

Pulse strategy for suppressing spreading on networks

Liu, Qiang; Zhou, Xiaoyu; Van Mieghem, Piet

DOI

[10.1209/0295-5075/127/38001](https://doi.org/10.1209/0295-5075/127/38001)

Publication date

2019

Document Version

Accepted author manuscript

Published in

EPL

Citation (APA)

Liu, Q., Zhou, X., & Van Mieghem, P. (2019). Pulse strategy for suppressing spreading on networks. *EPL*, 127(3), 38001-p1 - 38001-p4. Article 38001. <https://doi.org/10.1209/0295-5075/127/38001>

Important note

To cite this publication, please use the final published version (if applicable).
Please check the document version above.

Copyright

Other than for strictly personal use, it is not permitted to download, forward or distribute the text or part of it, without the consent of the author(s) and/or copyright holder(s), unless the work is under an open content license such as Creative Commons.

Takedown policy

Please contact us and provide details if you believe this document breaches copyrights.
We will remove access to the work immediately and investigate your claim.

Pulse strategy for suppressing spreading on networks

QIANG LIU¹, XIAOYU ZHOU¹ and PIET VAN MIEGHEM¹

¹ *Faculty of Electrical Engineering, Mathematics and Computer Science, Delft University of Technology, Delft, the Netherlands*

PACS 89.75.Hc – Networks and genealogical trees

Abstract – In previous modelling effort to understand the spreading process on networks, each node can infect its neighbors and cure spontaneously, and the curing is traditionally assumed to occur uniformly over time. This traditional curing is not optimal in terms of the trade-off between the effectiveness and cost. A pulse immunization/curing strategy is more efficient and broadly applied to suppress spreading process. We analyze the pulse curing strategy on networks with the Susceptible-Infected (SI) process. We analytically compute the mean-field epidemic threshold τ_c^p of the pulse SI model and show that $\tau_c^p = \frac{1}{\lambda_1} \ln \frac{1}{1-p}$, where λ_1 and p are the largest eigenvalue of the adjacency matrix of the contact graph and the fraction of nodes covered by each curing, respectively. These analytical results agree with simulations. Compared to the asynchronous curing process in the extensively studied Markovian SIS process, we show that the pulse curing strategy saves about 36.8%, i.e. $p \approx 0.632$, of the number of curing operations invariant to the network structure. Our results may help policymakers to design optimal containment strategies and minimize the controlling cost.

Background. – Viral spreading processes cause enormous losses of life. Due to the pandemic influenza A H1N1, 18500 laboratory-confirmed deaths are reported, while 284500 deaths are estimated during the period 2009.04 to 2010.08 [1]. Cyber-criminals earned around \$100 million per year by spreading an exploit kit, *Angler*, in computer systems [2]. A recent study shows that false news spreads faster and more broadly than true news online [3]. The suppression of spreading processes is thus necessary in many circumstances, but consumes resources, e.g. budget in disease control or computational resources in detecting computer viruses. Based on the data from the World Health Organization, around 19.9 million children under the age of one still cannot receive the basic diphtheria-tetanus-pertussis (DTP3) vaccine and the coverage level of DTP3 for infants is only about 85% in 2017. Cisco reported [2] that 83% of the Internet of Things devices are not patched to be immunized against cyber-attacks.

Suppressing spreading requires a strategic design to balance between the cost and performance. A straightforward strategy is the uniform, asynchronous strategy: each infected individual can be cured uniformly over time as a Poisson process and thus the curing is asynchronous among infected individuals. This strategy is weak in preventing reinfections between direct neighbors because an cured individual can still have an infected neighbor. A pulse/synchronous strategy, where two direct neighbors have a high probability to be cured at the same time, is more efficient. The pulse strategy was first proposed to control the epidemic of measles [4] by periodically and synchronously vaccinating several age cohorts

instead of uniformly and asynchronously vaccinating each individual at certain ages [5, 6]. In 1995, India introduced the National Immunization Days, which is a pulse strategy, to control the spread of polio [7]. Compared to the uniform, asynchronous strategy, the pulse strategy shows a better performance [8].

Furthermore, spreading processes are also focal in network science, because the underlying contact graph influences the spreading process non-trivially. For example, the epidemic threshold, which is determined by the network structure, of scale-free networks converges to zero with the network size under the mean-field approximation [9–13]. The spreading processes studied on networks are generally Markovian, which means that the infection and curing events occur both uniformly over time [14]. As mentioned earlier, the pulse strategy reduces the reinfections between neighboring nodes. If the curing occurs for all nodes at the same time, then no reinfection happens and the disease is immediately eradicated. If the curing only covers a fraction p of the whole population, synchronous curing with the pulse strategy still eliminates a substantial part of reinfections between neighbors and thus leads to better performance compared to a uniform, asynchronous curing strategy. Thus, one may wonder how efficient the pulse strategy is. The most reasonable way to quantify the effectiveness of the pulse strategy lies in assessing the reduction of the number of curing operations by using the asynchronous strategy as a benchmark. In the following, we consider the most basic spreading model on networks: the Susceptible-Infected (SI) process and evaluate the pulse strategy performed on the SI model. Here, we refer to the curing as a strategy, because we are focusing on curing actions that can be performed in synchronous/pulse or asynchronous manner by the public health department or cybersecurity team. In contrast and beyond our scope, individuals may be spontaneously cured by the immune system during an epidemic outbreak, which is essentially an asynchronous curing.

The Model. – In the networked spreading process, each node in the network is either infected or susceptible (healthy). Each infected node can infect each healthy neighbor by a Poisson process with rate β . We assume that each node is cured with rate δ . Thus, for the pulse curing strategy, the curing happens every $1/\delta$ time units, i.e. the nodes can only be synchronously cured at time k/δ for $k = 1, 2, \dots$. The curing has a successful probability p turning an infected node into a healthy one. Equivalently, each node can be cured certainly, but only a fraction p of nodes are randomly chosen to be cured. We define the effective infection rate $\tau \triangleq \beta/\delta$.

The difference between the above pulse curing SI model and the extensively studied Markovian Susceptible-Infected-Susceptible (SIS) model [15] is that each node in the Markovian SIS model is cured by a Poisson process with rate δ and $p = 1$, which represents an asynchronous curing strategy. In the Markovian SIS process on networks, there exists an epidemic threshold [12, 13] under the N -Intertwined mean-field approximation $\tau_c^{(1)} = \frac{1}{\lambda_1}$ where λ_1 is the largest eigenvalue of the adjacency matrix of the network. If $\tau > \tau_c^{(1)}$, then the process is in an endemic phase in the steady state, but if $\tau < \tau_c^{(1)}$, then the process converges to the all-healthy state. In the pulse curing strategy, limited resources or some other complications may lead to a partial coverage specified by a fraction $p < 1$. If $p = 1$, then synchronous curing destroys the spreading immediately. The average numbers of curing operations in the asynchronous Poisson curing and the pulse curing are δ and δp , respectively, for each node during one unit of time. In the following, we analyze the pulse curing effect on epidemic processes on networks under the mean-field theory to derive the epidemic threshold. Our main finding is that when $p = 1 - 1/e \approx 0.632$, the pulse curing is equally effective than Poisson curing process with the same curing rate δ .

Mathematical analysis. We represent the time t in the form of $t = k/\delta + t^*$, where $t^* \in [0, 1/\delta)$. For $t^* \neq 0$, only infection happens and the mean-field equation of node i is

$$\frac{dv_i(k/\delta + t^*)}{dt^*} = \beta [1 - v_i(k/\delta + t^*)] \sum_{j=1}^N a_{ij} v_j(k/\delta + t^*) \quad (1)$$

where $v_i(k/\delta + t^*)$ is the probability that node i is infected at time $t = k/\delta + t^*$ and $a_{ij} \in \{0, 1\}$ is the element of adjacency matrix of the network with N nodes. The probability $v_i(k/\delta + t^*)$ is discontinuous at $t^* = 0$ for all k when curing happens: $\lim_{t^* \rightarrow 0} v_i(k/\delta + t^*) = v_i(k/\delta)$ and $\lim_{t^* \rightarrow 1/\delta} v_i(k/\delta + t^*) \neq v_i((k+1)/\delta)$. Equation (1) is a mean-field approximation, because we omit the correlation of the infection state between neighbors just as in the Markovian SIS process [16]. Since the curing probability of each node at k/δ is p , the pulse curing process is governed by the following equation,

$$v_i\left(\frac{k+1}{\delta}\right) = (1-p) \lim_{t^* \rightarrow 1/\delta} v_i\left(\frac{k}{\delta} + t^*\right) \quad (2)$$

In our previous work [17], we introduced the bursty SIS model, where the infection happens periodically with rate β and the curing is a Poisson process. In the bursty SIS model, the relationship between the infection probability of each node at the start $t^* = 0$ and the end $t^* \rightarrow 1/\beta$ of the same time interval is explicitly known as an exponentially decreasing function. In pulse curing, the relationship between $v_i(k/\delta)$ and $\lim_{t^* \rightarrow 1/\delta} v_i(k/\delta + t^*)$ is described by (1), which does not have an explicit solution for general networks¹. However, since we only care about the regime where $v_i(k/\delta + t^*) \rightarrow 0$ to derive the epidemic threshold, we can first linearize Eq. (1) around $v_i(k/\delta + t^*) = 0$ for all i and obtain

$$\frac{d\mathbf{v}(k/\delta + t^*)}{dt^*} = \beta A \mathbf{v}(k/\delta + t^*) \quad (3)$$

where the infection probability vector $\mathbf{v}(k/\delta + t^*) \triangleq [v_1(k/\delta + t^*), \dots, v_N(k/\delta + t^*)]^T$. The general solution [14, p. 209] of (3) is $\mathbf{v}(k/\delta + t^*) = e^{\beta A t^*} C$ where $C = \mathbf{v}(k/\delta)$ is the initial value vector at $t^* = 0$. Thus, the solution of Eq. (3) evaluated at $t^* \rightarrow 1/\delta$ is

$$\lim_{t^* \rightarrow 1/\delta} \mathbf{v}(k/\delta + t^*) = e^{\tau A} \mathbf{v}(k/\delta) \quad (4)$$

Substituting (4) into the curing equation (2) yields

$$\mathbf{v}\left(\frac{k+1}{\delta}\right) = (1-p) e^{\tau A} \mathbf{v}\left(\frac{k}{\delta}\right) \quad (5)$$

When the largest eigenvalue of $(1-p)e^{\tau A}$, which is $(1-p)e^{\tau \lambda_1}$, is smaller than 1, eq. (5) illustrates that the infection probability $\mathbf{v}\left(\frac{k}{\delta}\right)$ converges to zero in the long run. Thus, for $(1-p)e^{\tau \lambda_1} = 1$, we obtain the epidemic threshold

$$\tau_c^p \triangleq \frac{1}{\lambda_1} \ln \frac{1}{1-p} \quad (6)$$

If $\tau > \tau_c^p$, then the spreading can persist in the network, which is the endemic phase. If $\tau < \tau_c^p$, then the spreading disappears in the long run, which is the all-healthy phase.

¹Only for the regular graph when the initial condition of each node is identical, there is an explicit solution for (1). One may verify for the d -regular graph that $v_i(k/\delta) = (1-p) - e^{-d\tau}$. Let $v_i(k/\delta) = 0$ and the threshold is $\frac{1}{d} \ln \frac{1}{1-p}$ which is consistent with (6)

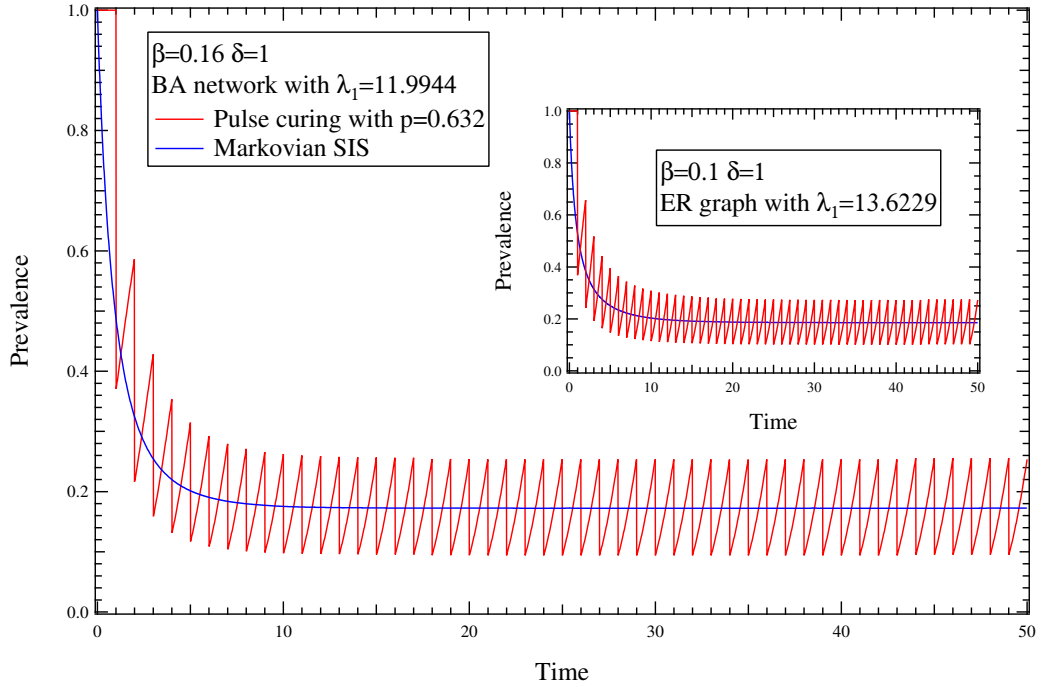


Fig. 1: The prevalence of the Markovian SIS model and the pulse curing model obtained by averaging 10^5 simulated realizations. The simulation is performed on a $N = 500$ -node network generated by the Barabási-Albert model and a $N = 500$ -node ER graph. The curing probability is set to be $p = 0.632$ for the pulse strategy.

The Markovian SIS process with a Poisson curing process has a mean-field epidemic threshold $\frac{1}{\lambda_1}$. When $\ln \frac{1}{1-p} = 1$ or $p = 1 - 1/e \approx 0.632$, the pulse curing is equivalent to the Poisson curing process in the traditional SIS model on any graph. Thus, to eliminate the spreading, the pulse strategy only consumes 63.2% of the number of curing operations of the asynchronous strategy, since the curing rates δ of the two strategies are equal. In the next section, two typical examples show that even above the epidemic threshold, the two strategies are comparable, if $p = 0.632$.

Simulations: above the epidemic threshold. In Fig. 1, we show the prevalence, which is the average fraction of the infected nodes, of the Markovian SIS model and the pulse curing model with $p = 0.632$, on a Barabási-Albert graph [18] and an Erdős-Rényi (ER) graph. The effective infection rates τ are above the epidemic thresholds $1/\lambda_1$. The prevalence of the Markovian SIS model is exactly centered in the middle of the prevalence generated by the pulse curing SI model. Figure 1 indicates that the two curing processes are equivalent to some extents at $p = 0.632$, even above the epidemic threshold.

The phase diagram and the parameter selection in spreading control. Figure 2 shows the phase diagram of the pulse curing strategy with the mean-field epidemic threshold calculated by (6). For small coverage p , the threshold τ_c^p increases slowly with p ; While for large p , there is an increased effectiveness of p in the pulse strategy.

For a spreading process in the endemic phase, one can tune both the curing rate δ and the curing coverage p to move the process from the endemic phase to the all-healthy phase. Fig. 2 shows that there are many different ways to achieve this. However, the optimal way is just increasing the curing coverage p and decreasing the curing rate δ along the red curve. The argument is as follows. From (6), we have that $\delta = \lambda_1 \beta / \ln[1/(1-p)]$ and thus $\delta p = \lambda_1 \beta p / \ln[1/(1-p)]$, when $\tau = \tau_c^p$. The goal is to minimize the average number of curing

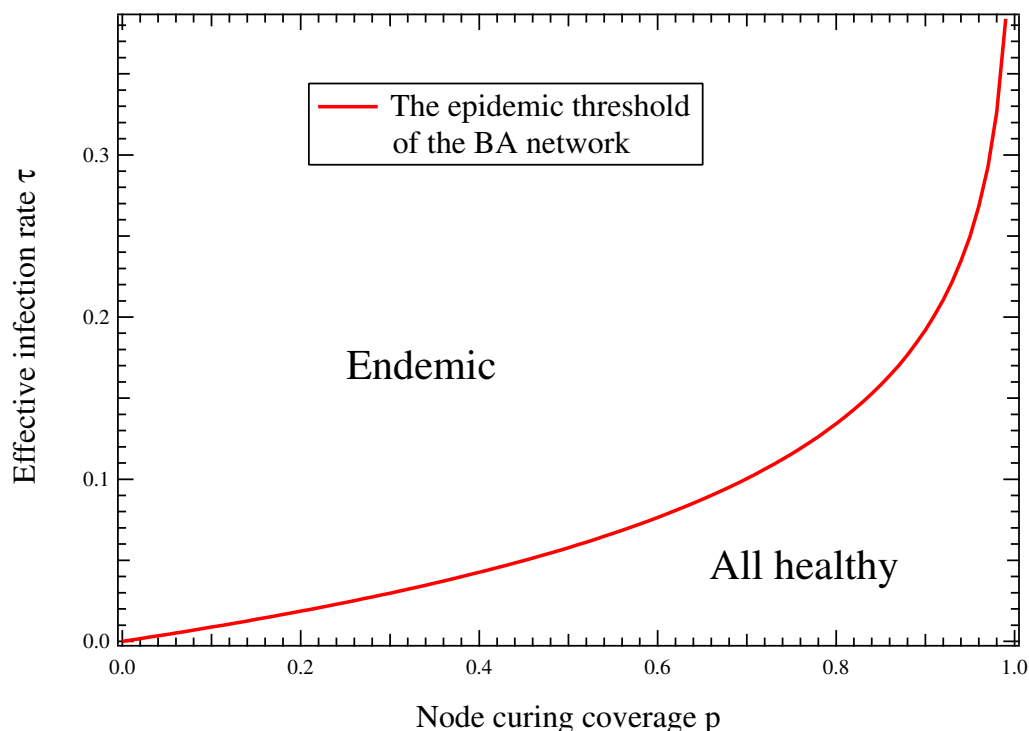


Fig. 2: The phase diagram of the BA network calculated by Eq. (6).

operations δpN during one time unit, which asks to minimize $p/\ln[1/(1-p)]$. One may verify that $p/\ln[1/(1-p)]$ is monotonically decreasing with p in $(0, 1)$ and thus increasing p along the red curve in the phase diagram is the optimal way of choosing δ and p to suppress spreading. The result is reasonable, because a large p can probably shut down the spreading within a few curing pulses. In real scenarios, the coverage p may be restricted and thus choosing the maximum possible p and a corresponding δ is an option.

Conclusion. – We quantified the effect of the pulse strategy for suppressing spreading processes on networks. To achieve an equivalent effect, the pulse strategy consumes 63.2% of the total number of curing operations, required by the uniform and asynchronous strategy, e.g. a Poisson curing process. This reduction of cost does not depend on the underlying contact graph in the mean-field approximation. Our results may help the agencies, e.g. disease control centers or computer security teams, to make policies or allocate resources.

Q. Liu is thankful for the support from China Scholarship Council.

REFERENCES

- [1] DAWOOD F. S., IULIANO A. D., REED C., MELTZER M. I., SHAY D. K., CHENG P.-Y., BANDARANAYAKE D., BREIMAN R. F., BROOKS W. A., BUCHY P., FEIKIN D. R., FOWLER K. B., GORDON A., HIEN N. T., HORBY P., HUANG Q. S., KATZ M. A., KRISHNAN A., LAL R., MONTGOMERY J. M., MLBAK K., PEBODY R., PRESANIS A. M., RAZURI H., STEENS A., TINOCO Y. O., WALLINGA J., YU H., VONG S., BRESEE J. and WIDDOWSON M.-A., *The Lancet Infectious Diseases*, **12** (2012) 687 .
<http://www.sciencedirect.com/science/article/pii/S1473309912701214>

- [2] CISCO SYSTEMS I., *Cisco 2018 annual cybersecurity report* (2018).
- [3] VOSOUGHI S., ROY D. and ARAL S., *Science*, **359** (2018) 1146.
<https://science.sciencemag.org/content/359/6380/1146>
- [4] DABBAGH A., LAWS R. L., STEULET C., DUMOLARD L., MULDER M. N., KRETSINGER K., ALEXANDER J. P., ROTA P. A. and GOODSON J. L., *MMWR. Morbidity and Mortality Weekly Report*, **67** (2018) 1323.
<https://doi.org/10.15585/mmwr.mm6747a6>
- [5] AGUR Z., COJOCARU L., MAZOR G., ANDERSON R. M. and DANON Y. L., *Proceedings of the National Academy of Sciences*, **90** (1993) 11698.
<https://www.pnas.org/content/90/24/11698>
- [6] STONE L., SHULGIN B. and AGUR Z., *Mathematical and Computer Modelling*, **31** (2000) 207
proceedings of the Conference on Dynamical Systems in Biology and Medicine.
<http://www.sciencedirect.com/science/article/pii/S0895717700000406>
- [7] OF HEALTH M. and OF INDIA F. W., *Operational guide for pulse polio immunization in india* (2006).
- [8] NOKES D. J. and SWINTON J., *Trends in Microbiology*, **5** (1997) 14 .
<http://www.sciencedirect.com/science/article/pii/S0966842X97817696>
- [9] PASTOR-SATORRAS R. and VESPIGNANI A., *Phys. Rev. Lett.*, **86** (2001) 3200.
<https://link.aps.org/doi/10.1103/PhysRevLett.86.3200>
- [10] CHATTERJEE S. and DURRETT R., *The Annals of Probability*, **37** (2009) 2332.
<http://www.jstor.org/stable/27795079>
- [11] CHUNG F., LU L. and VU V., *Annals of Combinatorics*, **7** (2003) 21.
<https://doi.org/10.1007/s000260300002>
- [12] VAN MIEGHEM P., OMIĆ J. and KOOIJ R., *IEEE/ACM Transactions on Networking*, **17** (2009) 1.
- [13] CASTELLANO C. and PASTOR-SATORRAS R., *Phys. Rev. Lett.*, **105** (2010) 218701.
<https://link.aps.org/doi/10.1103/PhysRevLett.105.218701>
- [14] VAN MIEGHEM P., *Performance Analysis of Complex Networks and Systems* (Cambridge University Press) 2014.
- [15] PASTOR-SATORRAS R., CASTELLANO C., VAN MIEGHEM P. and VESPIGNANI A., *Reviews of modern physics*, **87** (2015) 925.
- [16] CATOR E. and VAN MIEGHEM P., *Phys. Rev. E*, **89** (2014) 052802.
<https://link.aps.org/doi/10.1103/PhysRevE.89.052802>
- [17] LIU Q. and VAN MIEGHEM P., *Phys. Rev. E*, **97** (2018) 022309.
<https://link.aps.org/doi/10.1103/PhysRevE.97.022309>
- [18] BARABÁSI A.-L. and ALBERT R., *Science*, **286** (1999) 509.
<https://science.sciencemag.org/content/286/5439/509>