our result to susceptible-infected-recovered (SIR) models for infections as well. For the SIR model, the equation reported in Ref. [26] can be used instead of Eq. (5), i.e.,

$$\Theta_k(t) = \sum_{k'} \frac{k' - 1}{k'} P(k'|k) \rho_{k'}(t).$$
(18)

Consequently, Eq. (12) is replaced by

$$\frac{\lambda}{\langle k \rangle} \sum_{k=1}^{k_t-1} (k^2 - k) P(k) < 1.$$
(19)

In conclusion, we showed that the type reproduction number is a considerably useful metric to devise an optimal immunization strategy for a population. It should be noted that the main result of this study, i.e., Eq. (12), was obtained assuming no degree-degree correlation. However, if degree-degree correlation is considered, it is necessary to calculate the type reproduction number using the two matrices given by Eqs. (8) and (9). Lastly, the proposed method to calculate immunization threshold could also be used for various other extended epidemic models, such as in [27].

METHODS

Here, we prove that Eq. (10) gives the spectral radius of $C(I - A + I)^{-1}$ in the absence of degree-degree correlation. If Eq. (7) holds, then C and A - C are rewritten as follows:

$$C = \frac{\lambda}{\langle k \rangle} \begin{pmatrix} 0 \\ \vdots \\ 0 \\ k_t \\ \vdots \\ k_{\max} \end{pmatrix} (P(1), 2P(2), \cdots, k_{\max}P(k_{\max})), \qquad (20)$$

$$A - C = \frac{\lambda}{\langle k \rangle} \begin{pmatrix} k_1 \\ \vdots \\ k_t - 1 \\ 0 \\ \vdots \\ 0 \end{pmatrix} (P(1), 2P(2), \cdots, k_{\max}P(k_{\max})).$$
(21)

A simple matrix operation shows that the vector $(P(1), 2P(2), \dots, k_{\max}P(k_{\max}))$ is a left eigenvector of C and A - C, the corresponding eigenvalues of which are $\sum_{k=k_t}^{k_{\max}} k^2 P(k)$ and $\sum_{k=1}^{k_t-1} k^2 P(k)$, respectively. Since the rank of C and A - C is one, the other eigenvalues are zero. Thus, the above eigenvalues give the spectral radius. Moreover, the power of (A - C)can be expressed as follows:

$$(A-C)^{n} = \frac{\lambda^{n}}{\langle k \rangle^{n}} \left[\sum_{k=1}^{k_{t}-1} k^{2} P(k) \right]^{n-1} \begin{pmatrix} k_{1} \\ \vdots \\ k_{t}-1 \\ 0 \\ \vdots \\ 0 \end{pmatrix} (P(1), 2P(2), \cdots, k_{\max} P(k_{\max})) .$$
(22)

Thus, if the spectral radius of A - C is less than one $(\sum_{k=1}^{k_t-1} k^2 P(k) < 1), C(1 - A + C)^{-1}$ is rewritten as:

$$C(1 - A + C)^{-1} = C \sum_{n=0}^{\infty} (A - C)^{n}$$

$$= \frac{\frac{\lambda}{\langle k \rangle}}{1 - \frac{\lambda}{\langle k \rangle} \sum_{k=1}^{k_{t}-1} k^{2} P(k)} \begin{pmatrix} 0 \\ \vdots \\ 0 \\ k_{t} \\ \vdots \\ k_{\max} \end{pmatrix} (P(1), 2P(2), \cdots, k_{\max} P(k_{\max})).$$

$$(23)$$

As above, the vector $(P(1), 2P(2), \dots, k_{\max}P(k_{\max}))$ is a left eigenvector of $C(I - A + C)^{-1}$, and the corresponding eigenvalue is given as follows:

$$\frac{\frac{\lambda}{\langle k \rangle} \sum_{k=k_t}^{k_{\max}} k^2 P(k)}{1 - \frac{\lambda}{\langle k \rangle} \sum_{k=1}^{k_t-1} k^2 P(k)}.$$
(24)

As the other eigenvalues are zero, Eq. (24) gives the spectral radius of $C(I - A + C)^{-1}$.

This work was supported by the JSPS KAKENHI (no. 18K03453 and 21K03387). A part of this work was conducted at the Joint Usage / Research Center on Tropical Disease, Institute of Tropical Medicine, Nagasaki University (2020-Ippan-1), and at the Japan Science and Technology Agency Crest. I would like thank Hiromu Ito for his valuable inputs for this study. I would like to thank Editage (www.editage.com) for English language editing.

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- R. M. Anderson and R. M. May, *Infectious diseases of humans: dynamics and control* (Oxford University Press, Oxford; New York, 1991).
- [2] O. Diekmann and J. A. P. Heesterbeek, Mathematical Epidemiology of Infectious Diseases: Model Building, Analysis and Interpretation, Wiley Series in Mathematical & Computational Biology (Wiley, West Sussex, England, 2000).
- [3] P. van den Driessche and J. Watmough, Math. Biosci. 180, 29 (2002).
- [4] O. Diekmann, J. A. P. Heesterbeek, and M. G. Roberts, J. R. Soc. Interface 7, 873–885 (2010).
- [5] M. G. Roberts and J. A. P. Heesterbeek, Proc. R. Soc. Lond. B. 270 (2003).
- [6] J. Heesterbeek and M. Roberts, Math. Biosci. 206, 3 (2007).
- [7] H. Inaba, J. of Math. Biol. 66, 1065 (2013).
- [8] Z. Shuai, J. A. P. Heesterbeek, and P. van den Driessche, J. Math. Biol. 67, 1067 (2013).
- [9] M. A. Lewis, Z. Shuai, and P. van den Driessche, J. Math. Biol. 78, 2317 (2019).
- [10] M. Newman, A.-L. Barabasi, and D. J. Watts, The Structure and Dynamics of Networks: (Princeton Studies in Complexity) (Princeton University Press, USA, 2006).
- [11] A.-L. Barabási and M. Pósfai, *Network science* (Cambridge University Press, Cambridge, 2016).
- [12] R. Albert and A.-L. Barabási, Rev. Mod. Phys. 74, 47 (2002).

- [13] A.-L. Barabási and R. Albert, Science **286**, 509 (1999).
- [14] F. Liljeros, C. R. Edling, L. A. N. Amaral, H. E. Stanley, and Y. Åberg, Nature 411, 907 (2001).
- [15] A. Schneeberger, C. H. Mercer, S. Gregson, C. A. Ferguson, Neil M.and Nyamukapa, R. M. Anderson, A. M. Johnson, and G. P. Garnett, Sex. Transm. Dis. **31** (2004).
- [16] H. Ito, K. Tamura, T. Wada, T. Yamamoto, and S. Morita, PLOS ONE 14, 1 (2019).
- [17] M. S. Handcock and J. H. Jones, Theor. Popul. Biol. 65, 413 (2004).
- [18] D. T. Hamilton, M. S. Handcock, and M. Morris, Sex. Transm. Dis. 35, 30 (2008).
- [19] R. Pastor-Satorras and A. Vespignani, Phys. Rev. Lett. 86, 3200 (2001).
- [20] R. Pastor-Satorras and A. Vespignani, Phys. Rev. E 63, 066117 (2001).
- [21] R. Pastor-Satorras, C. Castellano, P. Van Mieghem, and A. Vespignani, Rev. Mod. Phys. 87, 925 (2015).
- [22] A. L. Lloyd and R. M. May, Science **292**, 1316 (2001).
- [23] R. Pastor-Satorras and A. Vespignani, Phys. Rev. E 65, 036104 (2002).
- [24] M. Boguñá and R. Pastor-Satorras, Phys. Rev. E 66, 047104 (2002).
- [25] M. Boguñá, R. Pastor-Satorras, and A. Vespignani, Phys. Rev. Lett. 90, 028701 (2003).
- [26] M. Boguñá, R. Pastor-Satorras, and A. Vespignani, Epidemic spreading in complex networks with degree correlations, in *Statistical Mechanics of Complex Networks*, edited by R. Pastor-Satorras, M. Rubi, and A. Diaz-Guilera (Springer Berlin Heidelberg, Berlin, Heidelberg, 2003) pp. 127–147.
- [27] S. Morita, Sci. Rep. 6, 22506 (2016).