Contact process on evolving inhomogeneous random graphs

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joint work with

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Contact process

Contact process or SIS infection is a popular model of infection on a finite or locally finite graph $G = (V, E)$. The infection is modeled by a Markov process (X_t) with values in $\{0,1\}^V$, with the interpretation

$$X_t(v) = \begin{cases} 1 & \text{if } v \text{ is infected at time t} \\ 0 & \text{if } v \text{ is healthy at time t} \end{cases}$$

Dynamics:

- An infected vertex infects every of its neighbours at rate λ.
- An infected vertex recovers (turns to healthy) at rate 1.

Remarks:

- Sometimes, we identify X_t with $\{v \in V, X_t(v) = 1\} \in \mathcal{P}(V)$.
- On a finite graph, this Markov process ends up in the absorbing state $\{0\}^V$, when every vertex is healthy and the infection is extinct.
- We will particularly focus on the extinction time T_{ext}.

Graphical representation

By attaching a Poisson point process of intensity λ on every edge and a Poisson point process of intensity 1 on every vertex, we construct the graphical representation of the process, from which we easily get

- A coupling of the processes $X_t^{(A)}$ for different starting infection sets $A \subset V$.
- The duality property of the process: One can construct $(\tilde{X}_t)_{t \in [0,T]}$ another contact process such that

$$\forall A, B \subset V, X_T^{(A)} \cap B = \emptyset \Leftrightarrow \tilde{X}_T^{(B)} \cap A = \emptyset.$$

Inhomogeneous random graphs

- Inhomogeneous random graph with kernel p:
  - $G_N = (V_N, E_N)$ random graph on vertex set $V = V_N = \{1/N, 2/N, \ldots, 1\}$.
  - Keep each edge $\{x, y\}$ in E_N independently and with probability

$$p_{x,y} = \frac{p(x,y)}{N} \wedge 1.$$

  - Factor kernel: $p(x,y) = \beta x^{-\gamma} y^{-\gamma}$ for some $\beta > 0$ gives Chung-Lu model (same class as configuration model).
  - Preferential attachment kernel: $p(x,y) = \beta (x \wedge y)^{-\gamma} (x \vee y)^{\gamma - 1}$ for some $\beta > 0$.
- The degree of vertex x is asymptotically Poisson with parameter $\int_0^1 p(x,y)\mathrm{d}y \sim cx^{-\gamma}$. The smaller x, the stronger the vertex...
- As $N \to +\infty$, the degree distribution in the graph converges (in probability) to a mixed Poisson distribution μ satisfying $\mu(k) = k^{-\tau + o(1)}$, with $\tau = 1 + 1/\gamma$. We say the network is scale-free, with power-law exponent $\tau = 1 + 1/\gamma \in (2, +\infty)$.

metastability versus fast extinction

- We say there is fast extinction for the contact process on G if $\mathbb{E}[T_{ext}]$ is at most polynomial in N.
- We say there is long survival for the contact process on G if $\mathbb{E}[T_{ext}]$ is exponential in N, namely if there exists $c > 0$ such that $\mathbb{E}[T_{ext}] \geq e^{cN}$.
- In the case of long survival, we say there is metastability with metastable density ρ if the expected number of infected vertices at time t_N converges to ρ as $N \to +\infty$, for any sequence t_N going to $+\infty$ slower than exponentially.

mean-field approximation

- A mean-field approximation of the contact process is obtained by considering the infection process on the complete graph, where the infection is transmitted along each edge $\{x, y\}$ with rate $\lambda p_{x,y}$.
- This mean-field model is easier to study. When $\tau \in (2, 3)$, there is always long survival, while when $\tau > 3$, there is fast extinction for small $\lambda > 0$.
- Based on this, Vespignani and Pastor-Satorras (early 2000's) predicted one should observe the same phenomenon for the true contact process (on the configuration model).
- This was disproved by Berger, Borgs, Chayes, Saberi ('05), and by the further works of Chatterjee and Durrett ('09), and later Mountford, Mourrat, Valesin, Yao ('11 to '15)

Metastability on static graph

A selected result:

Theorem MMVY 2013

- On the configuration model, for every $\tau > 2$ and every $\lambda > 0$, the contact process on the (static) configuration model exhibits long survival and metastability.
- The metastable density satisfies $\rho(\lambda) = \lambda^{\mathfrak{e}(\tau)+o(1)}$, where the exponent $\mathfrak{e}(\tau)$ has the form

$$\mathfrak{e}(\tau) = \begin{cases} \frac{1}{3-\tau} & \text{if } \tau < \frac{5}{2}, \\ 2\tau - 3 & \text{if } \tau \geq \frac{5}{2}. \end{cases} \tag{1}$$

The full result is even more precise and establishes the metastable density up to a constant multiplicative factor.

Evolving inhomogeneous random graphs

We now consider one of the following dynamics for our graph:

- Vertex updating dynamics: every vertex x independently updates its connections at rate $\kappa_x = x^{-\gamma\eta}$. When vertex x updates, all edges $\{x, y\}$ are updated, and thus belong to E_t, independently and independently of the past, with probability $p_{x,y}$.
- Edge updating dynamics: every edge $\{x, y\}$ is independently updated at rate $\kappa_{x,y} = x^{-\gamma\eta} + y^{-\gamma\eta}$.

It follows from these settings that:

- The graph is stationary. In particular, G_t has the same law as G_0.
- The degrees are not much affected by the updating events. A strong vertex x is always likely to have roughly degree $cx^{-\gamma}$.

In summary, the main parameters of the model are

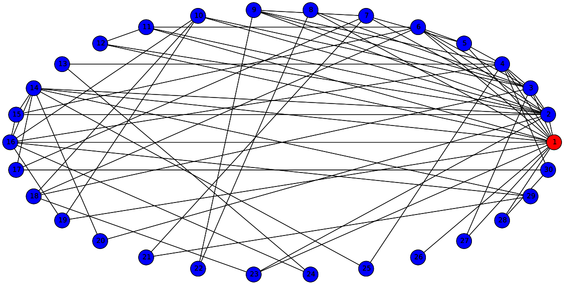
- The power-law exponent $\tau = 1 + 1/\gamma \in (2, +\infty)$.
- The updating exponent $\eta \in (-\infty, +\infty)$.
- The infection rate $\lambda > 0$.

Question: What is the effect of the graph dynamics on the metastability?

- Informally, this evolving graph should interpolate between the static case ($\eta = -\infty$) and the mean-field model ($\eta = +\infty$).

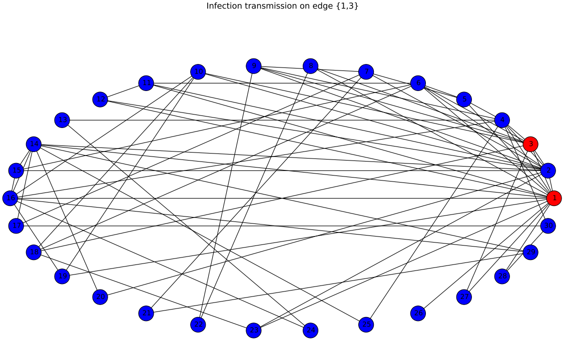
Example of the evolution of the contact process on the vertex updating inhomogeneous graph with factor kernel on 30 vertices.

Starting configuration



Dynamic on the dynamical network

Example of the evolution of the contact process on the vertex updating inhomogeneous graph with factor kernel on 30 vertices.



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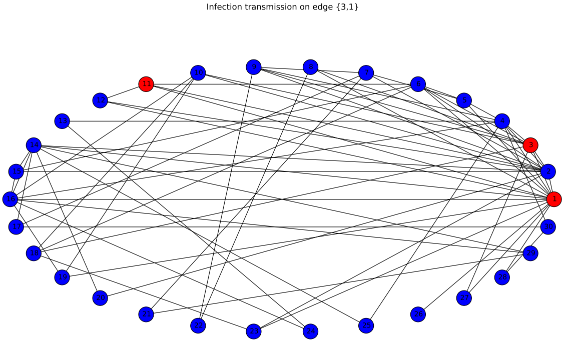

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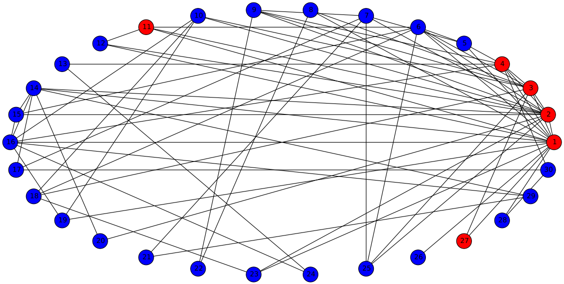
Infection transmission on edge {27,3}



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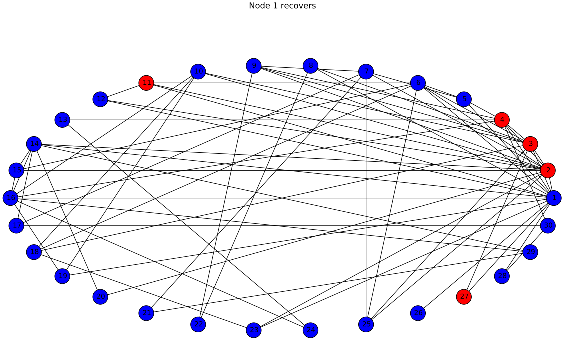
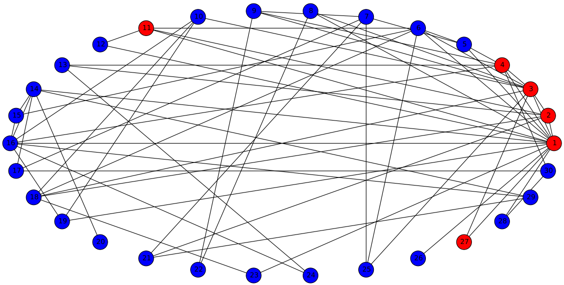


Dynamic on the dynamical network

Example of the evolution of the contact process on the vertex updating inhomogeneous graph with factor kernel on 30 vertices.

Infection transmission on edge {1,11}



Main result

Theorem J, Linker and Mörters (2018)

For vertex updating, factor kernel, and $\eta \geq 0$:
If $\eta < \frac{1}{2}$ and $\tau > 4 - 2\eta$, or if $\eta \geq \frac{1}{2}$ and $\tau > 3$, there is fast extinction for small λ.
On the contrary, if $\eta < \frac{1}{2}$ and $\tau < 4 - 2\eta$, or if $\eta \geq \frac{1}{2}$ and $\tau < 3$, there is long survival and metastable density satisfies $\rho(\lambda) = \lambda^{\mathfrak{e}(\tau,\eta)+o(1)}$, where $\mathfrak{e}(\tau,\eta)$ has the form

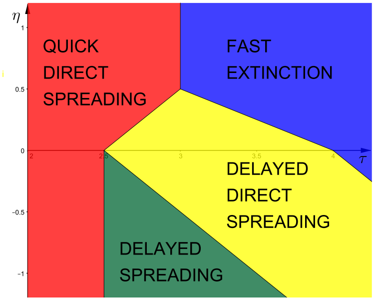
$$\mathfrak{e}(\tau,\eta) = \begin{cases} \frac{2\tau-2-2\eta}{4-2\eta-\tau} & \text{if } \tau > \frac{1}{2}+\eta, \\ \frac{1}{3-\tau} & \text{if } \gamma > \frac{1}{2}+\eta. \end{cases}$$

Main result

Theorem J, Linker and Mörters (2018)

For vertex updating, preferential attachment kernel, $\eta \geq 0$:
If $\eta \geq \frac{1}{2}$ and $\tau > 3$, there is fast extinction for small λ.
On the contrary, if $\eta < \frac{1}{2}$, or if $\eta \geq \frac{1}{2}$ and $\tau < 3$, there is long survival and the metastable density satisfies $\rho(\lambda) = \lambda^{\mathfrak{e}(\tau,\eta)+o(1)}$, where $\mathfrak{e}(\tau,\eta)$ has the form

$$\mathfrak{e}(\tau,\eta) = \begin{cases} \frac{3\tau-5-2\eta}{1-2\eta} & \text{if} \quad \eta < \frac{1}{2} \;\text{ and }\; \tau \geq \frac{8}{3} + \frac{2}{3}\eta, \\ \frac{3\tau-4-2\eta}{4-2\eta-\tau} & \text{if} \quad \eta < \frac{1}{2} \;\text{ and }\; 2+2\eta \leq \tau \leq \frac{8}{3} + \frac{2}{3}\eta, \\ \frac{\tau-1}{3-\tau} & \text{if} \quad \tau \leq 2+2\eta. \end{cases} \tag{2}$$



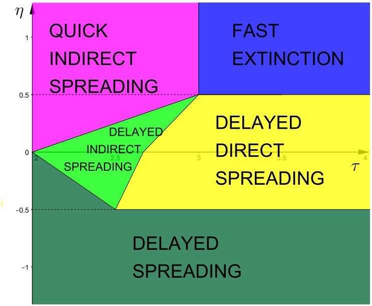


Figure: Phase diagram for vertex updating, for the factor kernel (left) and the preferential attachment kernel (right). The different metastable phases correspond to different expression for the metastable density exponent, and different mechanisms explaining these exponents.

$\eta \geq 0$ summarizes the theorem.

$\eta < 0$ is still ongoing work.

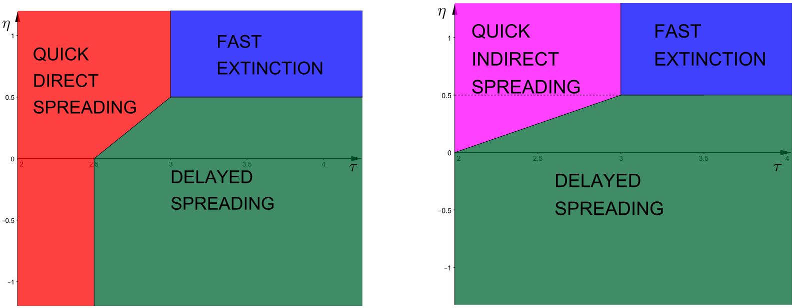


Figure: Phase diagram for edge updating, for the factor kernel (left) and the preferential attachment kernel (right). This is still ongoing work.

Local survival lemma for static graphs

- The mean-field model underestimates the effect of reinfections, and is unable to predict the following result, which is the starting point of the study of static graphs.

Local survival lemma

For a star graph around a central vertex of large degree k, the infection typically survives at least up to time

$$e^{c\lambda^2 k},$$

for c some explicit constant.

- Thus high degree vertices - we will call them stars - can maintain the infection much longer than the mean-field approach may suggest.
- With a careful geometric study of the network, one can ensure the infection travels to other stars, and is kept alive for exponential time.

Local survival lemma for evolving graphs

- The evolving star graph around vertex x is obtained by keeping only edges incident to x in our model.

Local survival lemma for evolving graphs

The typical extinction time of the evolving star graph around vertex x is at least up to logarithmic multiplicative factors of order $T_{loc}(x)$ where

$$T_{loc}(x) = \begin{cases} 1 \vee \exp(c\lambda^2 x^{-\gamma}) & \text{for edge updating, } \eta \leq 0, \\ 1 \vee \exp(c\lambda^2 x^{-\gamma(1-2\eta)}) & \text{for edge updating, } \eta > 0, \\ 1 \vee \lambda^2 x^{-\gamma(1-\eta)} & \text{for vertex updating, } \eta \leq 0, \\ 1 \vee \lambda^2 x^{-\gamma(1-2\eta)} & \text{for vertex updating, } \eta > 0. \end{cases}$$

Lower bounds

- Choose a threshold value $a \in (0, 1)$. Vertices in $(0, a)$ are called stars, those in $(a, 1)$ are called connectors.
- By preceding lemma, a typical infected star maintains the infection locally at least up to time $T_{loc}(a)$.
- During this time length, it can infect other stars either directly, or indirectly, using a connector.
- If you are lucky, this is enough to guarantee long survival of the contact process on stars.
- In that case, the metastable density has to be at least of order $\lambda a^{1-\gamma}$, which we obtain by considering that most of the stars are typically infected, as well as a proportion λ of their neighbours.
- Optimizing over the choice of a gives the desired lower bounds.
- There are actually additional difficulties when $\eta < 0$ because the subgraph of stars updates slowly and geometrical constraints should be taken into account...

Upper bounds

We developed two very different techniques to get the upper bounds:

- An abstract supermartingale technique inspired by the mean-field model.
- A more concrete approach based on the duality and the early evolution of the infection, and inspired by the static case and earlier work of MMVY.
- When η is too negative, the first upper bound is not accurate anymore (heuristically because updates become too rare).
- When η is too large, the second upper bound is not accurate anymore (heuristically because updates become too frequent).
- However, the smallest of these two upper bounds always match our lower bounds up to logarithmic multiplicative factors, yielding the result.

More on the supermartingale technique

The process is dominated by a mean-field infection process, defined similarly as the contact process, but on the complete graph, with edges holding an additional status revealed/unrevealed, with the following changes:

- An update turns the status of the edge to unrevealed.
- An infection along an edge turns the status of this edge to revealed.
- Infections hold:
  - at rate λ along revealed edges
  - but only at rate $\lambda p_{x,y}$ along unrevealed edges.

This mean-field process can now be analyzed by defining a score associated to a configuration and based on our understanding of the local survival mechanisms, so as to obtain a supermartingale.

More on the supermartingale technique

In particular, for vertex updating and $\eta \geq 0$, we obtain the following theorem for an explicit constant $\omega > 0$.

- For $\lambda > 0$, suppose there is some non-increasing function $s\colon (0,1] \to (0,\infty)$ with $\int_0^1 s(x)\,dx < \infty$ and

$$\lambda T_{loc}(x) \int_0^1 p(x,y)s(y)\mathrm{d}y \;\leq\; \omega s(x) \quad \forall x \in (0,1]. \tag{3}$$

  Then the expected extinction time is at most linear in N and in particular there is fast extinction.

- Suppose now that (4) holds only for $x \geq a$ for some $a \in (0,1)$. Then the metastable density (if defined) must satisfy

$$\rho(\lambda) \leq a(\lambda) + \frac{1}{s(a(\lambda))} \int_{a(\lambda)}^1 s(y)\,dy. \tag{4}$$

Summary

- We have defined a simple dynamic on the graph structure, susceptible to have a dramatic effect on the contact process run on the graph.
- Choosing this graph dynamic faster or slower provides a natural interpolation between the case of a static graph and that of its mean-field approximation.
- The network dynamic can help the infection spread faster. But more importantly, it can help the model escape metastable states, thus accelerating extinction...
- Understanding these effects allows to develop simple heuristics. These heuristics can in turn sometimes be turned to proofs more easily than in the static case. The graph dynamic helps by weakening the geometric constraints.
- An interesting new phase transition for the contact process has emerged.

Perspectives

- Get a more precise description of the metastability results. In particular, a proper characterization of the metastable density may need a generalization of the Benjamini-Schramm weak local limit to the settings of evolving graphs.
- We still have a gap between our lower and upper bounds, which may be interesting to fill. This gap is
  - a constant multiplicative factor in the phases of quick spreading.
  - a logarithmic multiplicative factor in the phases of delayed spreading
- What about other networks, other dynamics? What about adaptive dynamics?

Thank you

That's all folks!