Intrinsic Unpredictability of Epidemic Outbreaks on Networks

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It has been known that epidemic outbreaks in the SIR model on networks are described by phase transitions. Despite the similarity with percolation transitions, whether an epidemic outbreak occurs or not cannot be predicted with probability one in the thermodynamic limit. We elucidate its mechanism by deriving a simple Langevin equation that captures an essential aspect of the phenomenon. We also calculate the probability of epidemic outbreaks near the transition point.

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I. INTRODUCTION

We start with the following question: How can it be determined whether an epidemic outbreak has occurred. Obviously, this is hard to answer, because an accurate model of epidemic spread in real societies, which include complicated and heterogeneous human-to-human contact, cannot be constructed. Then, is it possible to predict the outbreak for a simple mathematical model? Even in this case, the manner of the early spread of disease may significantly influence states that manifest after a sufficiently long time. For example, it seems reasonable to conjecture that whether a single infected individual with a very high infection rate causes an outbreak may depend on the number of people infected by the individual, which is essentially stochastic. In the present paper, we attempt to formulate this conjecture.

Specifically, we study the stochastic SIR model as the simplest epidemic model, where an edge in the network represents a human-to-human contact and the infection rate $\lambda$ (the infection probability per unit time in each edge) is a parameter of the SIR model (see e.g. Ref. [1] for an introduction to the stochastic SIR model; see also Refs. [2, 3] for related social dynamics on complex networks). The SIR model may be defined for well-mixed cases [4–8], homogeneous networks [10–15], and scale-free networks [16, 17]. A remarkable phenomenon is that when $\lambda > \lambda_c$, a disease spreads to macroscopic scales from a single infected individual, which corresponds to an epidemic outbreak. This was found in well-mixed cases and random graphs, but $\lambda_c = 0$ for scale free networks. That is, epidemic outbreaks are described as phase transition phenomena. In addition to the interest in theoretical problems, recently, the SIR model on networks has been studied so as to identify influential spreaders [18] and so as to determine a better immunization strategy [19, 20].

Although the phase transition in the SIR model may be a sort of percolation transition, its property is different from that of standard percolation models. In the SIR model exhibiting the phase transition, the order parameter characterizing it may be the fraction of the infected population, which is denoted by $\rho$. Indeed, $\rho = 0$ in the non-outbreak phase ($\lambda < \lambda_c$), whereas the expectation of $\rho$ becomes continuously non-zero from 0 when $\lambda > \lambda_c$. This phenomenon is in accordance with the standard percolation transition. However, on one hand, the order parameter in the percolated phase, e.g. the fraction of the largest cluster, takes a definite value with probability one in the thermodynamic limit; on the other hand, the fraction of the infected population in the SIR model is not uniquely determined even in the thermodynamic limit. In fact, it has been reported that the distribution function of the order parameter in SIR models with finite sizes shows two peaks at $\rho = 0$ and $\rho = \rho_*$ for well-mixed cases [3–8], homogeneous networks [10, 11, 13], and scale-free networks [18]. Mathematically, the probability density of $\rho$ in the thermodynamic limit may be expressed as

$$P(\rho; \lambda) = (1 - q(\lambda))\delta(\rho) + q(\lambda)\delta(\rho - \rho_*),$$

where $q = 0$ for $\lambda \leq \lambda_c$ and $q \neq 0$ for $\lambda > \lambda_c$. This means that the value of the fraction of the infected population in the outbreak phase, which is either 0 or $\rho_*(\lambda)$, cannot be predicted with certainty. We call this phenomenon the intrinsic unpredictability of epidemic outbreaks.

In this paper, we clarify the meaning of (1). We first observe the phenomenon in the SIR model defined on a random regular graph. By employing a mean field approximation, we describe the epidemic spread dynamics in terms of a master equation for two variables. Then, with a system size expansion, we approximate the solutions to the master equation by those to a Langevin equation. Now we can analyze this Langevin equation and work out the mechanism of the appearance of the two peaks. We also calculate $q(\lambda)$ near the transition point.

II. MODEL

Let $G$ be a random $k$-regular graph consisting of $N$ nodes. For each $x \in G$, the state $\sigma(x) \in \{S, I, R\}$ is defined, where S, I, and R represent Susceptible, Infective, and Recovered, respectively. The state of the whole system is given by $(\sigma_x)_{x \in G}$, which is denoted by $\sigma$ collectively. The SIR model on networks is described by a continuous time Markov process with infection rate

$$\lambda \sigma(x) \sigma(y)
$$
\(\lambda\) and recovery rate \(\mu\). Concretely, the transition rate \(W(\sigma \to \sigma')\) of the Markov process is given as

\[
W(\sigma \to \sigma') = \sum_{x \in G} w(\sigma \to \sigma'|x),
\]

(2)

with

\[
w(\sigma \to \sigma'|x) = \lambda \left[ \delta(\sigma_x,S)\delta(\sigma'_x,I) \sum_{y \in B(x)} \delta(\sigma_y,I) \right] + \mu \delta(\sigma_x,I)\delta(\sigma'_x,R),
\]

(3)

where \(B(x)\) is a set of \(k\)-adjacent nodes to \(x \in G\). Hereinafter, without loss of generality, we use dimensionless time by setting \(\mu = 1\). For almost all time sequences, infective nodes vanish after a sufficiently long time, and then the system reaches a stationary state, which is called the final state. The ratio of the total number of recovered nodes to \(N\) in the final state is equivalent to the fraction of the infected population \(\rho\). This quantity measures the extent of the epidemic spread. At \(t = 0\), we assume that \(\sigma = 1\) for only one node selected randomly and that \(\sigma = S\) for the other nodes.

In Fig. 1 as an example, we show the result of numerical simulations for the model with \(k = 3\) and \(N = 8192\). We measured the probability density \(P(\rho;\lambda)\) of the fraction of the infected population \(\rho\) for various values of \(\lambda\). This figure suggests that the expectation of \(\rho\) becomes non-zero when \(\lambda\) exceeds a critical value. The important observation here is that \(\log P(\rho;\lambda)\) is exactly given as

\[
\sum \frac{\partial}{\partial t} P(s,i,t) = N \left( i + \frac{1}{N} \right) P\left( s, i, \frac{1}{N}, t \right) - NiP(s,i,t) + N\lambda \left( s + \frac{1}{N} \right) \left( i - \frac{1}{N} \right) P\left( s + \frac{1}{N}, i - \frac{1}{N}, t \right) - N\lambda siP(s,i,t).
\]

(4)

loops is \(O(\log N)\). Now, as an approximation, we assume that there are \(Ni(k - 2)\) edges connecting the tree-like cluster with susceptible nodes \([24,25]\). Therefore, \(\psi\) is estimated as the rate of \(Ni(k - 2)\) to the number of all edges \(Nk\) in the thermodynamic limit. That is, \(\psi = i(k - 2)/k\). Below, we focus on the case \(k = 3\).

Let \(P(s,i,t)\) be the probability density of \(s(t) = s\) and \(i(t) = i\). Then, \(P(s,i,t)\) obeys the master equation

\[
\frac{\partial P(s,i,t)}{\partial t} = N \left( i + \frac{1}{N} \right) P\left( s, i, \frac{1}{N}, t \right) - NiP(s,i,t) + N\lambda \left( s + \frac{1}{N} \right) \left( i - \frac{1}{N} \right) P\left( s + \frac{1}{N}, i - \frac{1}{N}, t \right) - N\lambda siP(s,i,t).
\]

(4)

III. ANALYSIS

Defining two variables \(s \equiv \sum_x \delta(\sigma_x,S)/N\) and \(i \equiv \sum_x \delta(\sigma_x,I)/N\), we consider a continuous-time Markov process of the two variables as an approximation of the SIR model on the network \([22,23]\). We expect the phenomenon we are concerned with to be reproduced within this approximation; we verify this at a later stage. The transition rate of \((s,i) \to (s,i+1/N)\) is exactly given as \(Ni_s\), and we approximate the rate \((s,i) \to (s-1/N,i+1/N)\) as \(\lambda kN\psi\), where \(\psi\) is the probability of finding \(y \in B(x)\) such that \(\sigma_y = I\) for any \(x\). Here, the infective nodes form a connected cluster, and this cluster is tree-like because the typical size of the

\[
\frac{\partial P}{\partial t} + \partial_s J_s + \partial_i J_i + O\left( \frac{1}{N^2} \right) = 0,
\]

(5)
The infected population is given by Refs. [22, 23]. In this description, the fraction of the rule. The same equations as (7) and (8) were presented of \( \xi \)

\[ J_i = (\lambda s - 1) i P - \partial_i \left[ \frac{(\lambda s + 1) i}{2N} P \right] + \partial_i \left( \frac{\lambda s i}{2N} P \right), \]

\[ J_s = -\lambda s i P - \partial_i \left( \frac{\lambda s}{2N} P \right) + \partial_i \left( \frac{\lambda s i}{2N} P \right). \]  

By assuming that \( O(1/N^2) \) terms can be ignored, we obtain the Fokker-Planck equation [26].

It can be confirmed by direct calculation that this Fokker-Planck equation describes the time evolution of the probability density for the following set of Langevin equations:

\[ \frac{ds}{dt} = -\lambda s i - \sqrt{\frac{\lambda s}{N}} \cdot \xi_1, \]  

\[ \frac{di}{dt} = \lambda s i - i + \sqrt{\frac{\lambda s}{N}} \cdot \xi_1 + \sqrt{\frac{1}{N}} \cdot \xi_2. \]

where \( \xi_1 \) is Gaussian white noise that satisfies \( \langle \xi_1 (t) \rangle = 0 \) and \( \langle \xi_1 (t) \xi_1 (t') \rangle = \delta_{ij} \delta (t - t') \). The symbol “\( \cdot \)” in front of \( \xi_1 \) and \( \xi_2 \) in (7) and (8) represents the Ito product rule. The same equations as (7) and (8) were presented in Refs. [22, 23]. In this description, the fraction of the infected population is given by

\[ \rho = 1 - s(\infty). \]  

In Fig. 3 we show the result of numerical simulations of the Langevin equations (7) and (8). Comparing Fig. 3 with Fig. 1, we find that the phenomenon under study is described by the Langevin equations (7) and (8). Thus, our problem may be solved by analyzing them.

Now, the key idea of our analysis is the introduction of a new variable \( Y = \sqrt{N} \). Then, (7) and (8) are re-written as

\[ \frac{ds}{dt} = \frac{1}{N} \left[ -\lambda s Y^2 - \sqrt{\lambda s Y^2} \cdot \xi_1 \right], \]

\[ \frac{dY}{dt} = \frac{1}{2} \left\{ (\lambda s - 1) Y - \frac{1}{4} (\lambda s + 1) \frac{1}{Y} \right\} \]  

\[ + \frac{1}{2} \sqrt{\lambda s} \cdot \xi_1 + \frac{1}{2} \sqrt{Y} \cdot \xi_2, \]  

where it should be noted that the multiplication of the variable \( Y \) and the noise does not appear in (11). We then consider the probability \( q(\lambda) \) in the thermodynamic limit as the probability of observing \( Y \approx N^{1/2} \), because it is equivalent to \( \rho > 0 \).

Here, from (10) and (11), we find that the characteristic time scale of \( s \) is \( N \) times that of \( Y \). Thus, when \( N \) is sufficiently large, \( s \) almost retains its value when \( Y \) changes over time. In particular, it is reasonable to set \( s = 1 \) when \( t \) is shorter than \( N \). In this time interval, (11) is expressed as

\[ \frac{dY}{dt} = -\partial_Y U(Y) + \sqrt{2D} \xi, \]  

where \( D = (\lambda + 1)/8 \) and the potential \( U(Y) \) is calculated as

\[ U(Y) = -\frac{1}{4} (\lambda - 1) Y^2 + \frac{1}{8} (\lambda + 1) \log(Y). \]

\( \xi \) is Gaussian white noise with unit variance, where we have used the relation \( \sqrt{2\xi_1 + 1/2\xi_2} = \sqrt{\lambda + 1/2\xi} \). The initial condition is given as \( Y(0) = 1 \). It should be noted that (12) is independent of \( N \). Thus, solutions satisfying \( Y \approx N^{1/2} \) in (10) and (11) correspond to solutions satisfying \( Y \to \infty \) in (12). We identify \( q(\lambda) \) with the probability of finding these solutions. We now derive this probability.

First, we investigate the shapes of the graph \( U(Y) \). We find that \( U(0_+) = -\infty \) for any \( \lambda \) and that \( U(Y) \) monotonically increases in \( Y \) for \( \lambda < 1 \), while \( U(Y) \) has a single maximum peak at \( Y = Y_* \) for \( \lambda > 1 \), where

\[ Y_* = \frac{1}{2} \sqrt{\frac{\lambda + 1}{\lambda - 1}}. \]

As a reference, in Fig. 4 we show the shapes of \( U(Y) \) for \( \lambda = 0.5 \) and 1.2.

Next, based on the shapes of the potential function, we discuss the expected behavior of solutions to (12). When \( \lambda < 1 \), the probability of \( Y \to \infty \) is obviously zero because \( U(Y) \) is a monotonically increasing function in \( Y \).
that \( Y > 1 \) is complicated. We thus focus on the case that \( \lambda = 1 + \epsilon \), where \( \epsilon \) is a small positive number. In this case, \( Y_\ast \simeq \epsilon^{-1/2} \). We then note that if a solution \( Y \) to (12) happens to exceed \( Y_\ast \), it is comparatively likely that \( Y \to \infty \). Assuming that the probability of \( Y \to \infty \) under the condition \( Y \geq Y_\ast \) at some time is unity, we estimate \( q(\lambda) \) as the probability that \( Y \) exceeds \( Y_\ast \). Furthermore, we express \( q(\lambda) \) in terms of the transition rate \( T \) from \( Y = 1 \) to \( Y = Y_\ast \). Noting that the transition rate from \( Y = 1 \) to \( Y = 0 \) is equal to the recovery rate in the original SIR model, we can write

\[
q = \frac{T}{1 + T}.
\]  

(15)

Since \( T \) is positive and finite, we obtain \( 0 < q(\lambda) < 1 \). In this manner, we have clearly explained the probabilistic nature in the outbreak phase, and we have obtained \( \lambda_c = 1 \).

Finally, we calculate \( q(\lambda) \) quantitatively near the transition point. From \( Y_\ast \simeq \epsilon^{-1/2} \) and \( U(Y_\ast) \simeq \log \epsilon \), we estimate the slope of the straight line connecting two points \((1, U(1))\) and \((Y_\ast, U(Y_\ast))\) in the \((Y, U)\) plane as \( U(Y_\ast) - U(1))/(Y_\ast - 1) \simeq \sqrt{\epsilon} \log \epsilon \), which approaches zero in the limit \( \epsilon \to 0 \). Thus, the transition from \( Y = 1 \) to \( Y = Y_\ast \) may be assumed to be free Brownian motion with the diffusion constant \( D = (\lambda + 1)/8 \). The transition rate from \( Y = 1 \) to \( Y_\ast \) is then estimated as \( T = 2D/Y_\ast^2 = \epsilon + O(\epsilon^2) \). We thus obtain

\[
q(\lambda) = \epsilon + O(\epsilon^2).
\]  

(16)

In Fig. 5 we compare the theoretical result with those obtained in numerical simulations of (10) and (11). We measured the probability that \( \rho > 0.003 \), which is denoted as \( p(\rho > 0.003) \). Recall that \( \lim_{N \to \infty} p(\rho > 0.003) = q(\lambda) \) when \( \rho_*(\lambda) > 0.003 \). Since the experimental result suggests \( p(\rho > 0.003) = \epsilon + O(\epsilon^2) \) in the limit \( N \to \infty \), we claim that the theoretical result (16) is in good agreement with the experimental result.

**IV. CONCLUDING REMARKS**

In this paper, we have achieved a novel understanding of the intrinsic unpredictability of epidemic outbreaks by analyzing the Langevin equation (12), which effectively describes this singular phenomenon. Further, trajectories in the outbreak phase are divided into two groups: trajectories in one group are absorbed into zero, and the others diverge in (12). The division corresponds to the non-trivial limiting density given in (1). On the basis of this description, we calculated the probability of an epidemic outbreak near the transition point. Before ending the paper, we make a few remarks.

First, the probability \( q(\lambda) \) was studied in the mathematical literature (see [27] and [28] as reviews.) To the best of our knowledge, the method proposed in this paper has never been used in previous studies. It might be interesting to connect our analysis with mathematical studies.

Second, although we have investigated the simplest model in this paper, similar analysis might be applied to various models. For example, we can consider the case that there are \( m \) infected nodes at time \( t = 0 \). Since the essence of the phenomenon is the existence of \( Y_\ast \), the same result is obtained when \( m \) is independent of \( N \). However, for the case \( m = cN \) with a small positive number \( c \), \( Y(t) \) is never adsorbed to zero in the outbreak phase, because \( Y(0) \) is infinitely far away from \( Y = Y_\ast \). This is qualitatively different from the case \( m = 1 \), which was reported in Refs. [29, 30]. In fact, as suggested in Fig. 6, \( q(\lambda) \) jumps discontinuously to \( q(\lambda) = 1 \) which is similar to the behavior observed in standard percolation transitions.

Finally, as another generalization, one may study the behavior of the SIR on more complex networks. In these cases, since the mean field approximation might not be effective, one needs to devise a new technique to describe the unpredictability of outbreaks. Moreover, one of the most interesting is to predict probabilistic epidemic out-
breaks from limited data on realistic networks. We hope that future studies will address these problems.

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[1] L. Allen, in Mathematical Epidemiology, edited by F. Brauer et al., (Springer, Berlin, 2008), §3, p. 81.
[2] S. Boccaletti, V. Latora, Y. Moreno, M. Chavez, and D. U. Hwang, Phys. Rep. 424, 175 (2006).
[3] C. Castellano, S. Fortunato, and V. Loreto Rev. Mod. Phys. 81, 1275 (2009).
[4] N. T. J. Bailey, Biometrika 37, 193 (1950).
[5] N. T. J. Bailey, Biometrika 40, 177 (1953).
[6] J. A. J. Metz, Acta Biotheor. 27, 75 (1978).
[7] A. Martin-Löf, J. Appl. Probab. 35, 671 (1998).
[8] D. A. Kessler and N. M. Shnerb, Phys. Rev. E 76, 010901 (2007).
[9] B. S. Bayati and P. A. Eckhoff, Phys. Rev. E 86, 062103 (2012).
[10] O. Diekmann, M. C. M. de Jong, and J. A. J. Metz, J. Appl. Probab. 35, 448 (1998).
[11] D. H. Zanette, Phys. Rev. E 64, 050901 (2001).
[12] M. E. Newman, Phys. Rev. E 66, 016128 (2002).
[13] A. Lančić, N. Antulov-Fantulin, M. Šikić, and H. Štefančič, Physica A 390, 65 (2011).
[14] T. Bohman and M. Picollelli, Random Struct. Algor. 41, 179 (2012).
[15] Y. Moreno, R. Pastor-Satorras, and A. Vespignani, Eur. Phys. J. B 26, 521 (2002).
[16] R. Pastor-Satorras and A. Vespignani, Phys. Rev. Lett. 86, 3200 (2001).
[17] R. M. May and A. L. Lloyd, Phys. Rev. E 64, 066112 (2001).
[18] L. K. Gallos and P. Argyrakis, Physica A 330, 117 (2003).
[19] M. Kitsak, L. K. Gallos, S. Havlin, F. Liljeros, L. Muchnik, H. E. Stanley, and H. A. Makse, Nature Phys. 6, 888 (2010).
[20] R. Cohen, S. Havlin, and D. Avraham, Phys. Rev. Lett. 91, 247901 (2003).
[21] Y. Chen, G. Paul, S. Havlin, F. Liljeros, and H. E. Stanley, Phys. Rev. Lett. 101, 058701 (2008).
[22] L. Hufnagel, D. Brockmann, and T. Geisel, Proc. Natl. Acad. Sci. 101, 15124 (2004).
[23] V. Colizza, A. Barrat, M. Barthélemy, and A. Vespignani, Bull. Math. Biol. 68, 1893 (2006).
[24] B. Derrida and Y. Pomeau, Europhys. Lett. 1, 45 (1986).
[25] M. J. Keeling and K. T. D. Eames, J. R. Soc. Interface 2, 295 (2005).
[26] C. Gardiner, Handbook of Stochastic Methods: for Physics, Chemistry and the Natural Sciences (Springer, Berlin, 2004).
[27] P. Yan, in Mathematical Epidemiology, edited by F. Brauer et al., (Springer, Berlin, 2008), §10.5, p. 261.
[28] T. Britton, Math. Biosci. 225, 24 (2010).
[29] A. D. Barbour, Adv. Appl. Probab. 6, 21 (1974).
[30] J. C. Miller, [arXiv:1208.3438] (2012).