J Syst Sci Complex (2015) 28: 96–104

Degree Distribution Dynamics for Disease Spreading with Individual Awareness*

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DOI: 10.1007/s11424-014-2186-x
Received: 7 September 2012 / Revised: 15 January 2013

Abstract Behavioral responses triggered by the perceived risk of experiencing the disease represent a key ingredient in the spread of epidemics across human population. In this paper, two forms of individual awareness (i.e., the risk perception of an emerging epidemic) are addressed: Contact awareness that increases with individual contact number, and local awareness that increases with the fraction of infected contacts. By extending the probability generating functionology, the author shows that it is possible to track the evolution of the degree distributions among susceptible and infected individuals when the underlying network of contacts is represented by a semi-random configuration model. It is hopefully to shed some light on the dynamic aspects of networked epidemiological models.

Keywords Behavioral response, complex network, epidemiology, generating function.

1 Introduction

Theoretical study of infectious disease transmission has recently been one of the most active and prolific fields in complex networks, largely improving the simple mathematical description of Kermack & McKendrick's mass action susceptible-infected-recovered (SIR) model[1]. Unlike mass-action models[1–3], random networks allow considering more realistic and accurate representation of heterogeneities in the number of contacts per individual. Various results such as the epidemic probability, endemic equilibrium, and expected final epidemic size have been obtained mainly by using the mean-field techniques and stochastic branching processes (see, e.g., [4–10]).

Due to the heterogeneity induced by the network model, the dynamic epidemic incidence, however, tends to be difficult to capture. Some viable approximation approaches have become heavily used. For example, the moment closure methods (or pairwise models) customarily ignore long-range state dependence between individuals[11–13]. For a network with n different degrees,

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*This research was supported by Air Force Office of Scientific Research under Grant No. FA9550-09-1-0165.
◇ *This paper was recommended for publication by Editor SUN Liuquan.*

these methods typically produce $O(n^2)$ coupled ordinary differential equations (ODE's). An algebraic lumping technique is developed in [14], which draws on symmetries and allows further reductions of the state space. Other researchers get around this difficulty through a mean-field assumption that all nodes of the same degree having the same infection probability at any given time[15, 16]. Recently, Volz[17] showed that the SIR epidemic dynamics on configuration model[18] can be modeled by a coupled system of only three nonlinear ODE's by using network-based variables such as the number of edges in a well-defined category rather than node-based quantities such as the numbers of susceptibles and infected individuals. This approach leads to excellent agreement with simulations and is further developed to treat a mixed SI(R) model[19]. Based on Volz's result, the degree distribution dynamics is derived in [20] for the paradigmatic susceptible-infected-susceptible (SIS) models.

Beyond the basic epidemic models, the elements of human behavioral responses to the presence of an infectious pathogen have recently gained great research attention. During an epidemic outbreak, people aware of the disease in their proximity may adopt measures, such as stay indoors, avoid public transportation, and wear surgical face masks, in order to reduce their susceptibility to infection, which in turn, can remarkably affect the epidemic dynamics[21–23]. The change of individual behavior to the risk of infection is referred to as individual awareness[24]. The effect of awareness is to decrease the infectivity. It is shown[25, 26] that the network architecture (homogeneous or heterogeneous) has significant impact on the existence of a critical level of awareness that halts the epidemics. In [27, 28], the role of different forms of awareness is further discussed in scale-free networks. However, it is still hard to quantify the impact of behavioral changes on the alteration of the epidemic spread of an emerging infectious disease in complex social networks. For a comprehensive survey of awareness in epidemic modeling of human to human infectious diseases, we refer readers to [29] and the references therein.

In this paper, we extend the previous work [20] by involving the increasingly relevant issue of awareness in the transmission of infectious diseases. Specifically, we consider an SIS model in which the infectivity is modulated by two types of awareness: Contact awareness, which increases with individual contact number (i.e., node degree), and local awareness, which increases with the fraction of infected contacts in an individual's neighborhood. The concept and theory of probability generating functions are extended. With these and the network-based quantities developed in [17], we show that it is possible to track the evolution of the degree distributions among susceptibles and infected individuals as well as the expected excess degrees in some well-defined categories for SIS epidemics on random networks represented by configuration model.

Unlike the previous work on awareness mostly at the mean-field level (see, e.g., [24–28, 30]), we derive rigorously the time-dependent distribution dynamics by using generating function formalism. Besides, no specific form of local awareness is assumed here. We mention that some nonlinear and linear functions of local awareness are used in [25, 26, 28] and [27], respectively. Therefore, our framework provides additional flexibility.

The rest of the paper is organized as follows. In Section 2, we present the SIS model with individual awareness. We report our main results in Section 3. Finally, we suggest some possible directions for future research in Section 4.

2 Model

We study the SIS epidemics of an infection spreading over a contact network of n individuals. The individuals are labeled $1, 2, \cdots, n$, and $\mathcal{N} = \{1, 2, \cdots, n\}$ represents the host population in question. Each individual is assigned a disease-related state of susceptible or infected. Denote by $\mathcal{S}$ and $\mathcal{I}$ the sets of individuals that are susceptible and infected, respectively. By definition, we have $\mathcal{N} = \mathcal{S} \cup \mathcal{I}$. Each susceptible individual $i \in \mathcal{S}$ is infected independently with a rate β_{ij} if it has a neighboring infected individual $j \in \mathcal{I}$. Below we will instantiate the infection rate β_{ij} to incorporate the effect of awareness. An infected individual is cured and become susceptible again (i.e., can still catch infections) at constant rate γ. As is customary, let S and I denote the fractions of individuals in the sets $\mathcal{S}$ and $\mathcal{I}$, respectively. Therefore, $S + I = 1$ at any time instant.

The network of contacts is defined in terms of configuration models[18], where random graphs can be seen as networks drawn uniformly at random from a family of all possible networks whose node degrees follow the specified one. Here, each individual is modeled as a node in the network. Let $d_1, d_2, \cdots, d_n$ be i.i.d random variables representing the degrees of nodes $1, 2, \cdots, n$ and $p_k = P(d_1 = k)$ $(k = 0, 1, \cdots)$ is the probability distribution of connectivity k. Make sure that $S_n = d_1 + d_2 + \cdots + d_n$ is even. Attach d_i stubs (half-edges) to individual i and pair up the S_n stubs completely at random to form complete edges. The resulting graph accounts for the interaction among individuals. Note that there may exist self-loops and multiple edges between pairs of individuals. However, subject to d_1 having finite variance, such imperfections are very rare in the network for large n; cf. [5, Theorem 3.1.2] and [31]. Therefore, we may safely ignore these effects.

To capture the impact of individual awareness, we make the transmission of the disease dependent on the quality of the information available to a given susceptible individual. Specifically, suppose that an individual $i \in \mathcal{S}$ has k neighbors, namely $d_i = k$, from which $l(\leq k)$ are infected at time t. We assume that the infection rate $\beta_{ij} = \beta_{ij}(t)$ take the following form:

$$\beta_{ij}(t) = \beta \cdot \psi_k \cdot \phi_{l/k}(t), \tag{1}$$

where β is the bare infection rate, the second multiplicative factor $\psi_k = k^{-\mu}$ stands for the contact awareness with $\mu \in [0, 1]$ denoting the level of precaution measures adopted[27, 28], and the third factor $\phi_{l/k}(t)$ stands for the local awareness, which is proportional to the fraction of contacts with infected neighbors, with respect to the total number of contacts, i.e., l/k. By definition, we have $0 \leq \psi_k \leq 1$, and it is taken as a decreasing function of k. This has an intuitive interpretation: Behavioral responses triggered by awareness of experiencing the disease depends on the amount of contact information individuals have access to and the risk of being infected is higher when the contact number is larger. Hence, the contact awareness of i can reduce its susceptibility (by taking precaution measures). On the other hand, we assume that the local awareness $\phi_{l/k}(t)$ is a decreasing function of l/k and $0 \leq \phi_{l/k}(t) \leq 1$. Its effect is also that of reducing the bare infection rate β when the quantity of local information l/k increases[29]. Some specific forms of local awareness have been used in the literature. Bagnoli, et al.[25], for example, took $\phi_{l/k} = \exp(J(l/k)^{\alpha})$ with $\alpha \in [0, 1]$ denoting the use of special

prophylaxis, while Shang[28] took $\phi_{l/k} = 1 - c(l/k)^\alpha$ with $\alpha \geq 1$ as an impact strength factor on the admission rate and $c \in [0, 1]$ representing the level of precaution measures. It is worth noting that, in (1), when $\mu = 0$ and $\phi_{l/k}(t) \equiv 1$, we reproduce the bare infection rate $\beta_{ij} = \beta$ independent of the infection levels of individuals.

For $\mu \in [0, 1]$ and $a \geq 0$, we define the μ-extended probability generating function $g_a(x)$ for the network's degree distribution as

$$g_a(x) = \sum_{k=0}^{\infty} p_k a^{k^{1-\mu}} x^k, \tag{2}$$

where p_k governs the probability that a node has degree k (i.e., k neighbors) as before. It can be seen that if the augmented variable $a = 1$, we obtain the usual probability generating function

$$g_1(x) = \sum_{k=0}^{\infty} p_k x^k. \tag{3}$$

Therefore, the definition of the μ-extended probability generating function can be regarded as a generalization of that of probability generating function. Given a, we have two extreme cases: $g_a(x) = a g_1(x)$ when $\mu = 1$, and $g_a(x) = g_1(ax)$ when $\mu = 0$. By virtue of the suitable normalization, we can derive that $g_1(1) = 1$, $g_1'(1) = \langle k \rangle$ (which is the average degree) and $g_1''(1) = \langle k^2 \rangle - \langle k \rangle$.

3 Degree Distribution Dynamics

The contact network, albeit undirected, is conveniently viewed as a directed graph so that we can keep track of who infects who. In particular, an edge is regarded as two arcs with opposite directions. The start node of an arc is called base, while the end node is called target. Following the notations in [17], let p_I be a measure of the probability that an arc with a susceptible base has an infected target. Likewise, let p_S be a measure of the probability that an arc with a susceptible base has a susceptible target. To formally define p_I, let $\mathcal{A}$ represent the set of all arcs in the contact network, $\mathcal{A}_X$ be the subset of arcs such that base $\in X$, and $\mathcal{A}_{XY}$ be the subset of arcs such that base $\in X$ and target $\in Y$. Define $M_X = \#\{\mathcal{A}_X\}/\#\{\mathcal{A}\}$ as the fraction of arcs in set $\mathcal{A}_X$, and $M_{XY} = \#\{\mathcal{A}_{XY}\}/\#\{\mathcal{A}\}$ as the fraction of arcs in set $\mathcal{A}_{XY}$. Thus we have

$$p_I = \frac{M_{SI}}{M_S} \quad \text{and} \quad p_S = 1 - p_I = \frac{M_{SS}}{M_S}, \tag{4}$$

respectively. It is worth noting that the sets $\mathcal{A}_X$, $\mathcal{A}_{XY}$, etc. change over time. Consequently, p_I and p_S are dynamical, not only dependent of connectivity structures but also coupled with epidemic information.

To derive the degree distribution among susceptibles or infected nodes, we consider a susceptible node with degree k at time t. We call this susceptible node as base and its k neighbors as $\text{target}_1, \text{target}_2, \cdots, \text{target}_k$. For $i = 1, 2, \cdots, k$, we assume that for each arc from base to target_i there will be a uniform probability p_I that target_i is infected. Then the hazard for base

becoming infected at time t can be shown as[17]

$$\lambda_k(t) = kp_I(t) \cdot \beta\psi_k\phi_{p_I}(t). \tag{5}$$

Let $u_k(t)$ be the probability that base remains susceptible at time t, and then

$$u_k(t) = \mathrm{e}^{-\int_0^t \lambda_k(\tau)\mathrm{d}\tau} = \left(\mathrm{e}^{-\int_0^t \beta p_I(\tau)\phi_{p_I}(\tau)\mathrm{d}\tau}\right)^{k^{1-\mu}} := \theta^{k^{1-\mu}}, \tag{6}$$

where $\theta = u_1$ is the fraction of degree one nodes that remain susceptible at time t. It follows from the μ-extended generating function (2), the fraction of nodes which is susceptible at time t is given by

$$S = \sum_{k=0}^{\infty} p_k u_k = \sum_{k=0}^{\infty} p_k \theta^{k^{1-\mu}} = g_\theta(1). \tag{7}$$

We establish the following results regarding the degree distribution dynamics.

Theorem 3.1 *Let dummy variables x_S and x_I correspond to the number of arcs from a base to a target in sets $\mathcal{S}$ and $\mathcal{I}$, respectively. We have*

(i) *the degree distribution for susceptible nodes at time t is generated by*

$$g_S(x_S, x_I) = \frac{g_\theta(x_S p_S + x_I(1-p_S))}{g_\theta(1)}; \tag{8}$$

(ii) *the degree distribution for infected nodes at time t is generated by*

$$g_I(x_S, x_I) = \frac{g_1(x_S p_S + x_I(1-p_S)) - g_\theta(x_S p_S + x_I(1-p_S))}{1 - g_\theta(1)}; \tag{9}$$

(iii) *the excess degree distribution*[8] *for susceptible nodes selected with probability proportional to the number of arcs to infected nodes at time t is generated by*

$$g_{SI}(x_S, x_I) = \frac{g'_\theta(x_S p_S + x_I(1-p_S))}{g'_\theta(1)}, \tag{10}$$

and what's more, $g_{SS}(x_S, x_I) = g_{SI}(x_S, x_I)$;

(iv) *the excess degree distribution for infected nodes selected with probability proportional to the number of arcs to susceptible nodes at time t is generated by*

$$g_{IS}(x_S, x_I) = \frac{g'_1(x_S p_S + x_I(1-p_S)) - g'_\theta(x_S p_S + x_I(1-p_S))}{\langle k\rangle - g'_\theta(1)}, \tag{11}$$

and what's more, $g_{II}(x_S, x_I) = g_{IS}(x_S, x_I)$.

Define δ_{XY} as the average degree of nodes in set X, selected with probability proportional to the number of arcs to nodes in set Y, not counting one arc to nodes of type Y. The notion of δ_{XY} is commonly called average excess degree[8]. Furthermore, define $\delta_{XY}(Z)$ as δ_{XY} but counting only arcs directed to nodes in set Z. By taking derivative of the probability generating functions, we obtain the following corollary immediately.

Corollary 3.2 *The following statements for average excess degrees at time t hold.*
(i) $\delta_{SI} = \frac{d}{dx}\left(g_{SI}(x,x)\right)\big|_{x=1} = \frac{g''_\theta(1)}{g'_\theta(1)}$;
(ii) $\delta_{SI}(I) = \frac{d}{dx_I}\left(g_{SI}(x_S,x_I)\right)\Big|_{x_S=x_I=1} = \frac{(1-p_S)g''_\theta(1)}{g'_\theta(1)}$;
(iii) $\delta_{SI}(S) = \frac{d}{dx_S}\left(g_{SI}(x_S,x_I)\right)\Big|_{x_S=x_I=1} = \frac{p_S g''_\theta(1)}{g'_\theta(1)}$;
(iv) $\delta_{IS} = \frac{d}{dx}\left(g_{IS}(x,x)\right)\big|_{x=1} = \frac{g''_1(1)-g''_\theta(1)}{g'_1(1)-g'_\theta(1)} = \frac{\langle k^2\rangle-\langle k\rangle-g''_\theta(1)}{\langle k\rangle-g'_\theta(1)}$;
(v) $\delta_{IS}(I) = \frac{d}{dx_I}\left(g_{IS}(x_S,x_I)\right)\Big|_{x_S=x_I=1} = \frac{(g''_1(1)-g''_\theta(1))(1-p_S)}{g'_1(1)-g'_\theta(1)} = \frac{(\langle k^2\rangle-\langle k\rangle-g''_\theta(1))(1-p_S)}{\langle k\rangle-g'_\theta(1)}$;
(vi) $\delta_{IS}(S) = \frac{d}{dx_S}\left(g_{IS}(x_S,x_I)\right)\Big|_{x_S=x_I=1} = \frac{(g''_1(1)-g''_\theta(1))p_S}{g'_1(1)-g'_\theta(1)} = \frac{(\langle k^2\rangle-\langle k\rangle-g''_\theta(1))p_S}{\langle k\rangle-g'_\theta(1)}$.

Now we present the proof of Theorem 3.1.

Proof of Theorem 3.1 Considering two arcs from a base to two targets: target_1 and target_2. For two sets X and Y, we assume that the event that $\text{target}_1 \in X$ is independent of the event that $\text{target}_2 \in Y$ [17]. Accordingly, arcs from a base to nodes in sets $\mathcal{S}$ and $\mathcal{I}$ are distributed according to a binomial distribution with probabilities p_S and $p_I = 1 - p_S$. Let $d_{\text{base}}(X)$ be the random variable denoting the number of arcs from base to nodes in set X. Suppose that c normalizes the distribution. In what follows, we prove the four statements separately.

For (i), by using (2) and (6), the generating function of degree distribution for susceptible nodes can be written as

$$\begin{aligned} g_S(x_S,x_I) &= \sum_{k=0}^{\infty} p_k u_k \sum_{i=0}^{k} x_S^i x_I^{k-i} P(d_{\text{base}}(\mathcal{S}) = i|p_S)/c \\ &= \sum_{k=0}^{\infty} p_k \theta^{k^{1-\mu}} (x_S p_S + x_I(1-p_S))^k/c \\ &= g_\theta(x_S p_S + x_I(1-p_S))/g_\theta(1), \end{aligned} \tag{12}$$

where $c = \sum_{k=0}^{\infty} p_k \theta^{k^{1-\mu}} (p_S + (1-p_S))^k = g_\theta(1)$ normalizes the distribution.

For (ii), similar derivation can be applied by taking the complement. Indeed, we obtain

$$\begin{aligned} g_I(x_S,x_I) &= \sum_{k=0}^{\infty} p_k (1-u_k) \sum_{i=0}^{k} x_S^i x_I^{k-i} P(d_{\text{base}}(\mathcal{S}) = i|p_S)/c \\ &= \frac{g_1(x_S p_S + x_I(1-p_S)) - g_\theta(x_S p_S + x_I(1-p_S))}{1 - g_\theta(1)}. \end{aligned} \tag{13}$$

For (iii), the excess degree distribution can be though of as choosing a random arc in set $\mathcal{A}_{SI}$, following it to the susceptible node, and then counting the number of arcs out of that node except the one we arrived on. Hence, we have

$$\begin{aligned} g_{SI}(x_S,x_I) &= \frac{\sum_{k=0}^{\infty} p_k u_k \sum_{i=0}^{k} (k-i) x_S^i x_I^{k-i-1} P(d_{\text{base}}(\mathcal{S}) = i|p_S)}{\sum_{k=0}^{\infty} p_k u_k \sum_{i=0}^{k} (k-i) P(d_{\text{base}}(\mathcal{S}) = i|p_S)} \\ &= \left(\frac{d}{dx_I} g_S(x_S,x_I)\right) \Big/ \left(\frac{d}{dx_I} g_S(x_S,x_I)\right)\Big|_{x_S=x_I=1} \\ &= \frac{g'_\theta(x_S p_S + x_I(1-p_S))}{g'_\theta(1)}, \end{aligned} \tag{14}$$

in light of the binomial theorem and (12). Similarly, we obtain

$$\begin{aligned} g_{SS}(x_S, x_I) &= \frac{\sum_{k=0}^{\infty} p_k u_k \sum_{i=0}^{k} i x_S^{i-1} x_I^{k-i} P(d_{\text{base}}(\mathcal{S}) = i | p_S)}{\sum_{k=0}^{\infty} p_k u_k \sum_{i=0}^{k} i P(d_{\text{base}}(\mathcal{S}) = i | p_S)} \\ &= \left(\frac{d}{dx_S} g_S(x_S, x_I) \right) \Big/ \left(\frac{d}{dx_S} g_S(x_S, x_I) \right) \Big|_{x_S = x_I = 1} \\ &= \frac{g'_\theta(x_S p_S + x_I(1 - p_S))}{g'_\theta(1)} \\ &= g_{SI}(x_S, x_I). \end{aligned} \tag{15}$$

Finally, for (iv), we obtain by using (13),

$$\begin{aligned} g_{IS}(x_S, x_I) &= \frac{\sum_{k=0}^{\infty} p_k (1 - u_k) \sum_{i=0}^{k} i x_S^{i-1} x_I^{k-i} P(d_{\text{base}}(\mathcal{S}) = i | p_S)}{\sum_{k=0}^{\infty} p_k (1 - u_k) \sum_{i=0}^{k} i P(d_{\text{base}}(\mathcal{S}) = i | p_S)} \\ &= \left(\frac{d}{dx_S} g_I(x_S, x_I) \right) \Big/ \left(\frac{d}{dx_S} g_I(x_S, x_I) \right) \Big|_{x_S = x_I = 1} \\ &= \frac{g'_1(x_S p_S + x_I(1 - p_S)) - g'_\theta(x_S p_S + x_I(1 - p_S))}{g'_1(1) - g'_\theta(1)}, \end{aligned} \tag{16}$$

which yields (11) noting that $g'_1(1) = \langle k \rangle$. We have

$$\begin{aligned} g_{II}(x_S, x_I) &= \left(\frac{d}{dx_I} g_I(x_S, x_I) \right) \Big/ \left(\frac{d}{dx_I} g_I(x_S, x_I) \right) \Big|_{x_S = x_I = 1} \\ &= g_{IS}(x_S, x_I). \end{aligned} \tag{17}$$

The proof is thus complete. ∎

We remark that the infection rate β_{ij} and recovery rate γ are presumably involved in the probability p_S (as well as p_I), and the awareness affects p_S (as well as p_I) in an implicit manner. The same thing is true for the quantity θ as indicated by (6).

4 Discussions

In this paper, we present an analytical framework for studying the dynamical aspects of degree distributions among susceptible and infected individuals in a networked SIS model. Two forms of epidemiological awareness, i.e., contact awareness which increases with individual contact number, and local awareness which increases with the fraction of infected contacts, are considered. By extending probability generating function methods, we show that it is possible to track the evolution of the degree distributions among susceptible and infected individuals when the underlying contact network is modeled by the configuration model.

The results we obtain is somehow initial steps towards understanding analytically the time-dependent evolution of networked SIS model with awareness. What we really want is to write down the equations that actually track degree distributions. It is hoped that the distribution dynamics described in this paper will shed some light on the final solution of this issue. Other directions worthy of further investigation include awareness based on some memory mechanism,

misperception of risk induced by partial or incorrect information [32], and awareness concerning the prevalence of infection during an outbreak. We will work on some of these topics in the future.

References

[1] Kermack W O and McKendrick A G, A contribution to the mathematical theory of epidemics, *R. Soc. Lond. Proc. Ser. A*, 1927, **115**: 700–721.

[2] Brauer F and Castillo-Chávez C, *Mathematical Models in Population Biology and Epidemiology*, Springer, New York, 2001.

[3] Shang Y, A Lie algebra approach to susceptible-infected-susceptible epidemics, *Electron. J. Differ. Equ.*, 2012, **2012**(233): 1–7.

[4] Allard A, Noël P A, Dubé L J, and Pourbohloul B, Heterogeneous bond percolation on multitype networks with an application to epidemic dynamics, *Phys. Rev. E*, 2009, **79**: 036113.

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

[6] Keeling M, The implications of network structure for epidemic dynamics, *Theor. Popul. Biol.*, 2005, **67**: 1–8.

[7] Keeling M and Eames K T D, Networks and epidemic models, *J. R. Soc. Interface*, 2005, **2**: 295–307.

[8] Meyers L A, Pourbohloul B, Newman M E J, Skowronski D M, and Brunham R C, Network theroy and SARS: Predicting outbreak diversity, *J. Theor. Biol.*, 2005, **232**: 71–81.

[9] Pautasso M, Moslonka-Lefebvre M, and Jeger M J, The number of links to and from the starting node as a predictor of epidemic size in small-size directed networks, *Ecol. Complexity*, 2010, **7**: 424–432.

[10] Shang Y, Asymptotic behavior of estimates of link probability in random networks, *Rep. Math. Phys.*, 2011, **67**: 255–257.

[11] Altmann M, Susceptible-infected-removed epidemic models with dynamic partnerships, *J. Math. Biol.*, 1995, **33**: 661–675.

[12] Bauch C T, A versatile ODE approximation to a network model for the spread of sexually transmitted diseases, *J. Math. Biol.*, 2002, **45**: 375–395.

[13] Eames K T D and Keeling M J, Modeling dynamic and network heterogeneities in the spread of sexually transmitted disease, *Proc. Natl. Acad. Sci. USA*, 2002, **99**: 13330–13335.

[14] Simon P L, Taylor M, and Kiss I Z, Exact epidemic models on graphs using graph-automorphism driven lumping, *J. Math. Biol.*, 2011, **62**: 479–508.

[15] Barthélémy M, Barrat A, Pastor-Satorras R, and Vespignani A, Dynamical patterns of epidemic outbreaks in complex heterogeneous networks, *J. Theor. Biol.*, 2005, **235**: 275–288.

[16] Pastor-Satorras R and Vespignani A, Epidemic spreading in scale-free networks, *Phys. Rev. Lett.*, 2001, **86**: 3200–3203.

[17] Volz E, SIR dynamics in random networks with heterogeneous connectivity, *J. Math. Biol.*, 2008, **56**: 293–310.

[18] Molloy M and Reed B, A critical point for random graphs with a given degree sequence, *Random*

Struct. Algor., 1995, **6**: 161–179.

[19] Shang Y, Mixed SI(R) epidemic dynamics in random graphs with general degree distributions, *Appl. Math. Comput.*, 2013, **219**: 5042–5048.

[20] Shang Y, Distribution dynamics for SIS model on random networks, *J. Biol. Syst.*, 2012, **20**: 213–220.

[21] Ferguson N, Capturing human behaviour, *Nature*, 2007, **446**(7137): 733.

[22] Reluga T C, Game theory of social distancing in response to an epidemic, *PLoS Comput. Biol.*, 2010, **6**: e1000793.

[23] Tracht S M, Del Valle S Y, and Hyman J M, Mathematical modelling of the effectiveness of facemasks in reducing the spread of novel influenza A(H1N1), *PLoS One*, 2010, **5**: e9018.

[24] Funk S, Gilad E, Watkins C, and Jansen V A A, The spread of awareness and its impact on epidemic outbreaks, *Proc. Natl. Acad. Sci. U.S.A*, 2009, **106**: 6872–6877.

[25] Bagnoli F, Liò P, and Sguanci L, Risk perception in epidemic modeling, *Phys. Rev. E*, 2007, **76**: 061904.

[26] Kitchovitch S and Liò P, Risk perception and disease spread on social networks, *Procedia Comput. Sci.*, 2010, **1**: 2339–2348.

[27] Wu Q, Fu X, Small M, and Xu X J, The impact of awareness on epidemic spreading in networks, *Chaos*, 2012, **22**: 013101.

[28] Shang Y, Modeling epidemic spread with awareness and heterogeneous transmission rates in networks, *J. Biol. Phys.*, 2013, **39**(3): 489–500.

[29] Funk S, Salathé M, and Jansen V A A, Modelling the influence of human behaviour on the spread of infectious diseases: A review, *J. R. Soc. Interface*, 2010, **7**: 1247–1256.

[30] Funk S, Gilad E, and Jansen V A A, Endemic disease, awareness, and local behavioural response, *J. Theor. Biol.*, 2010, **264**: 501–509.

[31] Janson S, The probability that a random multigraph is simple, *Comb. Probab. Comput.*, 2009, **18**: 205–225.

[32] Shang Y, Efficient strategies for attack via partial information in scale-free networks, *Inf. Sci. Lett.*, 2012, **1**: 1–5.