

# Spreading on Networks

Modelling spreading phenomena and understanding its complexity

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# Introduction: motivations, SIS processes, networks

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# The spreading phenomena

Spreading is a basic dynamical process in socio-technical systems and broadly exists:

- Diseases; virus
- Spreading of information; cultural norms; social behavior
- Error propagation; cascading failure; propagation of neuronal activity

Exhibiting non-trivial phenomenon:

- Phase transition

# Motivations of my research

## 1. Mathematical modelling of spreading phenomena

- *Understanding spreading*
- *Prediction*
- *Control*

*I simply wish that, in a matter which so closely concerns the wellbeing of the human race, no decision shall be made without all the knowledge which a little analysis and calculation can provide.*

---Daniel Bernoulli, 1760.



## 2. Understanding complex systems/networks



- *Emergent phenomena, e.g. phase transition*
- *Critical properties of complex systems.*
- *Spreading processes as a probe to exam network structure.*

Fitzgerald: *The rich are different from us.*

Hemingway: *Yes, they have more money.*

# Networks

**Graphs**  $\xrightarrow{\text{Scale increases}}$  **Networks**  $\xrightarrow{\text{Go to infinity}}$  **Thermodynamic limits:**

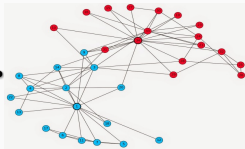
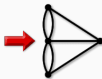
**Static and small system**  
*(pure random or regular):*  
Algorithms; Combinatorics.  
Examples:  
---Shortest path  
---max flow min cut

**Dynamical and large systems**  
*(between random and regular):*  
Data; Machine learning;  
Infrastructures;  
Cyber-physical System;  
Society; Brain. Examples:  
---Community structure  
---Centrality  
---Robustness

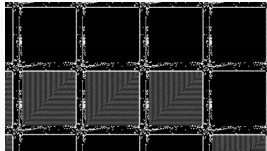
Physics:  
---Phase transition;  
---Emergence;  
---Power-law (Scale-free).



Euler's Königsberg seven bridge



Zachary's karate club



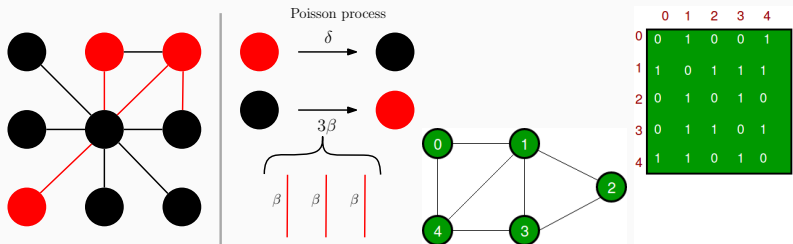
Conway's Game of Life

# The SIS model on networks

The Susceptible-Infected-Susceptible (SIS) process on networks  
(represented by the **adjacency matrix**):

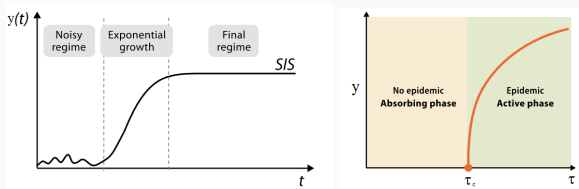
Each node is either **Infected** or **Susceptible** (healthy);

The infection and curing process are **Poisson processes** (uniform and memoryless, an assumption for simplicity)



The SIS model is a  $2^N$ -state continuous-time Markov process with an **absorbing all-healthy state**.

# Basic results about the SIS model



Time-dependent fraction of infected nodes (prevalence) and its steady-state

Where  $\tau = \frac{\text{infection rate}}{\text{curing rate}}$ . Mean-field analysis:

- Heterogeneous mean-field theory:  $\lambda_c = \frac{E[D]}{E[D^2]}$
- The  $N$ -intertwined (quenched) mean-field theory:  $\lambda_c = \frac{1}{\lambda_1}$

**In power-law networks,  $\lambda_c \rightarrow 0$  with network size. (NIMFA and a proof by Chatterjee and Durrett)**

**In homogeneous networks,  $\lambda_c \rightarrow c > 0$ .**



## New understandings about the SIS model

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## 2.1 Temporal correlations in spreading processes

The **temporal correlations** are **generally omitted** in most of the research on spreading processes. Previous studies only care about:

- **First-moment** and **metastable** properties: epidemic threshold, prevalence...
- Influence of  $\tau \triangleq \beta/\delta$ , but not  $\beta$  and  $\delta$ .

However, a lot of **dynamical details** are lost:

- $\beta$  and  $\delta$ : the **speed of evolution**?
- Dynamics of the **transient state** (before metastable)?
- Higher-moment differences (other than infection probability  $E[X_i(t)]$ ) among nodes, e.g. **state flipping**.

We study the temporal correlation of the infection state of each node by considering **the stochastic process defined by mean-field method**.

## 2.1 Some new insights

1. The mean-field steady-state autocorrelation of nodal infection state is

$$R_i(h) = \exp\left(-\frac{\delta}{1 - v_{i\infty}}h\right)$$

Thus, the infection state **flips fast for** nodes with high infection probability (**hubs**) and  $R_i(h)$  can be arbitrarily small. (**How about the experiments in brain?**)

2. Interestingly, in regular graphs with degree  $k$ , the autocorrelation

$$R(h) = \exp(-\beta kh)$$

is irrelevant to curing rate!

3. In the transient state, the autocorrelations can be calculated by Magnus expansion involving neighbors with certain hops determined by the accuracy.

## 2.1 Calculating the infection and curing rate by measuring state flips

**The reverse problem:** In the metastable state, measuring the nodal infection state  $X_i(t + \Delta), X_i(t + 2\Delta), \dots, X_i(t + n\Delta)$  as a binary sequence, the autocorrelation  $R_i(h)$  is the correlation between the state sequence and its shifting sequence.

**Calculating the curing rate:**

$$\delta = -(1 - v_{i\infty}) \frac{\ln[R_i(h)]}{h}$$

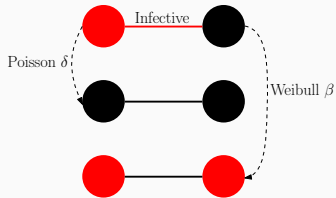
With local topological information, **the infection rate** is

$$\beta = -\frac{v_{i\infty}}{\sum_{j \in \mathcal{N}_i} v_{j\infty}} \frac{\ln[R_i(h)]}{h}$$

or with global information (degree sequence), **the infection rate** is

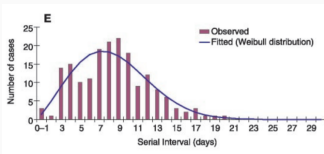
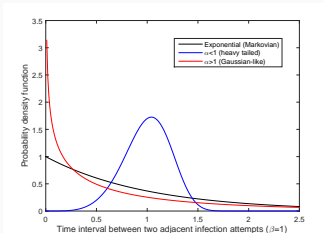
$$\beta = -\frac{\sum_{i=1}^N v_{i\infty}}{\sum_{i=1}^N d_i v_{i\infty}} \frac{\ln[R_i(h)]}{h}$$

## 2.2 Non-Markovian SIS model: The Weibull renewal infection as an example

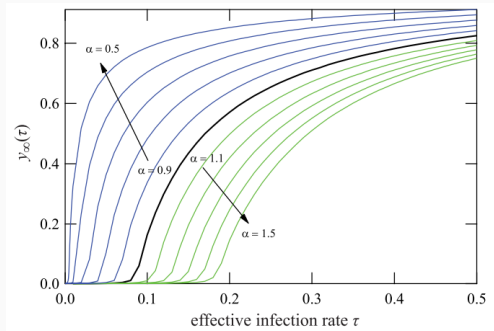


The Poisson infection process is a special case. The infected nodes can be cured with rate  $\delta$  (Poisson).

The Weibull infection process can represent very different infections: from long-tailed to Gaussian-like.



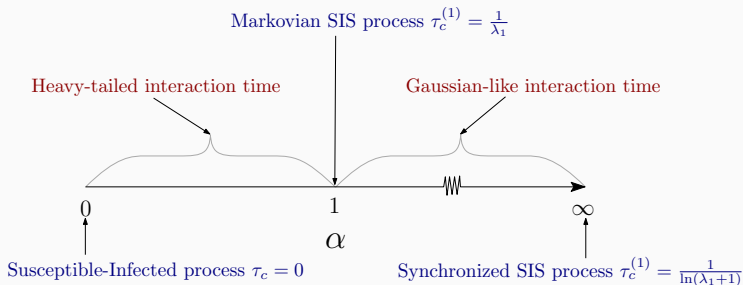
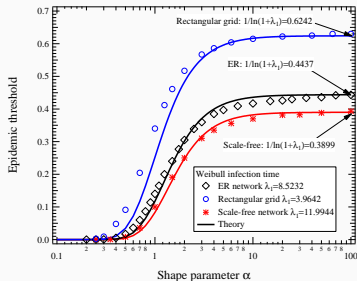
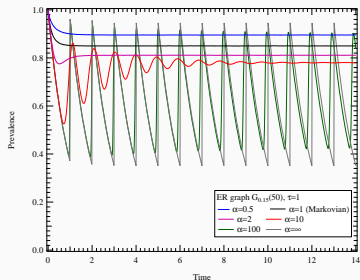
## 2.2 The epidemic threshold is altered



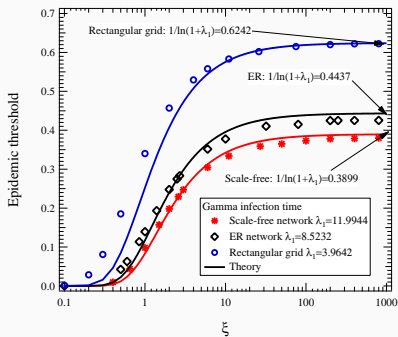
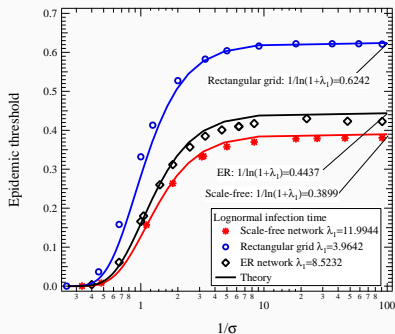
Non-Markovian spread dramatically alters the epidemic threshold ( $1/\lambda_1$  for Markovian) in networks:

- How does the spreading look like?
- How much does the threshold change?

## 2.2 Non-unimodal prevalence and threshold



## 2.2 Universal for other distributions



For gamma infection, the explicit threshold is

$$\tau_c^{(1)} = \frac{1}{\xi[(1 + \lambda_1)^{\frac{1}{\xi}} - 1]}$$



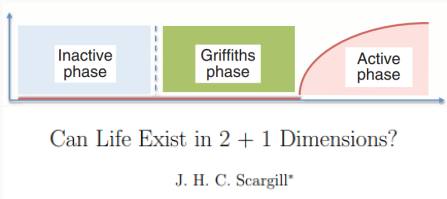
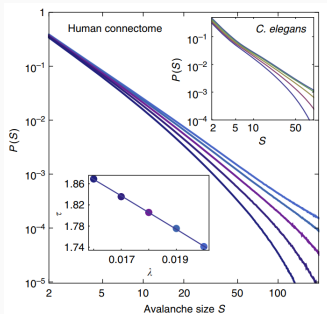
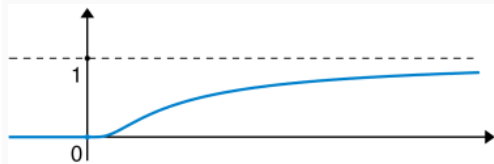
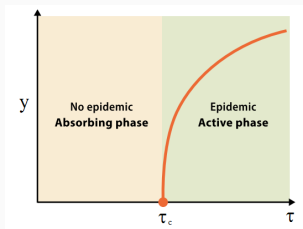
## 2.3 Localization in various systems

Localization is the phenomenon that the dynamics are restricted within a small portion of a system due to randomness or heterogeneity.

- Wave (Anderson localization)
- Maximum-entropy random walk
- Google matrix (Markov chain)
- Spreading processes on networks  
(Near the threshold, imagine an infinite large network where the SIS infection persists but the prevalence is zero)

Localization of spreading happens just above the threshold;  
**We need to rethink the vanishing threshold.**

## 2.3 SIS (de-)Localization and neural/brain networks



## 2.3 Mean-field analysis of localization

When  $\tau \triangleq \frac{\beta}{\delta}$  is just above the epidemic threshold  $\tau_c^{(1)} = \frac{1}{\lambda_1}$

$$y_\infty(\tau) = \frac{1}{N} \underbrace{\sum_{j=1}^N X_j}_{a} \left( \frac{\tau}{\tau_c^{(1)}} - 1 \right) + O \left( \frac{\tau}{\tau_c^{(1)}} - 1 \right)^2$$

where  $x$  is the principle eigenvector  $Ax = \lambda_1 x$ .



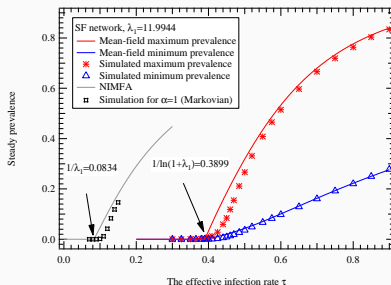
**In the thermodynamic limit  $N \rightarrow \infty$  and  $\frac{\tau}{\tau_c^{(1)}} \ll 1$ :**

If  $a = O(1)$  (homogeneous networks), then the prevalence  $y_\infty(\tau) > 0$  (order parameter) just above the threshold.

While if  $a \rightarrow 0$ , then  $y_\infty(\tau) = 0$  but the number of infected node  $y_\infty(\tau)N$  is non-zero.

## 2.3 Is the network localization alterable by dynamics?

We need a spreading mechanism to amplify the prevalence to the maximum extent.



- When  $\frac{\tau}{\tau_c^{(1)}} \rightarrow 1$ , where  $\tau_c^{(1)} = \frac{1}{\ln(1+\lambda_1)}$ ,  $\frac{\text{Maximum prevalence}}{\text{Minimum prevalence}} = 1 + \lambda_1$ .
- Divergent  $\lambda_1 = \infty$  for heterogeneous networks when  $N \rightarrow \infty$

Amplifying the prevalence into high order.

## 2.3 Localization seems unalterable

- The maximum:  $y_{\infty}^+(\tilde{\tau}) = a_{\max}\tilde{\tau} + o(\tilde{\tau})$
- The minimum:  $y_{\infty}^-(\tilde{\tau}) = a_{\min}\tilde{\tau} + o(\tilde{\tau})$

where

$$a_{\max} = \frac{2(\lambda_1 + 1) \ln(\lambda_1 + 1)}{\lambda_1} \underbrace{\frac{1}{N} \frac{\sum_{j=1}^N X_j}{\sum_{j=1}^N X_j^3}}_a = O(a \ln N) \quad (1)$$

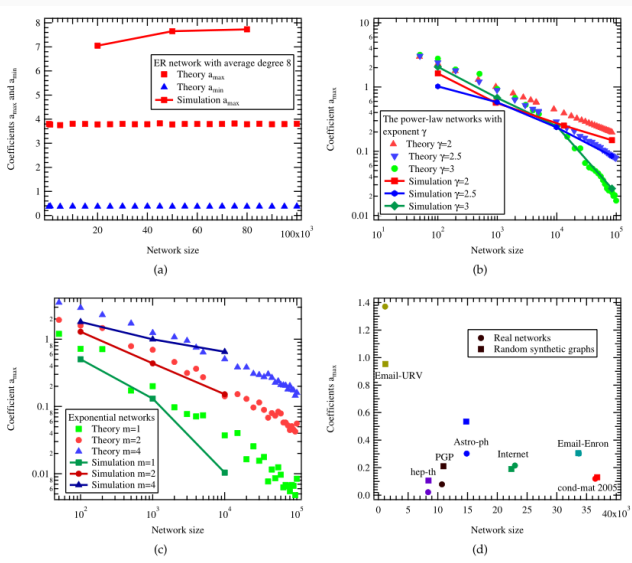
and

$$a_{\min} = a_{\max}/(\lambda_1 + 1)$$

**Result:**  $a = O(N^{-c})$  and thus  $a_{\max} = \frac{\ln N}{N^c}$ ,  $a_{\max} \rightarrow 0$  with  $N \rightarrow \infty$ .

*Structural localization of network is unalterable even for such a maximum amplification of spreading.*

## 2.3 Evaluating localization for different networks

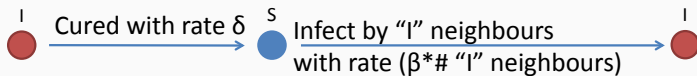


## 2.4 Control of spreading costs

- 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.  
2017: Netherlands, 94%; China, 99%; South Sudan, 26%.
- Cisco reported that 83% of the Internet of Things devices are not patched to be immunized against cyber-attacks.

## 2.4 Effectiveness of the pulse curing

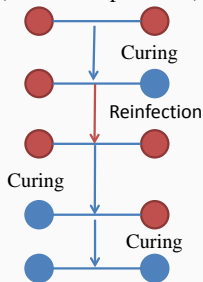
### SIS process:



### Curing process:

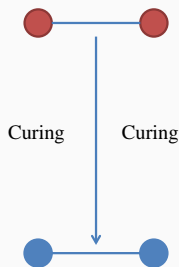
#### asynchronous:

3 operations  
(At least 2 operations)



#### Synchronous (pulse):

2 operations

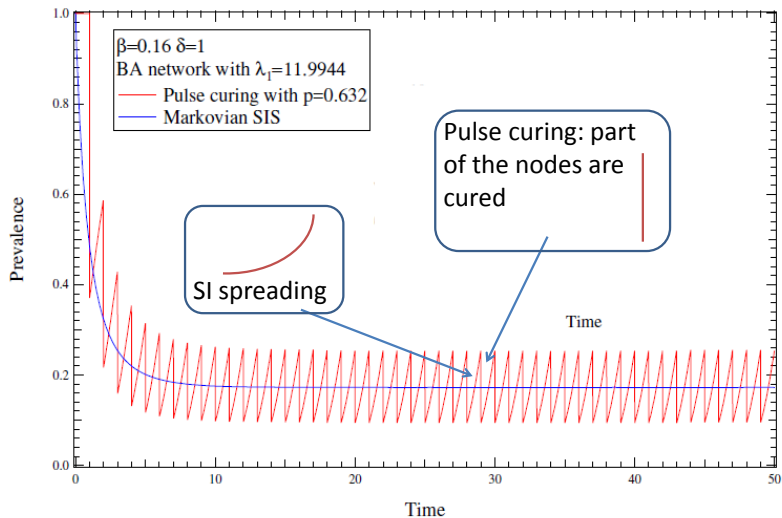




## 2.4 Pulse strategy in reality

- In **measles** vaccination, by periodically and synchronously vaccinating several age cohorts, instead of uniformly and asynchronously vaccinating each individual at certain ages.
- **Computer network**: synchronous virus check of all computers instead of asynchronous check of each computer.
- India introduced the **National Immunization Days** to control the spread of **polio**.

## 2.4 Pulse strategy to control spreading



## 2.4 Pulse strategy to control spreading

- The pulse curing arrives with rate  $\delta$ . Each pulse curing covers a fraction of all infected nodes in the network, which is  $0 \leq p \leq 1$ .
- If  $p = 1$ , then no infected node exists and the spreading is eliminated instantly; if  $p = 0$ , then the SIS process is an SI process and eventually all nodes are infected. (pulse curing + SI model for non-zero  $p$ )

Compare to the Markovian SIS process (the straightforward curing strategy):

- The curing rates are the same; Each node is cured **asynchronously in SIS** while **synchronously in the pulse strategy**.
- The number of curing operations for each node during one time unit:  **$\delta p$  for pulse**;  **$\delta$  for SIS**. (Pulse strategy saves costs)

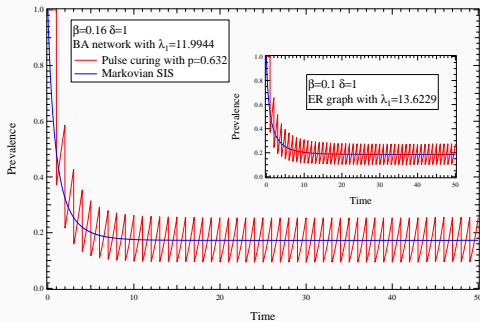
## 2.4 The epidemic threshold and the effect of pulse curing

The epidemic threshold of Pulse+SI is  $\frac{1}{\lambda_1} \ln \frac{1}{1-p}$ . Let  $\frac{1}{\lambda_1} \ln \frac{1}{1-p} = \frac{1}{\lambda_1}$  the SIS threshold, we obtain

$$p = 0.632$$

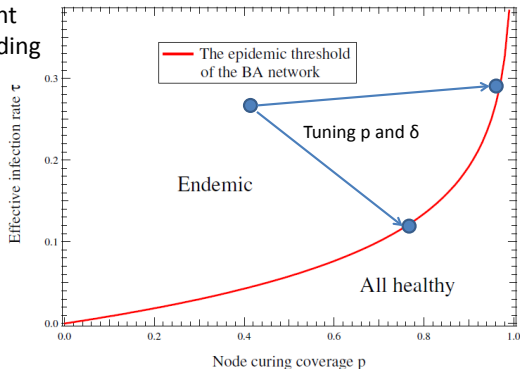
invariant to the network.

Above the threshold, let  $p = 0.632$ , we have



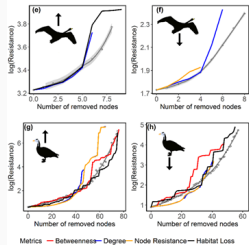
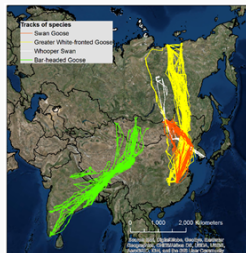
### The phase diagram

$\tau = \beta/\delta$ :  $\beta$  is the inherent property of the spreading

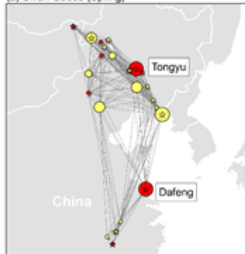


Calculations indicates that larger p saves more curing operations (smaller  $\delta p$ ). If p is restricted, then choosing the largest possible p along the red curve.

## 2.5 Prioritizing conservation efforts for migratory birds



(a) Swan Goose (Spring)



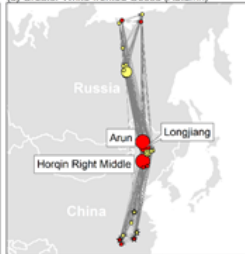
(b) Swan Goose (Autumn)



(c) Greater White-fronted Goose (Spring)



(d) Greater White-fronted Goose (Autumn)

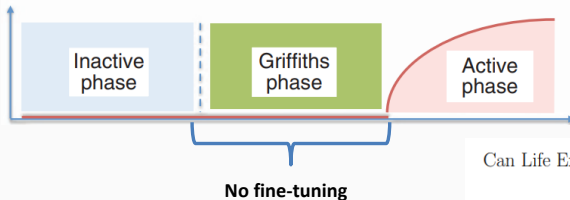
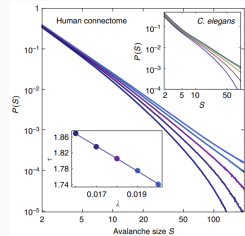
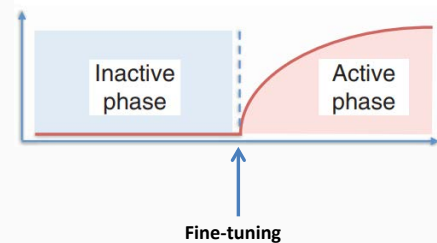


## The SIS processes and brain networks: connections and potential research

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# 3.1 Criticality of brain

Non-trivial network structures introduce non-trivial properties, e.g. Griffiths phase and localizations, which explains the critical nature of brain.  
Power-law: minimum energy consumption while the whole network is functional.



Can Life Exist in 2 + 1 Dimensions?

J. H. C. Scargill\*



## 3.2 Initial conditions and their consequences

The initial conditions in many studies about SIS processes are **ignored**. Most simulations just randomly select a certain number of nodes to be infected initially. For finite-size networks, the initial condition influences the metastable state properties obtained by simulation. The initial condition should be carefully evaluated.

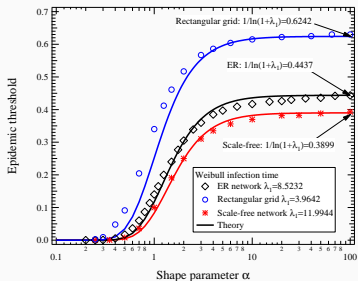
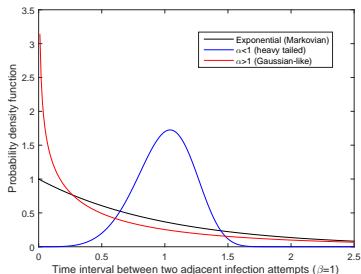
However, stimulating initial nodes provides a way of experimenting the spreading process. The different selection of initially stimulated nodes will lead to different avalanche distributions.

With SIS processes, it might be possible to **reproduce** the brain activity from data and then to **predict** the further evolutions **by stimulating different initial nodes**.

## 3.2 Non-Markovian processes

Most real spreading processes have a non-exponential generation time (time between the onsets of infections between primary and the secondary case).

For brain network, can we extract the signal delay between two nodes and check which distribution fitting the time? Once the fitting parameter is obtained, the non-Markovian SIS theories may provide more insights.



- Some new insights about the SIS process and networks are introduced.
- Applying SIS processes to study neural networks.