J. Math. Biol. (2011) 62:349–358
DOI 10.1007/s00285-010-0337-9

Mathematical Biology

A note on a paper by Erik Volz: SIR dynamics in random networks

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Received: 28 September 2009 / Revised: 3 February 2010 / Published online: 23 March 2010

Abstract Recent work by Volz (J Math Biol 56:293–310, 2008) has shown how to calculate the growth and eventual decay of an SIR epidemic on a static random network, assuming infection and recovery each happen at constant rates. This calculation allows us to account for effects due to heterogeneity and finiteness of degree that are neglected in the standard mass-action SIR equations. In this note we offer an alternate derivation which arrives at a simpler—though equivalent—system of governing equations to that of Volz. This new derivation is more closely connected to the underlying physical processes, and the resulting equations are of comparable complexity to the mass-action SIR equations. We further show that earlier derivations of the final size of epidemics on networks can be reproduced using the same approach, thereby providing a common framework for calculating both the dynamics and the final size of an epidemic spreading on a random network. Under appropriate assumptions these equations reduce to the standard SIR equations, and we are able to estimate the magnitude of the error introduced by assuming the SIR equations.

Mathematics Subject Classification (2000) 92D25 · 92D30

1 Introduction

Infectious diseases are constrained to spread along the contacts of a population. Mathematical models investigating epidemics typically assume that the contacts occur through mass action mixing (Kermack and McKendrick 1927; Anderson and May 1991). However, true populations violate some mass-action assumptions in a manner affecting the epidemic dynamics. Recently a number of investigations have been

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performed using random networks (Newman 2002; Kenah and Robins 2007; Miller 2007; Noël et al. 2009; Meyers et al. 2005) which allow for a better accounting of mixing in the population.

Unlike mass-action models, random networks allow for the number of contacts individuals have to remain bounded as the population size increases. Thus, once an individual infects a contact, the number of available contacts to infect decreases by a non-negligible amount. Random networks also allow for more accurate representation of heterogeneities in the number of contacts compared with mass-action models. In a population with heterogeneous contact levels, individuals with more contacts are preferentially infected early in the epidemic (and in turn cause more infections), while at the end of the epidemic the remaining susceptibles tend to have fewer contacts.

A number of analytic results have been found for epidemic probability or size in random networks, but with only a few exceptions (notably Noël et al. 2009; Volz 2008), no analytic attention has been paid to the dynamics of the growth in networks. However, some attempts have been made using *pair approximations* which track the number of joined pairs of individuals with k_1 contacts and k_2 contacts in each infection state Eames and Keeling (2002) (assuming infection and recovery occur at constant rates). For a network with n different degrees, such a model results in an approximate system with $\mathcal{O}(n^2)$ coupled differential equations.

Recent work by Volz (2008) has shown that it is possible to investigate the dynamics of epidemic spread on Configuration Model networks (described below) using a coupled system of only three ODEs (again assuming infection and recovery occur at constant rates). The resulting system has many nonlinear terms, but the number of equations does not grow with the number of different degrees. In this note we derive a single differential equation with only a single higher order term that governs the dynamics. The framework we develop to calculate the dynamics can also be applied to predicting the final size of an epidemic in a concise way. We reproduce earlier results in this context.

Although our results are equivalent to pre-existing results, we place previous calculations of epidemic size and epidemic dynamics into a common framework. The equations we derive are simpler, and the terms in the equations are more easily interpreted. The resulting calculations for the numbers of susceptible, infected, and recovered individuals are of comparable complexity to the standard mass-action SIR equations, but allow for more realistic population interactions.

In Sect. 2, we develop the framework for the later sections. In Sect. 3 we apply this framework to calculating the time course of an epidemic. In Sect. 4 we apply this framework to calculating the final size of an epidemic. Finally in Sect. 5 we discuss the significance of these calculations.

2 The framework

We represent the population by a network. Each individual is thought of as a node joined to other nodes by edges through which disease can spread. We use configuration model (CM) networks (Newman 2003) to model the population. To generate a CM network, the degree or number of edges of each node, k, is assigned with

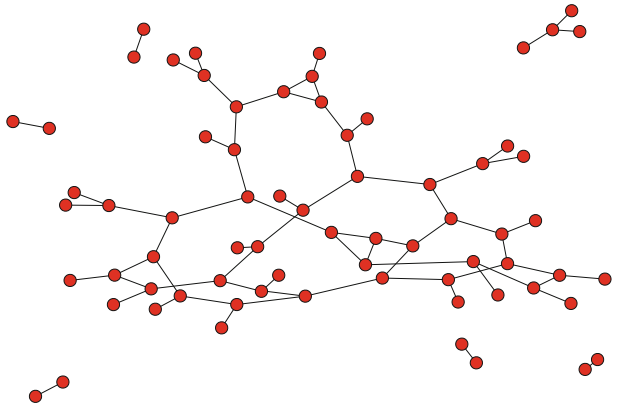

Fig. 1 A sample Configuration Model network with 70 nodes. The degrees are chosen using $P(3) = 0.5$ and $P(1) = 0.5$. Thus $\psi(x) = (x^3 + x)/2$

probability $P(k)$ based on a given degree distribution. If the sum of degrees is odd, all degrees are reassigned until the sum is even. Then each node is placed into a list with repetition equal to its degree, the list is randomized, and each node in position $2n$ ($n = 0, 1, \ldots$) is connected with the node in position $2n + 1$. The resulting network constitutes a uniform choice from the networks with the given degree distribution. In general the network may have self-loops or repeated edges. For degree distributions with finite mean, the density of loops and repeated edges goes to zero as the network size approaches infinity. Consequently, we ignore this effect. We define

$$\psi(x) = \sum_{k=0}^{\infty} P(k)x^k,$$

the probability generating function of the degree distribution. Note that $\psi'(1) = \langle K \rangle$ is the average degree. An example CM network is shown in Fig. 1. For many important distributions, ψ takes a simple form; for example, a Poisson distribution with parameter λ has $\psi(x) = e^{\lambda(x-1)}$.

Nodes in the network are assigned to one of three classes: susceptible, infected, or recovered. We denote the fraction of the population in each class by S, I, and R, respectively. A susceptible node becomes infected at rate $n\beta$ where n is the number of infected neighbors it has. Once infected, a node recovers at rate γ. A recovered node plays no further role in the spread. Typically an outbreak is initiated with a single randomly chosen infected individual in an otherwise susceptible population.

We define an *infectious contact* from v to its neighbor u to be a contact when v is infectious that would cause infection of u if u were susceptible. Physically this is the transmission of an infectious dose from v to u. An individual can cause infectious contact only when infected. However, an individual can receive an infectious contact regardless of his/her state, and so an infectious contact does not necessarily lead to infection.

We use θ as a measure of the probability that a random edge has not transmitted an infectious contact. Its precise definition is subtle, but important. To define θ, we

choose an edge uniformly from all edges. We then choose a direction for that edge, say from v to u. We refer to v as the base and u as the *target*. We modify the spread of the disease by disallowing infectious contacts from u to v. Then $\theta(\infty)$ is the probability that there is never an infectious contact from v to u, while $\theta(t)$ is the probability that at time t there has not been infectious contact from v to u. If we did not disallow infection from the target then an infection of u from some other source would in turn make infection of v more likely, which in turn makes infectious contact from v to u more likely, and so transmission along different edges to the same target would not be independent, thereby complicating the analysis.

Under the assumption that the spread is deterministic, the cumulative size of an epidemic at a given time is equal to the probability a randomly chosen node has been infected. Disallowing infection from that single randomly chosen node may impact the dynamics *after* that node is infected, but it does not modify the probability that that single node has become infected. Consequently, to calculate the size at a given time, it suffices to calculate the probability a randomly chosen node that cannot infect its neighbors has been infected, or alternately, is still susceptible. The important consequence of the definition of θ is that if a node has k contacts, its probability of being infected is θ^k. An alternate (equivalent) definition of θ is the probability that a node of degree 1 is susceptible Volz (2008).

3 Dynamics

To calculate the dynamics, we calculate the fraction of the population that has not yet been infected. To do this, we look at the probability that a randomly chosen node is not yet infected at time t. We choose a random target u and disallow infection from u to all of its neighbors. Using θ as defined above, if the degree of u is k, then the probability that u is still susceptible is $\theta(t)^k$. Thus the fraction of susceptibles is

$$S(t) = \sum_{k=0}^{\infty} P(k)\theta(t)^k = \psi(\theta(t)). \tag{1}$$

To calculate the rate of change of θ, we will need to know how many of those edges that have not transmitted an infectious contact have the opportunity to transmit infection at any given time. That is, we need to know what proportion of all edges have not had an infectious contact but come from an infected base node. We set ϕ to be the probability that the base node of an edge is infected but the edge has not transmitted infection (assuming as for θ that the target node does not cause infection). Those edges which satisfy the definition for ϕ are a subset of those which satisfy the definition for θ.

We derive coupled differential equations for θ and ϕ. The rate of change in the probability a random edge has not transmitted infection is equal to the rate at which infection crosses edges

$$\dot{\theta} = -\beta\phi. \tag{2}$$

An edge no longer satisfies the definition for ϕ when infection crosses the edge or when the base node recovers. An edge from v to u begins to satisfy the definition if v becomes infectious. The rate at which neighbors become infectious matches the rate at which neighbors stop being susceptible. We use $h(t)$ to denote the probability that a neighbor is susceptible, so $\dot{\phi} = -(\beta + \gamma)\phi - (d/dt)h(t)$.

We now find $h(t)$. A node is more likely to be a neighbor if it has more contacts Feld (1991), and so the probability the neighbor has degree k is $kP(k)/\langle K\rangle$. The neighbor can only be infected by an edge other than the one from the target node. Thus

$$h(t) = \frac{\sum_{k=0}^{\infty} kP(k)\theta^{k-1}}{\langle K\rangle} = \frac{\psi'(\theta)}{\psi'(1)}.$$

Thus the neighbor becomes infectious at rate $-(d/dt)h(t) = \beta\phi\psi''(\theta)/\psi'(1)$. We finally get

$$\dot{\phi} = \left[-\beta - \gamma + \beta\frac{\psi''(\theta)}{\psi'(1)}\right]\phi. \tag{3}$$

In fact, we can integrate this equation using Eq. (2) to get

$$\phi = \theta - \frac{\gamma}{\beta}(1-\theta) - \frac{\psi'(\theta)}{\psi'(1)},$$

where the constant of integration is found by taking $\phi(-\infty) = 0, \theta(-\infty) = 1$. The term θ represents the proportion of edges which have not yet transmitted infection, and the remaining terms subtract out the probability that an edge has not transmitted infection, but the base node is not infected. The term $(\gamma/\beta)(1-\theta)$ is the probability that the base node has been infected but recovered without an infectious contact, and $\psi'(\theta)/\psi'(1)$ is the probability that the base node is still susceptible. Consequently we arrive at

$$\dot{\theta} = -\beta\theta + \gamma(1-\theta) + \beta\frac{\psi'(\theta)}{\psi'(1)}. \tag{4}$$

The epidemiological quantity of interest is only rarely the proportion of edges which have or have not transmitted infection, but rather it is usually the values of S, I, and R. We can calculate $S(t) = \psi(\theta(t))$ directly. It is not difficult to show that $\dot{R} = \gamma I$, and conservation of individuals gives $I = 1 - S - R$. Consequently, we can augment Eq. (4) with

$$\dot{R} = \gamma I, \tag{5}$$
$$S = \psi(\theta), \tag{6}$$
$$I = 1 - R - S. \tag{7}$$

to find S, I, and R.

In order to solve our equations, we need to find appropriate initial conditions. At the earliest stages, the outbreak grows stochastically, and so the deterministic equations are not yet appropriate. If an epidemic occurs, eventually the outbreak infects a large number of nodes and then behaves effectively deterministically. In a sufficiently large population we can assume that deterministic behavior begins while the proportion infected is still small compared to the population.

Once the stochastic phase is over, we have $\theta = 1 - \epsilon$ with $\epsilon \ll 1$. At early time $\epsilon \propto \exp[(-\beta - \gamma + \beta\psi''(1)/\psi'(1))t]$ unless $\psi''(1)$ is infinite (which corresponds to an infinite variance in the degree distribution such as occurs in some power-law distributions). For simplicity we assume the $\psi''(1)$ is finite (if it were not, growth would not be exponential initially and this calculation would require more attention). We define $t = 0$ to correspond to a time when the epidemic is sufficiently large that the outbreak proceeds deterministically, but the proportion affected is still small. From the value of θ we can easily calculate $S(t) = \psi(\theta(t))$, and thus we can also calculate $I + R$.

To distinguish the number of current infections (I) from recovered infections (R) requires somewhat more effort. To find the early behavior for R, we note that I and ϵ are linearly related at early time, so that $I \propto \exp[(-\beta - \gamma + \beta\psi''(1)/\psi'(1))t]$. Then $\dot{R} = \gamma I$ gives $R = \gamma I/[-\beta - \gamma + \beta\psi''(1)/\psi'(1)]$. Combined with $I + R = 1 - S = 1 - \psi(1 - \epsilon)$ this gives R at $t = 0$.

We show a comparison of simulation with results calculated using Eq. (4) in Fig. 2. The results show good agreement, except for time shifts resulting from stochastic effects in the simulations while the outbreak size is still small. The final size (derived later) also matches the simulation.

3.1 Discussion

Equation (4) contrasts with the original system of Volz (2008) which uses three equations. In addition to the variable θ, the system of Volz (2008) uses $p_I = \phi/\theta$ (the probability that an edge is connected to an infected node given that it has not transmitted infection to the target node) in place of ϕ and an additional variable p_S (the probability that an edge is connected to a susceptible node given that it has not transmitted infection to the target node):

$$\begin{aligned}
\dot{\theta} &= -\beta p_I \theta, \\
\dot{p}_I &= p_I \left[\beta p_S \theta \frac{\psi''(\theta)}{\psi'(\theta)} - (\beta + \gamma) + \beta p_I \right], \\
\dot{p}_S &= \beta p_S p_I \left[1 - \theta \frac{\psi''(\theta)}{\psi'(\theta)} \right].
\end{aligned}$$

We have replaced this system by the single equation (4) with only one higher order term. To see that these systems are equivalent, we note the $\dot{p}_S$ equation can be eliminated by observing that the probability the neighbor has not been infected is $\psi'(\theta)/\psi'(1)$ and so $p_S = \psi'(\theta)/[\theta\psi'(1)]$. Equation (3) can be modified by using $\psi'(1) = \psi'(\theta)/[\theta p_S]$ and $\phi = \theta p_I$ to arrive at the same $\dot{p}_I$ equation.

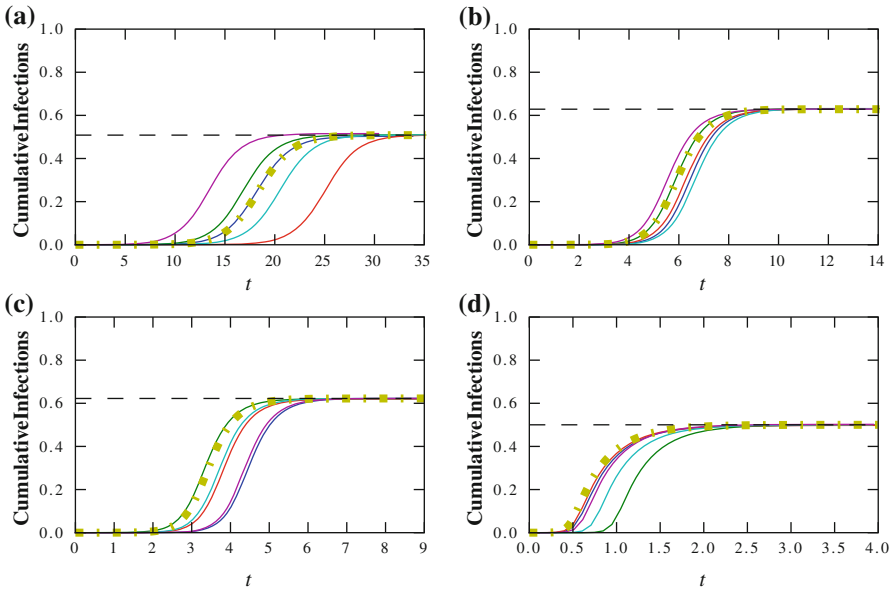

Fig. 2 Plots of cumulative infections $I + R$ against time. Predicted epidemic dynamics (*thick, broken curves*) and final sizes (*horizontal dashed lines*) compared with simulations (*solid curves*) in CM networks of 500,000 individuals with $\beta = 1.3$, $\gamma = 2$, and $\langle K \rangle = 4.0$. **a** Every node has the same degree. **b** Poisson degree distribution. **c** A bimodal distribution: $P(1) = 5/12$, $P(2) = 1/12$, $P(6) = 1/12$, and $P(7) = 5/12$. **d** A truncated powerlaw with $P(k) = 0.566e^{-k/40}k^{-1.728}$ for $1 \leq k < 80$

4 Final epidemic size

We now reproduce some of the earliest results for epidemics on networks by calculating the final size of epidemics in the limit of infinite networks (under the assumption that the outbreak does not die out during the stochastic phase) (Andersson 1999; Miller 2007; Newman 2002). Our approach is effectively the same as in Andersson (1999) and Miller (2007). We include it here to show the parallels with the method for calculating epidemic dynamics and to demonstrate a common framework for both.

We can find this by solving Eq. (4) for $\dot{\theta} = 0$. However, this approach is unnecessarily specific and we can easily generalize to disease processes that do not depend on constant infection and recovery rates by calculating $\theta(\infty)$ directly rather than through equations for the intermediate dynamics. To simplify notation in this section we use θ_∞ to represent $\theta(\infty)$ as we are not interested in the epidemic state at intermediate time.

To calculate the epidemic size, we look for the probability that a randomly chosen node u is never infected. If a node has degree k, then the probability that it is never infected is θ_∞^k. From this we get

$$S(\infty) = \sum_{k=0}^{\infty} P(k)\theta_\infty^k = \psi(\theta_\infty). \tag{8}$$

We must calculate θ_∞. For constant infection and recovery rates we set $T = \int_0^\infty \gamma e^{-\gamma\tau}(1 - e^{-\beta\tau})\,\mathrm{d}\tau = \beta/(\gamma + \beta)$. This is the probability that a randomly chosen neighbor has an infectious contact with u given that the neighbor becomes infected. If h is the probability that the neighbor does not become infected (given that u does not transmit infection), the probability of infectious contact is $T(1 - h)$. Thus the probability of not transmitting infection to u along the chosen edge is

$$\theta_\infty = 1 - T(1 - h) = 1 - T + Th.$$

An argument in the previous section shows that $h = \psi'(\theta_\infty)/\psi'(1)$, and so θ_∞ solves the implicit relation

$$\theta_\infty = 1 - T + T\frac{\psi'(\theta_\infty)}{\psi'(1)}. \tag{9}$$

The solution is easily found by iteration starting with $\theta_\infty < 1$ ($\theta_\infty = 1$ is always a solution but corresponds to outbreaks that die out). Using Eqs. (8) and (9) together gives $S(\infty)$, and the final size of an epidemic is simply $1 - S(\infty)$.

Note that the ability of a base node to infect a neighbor depends on duration of infection and whether the base node becomes infected. Consequently, infectious contacts along different edges out of the same node are not independent events (they both depend on the base node's properties). However, this does not affect our calculations because infectious contacts along different edges into the same node are independent events. If there were variation in susceptibility, more work would be needed (Miller 2007). Also the independence assumption will fail if short cycles are not negligible because infection of one neighbor is correlated with infection of another.

5 Discussion

We have shown that calculations for both the final size and the dynamics of an epidemic on a random network can be placed into a common framework. This framework allows us to simplify previous calculations of the dynamics Volz (2008). Our calculations closely match simulations, except for time shifts that result from stochastic effects when the infected population is still small. Our model is of similar complexity to the standard mass-action SIR equations but captures important details of the network structure.

The assumption that the network is random with no degree correlations is central to this derivation. If there is a tendency for high degree individuals to preferentially contact high degree individuals, these approaches do not directly apply. Similarly the presence of many short cycles will also affect these calculations. When a short cycle exists, whether or not one neighbor of the target node is still susceptible may no longer be independent of whether another neighbor is still susceptible.

Acknowledgments I am grateful to Erik Volz for useful comments. This work was developed while preparing a talk for the China–Canada Colloquium on Modeling Infectious Diseases in Xi'an, China September 2009. The work was supported by the RAPIDD program of the Science and Technology Directorate, Department of Homeland Security and the Fogarty International Center, National Institutes of Health.

Appendix A: Reduction to the standard SIR equations

Let us consider a large population of N individuals in which every individual has $C \leq N-1$ contacts. We will show that as $C \to \infty$ (and therefore $N \to \infty$ as well) with $\hat{\beta} = \beta C$ constant, the Eqs. (4)–(7) reduce to the standard SIR equations with

$$\begin{aligned} \dot{S} &= -\hat{\beta} I S \\ \dot{I} &= \hat{\beta} I S - \gamma I \\ \dot{R} &= \gamma I \end{aligned}$$

We already have the equation for $\dot{R}$. For I we have $I = 1-S-R$ and so $\dot{I} = -\dot{S}-\dot{R}$. Thus if we can show that $\dot{S} \to -\hat{\beta} I S$ as $C \to \infty$ we will be done. We have $S = \psi(\theta)$, so

$$\begin{aligned} \dot{S} &= \psi'(\theta)\dot{\theta} \\ &= -\psi'(\theta)\beta\phi \end{aligned}$$

However, $S = \psi(\theta) = \theta^C$ and thus $\psi'(\theta) = CS/\theta$. Consequently $\dot{S} = -C\beta S\phi/\theta = -\hat{\beta} S\phi/\theta$. We now show that $\phi/\theta \approx I$.

The probability an edge to a randomly chosen target node has not transmitted infection is θ (given that the target node does not cause any infection), and the probability that it satisfies this condition and it is connected to an infected node is ϕ. Because $C\beta = \hat{\beta}$ is taken to be constant [and thus $\mathcal{O}(1)$ as C grows], we know that only an $\mathcal{O}(I/C)$ proportion of edges to the target node may have transmitted infection from a node that is still infected. At large values of C, ϕ is $I - \mathcal{O}(I/C)$. Similarly, we can approximate θ as $1 - \mathcal{O}(1/C)$. Consequently, $\phi/\theta = I + \mathcal{O}(I/C)$, and so

$$\dot{S} = -\hat{\beta} I S + \mathcal{O}\left(\frac{IS}{C}\right)$$

giving the standard SIR equations at leading order in C. Note that if C is not large, the correction may be significant. This represents the fact that if the number of contacts an individual has is not large, then once one of those contacts has been infected, the reduction in susceptible contacts is not negligible.

If not everyone has the same degree, but the degrees are all sufficiently close to C, then the same approach will apply, so the restriction to constant degree is only a convenience that simplifies the calculations.

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