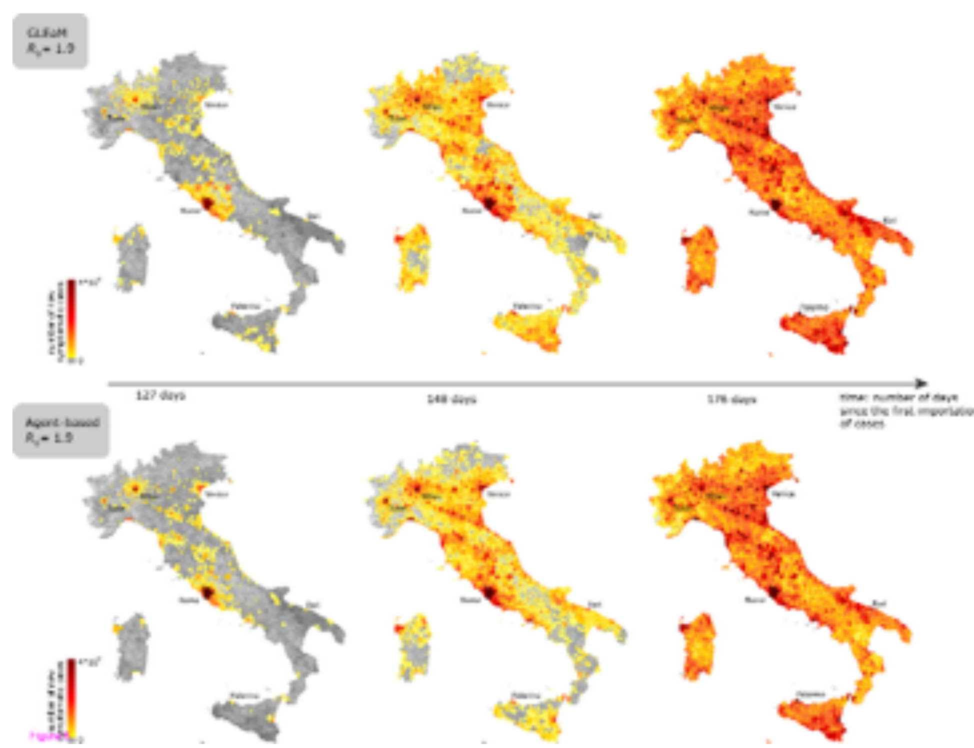
Spreading processes on complex networks



GLEaM

Dr. Márton Karsai

ResCom 2014 Summer School

12th May 2014

Practical matters

Lecturer: Márton Karsai, PhD

Maître de Conferences with INRIA chair
Ecole Normale Supérieure de Lyon
Computer Science Department - LIP
IXXI - Rhône Alpes Complex Systems Institute
INRIA Dante team

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Coords: 12th May, 15:00-18:00

Expériences de recherche

- Condensed Matters Physics
  - Critical dynamics of geometrically frustrated systems
  - Conformal invariance in strongly disordered systems
  - Surface equivalence between disordered spin systems
- Processes on complex networks
  - Optimal cooperation on scale-free networks
  - Non-equilibrium phase transitions in scale-free networks
  - Influence of temporal and topological correlations on information diffusion in social networks
- Temporal and dynamical networks
  - Entropy of dynamical social networks
  - Mesoscopic analysis of community structures in large social networks
  - Causal motifs in temporal networks
  - Evolution of the structure of communication networks
  - Universal characteristics of correlated temporal behaviour
- Human dynamics and social contagion phenomena
  - Circadian fluctuations and burstiness in human communication dynamics
  - Correlated dynamics of egocentric networks
  - Spatio-temporal correlations of mobile service usage
  - Heterogeneous evolution of egocentric networks in Skype
  - Role of strong ties in information diffusion processes
  - Dynamics of online adoption spreading
  - Cascading behaviour of online adoption behaviour

Outline

Brief introduction to complex networks

- The network representation
- Basic network properties
- Random networks - the Erdős-Rényi model
- Scale-free networks - the Barabási-Albert model
- Temporal networks

Spreading processes

- Examples and motivations
- General spreading models

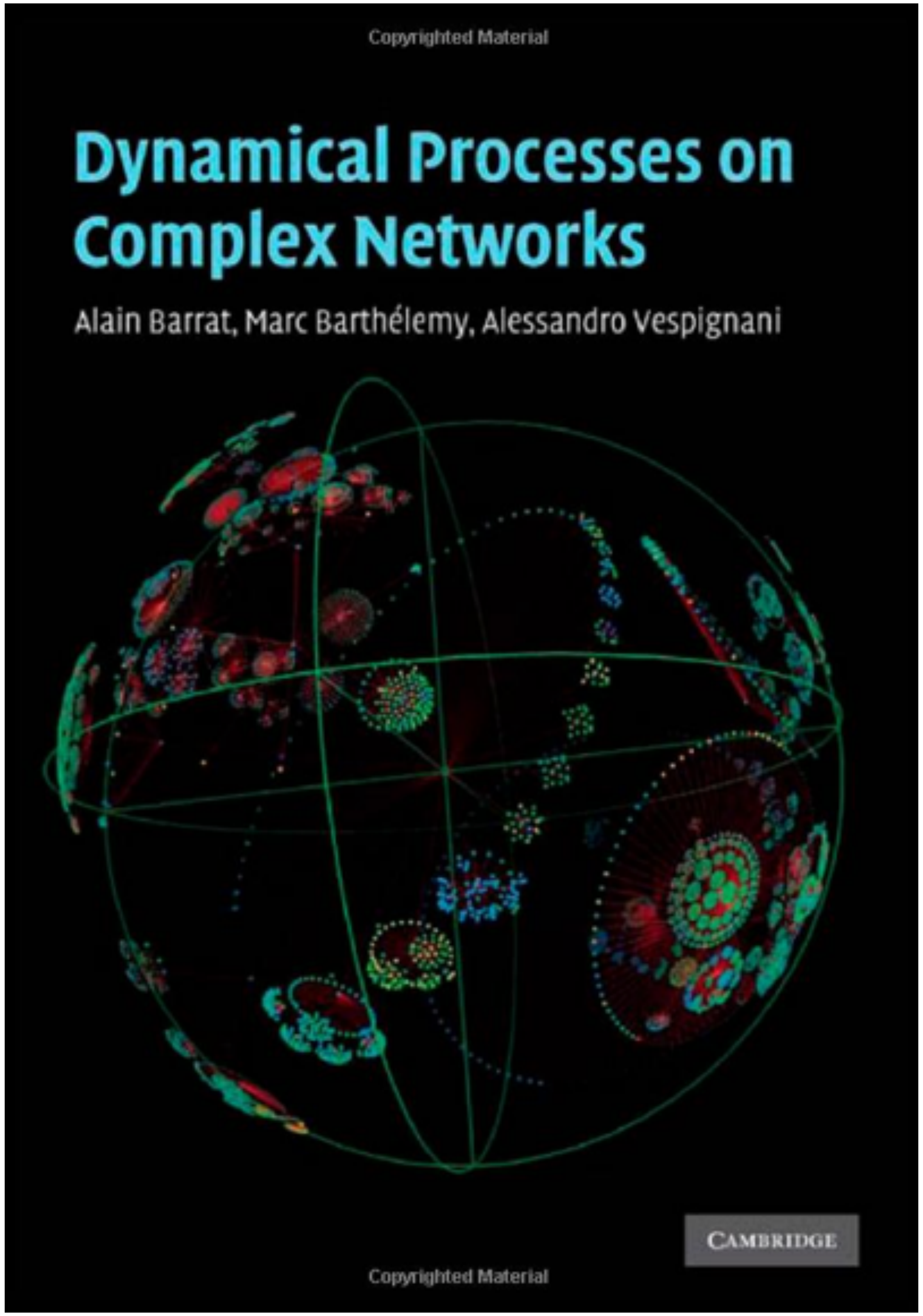
Spreading processes on networks

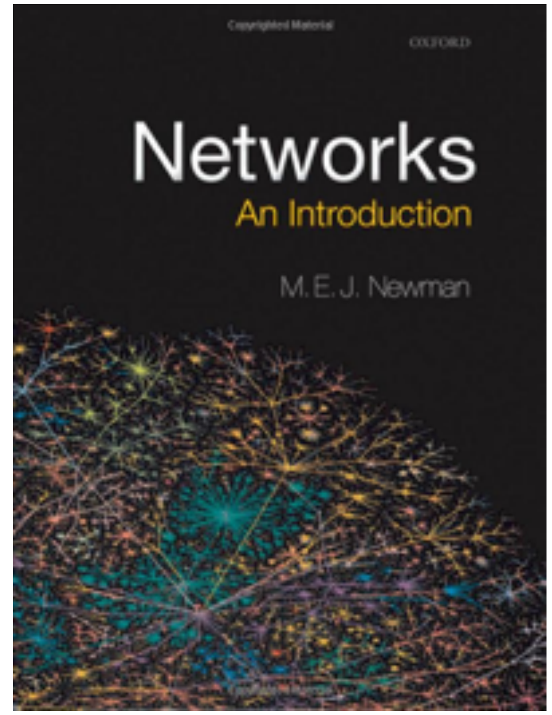
- homogeneous networks
- heterogeneous networks
- Immunisation strategies

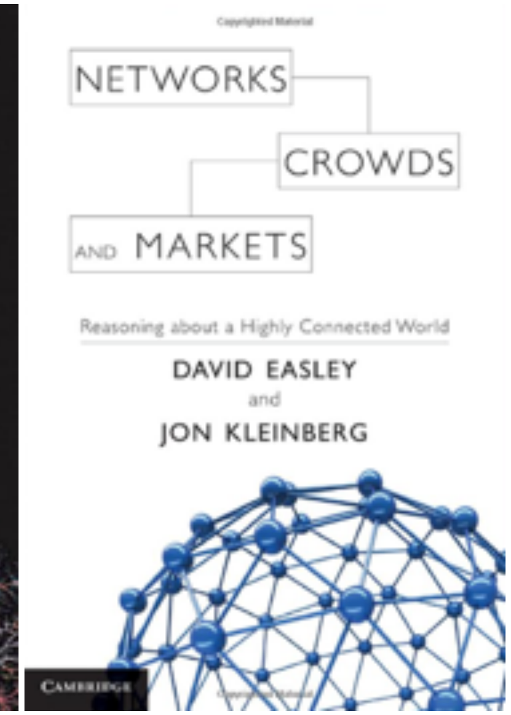
Spreading processes on temporal networks

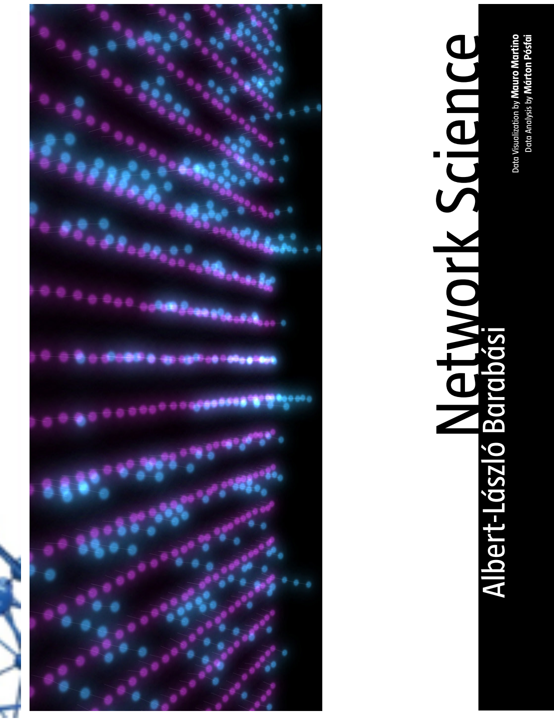
- null model approach
- activity driven approach

Literature









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The Structure and Function of Complex Networks*

M. E. J. Newman†

REVIEWS OF MODERN PHYSICS, VOLUME 74, JANUARY 2002

Statistical mechanics of complex networks

Réka Albert* and Albert-László Barabási

Department of Physics, University of Notre Dame, Notre Dame, Indiana 46556

Temporal networks

Petter Holme [a,b,c,*], Jari Saramäki [d]

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Brief introduction to complex networks

Complex

[adj., v. kuh m-pleks, kom-pleks; n. kom-pleks]
–adjective

1.
composed of many interconnected parts; compound; composite: a complex highway system.

2.
characterized by a very complicated or involved arrangement of parts, units, etc.: complex machinery.

3.
so complicated or intricate as to be hard to understand or deal with: a complex problem.

Source: Dictionary.com

Complexity, a **scientific theory** which asserts that some systems display behavioral phenomena that are completely inexplicable by any conventional analysis of the systems' constituent parts. These phenomena, commonly referred to as emergent behaviour, seem to occur in many complex systems involving living organisms, such as a stock market or the human brain.

Source: John L. Casti, Encyclopædia Britannica

Complexity

Complex Systems

- Self-organised
- Evolving
- Adaptive

- No central organising mind
- No conventional way of description

Complex Systems: how to approach

Statistical description

- Systems with random features
- One sample does not characterise the typical behaviour
- Statistical averages of quantities

Empirical data analysis

- How to detect patterns and structure in information?
- How to characterize the system instead of its building blocks?
- Multivariate methods etc

Analytical approach

- Write down (coupled) differential equations for interactions
- Attempt to solve
- Usually no closed-form solutions; numerical solutions, phase space analysis, etc

Simulations

- Postulate rules (e.g. the ant raids)
- Simulate and observe system behaviour
- Try to match empirical observations

OR

Complex Networks

…a way of mapping complexity

Each complex system can be interpreted as a complex network, which identifies the interactions between the interconnected components

The network approach

- Combines the elements of all the other approach
- Disregards (unnecessary) details of the system
- Focuses on the structure of interactions
- Statistical characterisation of system

The network approach

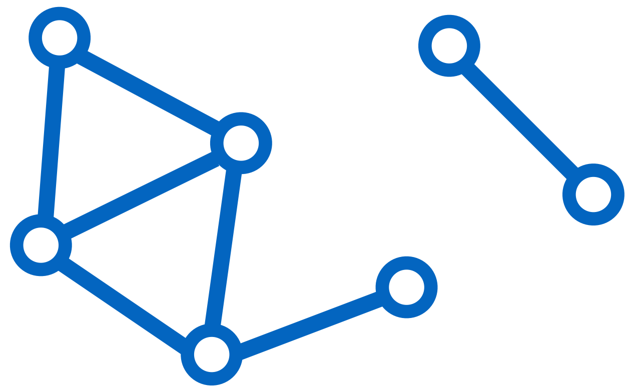
1. **Measuring** - make observations on Nature
2. **Modelling** - attempt to explain observations:
   2.1. Choose the right level of coarse-graining
   - Units: Vertices or nodes ⇔ interacting elements
   - Edges or links ⇔ interactions

   2.2. Strip the problem to its simplest form
   - Interaction structure ⇔ evolution and behaviour of system

   2.3. Formulate the problem in mathematical terms
   - Statistical analysis of network structure
   - Dynamics of processes taking place on networks
3. **Validating** - check if calculations or simulations can
   - reproduce the observations
   - explain the observations
4. Go back to 1. & 2. and rethink

Complex systems as networks

Networks are interpreted as graphs



$$G=(V, E)$$

- Components ⇔ vertices $v \in V$
- Interactions between components ⇔ edges $(u,v) \in E$
- Identification of vertices and edges defines the type of the actual network

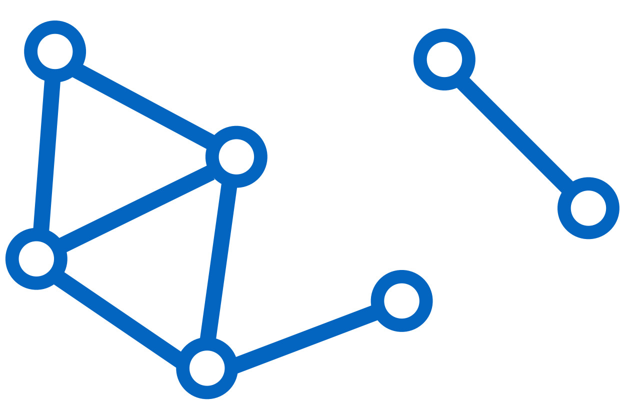
Vertex	Edge
person	friendship
neuron	synapse
WWW	hyperlink
company	ownership
gene	regulation

Undirected networks

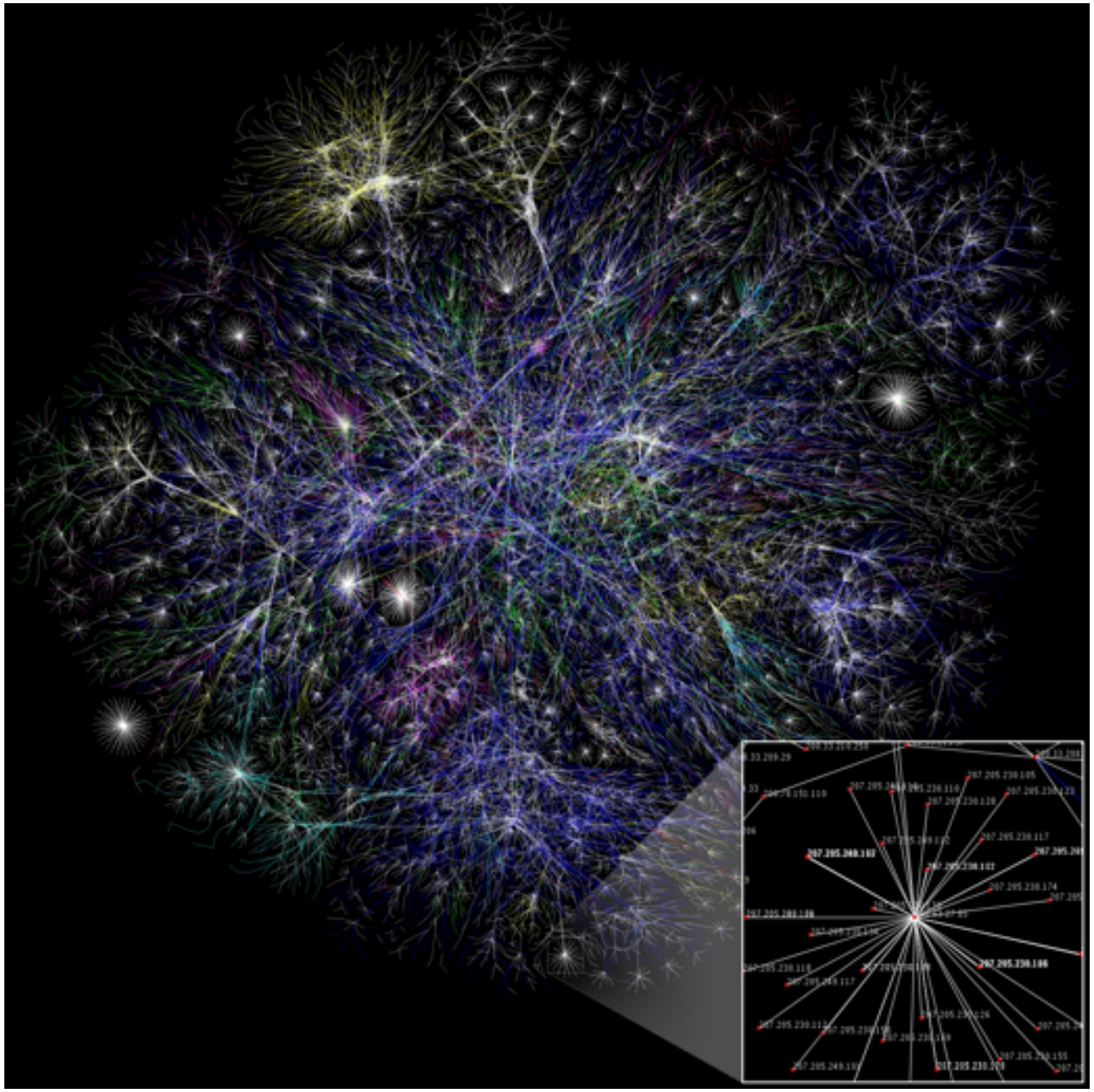
$G=(V, E)$

$(u,v) \in E \equiv (v,u) \in E$

- The directions of edges do not matter
- Interactions are possible between connected entities in both directions



Opte project



The Internet: Nodes - routers, Links - physical wires

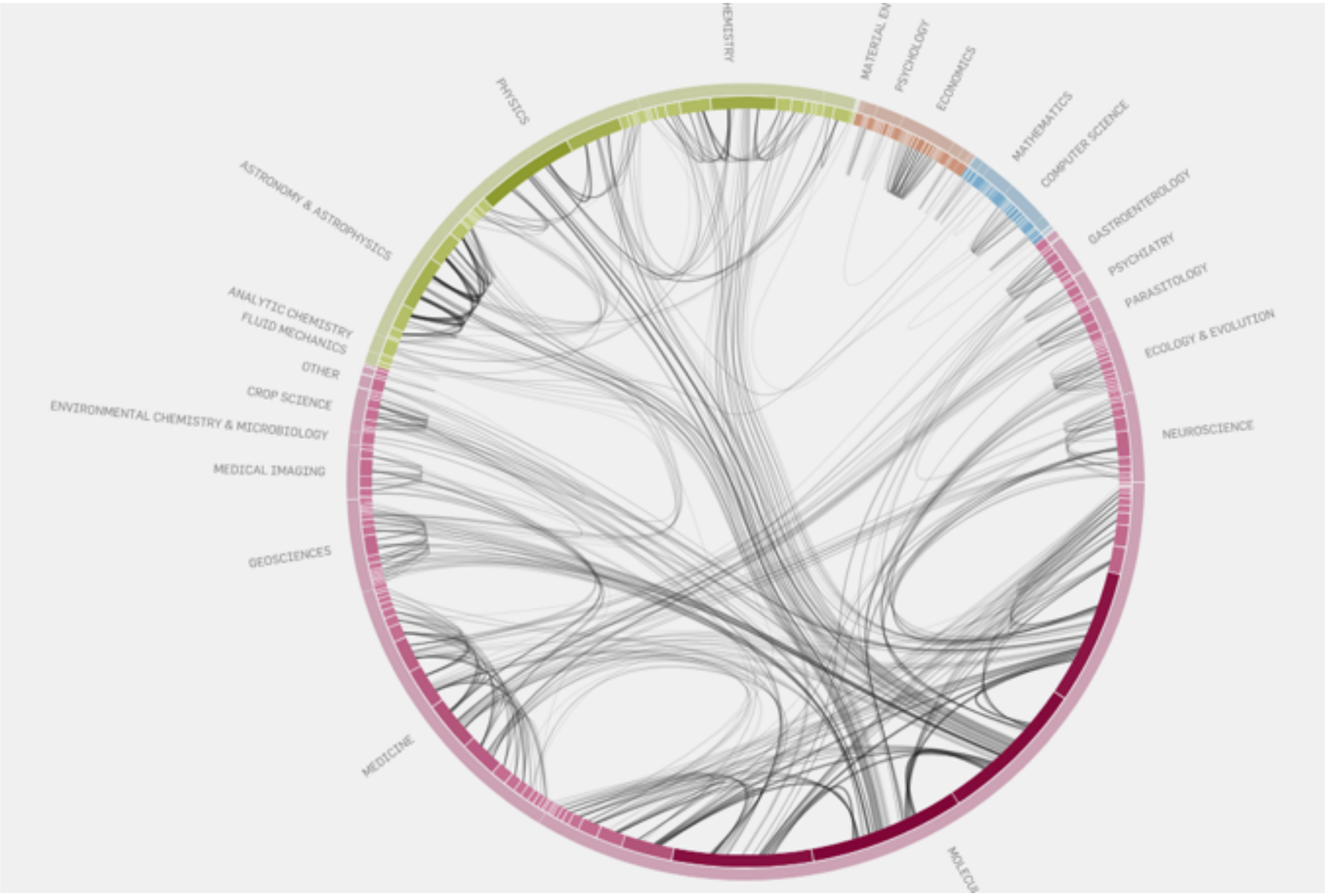
Directed networks

Moritz Stefaner, eigenfactor.com

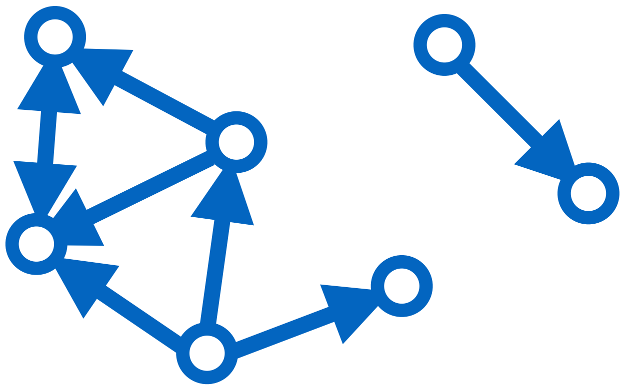
$G=(V, E)$

$(u,v) \in E \neq (v,u) \in E$

- The directions of edges matter
- Interactions are possible between connected entities only in specified directions



Citation network: Nodes - publications, Links - references



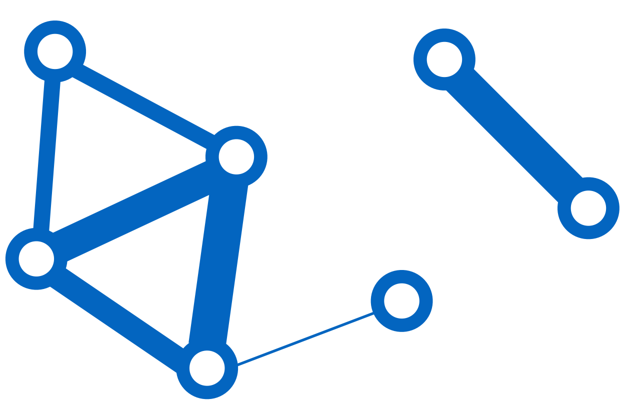
Weighted networks

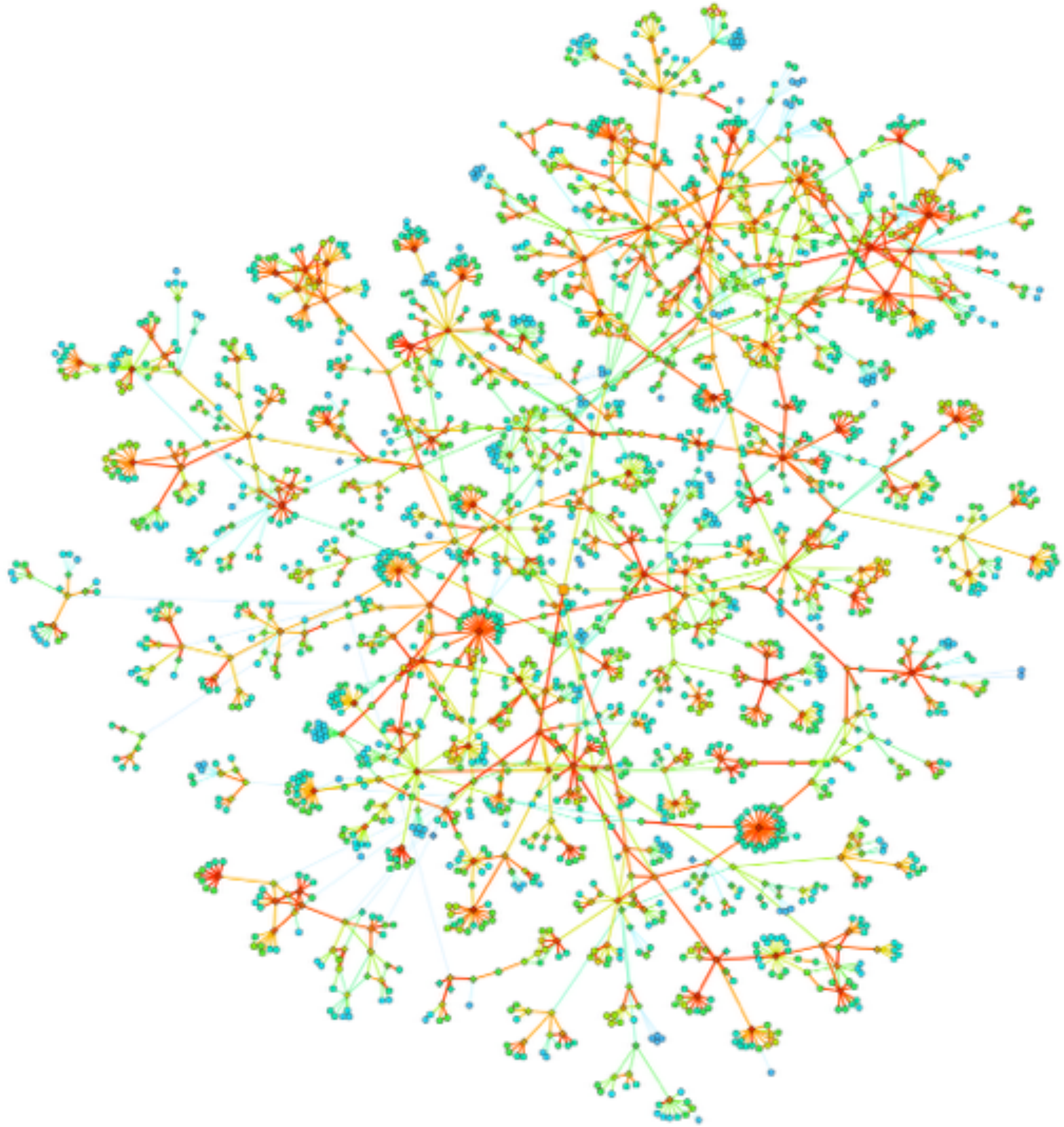
Onnela et.al. New Journal of Physics 9, 179 (2007).

$G=(V, E, w)$

$w: (u,v) \Rightarrow R$

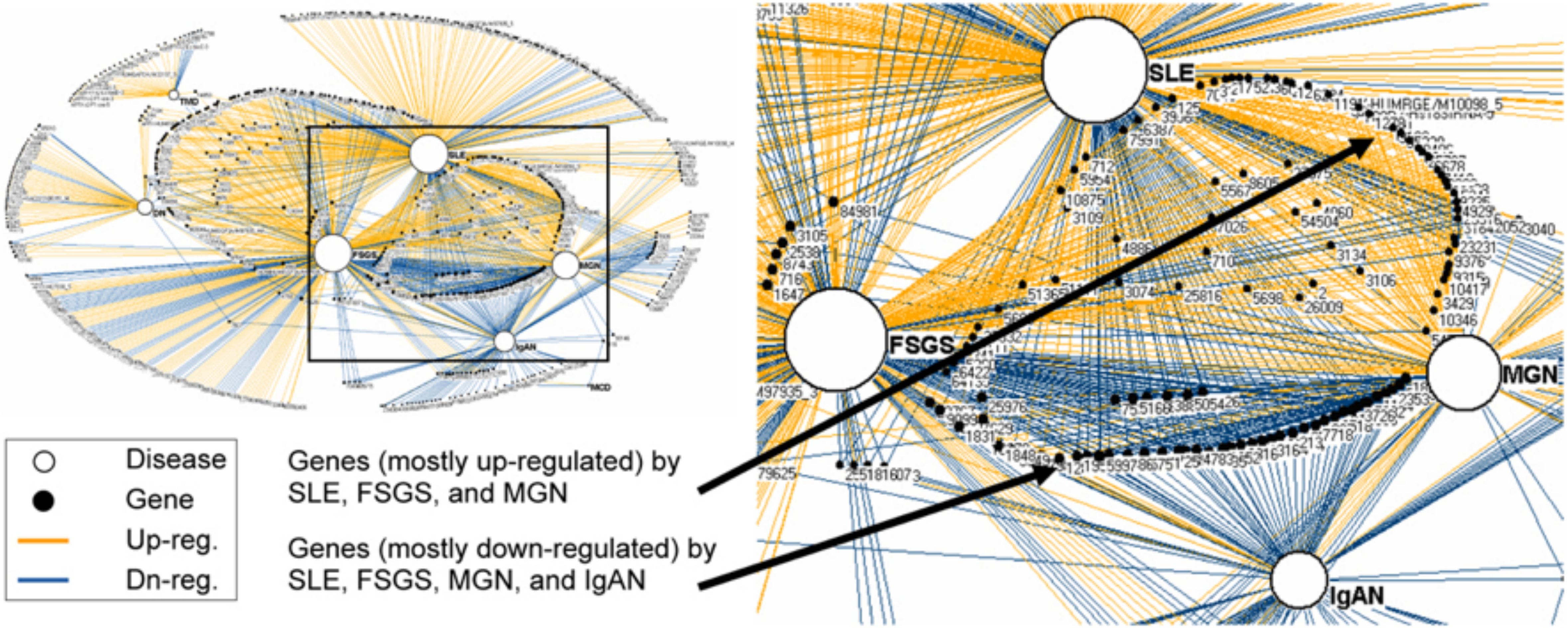
- Strength of interactions are assigned by the weight of links





Social interaction network: Nodes - individuals
Links - social interactions

Bipartite network

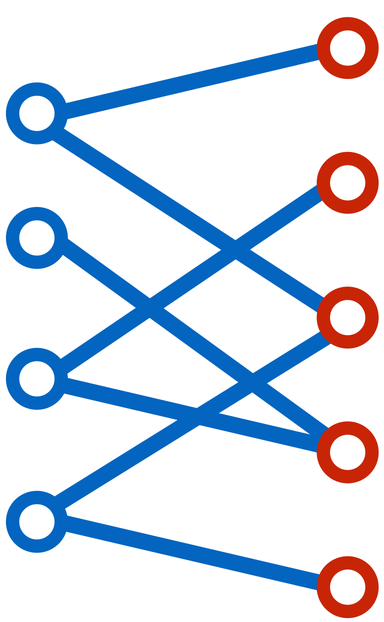


Bhavnani et.al. BMC Bioinformatics 2009, **10**(Suppl 9):S3

Gene-desease network:

Nodes - Desease (7)&Genes (747)

Links - gene-desease relationship



$$G=(U, V, E)$$

$$U \cap V = \emptyset$$

$$\forall (u,v) \in E,\ u \in U \text{ and } v \in V$$

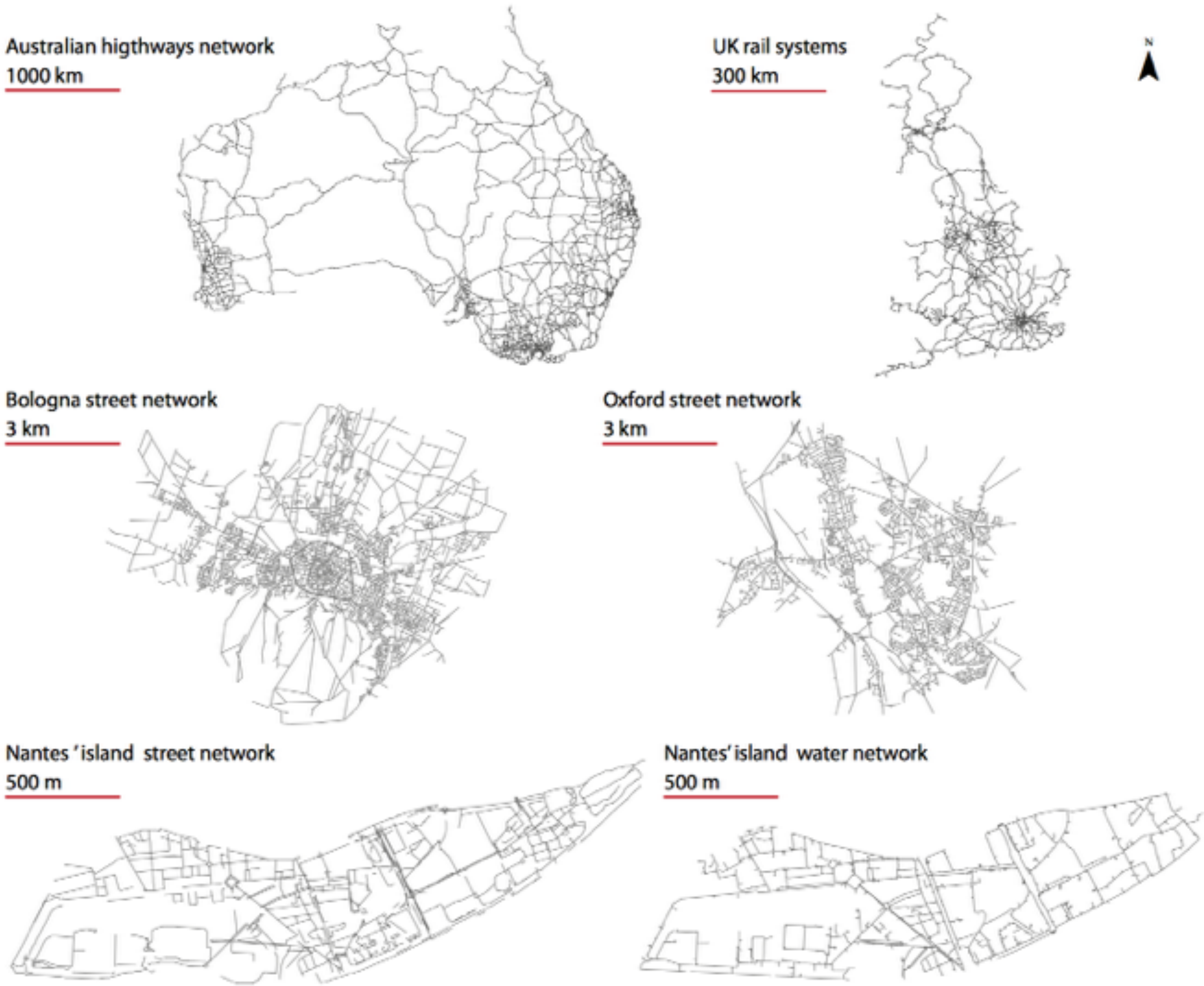
Planar networks

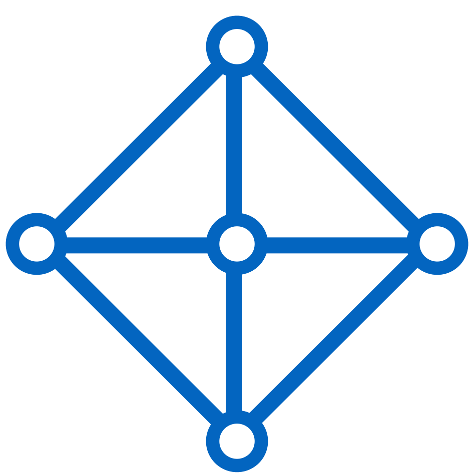
Viana et.al. Nature Scientific Reports 3:3495 (2013)

$G=(V, E, loc)$

$loc: v \Rightarrow (x,y)$

- Nodes can be embedded in a plane
- Geo-localised networks
- Spatial networks





Street networks:

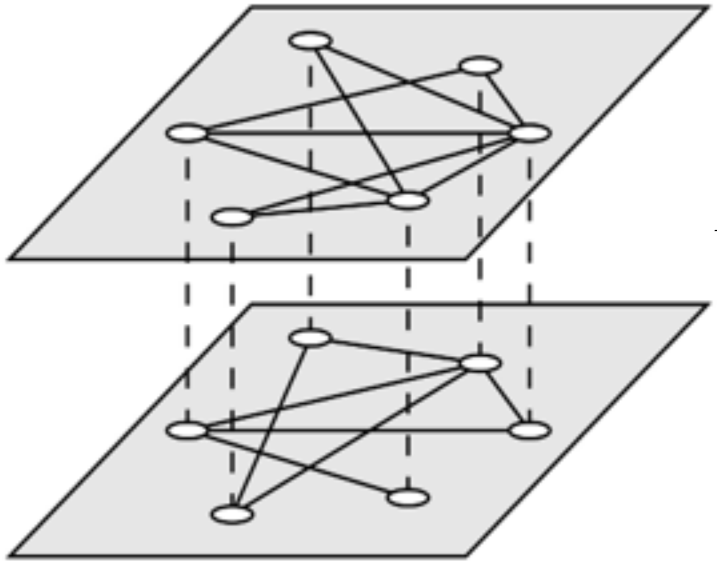
Nodes - junctions, Links - streets

Multiplex and multilayer networks

Karsai et.al. (submitted)

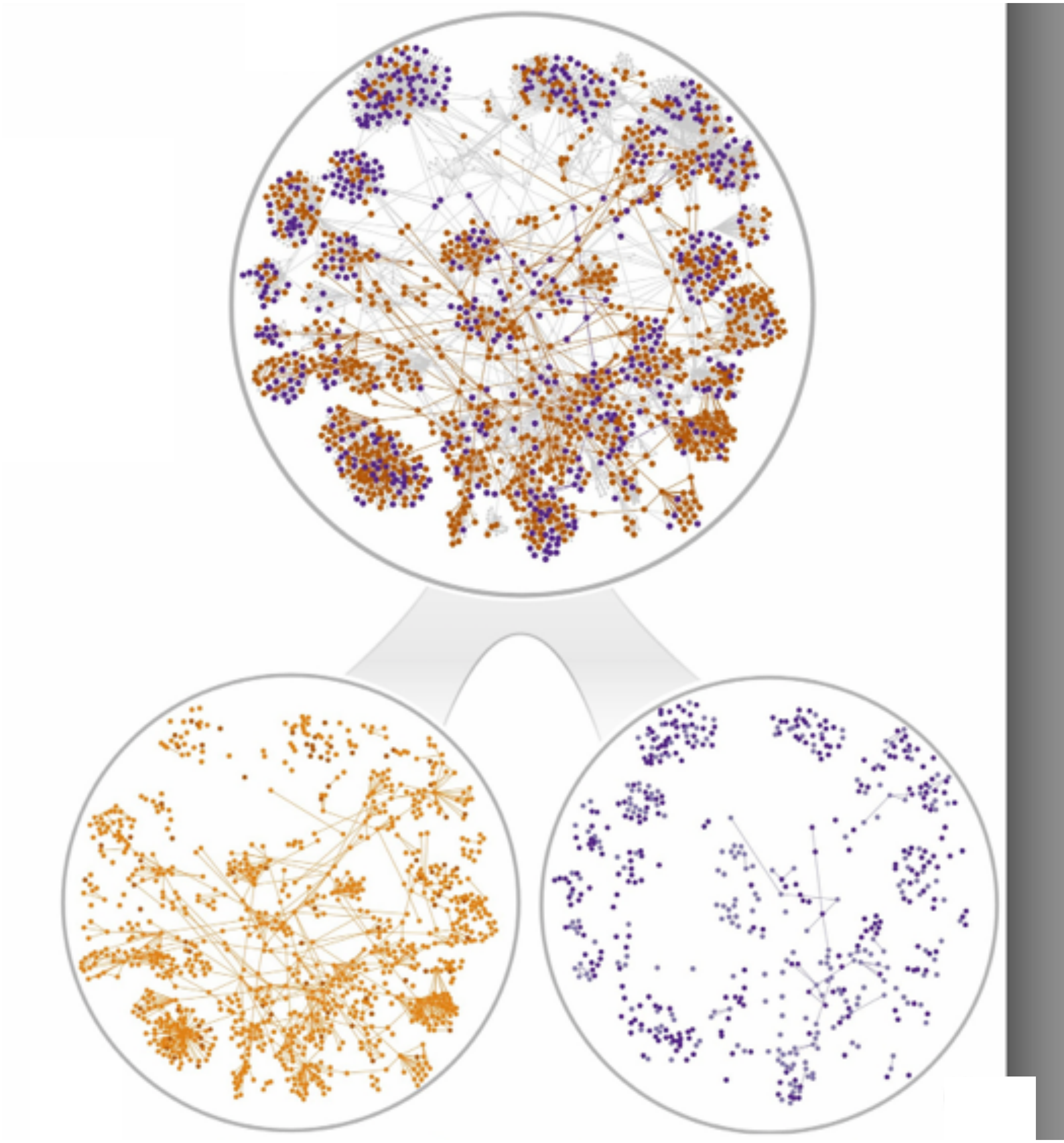
$G=(V, E_i),\ i=1\ldots M$

- Nodes can be present in multiple networks simultaneously
- These networks are connected (can influence each other) via the common nodes



$M=2$

Gomes et.al. Phys. Rev. Lett. 110, 028701 (2013)



Skype adoption network

Nodes - users, Links - social ties,

Colours - service adoption/termination

Temporal and evolving networks

$G=(V, E_t), (u,v,t,d) \in E_t$

t - time of interaction (u,v)
d - duration of interaction (u,v,t)

- Temporal links encode time varying interactions

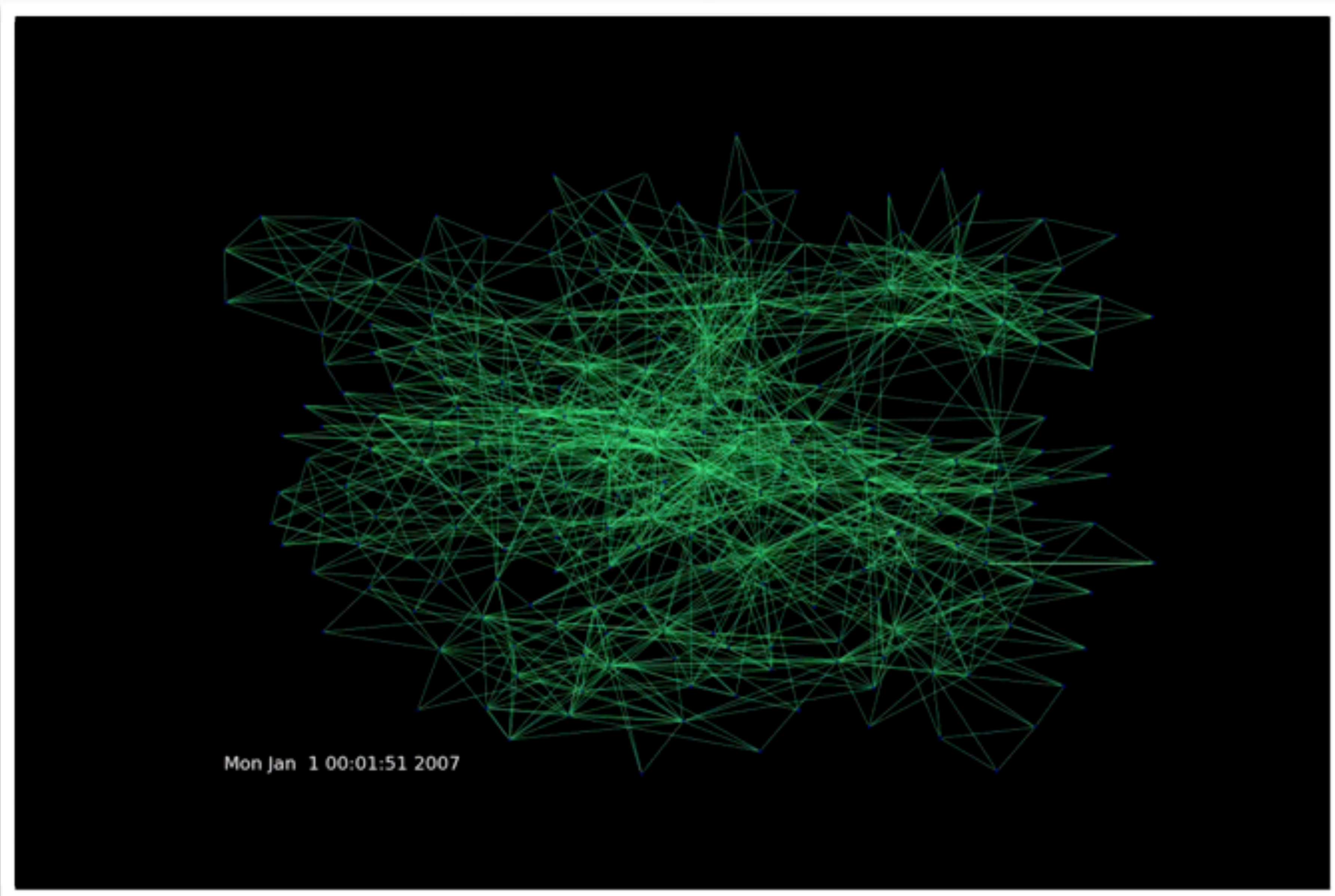
Mikko Kivela

$G=(V_{t'}, E_{t'})$

$v(t) \in V_{t'}$

$(u,v,t) \in E_{t'}$

- Dynamical nodes and links encode the evolution of the network
- Usually t<<t'



Mobile communication network
Nodes - individuals
Links - calls and SMS

WHY

and

WHY NOW?

Why?

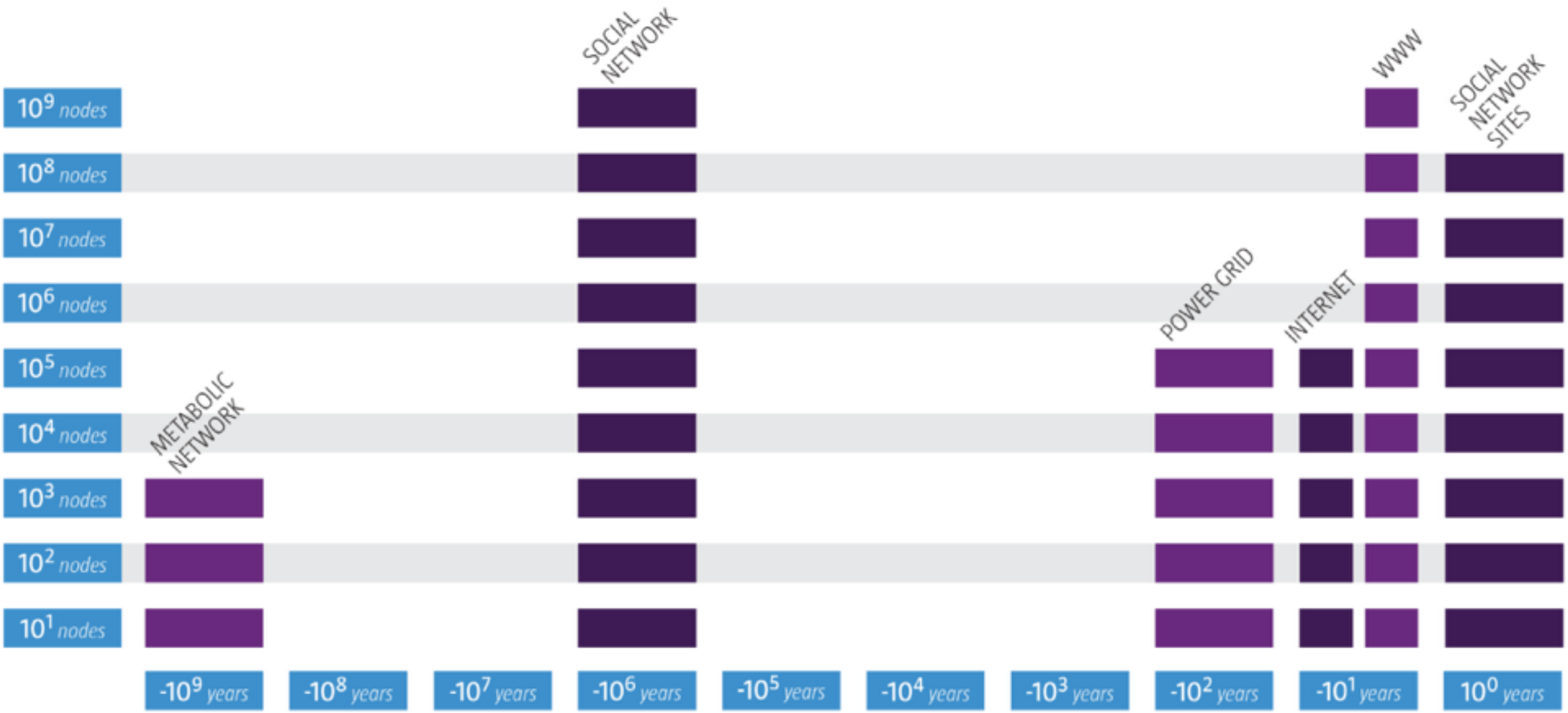
- A common framework applicable to many systems
- Different systems can be studied with same methods
- A “birds-eye” view on the system

MANY NETWORKS SHARE SIMILAR CHARACTERISTICS

- Similar processes shape the networks

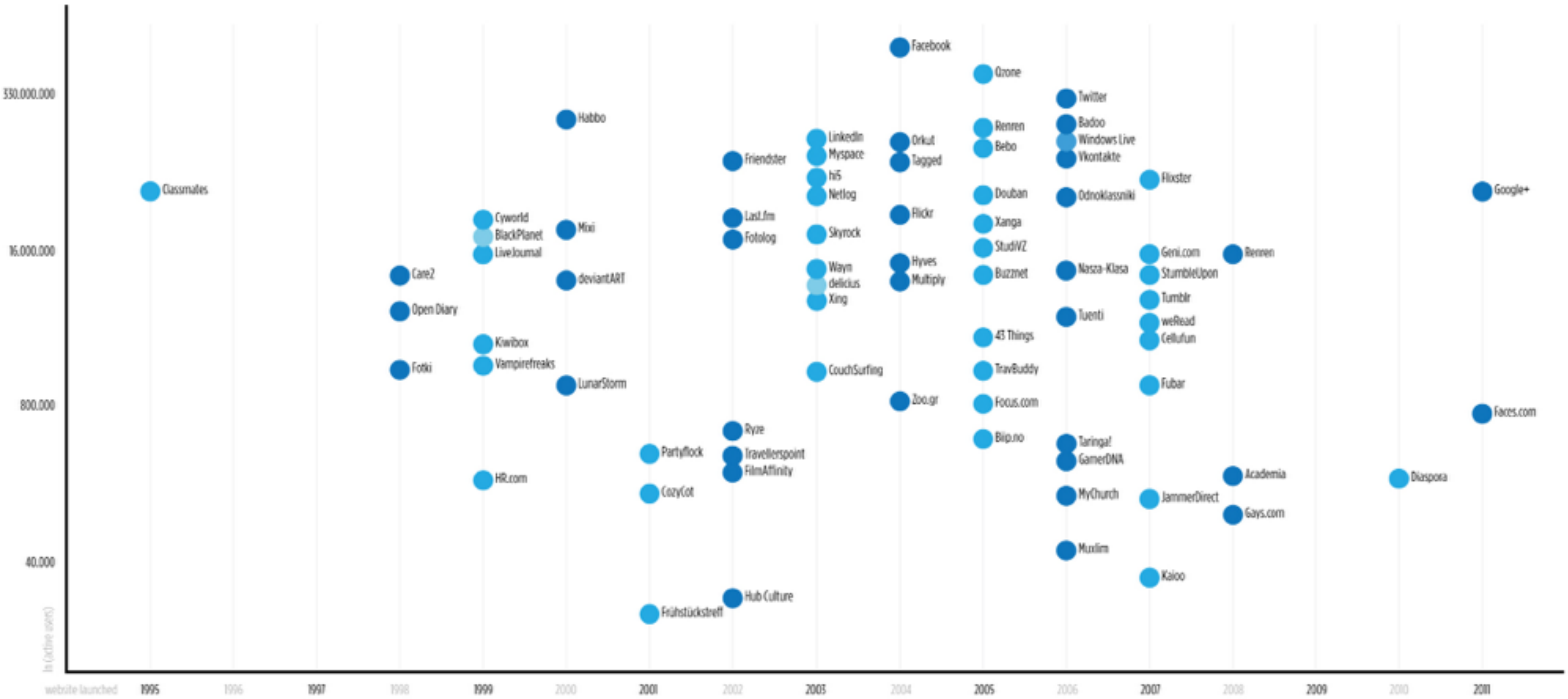
WE WILL NEVER UNDERSTAND COMPLEX SYSTEMS UNLESS WE MAP OUT AND UNDERSTAND THE NETWORKS BEHIND THEM *AL Barabási*

Why now?



Slide from CCNR Complex Networks Course
A. L. Barabási 2012

Why now?



Slide from CCNR Complex Networks Course
A. L. Barabási 2012

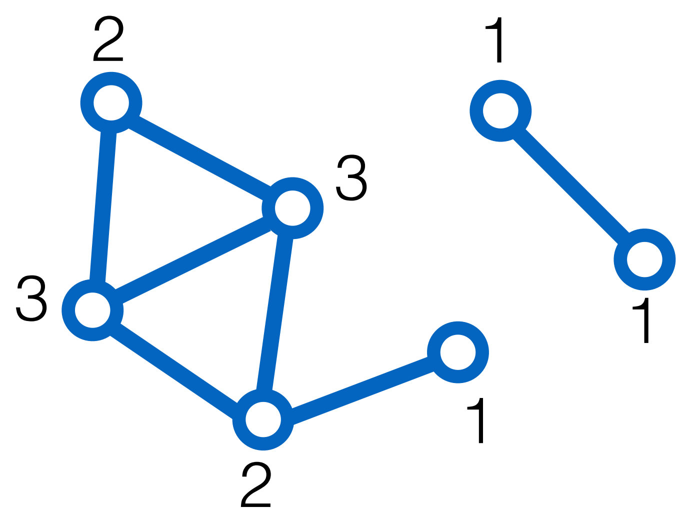
Why now?

- Data availability - the Big Data Revolution
- Universality - similar features of very similar systems
- Urgent need to understand complexity
  - Economic impact
  - Drug design, metabolic engineering
  - Human decease network
  - Fighting, terrorism and military
  - Epidemic forecast
  - Brain research

Characteristics:
The node degree

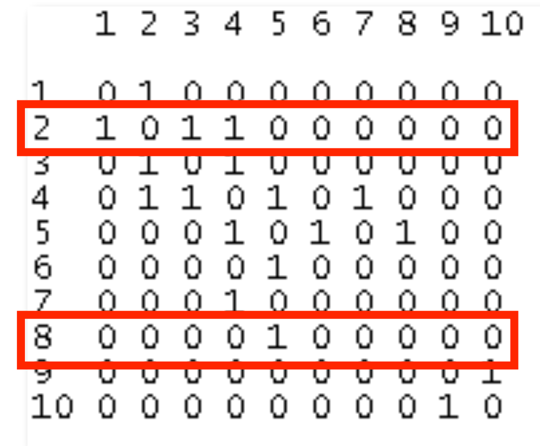
Node degree

- Undirected network



Number of connections of a node

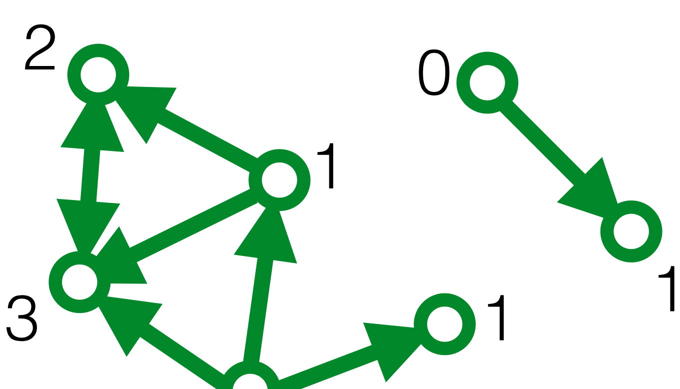
$$k_i = A_{i1} + A_{i2} + \ldots + A_{iN} = \Sigma_j A_{ij}$$



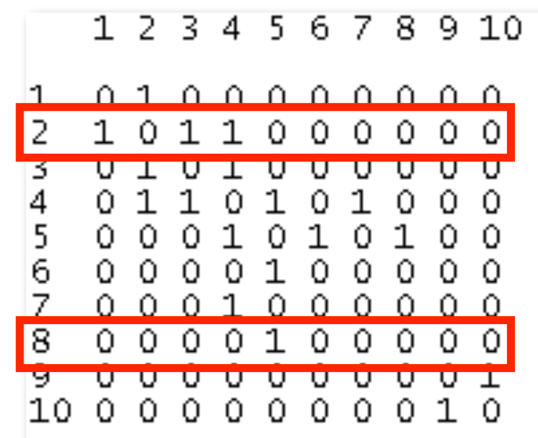
	1	2	3	4	5	6	7	8	9	10
1	0	1	0	0	0	0	0	0	0	0
2	1	0	1	1	0	0	0	0	0	0
3	0	1	0	1	0	0	0	0	0	0
4	0	1	1	0	1	0	1	0	0	0
5	0	0	0	1	0	1	0	1	0	0
6	0	0	0	0	1	0	0	0	0	0
7	0	0	0	1	0	0	0	0	0	0
8	0	0	0	0	1	0	0	0	0	0
9	0	0	0	0	0	0	0	0	0	1
10	0	0	0	0	0	0	0	0	1	0

- Directed network

In degree

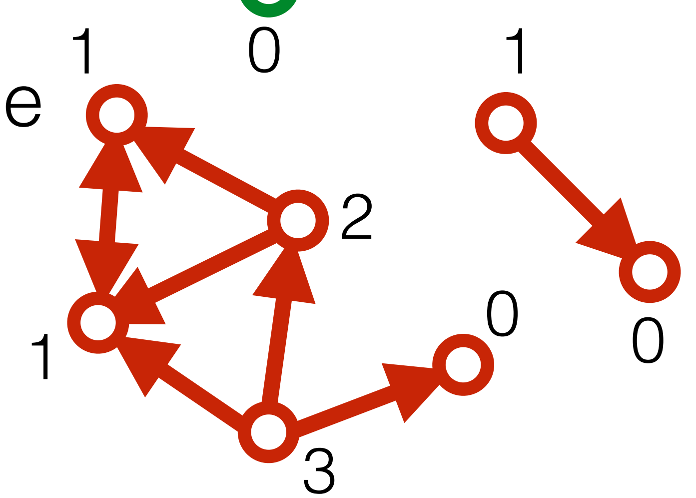


$$k_{in,i} = \Sigma_j A_{ji}$$

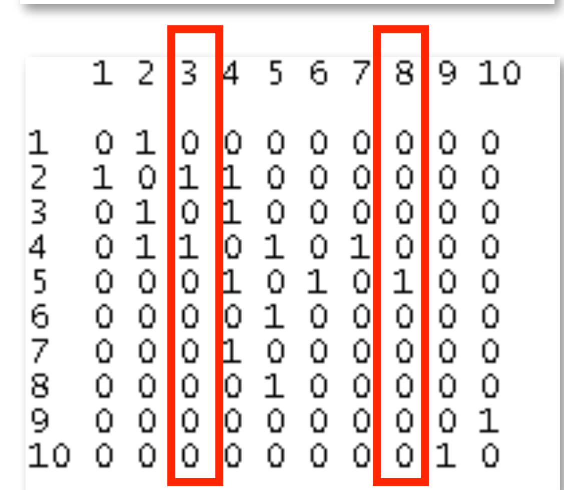


	1	2	3	4	5	6	7	8	9	10
1	0	1	0	0	0	0	0	0	0	0
2	1	0	1	1	0	0	0	0	0	0
3	0	1	0	1	0	0	0	0	0	0
4	0	1	1	0	1	0	1	0	0	0
5	0	0	0	1	0	1	0	1	0	0
6	0	0	0	0	1	0	0	0	0	0
7	0	0	0	1	0	0	0	0	0	0
8	0	0	0	0	1	0	0	0	0	0
9	0	0	0	0	0	0	0	0	0	1
10	0	0	0	0	0	0	0	0	1	0

Out degree

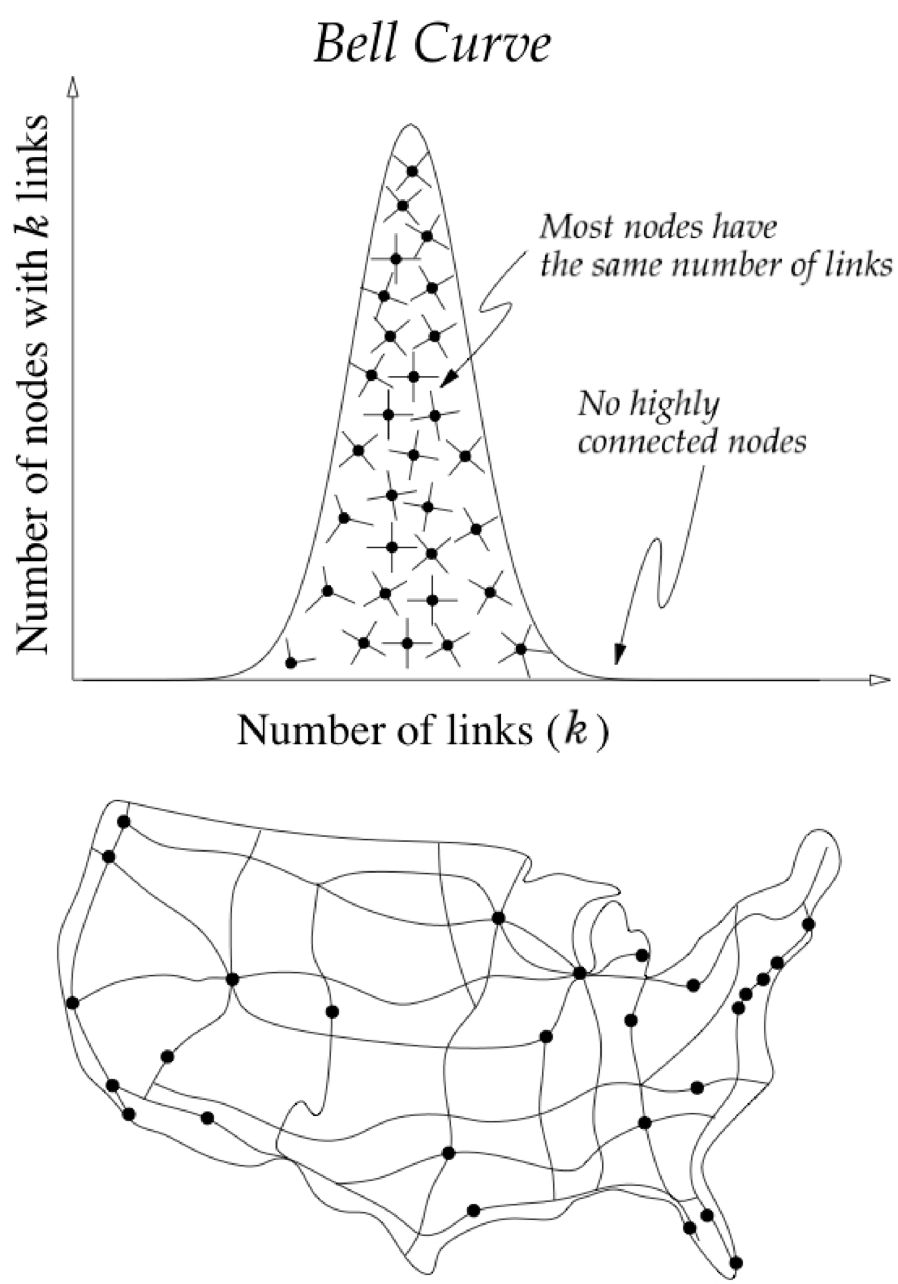


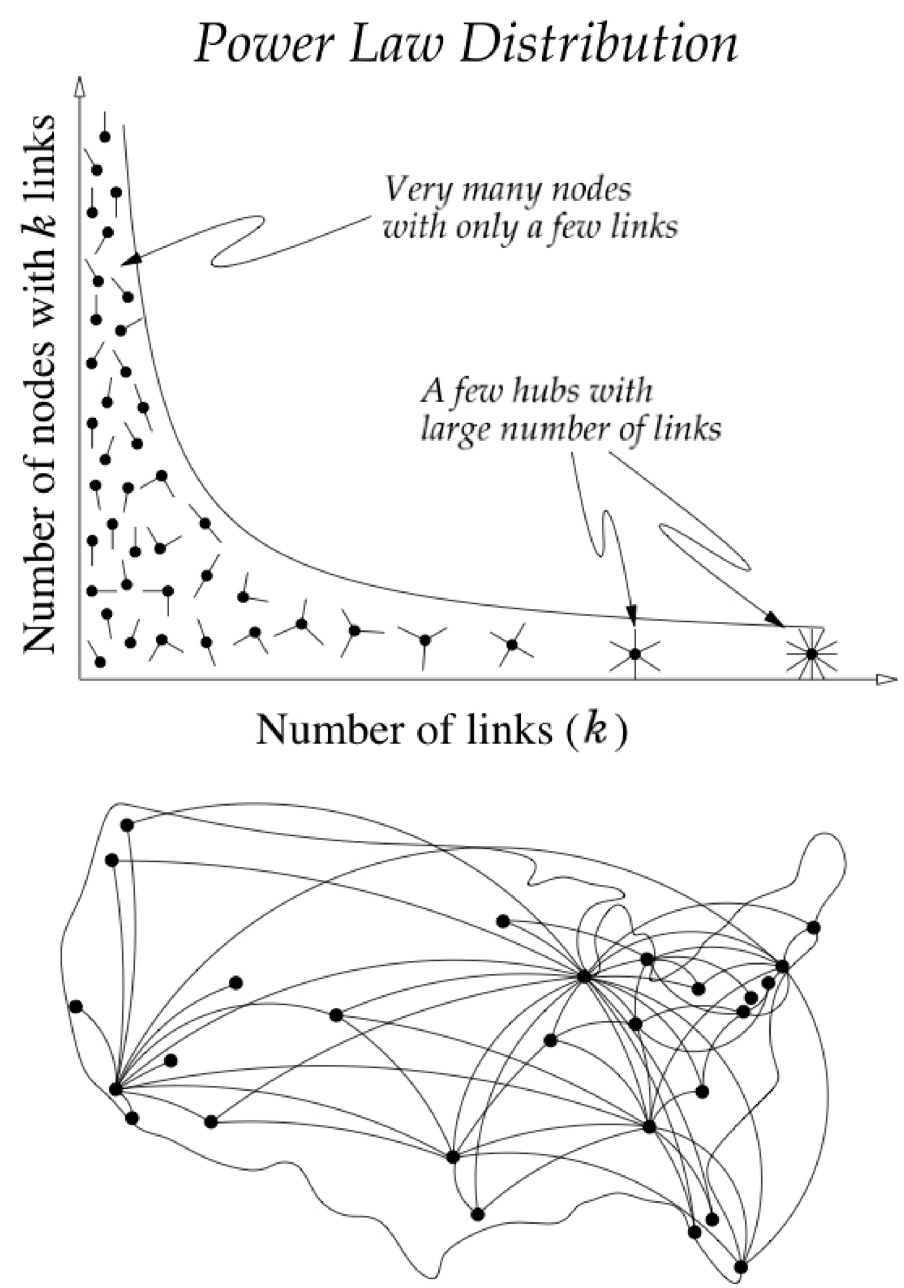
$$k_{out,i} = \Sigma_j A_{ij}$$



	1	2	3	4	5	6	7	8	9	10
1	0	1	0	0	0	0	0	0	0	0
2	1	0	1	1	0	0	0	0	0	0
3	0	1	0	1	0	0	0	0	0	0
4	0	1	1	0	1	0	1	0	0	0
5	0	0	0	1	0	1	0	1	0	0
6	0	0	0	0	1	0	0	0	0	0
7	0	0	0	1	0	0	0	0	0	0
8	0	0	0	0	1	0	0	0	0	0
9	0	0	0	0	0	0	0	0	0	1
10	0	0	0	0	0	0	0	0	1	0

Degree-distribution





- Quasy-regular structures
- Random graphs
- Erdős-Rényi model

- Most of real networks
- Scale-free networks
- Barabási-Albert model

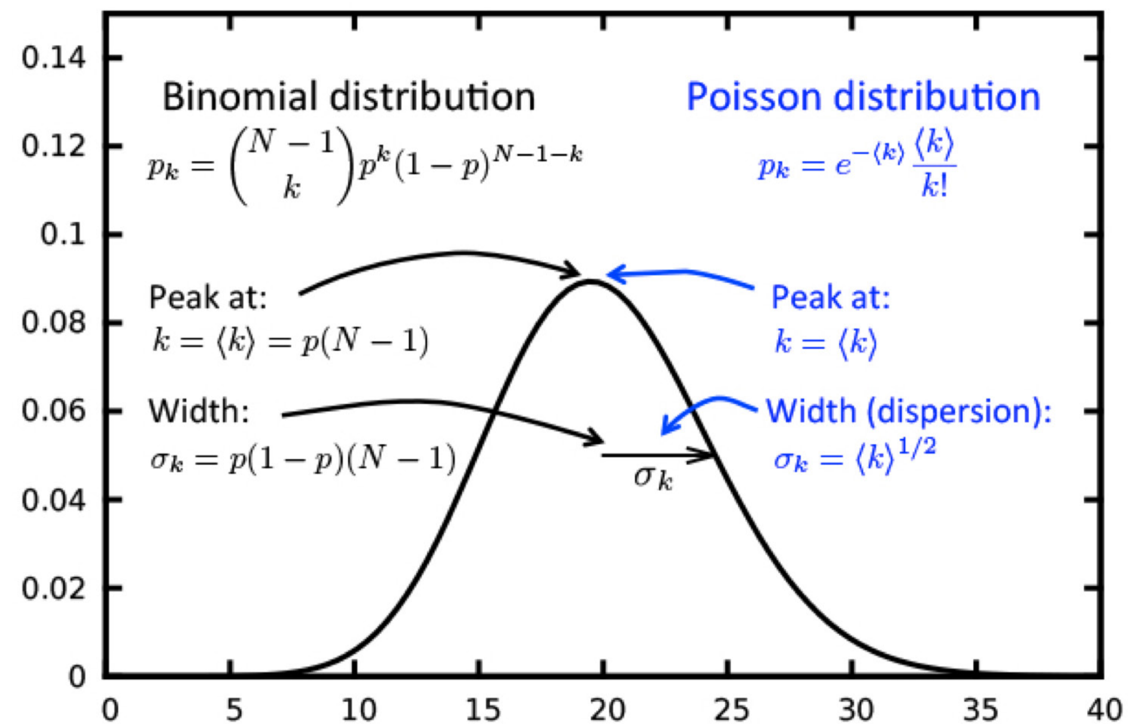
Random Graphs

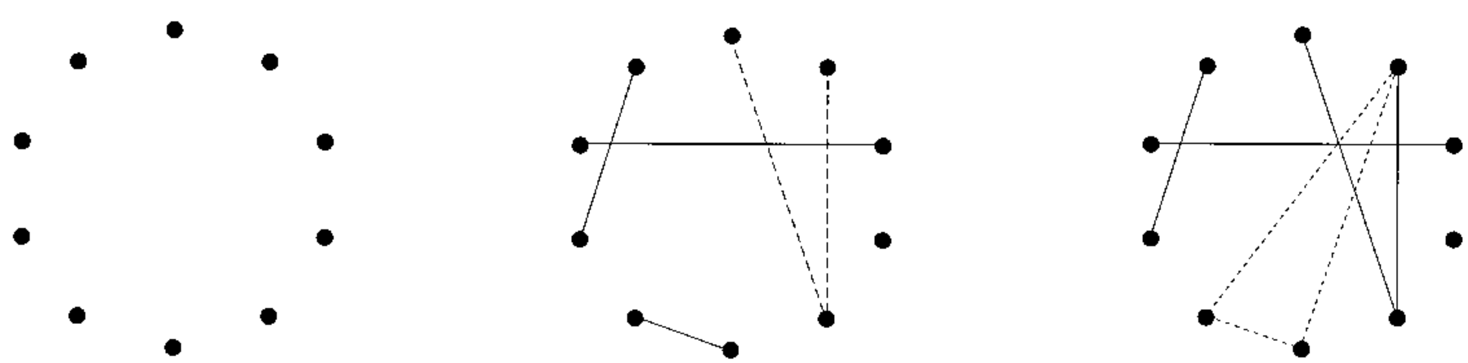
Erdős-Rényi model: simple way to generate random graphs

- The $G(n,p)$ definition
  1. Take n disconnected nodes
  2. Add an edge between any of the nodes independently with probability p

Alternatively:

- pick with probability $p^m(1-p)^{\binom{n}{2}-m}$ a network from the set of all networks with size n





p=0 p=0.1 p=0.15

Degree distribution

$$p_k = \frac{\langle k \rangle^k}{k!} e^{-\langle k \rangle}$$

Poisson distribution

Scale-free networks - first observations

Networks of scientific papers **Derek J. de Solla Price**, Science (1965)

- Nodes: scientific papers, Links: citations between them
- Number of citations to scientific papers shows a heavy-tailed distribution
- It can be characterised as a Pareto distribution or power-law distribution

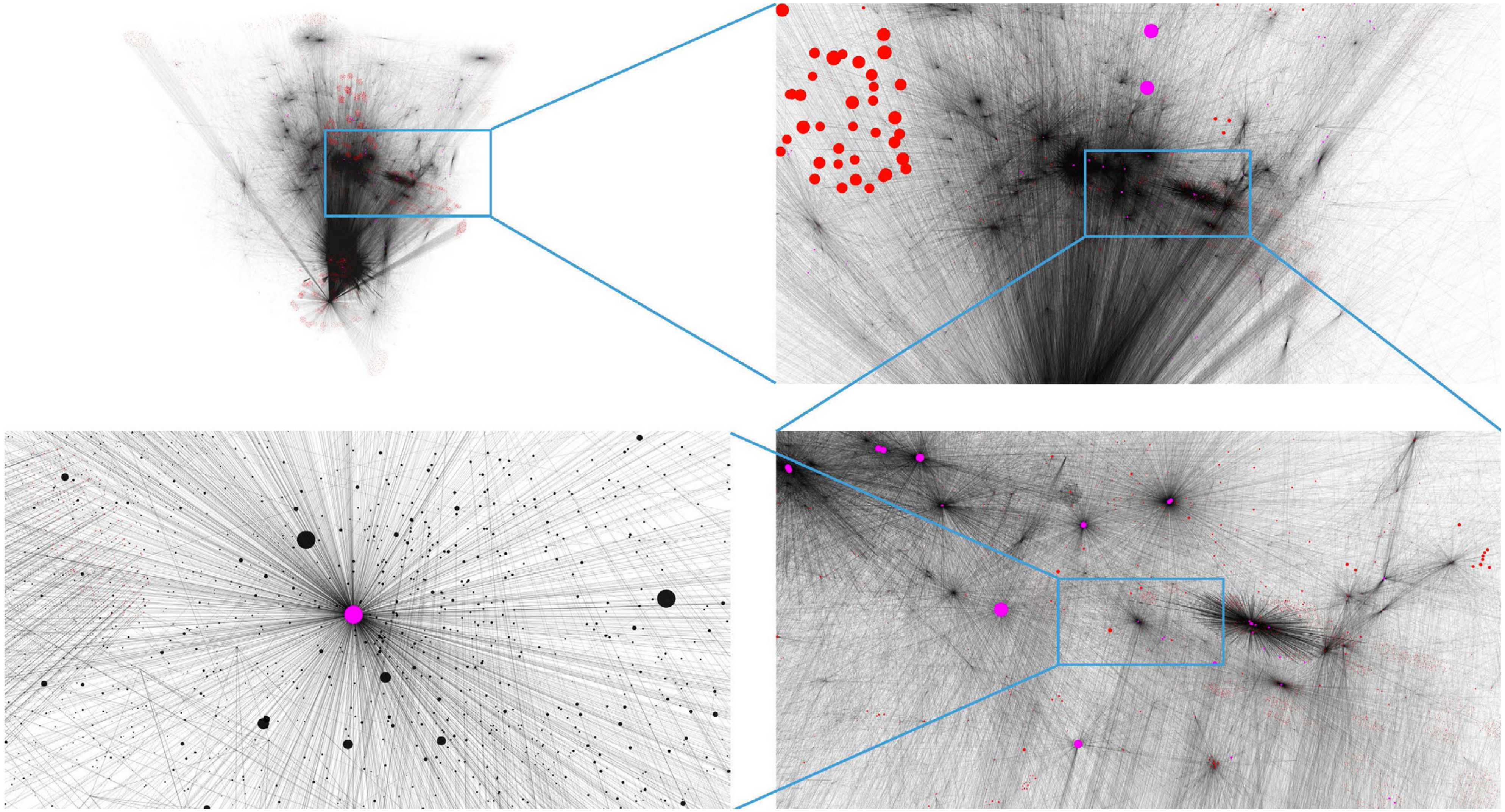
Structure of the WWW **R. Albert, H. Jeong, A-L Barabási**, Nature (1999)

- Nodes: WWW documents, Links: URL links
- More than 3 billions of documents
- Collection by a robot which explores all URL links in a document (web site) and follow them recursively
- They found a heavy-tailed degree distribution which could be well approximated with a power-law function

$$P(k) \sim k^{-\gamma}$$

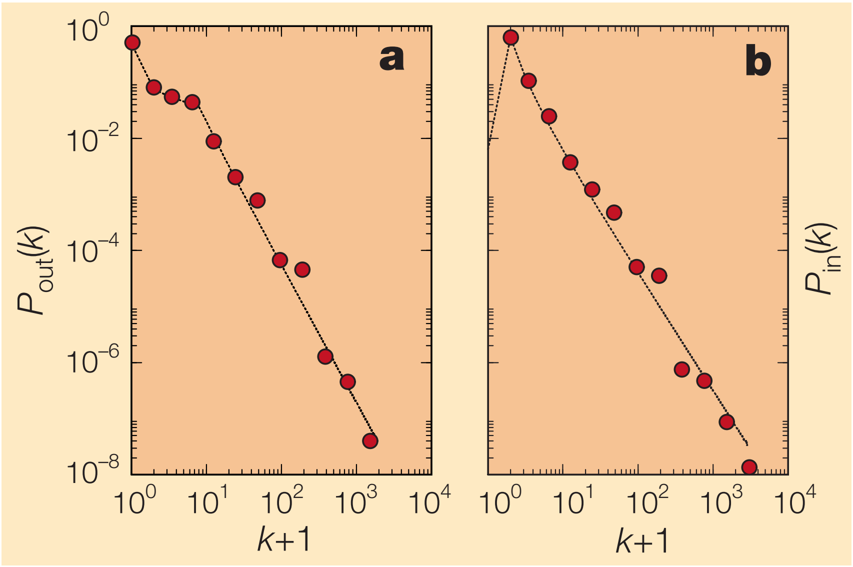
- It is a scale-free network

Scale-free networks - first observations



AL Barabási, Network Science Book (2013)

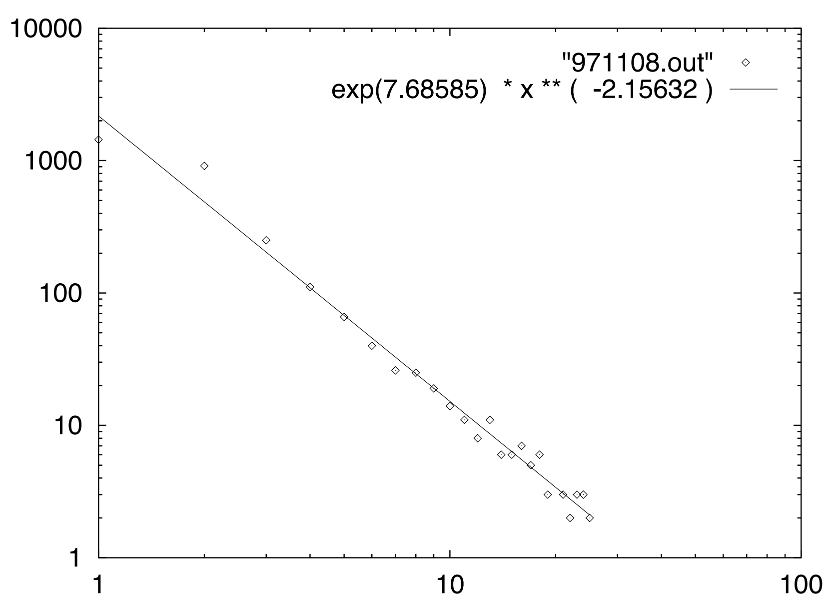
R. Albert, H. Jeong, A-L Barabási, Nature (1999)



Scale-free networks - other examples

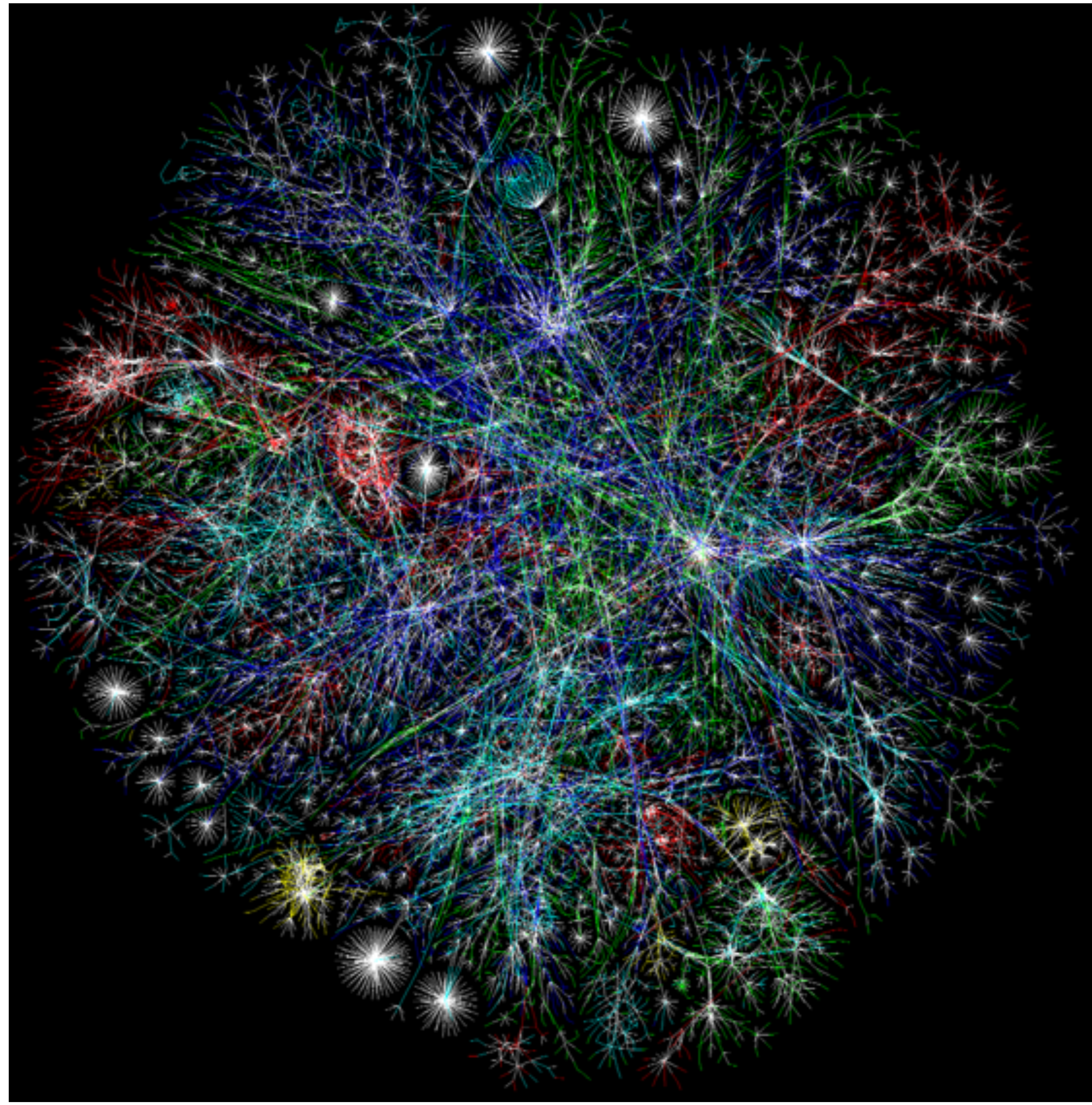
The internet

- Nodes: routers
- Links: Physical wires



(a) Int-11-97

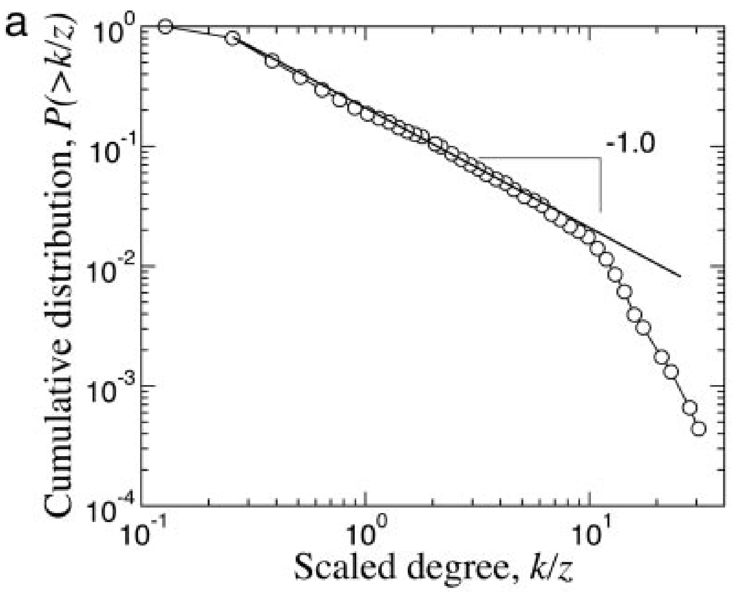
Faloutsos, Faloutsos and Faloutsos (1999)



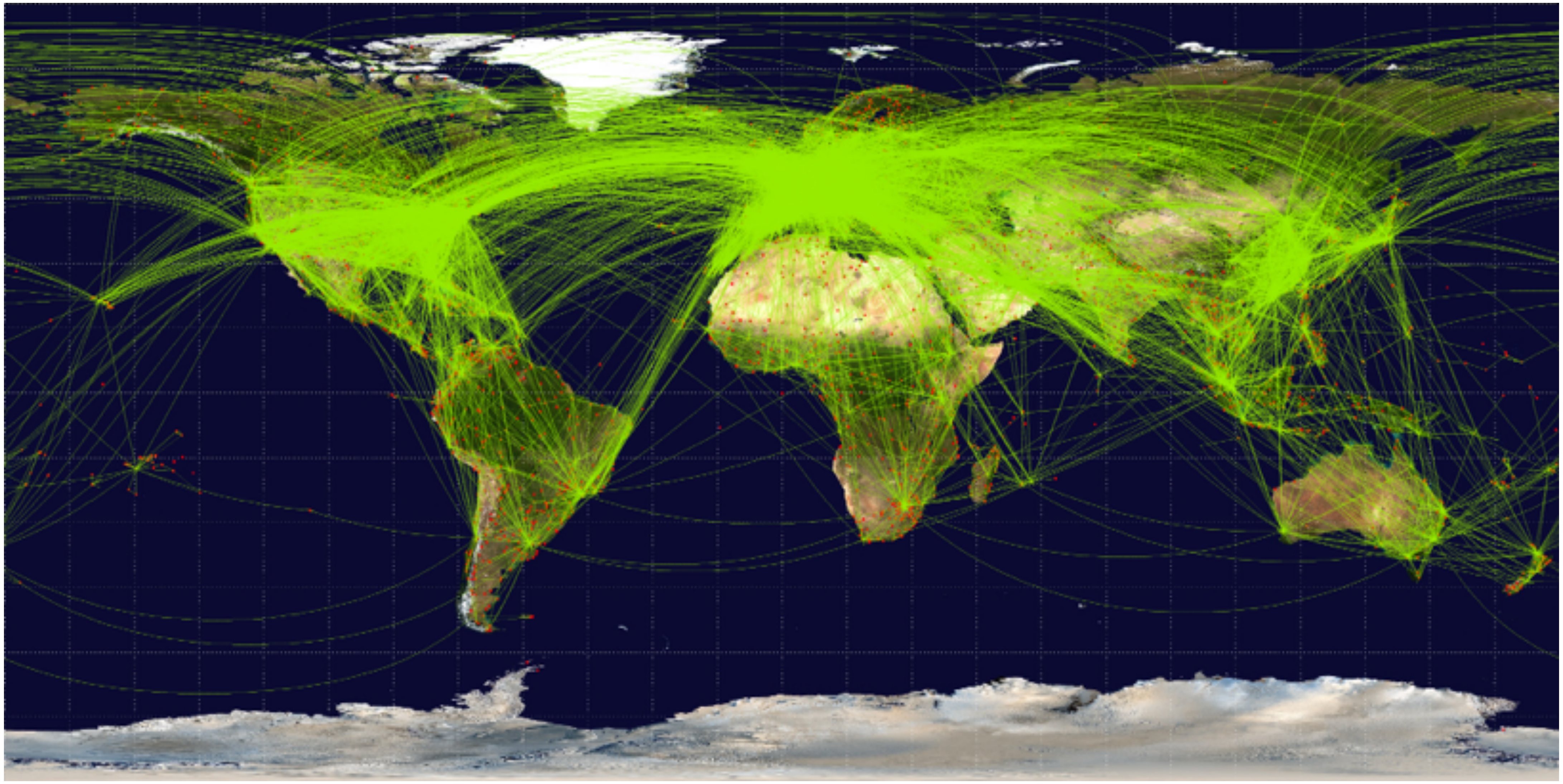
Scale-free networks - other examples

Airline route map network

- Nodes: airports
- Links: airplane connections



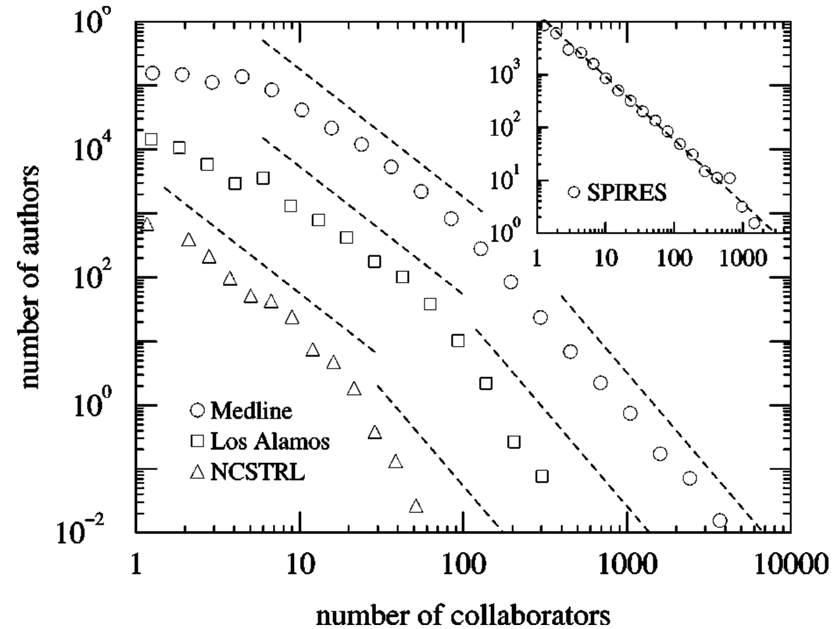
Guimera et.al. (2004)



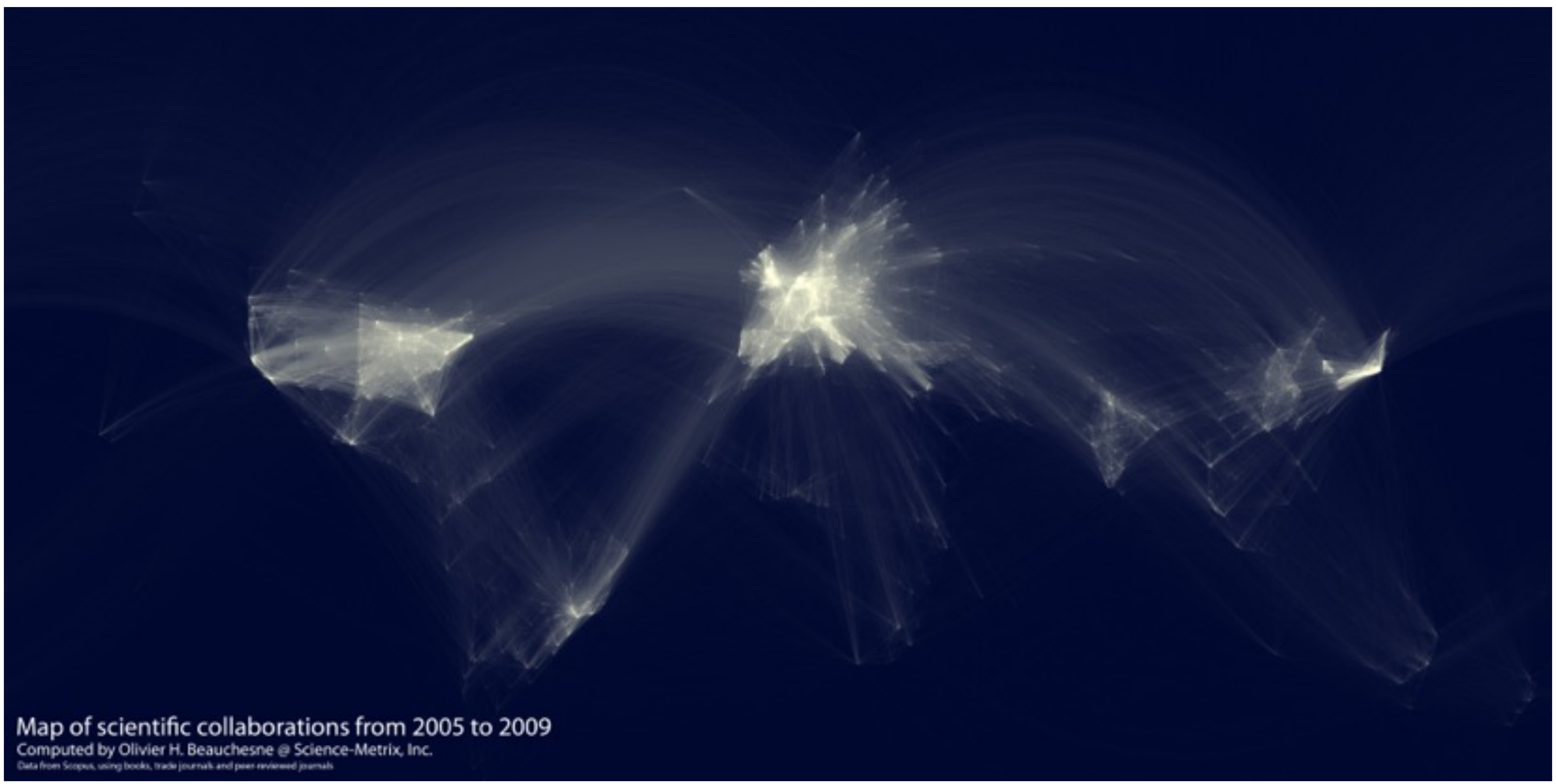
Scale-free networks - other examples

Scientific collaborations

- Nodes: scientists (here geo-localised)
- Links: common papers



Newman (2001)

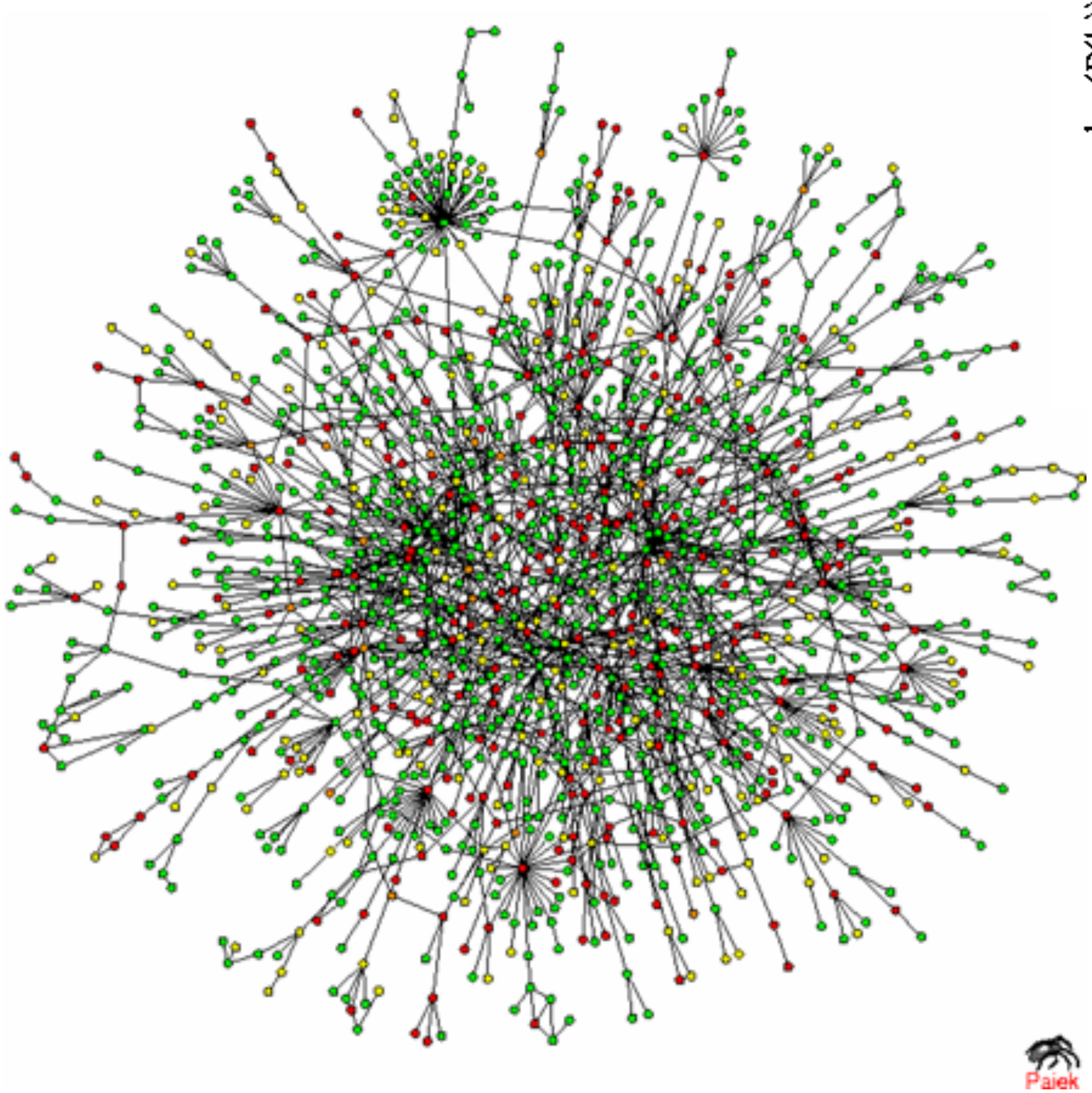


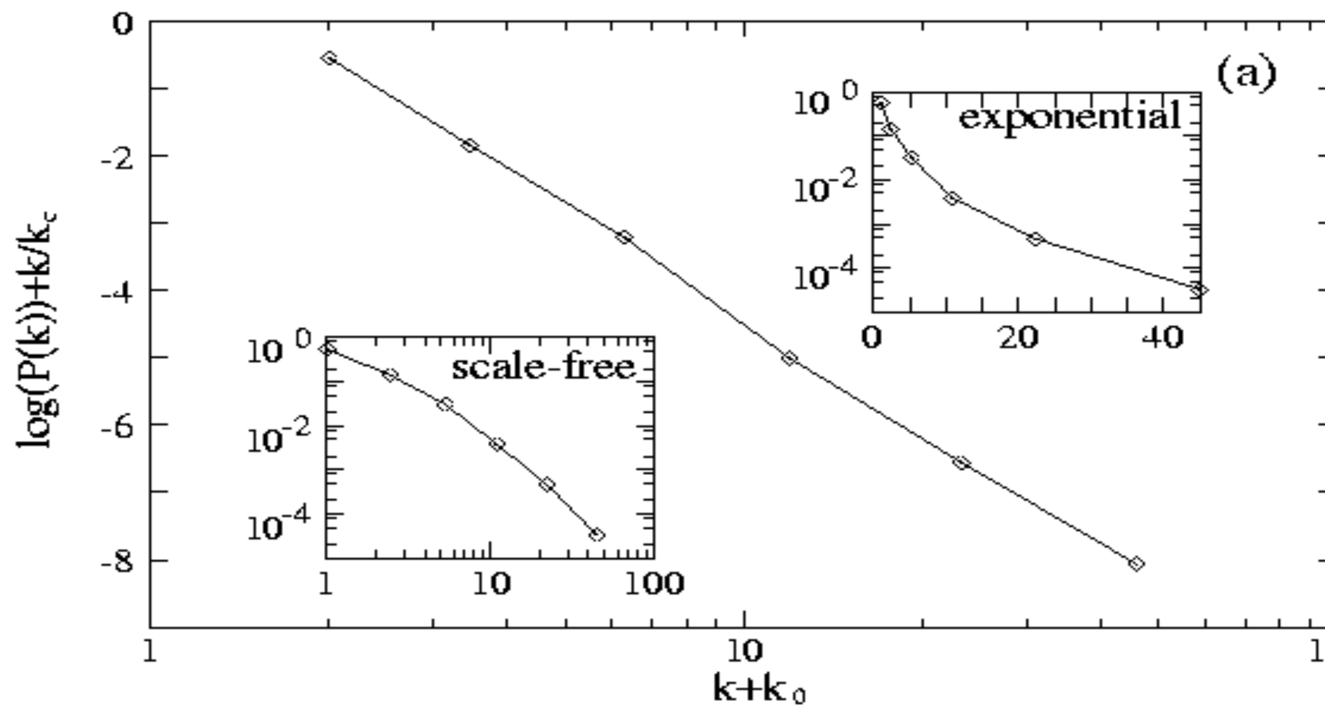
Scale-free networks - other examples

Protein networks

Jeong et.al. (2001)

- Nodes: proteins
- Links: physical interactions-binding





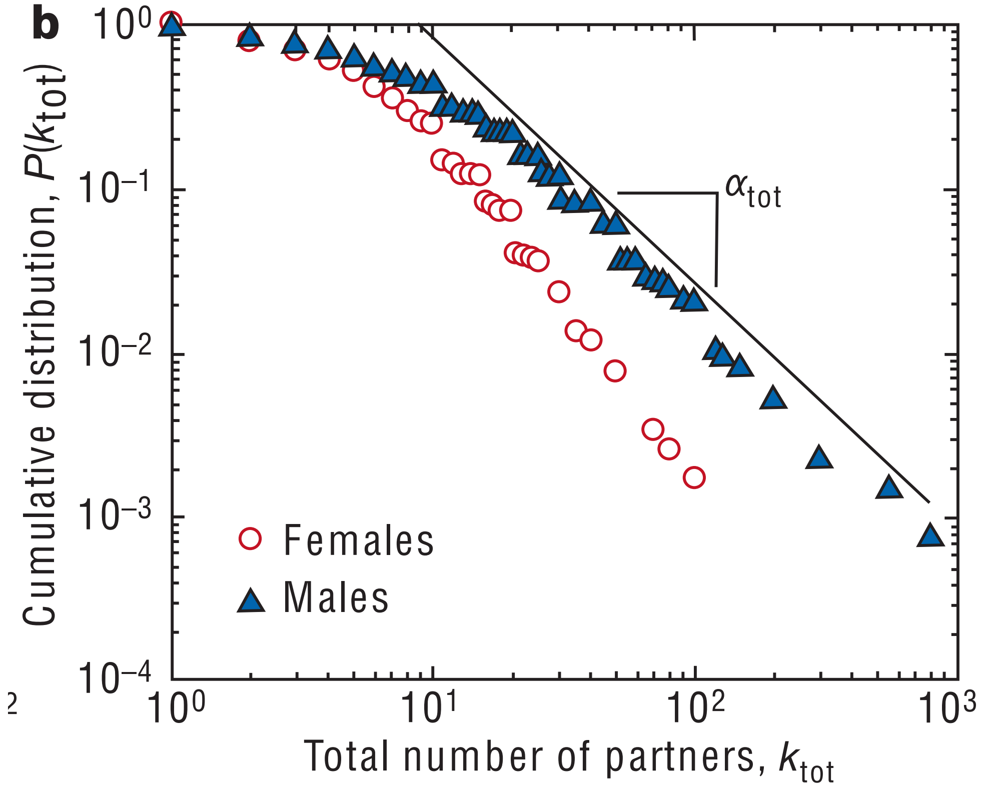
$$P(k) \sim (k + k_0)^{-\gamma} \exp(-\frac{k + k_0}{k_\tau})$$

Scale-free networks - other examples

Sexual-interaction networks

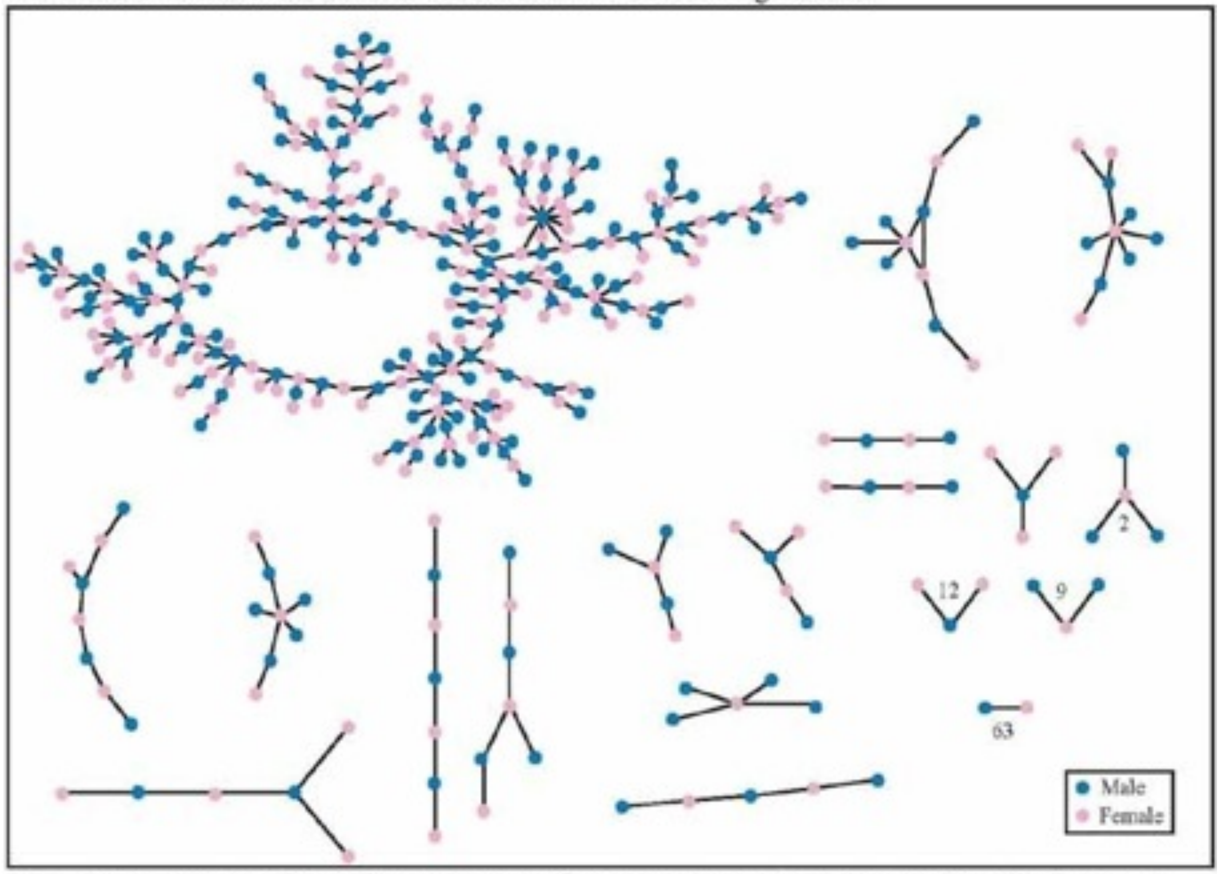
- Nodes: individuals
- Links: sexual incursion

Liljeros et.al. (2001)



Bearman et.al. (2004)

The Structure of Romantic and Sexual Relations at "Jefferson High School"

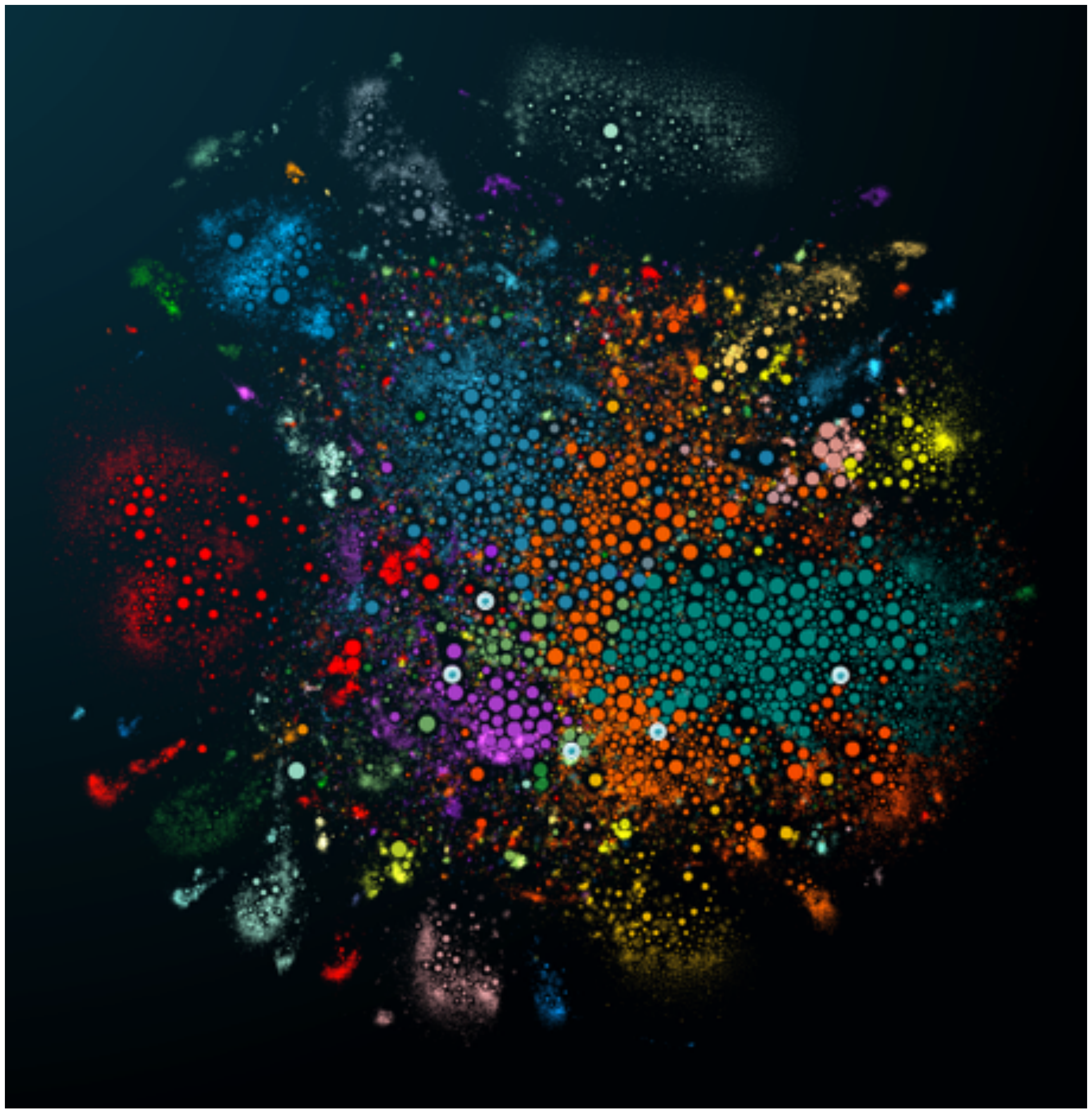


Each circle represents a student and lines connecting students represent romantic relations occuring within the 6 months preceding the interview. Numbers under the figure count the number of times that pattern was observed (i.e. we found 63 pairs unconnected to anyone else).

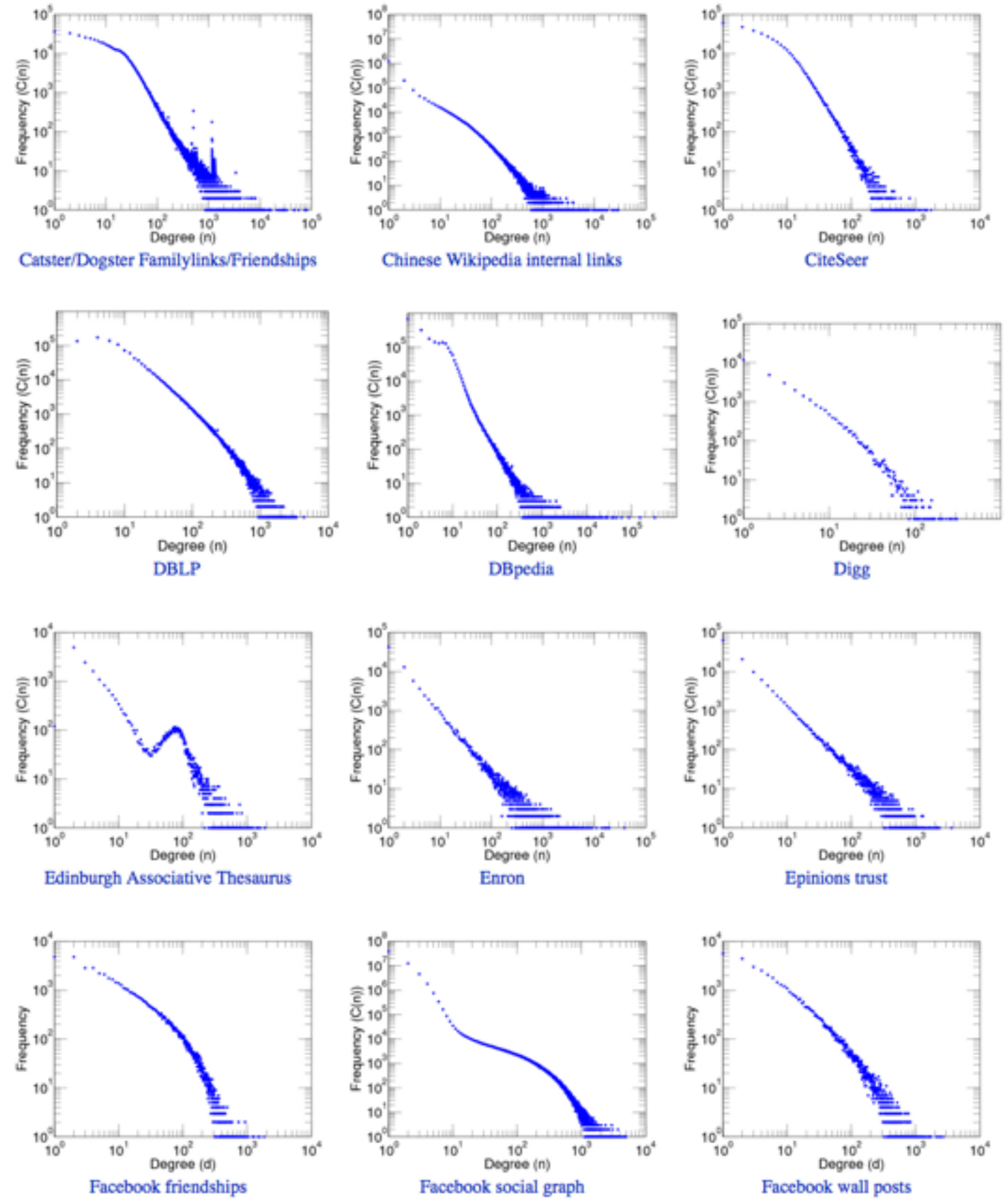
Scale-free networks - other examples

Online social networks

- Nodes: individuals
- Links: online interactions



Social network of Steam
http://85.25.226.110/mapper



Scale-free networks

Network	Size	$\langle k \rangle$	κ	γ_{out}	γ_{in}	ℓ_{real}	ℓ_{rand}	ℓ_{pow}	Reference
WWW	325 729	4.51	900	2.45	2.1	11.2	8.32	4.77	Albert, Jeong, and Barabási 1999
WWW	4×10^7	7		2.38	2.1				Kumar *et al.*, 1999
WWW	2×10^8	7.5	4000	2.72	2.1	16	8.85	7.61	Broder *et al.*, 2000
WWW, site	260 000				1.94				Huberman and Adamic, 2000
Internet, domain*	3015–4389	3.42–3.76	30–40	2.1–2.2	2.1–2.2	4	6.3	5.2	Faloutsos, 1999
Internet, router*	3888	2.57	30	2.48	2.48	12.15	8.75	7.67	Faloutsos, 1999
Internet, router*	150 000	2.66	60	2.4	2.4	11	12.8	7.47	Govindan, 2000
Movie actors*	212 250	28.78	900	2.3	2.3	4.54	3.65	4.01	Barabási and Albert, 1999
Co-authors, SPIRES*	56 627	173	1100	1.2	1.2	4	2.12	1.95	Newman, 2001b
Co-authors, neuro.*	209 293	11.54	400	2.1	2.1	6	5.01	3.86	Barabási *et al.*, 2001
Co-authors, math.*	70 975	3.9	120	2.5	2.5	9.5	8.2	6.53	Barabási *et al.*, 2001
Sexual contacts*	2810			3.4	3.4				Liljeros *et al.*, 2001
Metabolic, *E. coli*	778	7.4	110	2.2	2.2	3.2	3.32	2.89	Jeong *et al.*, 2000
Protein, *S. cerev.**	1870	2.39		2.4	2.4				Jeong, Mason, *et al.*, 2001
Ythan estuary*	134	8.7	35	1.05	1.05	2.43	2.26	1.71	Montoya and Solé, 2000
Silwood Park*	154	4.75	27	1.13	1.13	3.4	3.23	2	Montoya and Solé, 2000
Citation	783 339	8.57			3				Redner, 1998
Phone call	53×10^6	3.16		2.1	2.1				Aiello *et al.*, 2000
Words, co-occurrence*	460 902	70.13		2.7	2.7				Ferrer i Cancho and Solé, 2001
Words, synonyms*	22 311	13.48		2.8	2.8				Yook *et al.*, 2001b

Albert, R. et.al. Rev. Mod. Phy. (2002)

Exponents of real-world networks are usually between 2 and 3

Scale-free distribution - continuous formalism

$$P(k) = Ck^{-\gamma} \qquad k = [K_{\min}, \infty)$$

$$\int_{K_{\min}}^{\infty} P(k)dk = 1 \qquad C = \frac{1}{\int_{K_{\min}}^{\infty} k^{-\gamma}\, dk} = (\gamma - 1)K_{\min}^{\gamma-1}$$

$$P(k) = (\gamma - 1)K_{\min}^{\gamma-1}k^{-\gamma}$$

Scale-free distribution - continuous formalism

m-th moment of the degree distribution: $$< k^m >= \int_{K_{\min}}^{\infty} k^m P(k) dk$$

$$P(k) = (\gamma - 1) K_{\min}^{\gamma - 1} k^{-\gamma} \qquad k = [K_{\min}, \infty)$$

$$< k^m >= (\gamma - 1) K_{\min}^{\gamma - 1} \int_{K_{\min}}^{\infty} k^{m-\gamma} dk = \frac{(\gamma - 1)}{(m - \gamma + 1)} K_{\min}^{\gamma - 1} \left[k^{m-\gamma+1} \right]_{K_{\min}}^{\infty}$$

If m-γ+1<0: $$< k^m >= -\frac{(\gamma - 1)}{(m - \gamma + 1)} K_{\min}^{m}$$

If m-γ+1>0, the integral diverges.
For a fixed γ this means that all moments with m>γ-1 diverge.

Scale-free distribution - continuous formalism

Network	Size	$\langle k \rangle$	κ	γ_{out}	γ_{in}
WWW	325 729	4.51	900	2.45	2.1
WWW	4×10^7	7		2.38	2.1
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Albert, R. et.al. Rev. Mod. Phy. (2002)

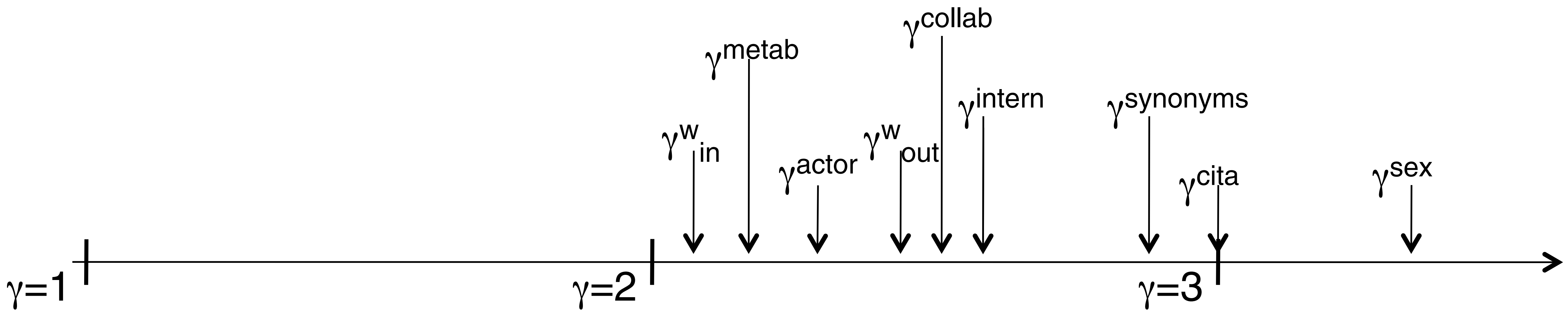
- Exponents of real-world networks are usually between 2 and 3
- $\Rightarrow$ $<k^2>$ diverges if $N\to\infty$
- Consequently:

$$\sigma_k = (\langle k^2 \rangle - \langle k \rangle^2)^{1/2} \to \infty$$

$$k = \langle k \rangle \pm \sigma_k = \langle k \rangle \pm \infty$$

- Average values are meaningless since the fluctuations are infinitely large

Scale-free networks - summary



$\langle k^2 \rangle$ diverges		$\langle k^2 \rangle$ finite
$\langle k \rangle$ diverges	$\langle k \rangle$ finite	
Ultra small world behavior		Small world
Regime full of anomalies…	The scale-free behavior is relevant	Behaves like a random network

Slide from CCNR Complex Networks Course
A. L. Barabási 2012

The Barabási-Albert model

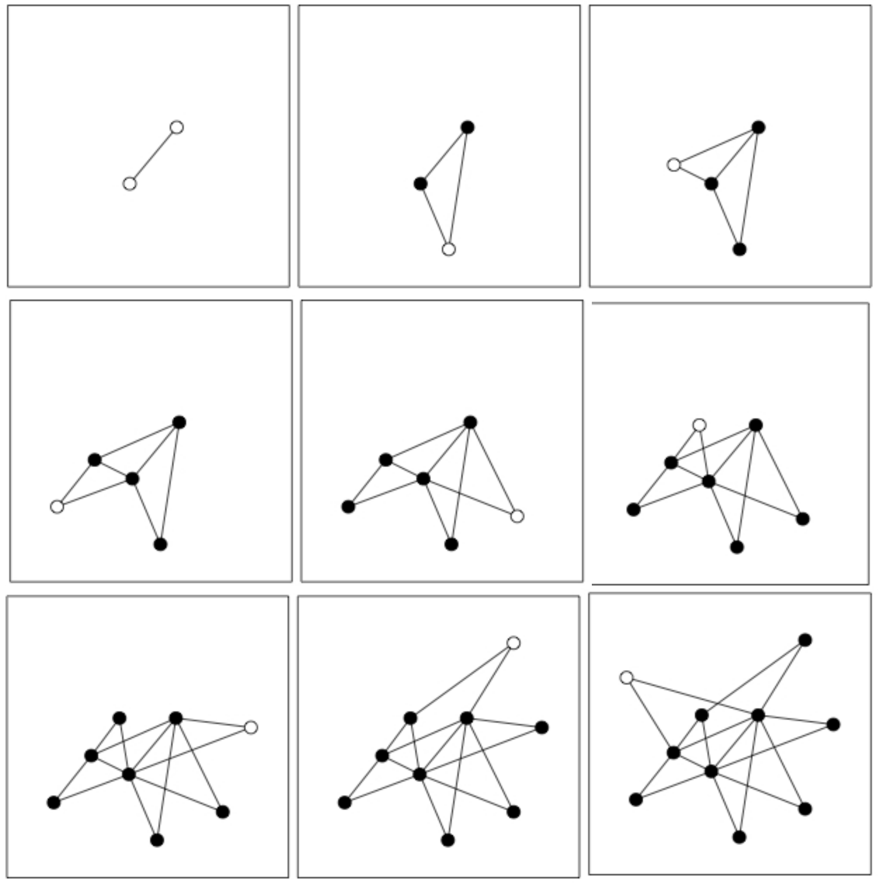
1. Start with m_0 connected nodes

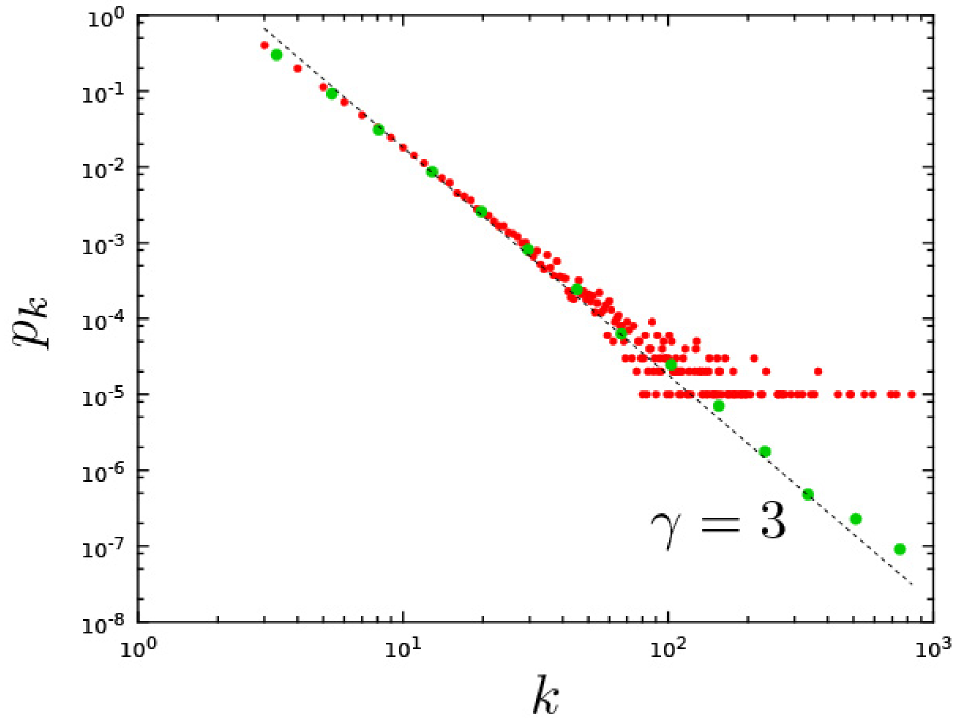
2. At each timestep we add a new node with m ($\leq m_0$) links that connect the new node to m nodes already in the network.

3. The probability $\pi(k)$ that one of the links of the new node connects to node i depends on the degree k_i of node i as

$$\Pi(k_i) = \frac{k_i}{\sum_j k_j}$$

- The emerging network will be scale-free with degree exponent $\gamma=3$ independently from the choice of m_0 and m



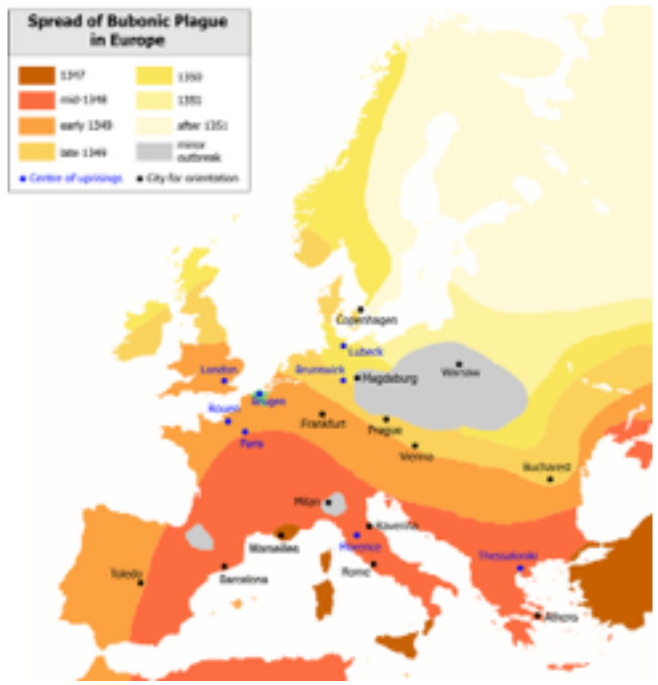


AL Barabási, Network Science Book (2013)

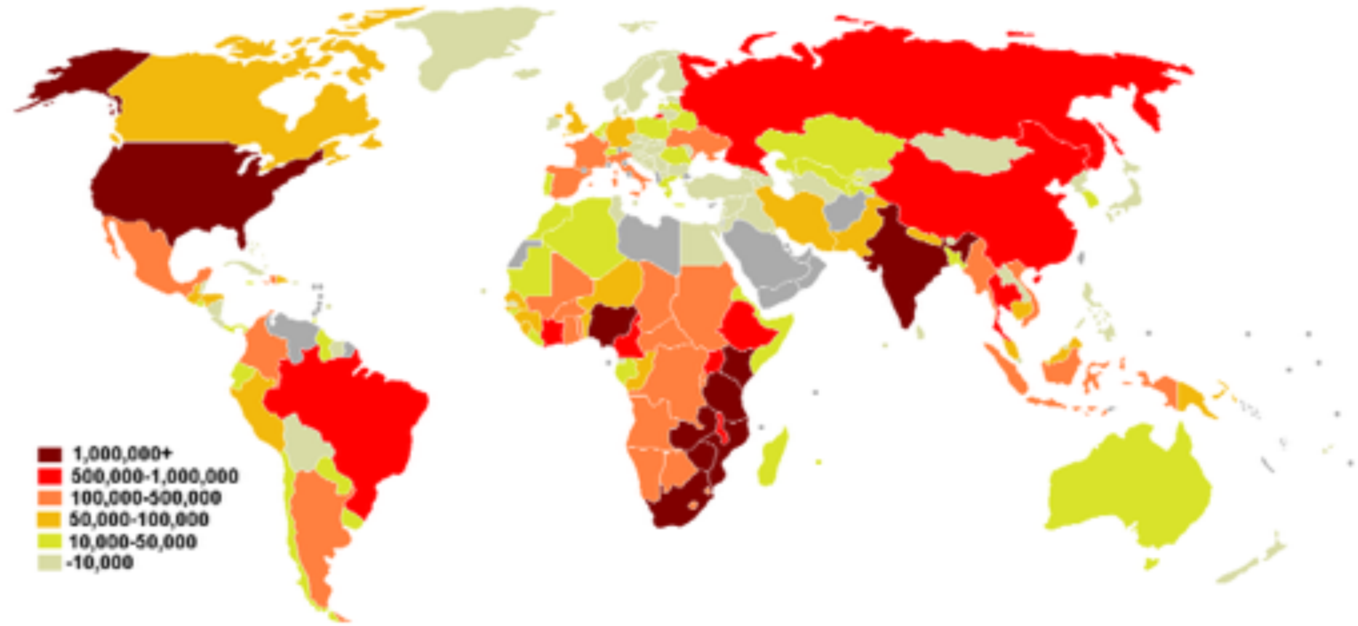
Spreading processes

Spreading processes

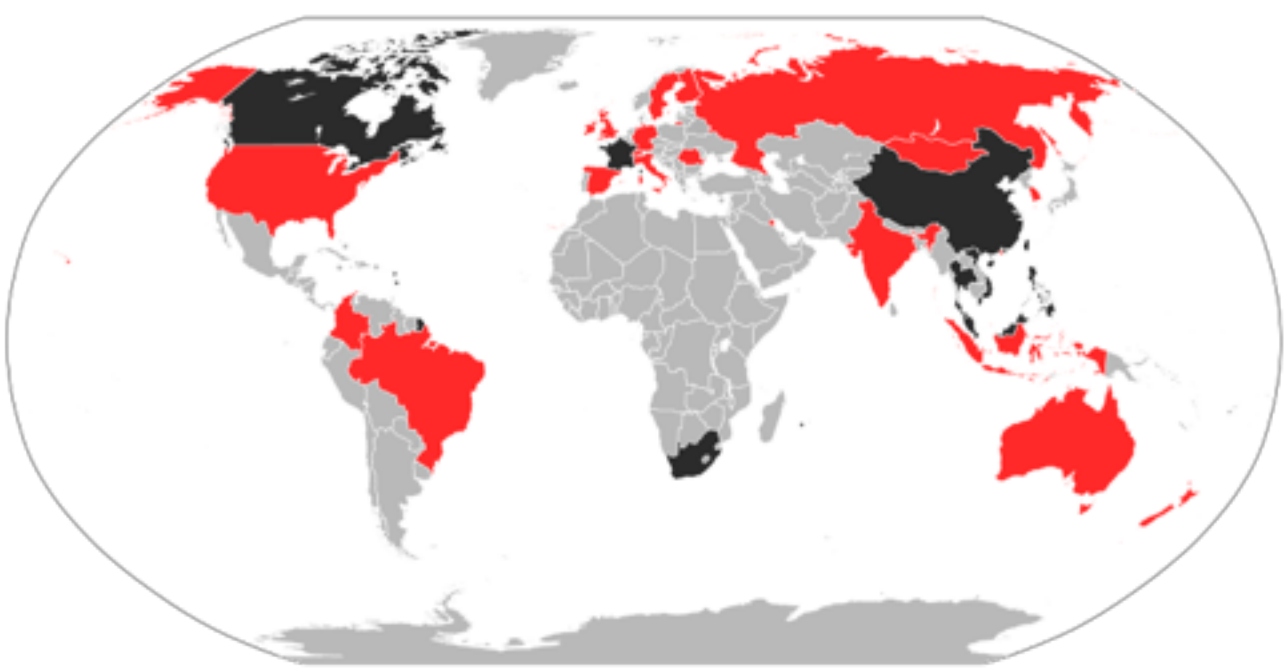
Biological epidemic spreading



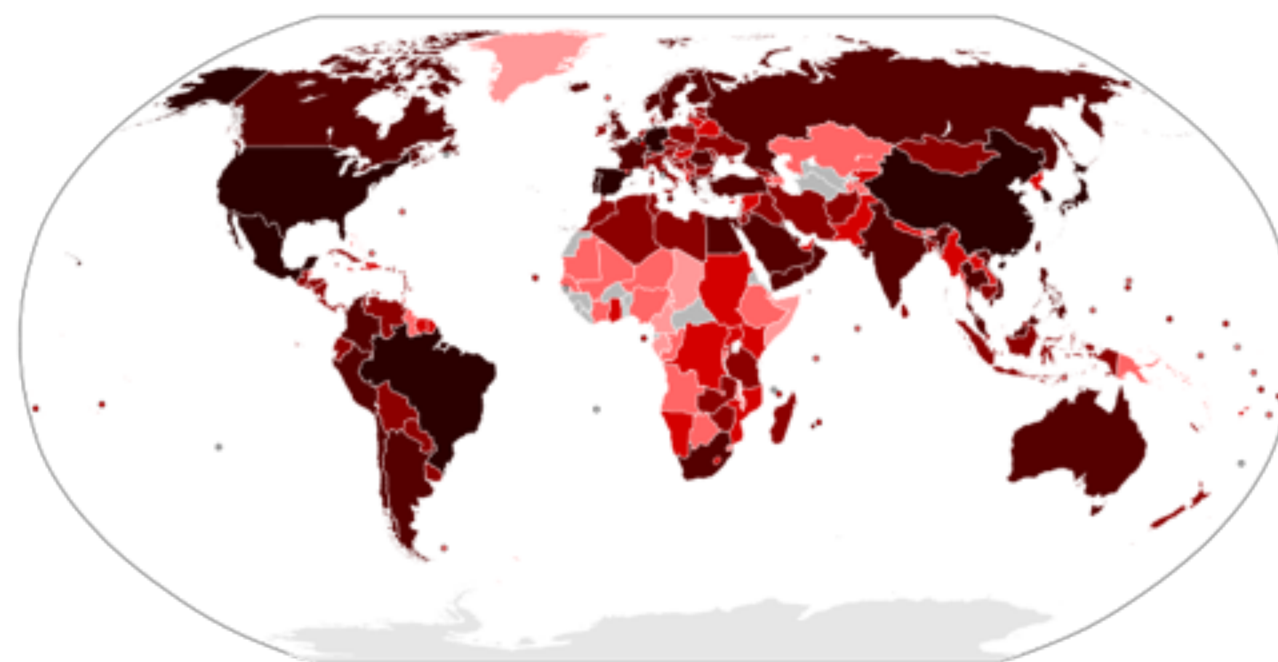
The great plague (14th century)



HIV (2008)



SARS (2008)

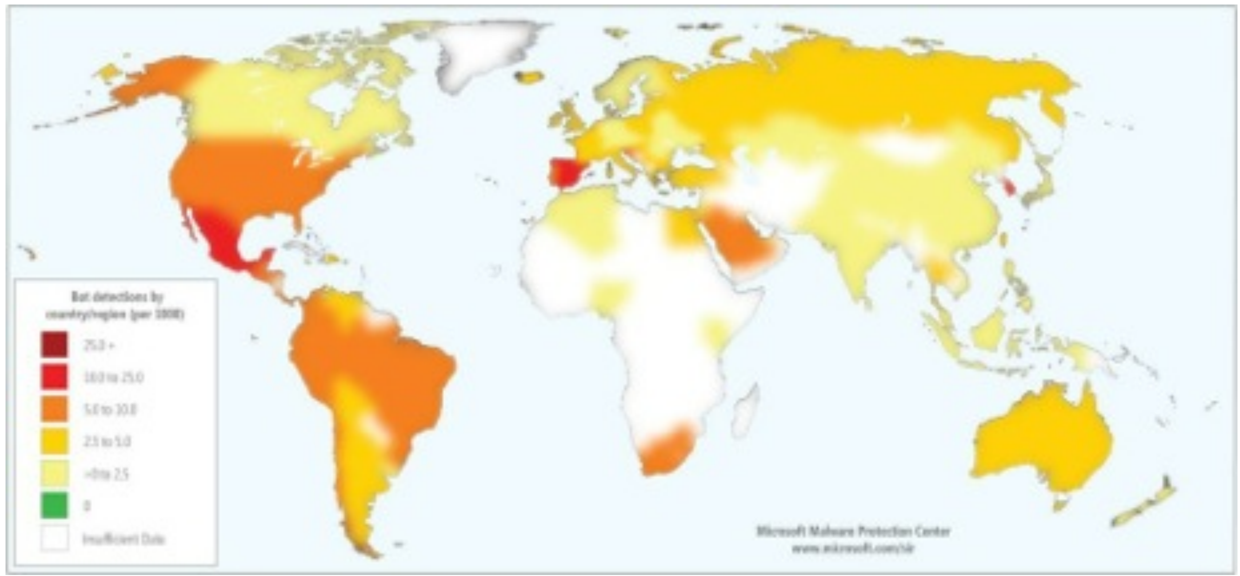


H1N1 (2011)

Wikipedia

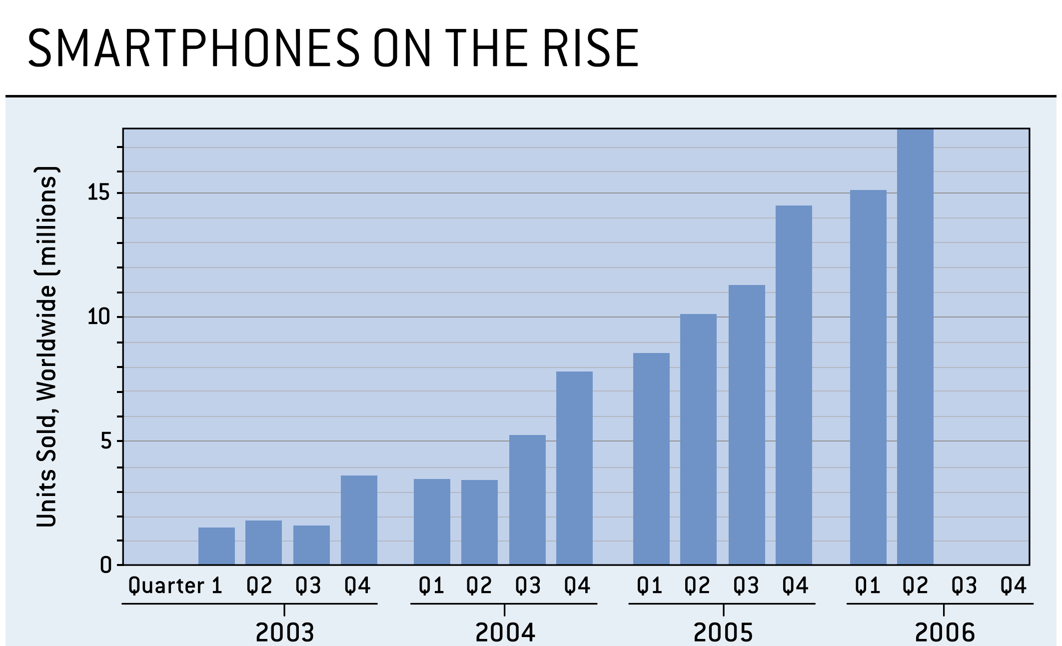
Spreading processes

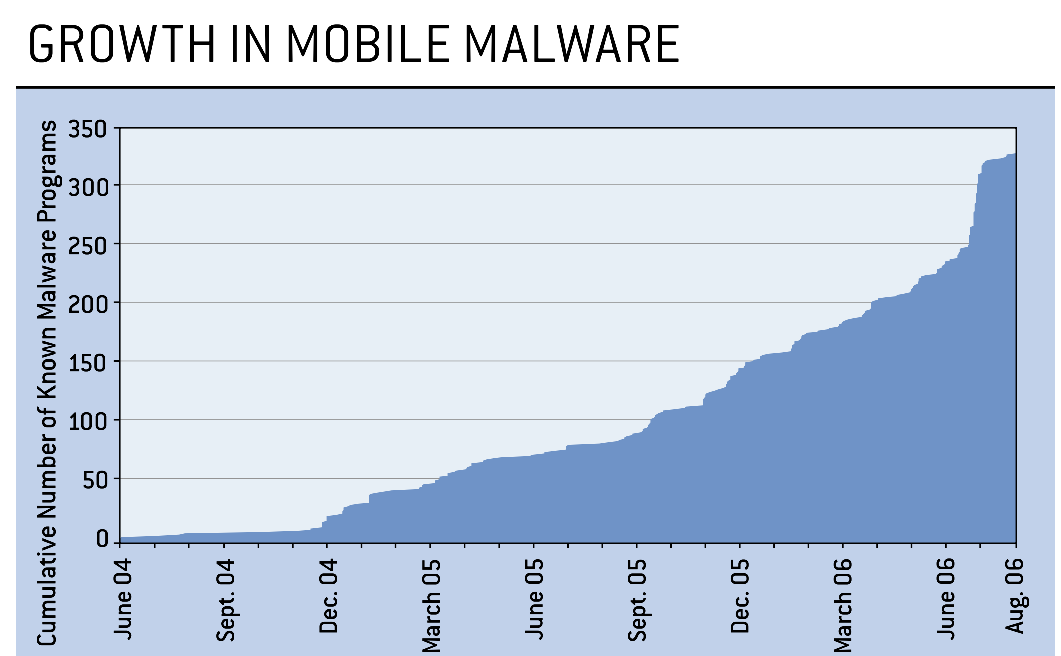
Malware spreading



Botnet infections (2010)

Mobile malware (2011)



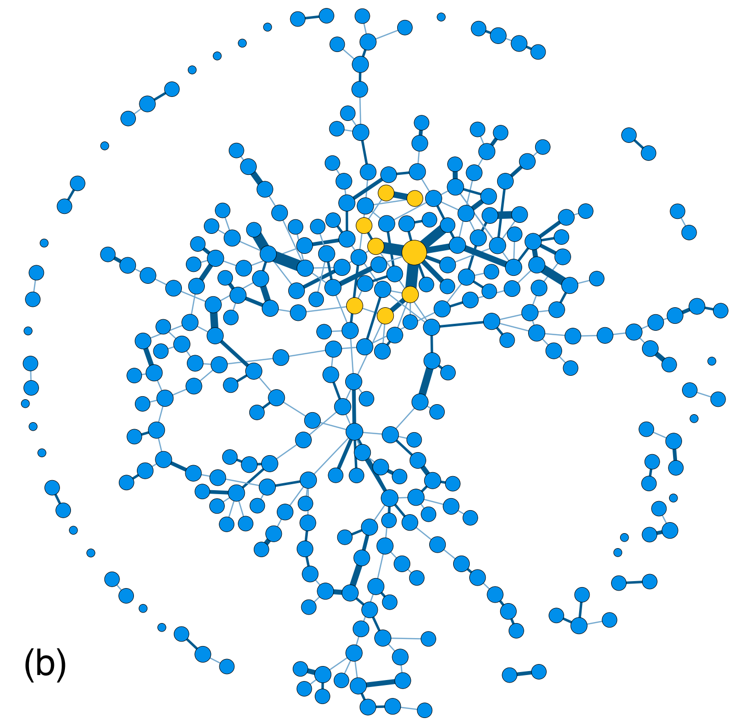


Hypponen M. *Scientific American* Nov. 70-77 (2006).

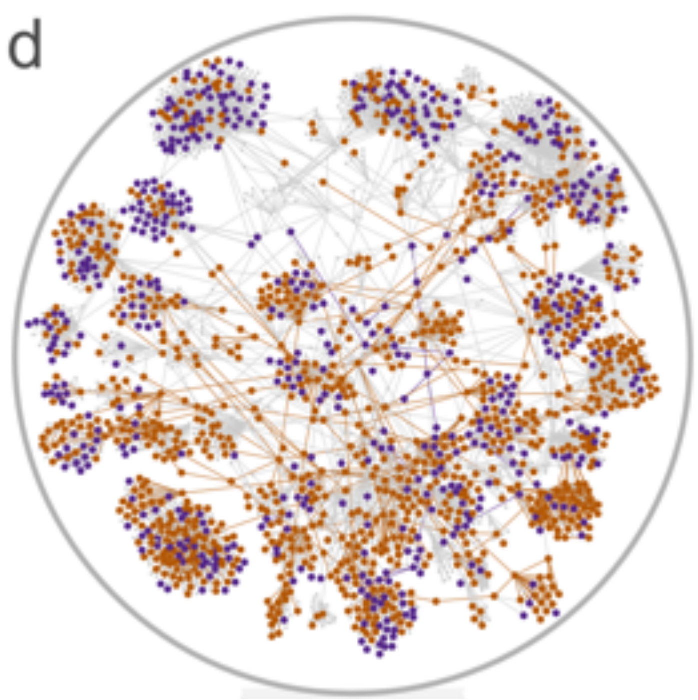
Spreading processes

Social contagion

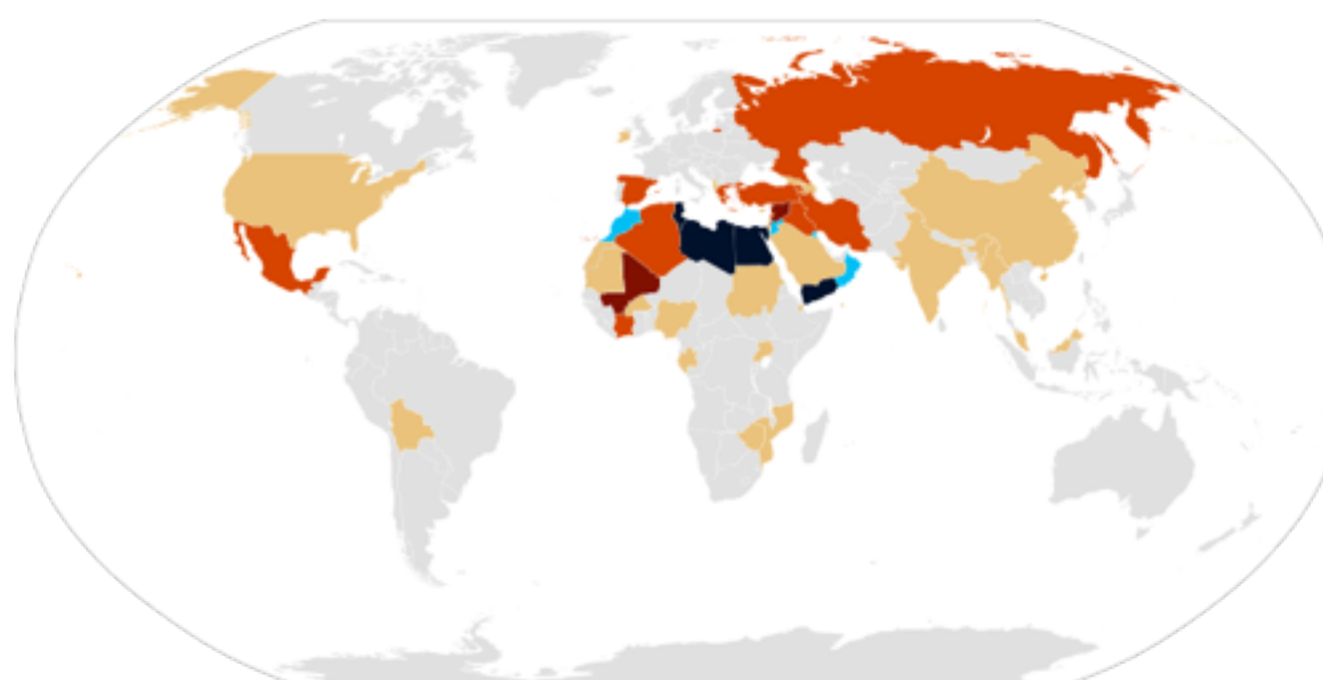
Information spreading



Rumour spreading
Karsai et.al. (2014)



Adoption spreading (Skype)
Karsai et.al. (2014)



Wikipedia

Protest diffusion (Arabian spring)

Spreading processes

Why?

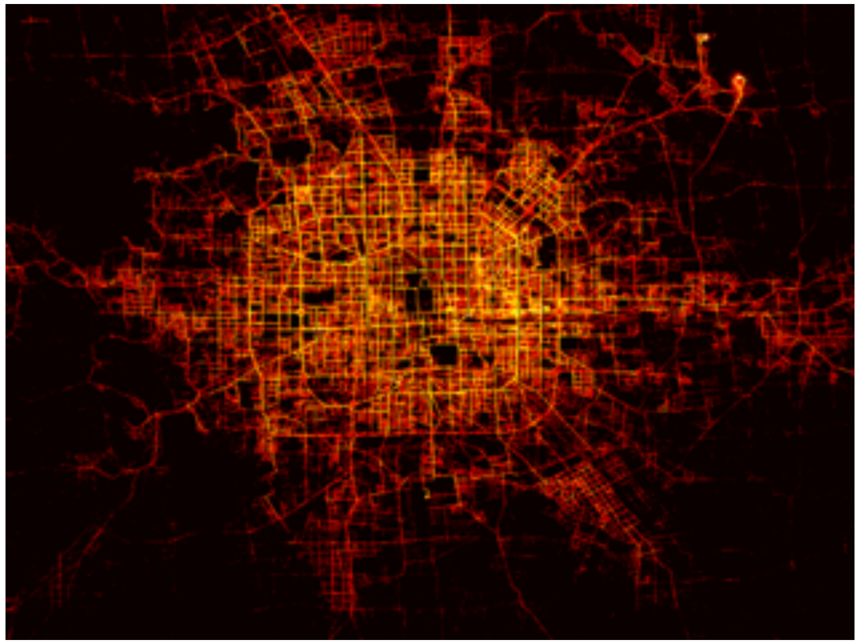
- High population density
- Interconnected and mixing population
- Dynamical mobility patterns

Why on networks?

- Spreading can happen only through interactions between agents
    - Geographic vicinity
    - Physical connection
    - Social interaction
    - etc.
- Network structure critically influence the dynamics of spreading processes

PPD blog, Jooseery (2011)

Freese (2009)



Unknown

Models of Spreading processes

Spreading processes

Model assumptions of spreading processes

- Constant set of interacting agents
- Nodes are partitioned into distinct compartments based on their actual states
- States are defined by the distinct stages of the epidemics:

S+I+R=N

Susceptible (S)
(Healthy)

Infected (I)
(Sick)

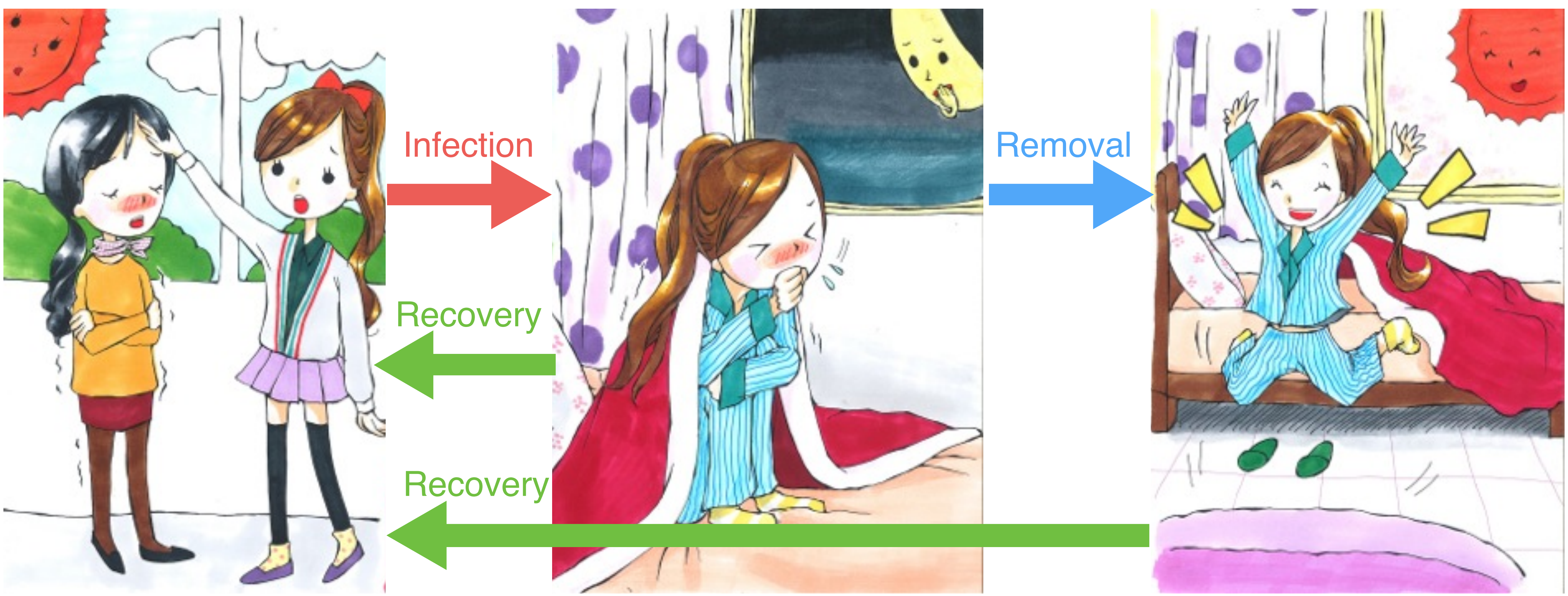
Recovered (R)
(Removed, dead)

Mi jin Lee's cartoons, (after P. Holme)

Spreading processes

Simple spreading phenomena

- Lack of decision to become infected
- State change depends on the absolute number of stimuli coming from neighbours
- Examples: epidemic spreading, biological contagion, information spreading, etc.



Mi jin Lee's cartoons, (after P. Holme)

Susceptible (S)
(Healthy)

Infected (I)
(Sick)

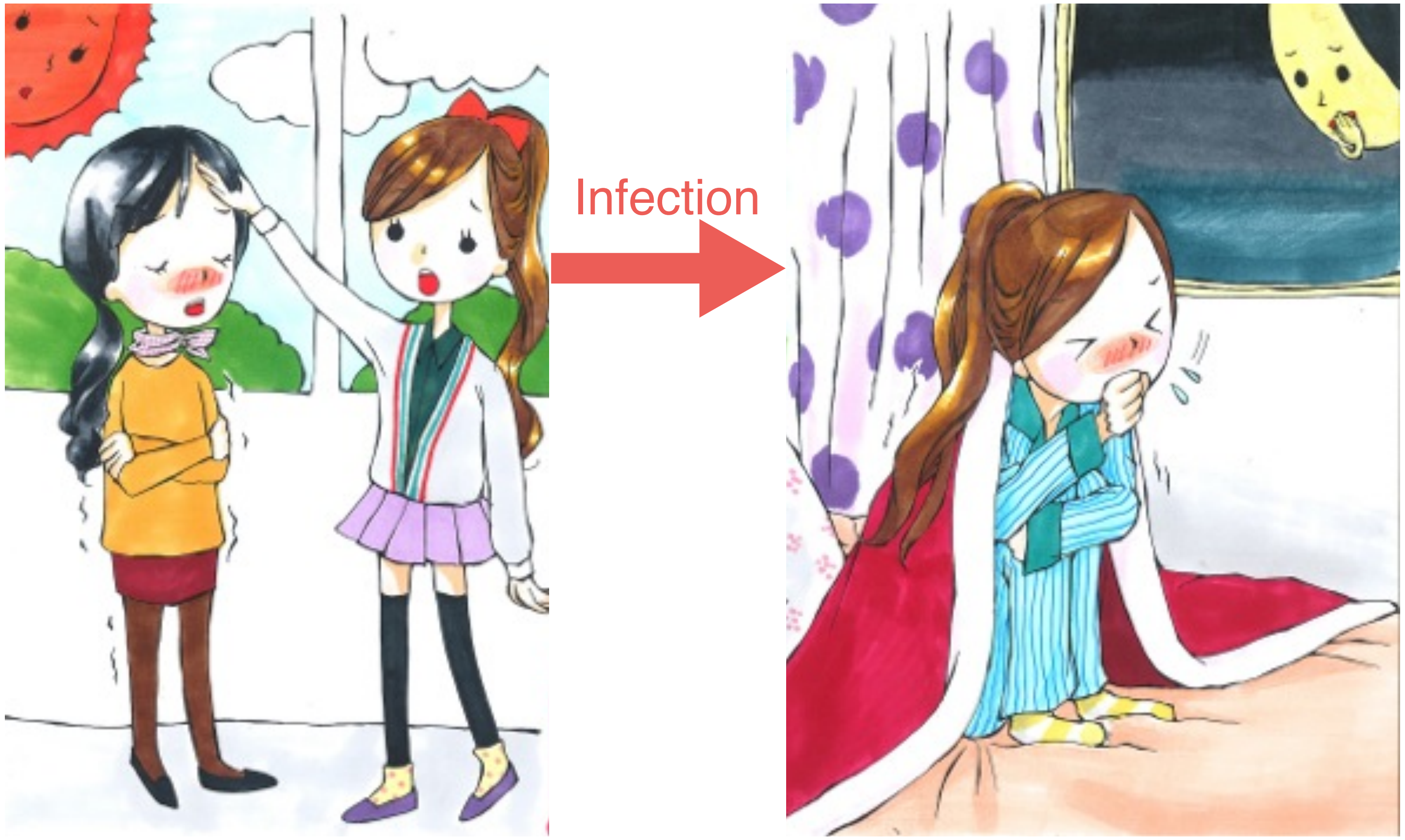
Recovered (R)
(Removed, dead)

The SI model

Susceptible-Infected model

- Take a population of N nodes
- Assign by I the number of infected and by S the number of susceptible nodes
- $I+S=N$ any time
- We infect a single seed node thus at $t=0$ $I=1$

Mi jin Lee's cartoons, (after P. Holme)



Susceptible (S)
(Healthy)

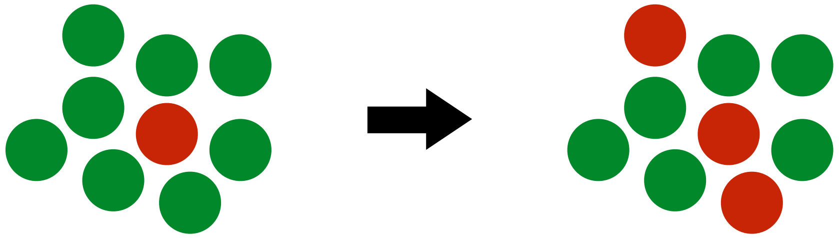
Infected (I)
(Sick)

Homogeneous mixing (no network)

- Each node has β contacts with randomly selected other nodes per unit time
- An infected node on average contacts a susceptible with probability $\beta S/N$
- Average number of infection per unit time is $\beta IS/N$
- Consider the fractions instead of absolute numbers:

$$s=S/N \quad \text{and} \quad i=I/N$$

such as $s+i=1$



The SI model

- Time evolution of the infected and susceptible fractions:

$$\frac{di(t)}{dt} = \beta si \qquad \frac{ds(t)}{dt} = -\beta si$$

- Since $s(t)+i(t)=1$ the fraction of susceptible nodes can be written as $s(t)=1-i(t)$

$$\frac{di(t)}{dt} = \beta(1-i)i \quad \Rightarrow$$

$$\Rightarrow \quad \frac{di}{i} + \frac{di}{(1-i)} = \beta dt \quad \Rightarrow \quad ln(i) - ln(1-i) + c = \beta t$$

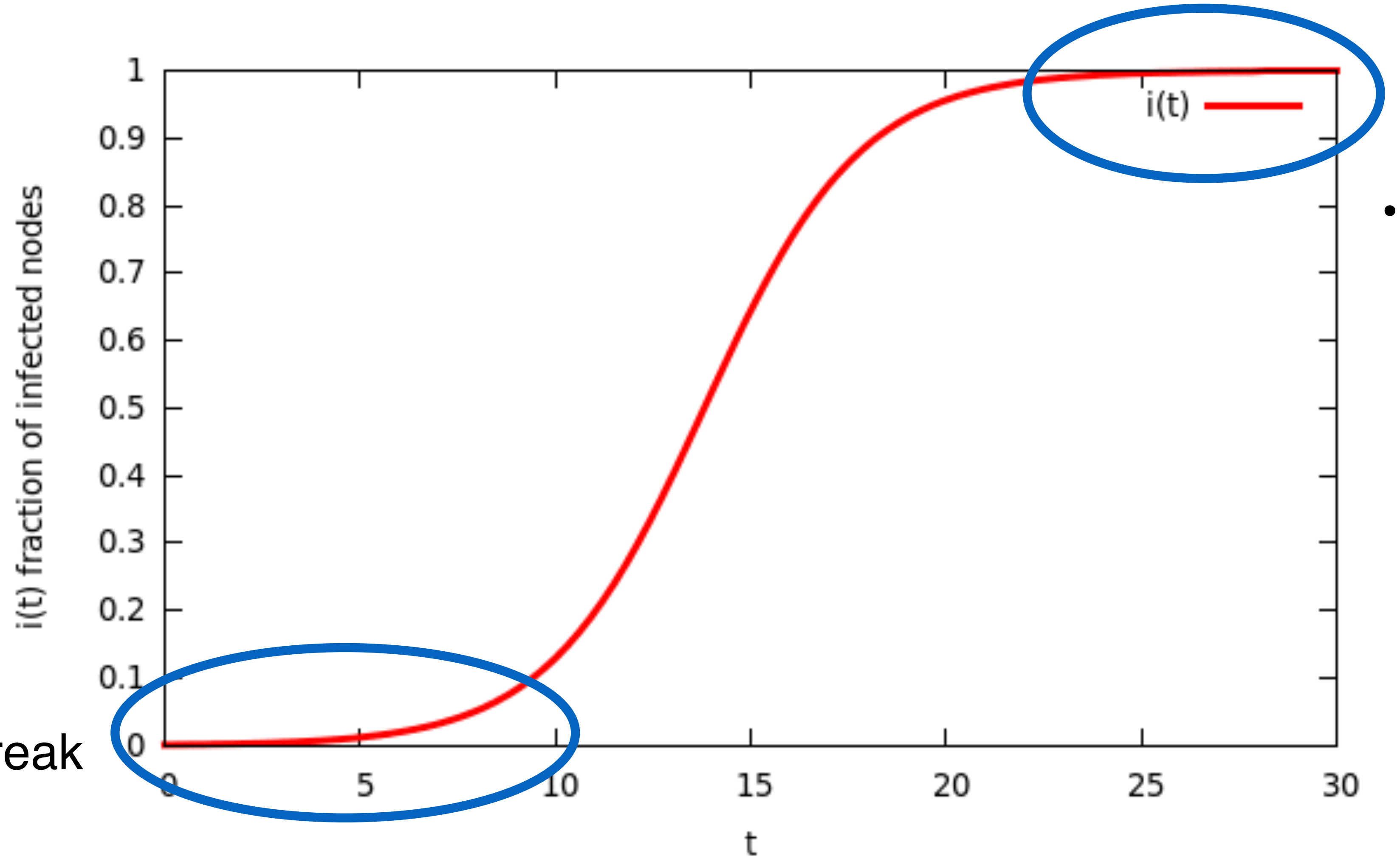
$$\Rightarrow \quad ln\frac{i}{1-i} + c = \beta t \quad \Rightarrow \quad \frac{i}{1-i} = Ce^{\beta t}$$

- If we take t=0 then $C = \frac{i_0}{1-i_0}$ and $i(t) = \frac{i_0 e^{\beta t}}{1 - i_0 + i_0 e^{\beta t}}$

Logistic equation: a basic model of population growth

The SI model

$$\frac{di(t)}{dt} = \beta(1-i)i \qquad i(t) = \frac{i_0 e^{\beta t}}{1 - i_0 + i_0 e^{\beta t}}$$



- For early times
  (i(t) is small)

$$\frac{di}{dt} \approx \beta i$$

exponential outbreak

$$i \approx i_0 e^{\beta t}$$

- For large times

$i(t) \to 1$

$$\frac{di(t)}{dt} = 0$$

- Saturation

SI model: in the end of the process always everyone get infected if $\beta > 0$

The SIS model

Susceptible-Infected-Susceptible model

- SI process + infected nodes recover with rate μ per unit time

$$\frac{di}{dt} = \beta i(1-i) - \mu i = i(\beta - \mu - \beta i)$$

S→I I→S

$$\frac{di}{i} + \frac{di}{1-\mu/\beta - i} = (\beta - \mu)dt$$

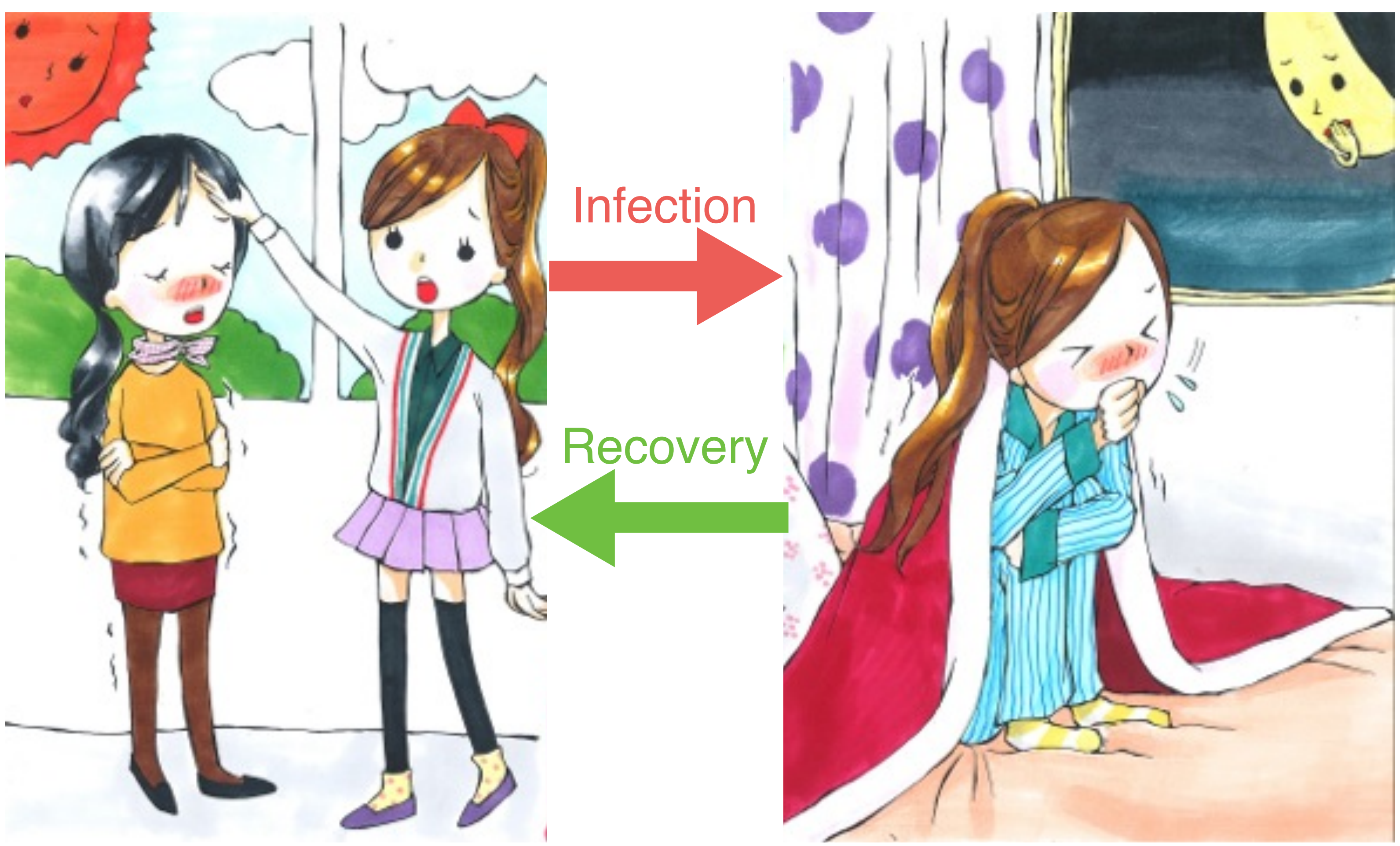
$$\ln(i) - \ln(1-\mu/\beta - i) = (\beta - \mu)t + c$$

$$\frac{i}{1-\mu/\beta - i} = Ce^{(\beta-\mu)t}$$

$$i(t) = \left(1 - \frac{\mu}{\beta}\right)\frac{Ce^{(\beta-\mu)t}}{1+Ce^{(\beta-\mu)t}}$$

$$C = \frac{i_0}{1-\mu/\beta - i_0}$$

Mi jin Lee's cartoons, (after P. Holme)

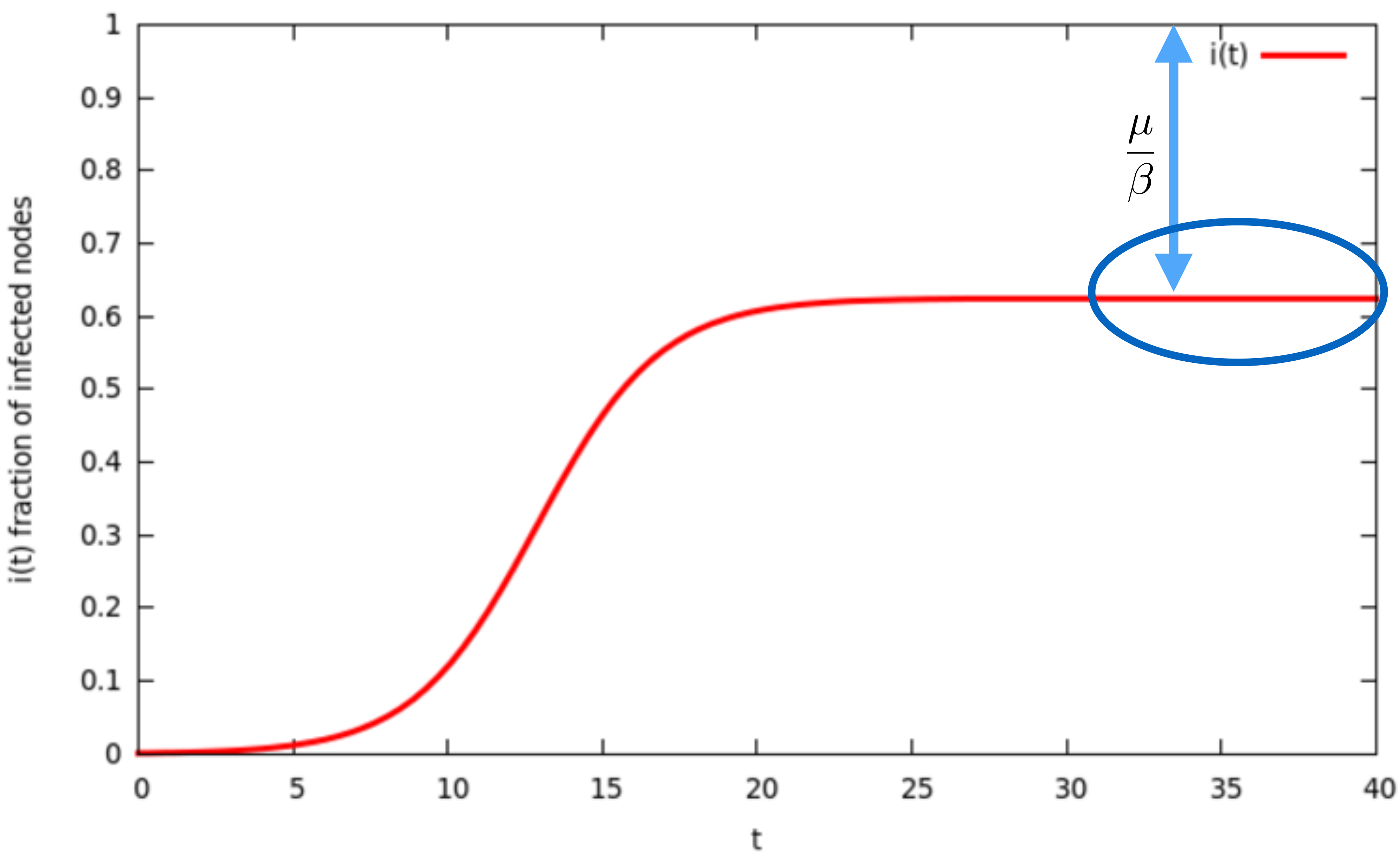


Susceptible (S)
(Healthy)

Infected (I)
(Sick)

The SIS model

$$i(t) = \left(1 - \frac{\mu}{\beta}\right)\frac{Ce^{(\beta-\mu)t}}{1+Ce^{(\beta-\mu)t}}$$



- For large times

$$i(t) \rightarrow 1 - \frac{\mu}{\beta}$$

$$\frac{di}{dt} = \beta i(1-i) - \mu i = 0$$

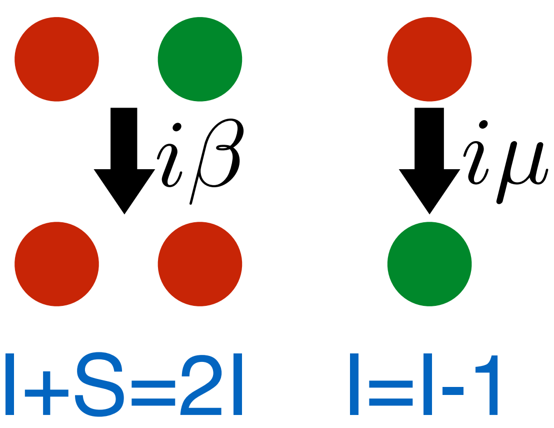
- For SIS model the saturation of infected nodes is below 1
- If it saturates to 1 then the model is equivalent with SI (μ=0)

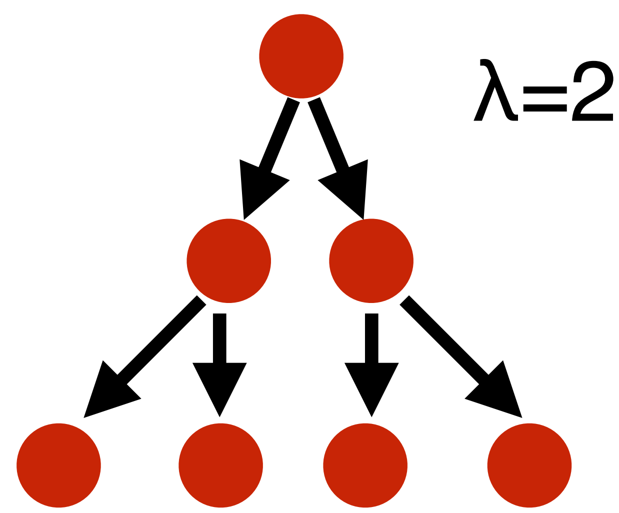
The SIS model

$$i(t) = \left(1 - \frac{\mu}{\beta}\right) \frac{Ce^{(\beta-\mu)t}}{1 + Ce^{(\beta-\mu)t}}$$

Basic reproduction number $\lambda = \beta/\mu$

- On average how many nodes will be infected by a single infected node in a fully susceptible population





if $\lambda > 1$ - outbreak

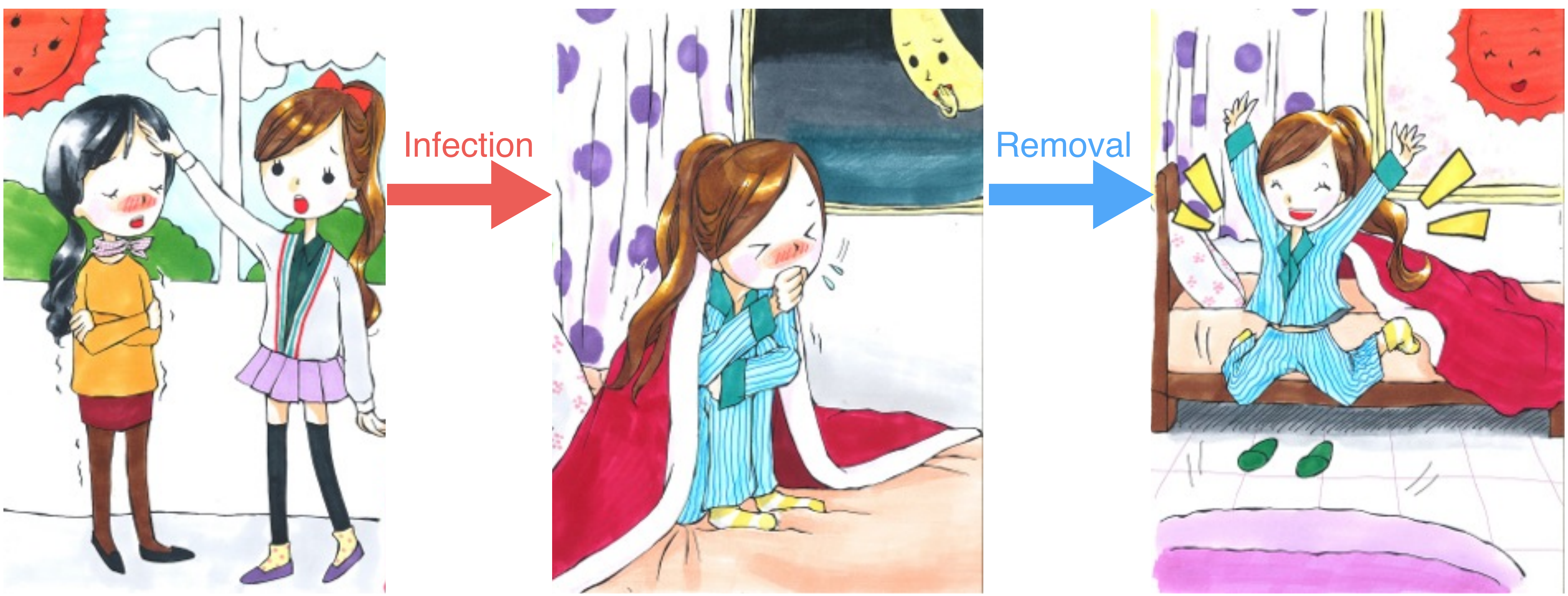
if $\lambda_c = 1$ - epidemic threshold

if $\lambda < 1$ - vanish

Spreading processes

Susceptible-Infected-Removed model

- SI process + infected nodes removed with rate μ per unit time



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(Removed, dead)

Mi jin Lee's cartoons, (after P. Holme)

Spreading processes

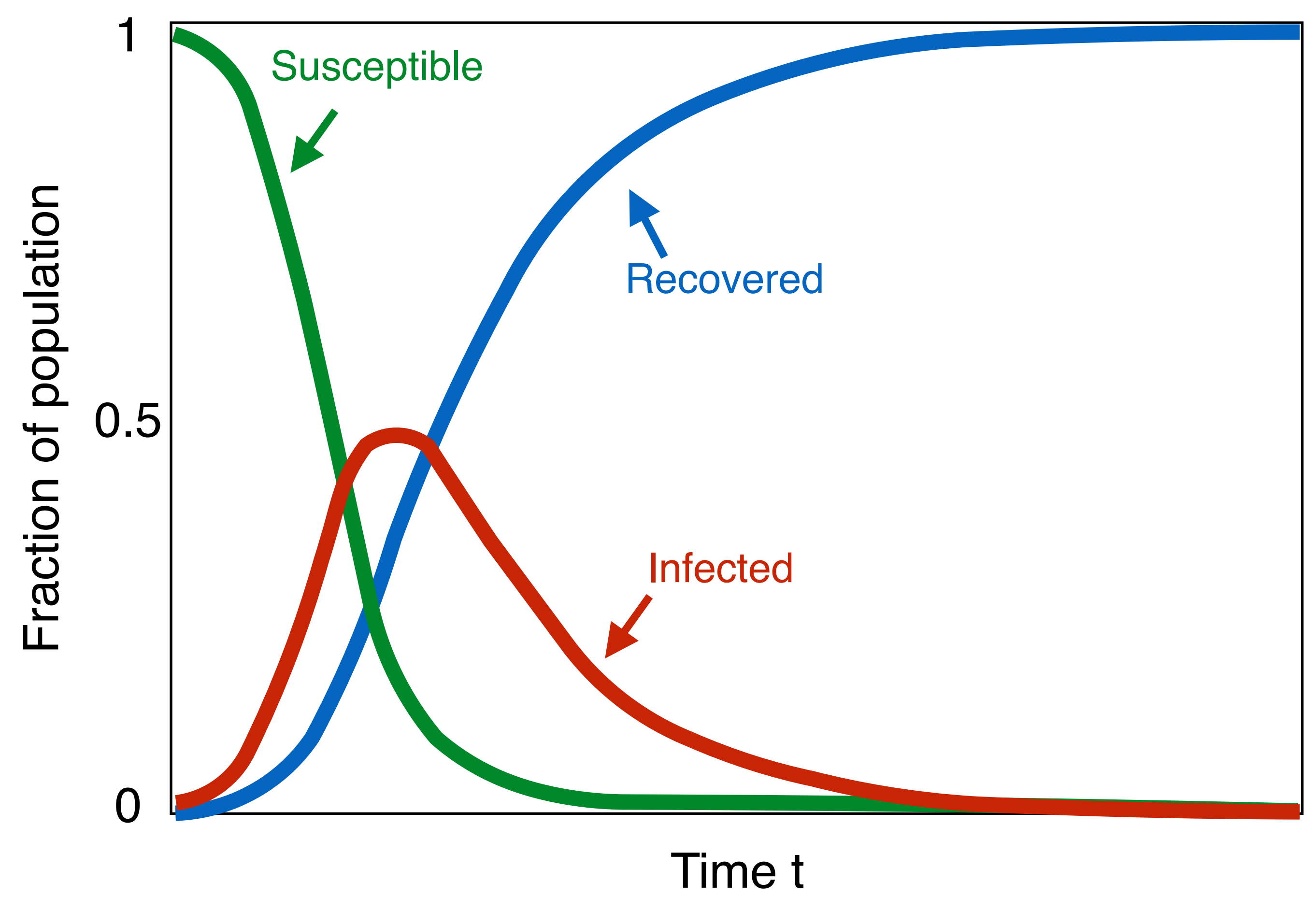
Susceptible-Infected-Removed model

- SI process + infected nodes removed with rate μ per unit time

$$\frac{s(t)}{dt} = -\beta i(t)(1 - r(t) - i(t))$$

$$\frac{i(t)}{dt} = -\mu i(t) + \beta i(t)(1 - r(t) - i(t))$$

$$\frac{r(t)}{dt} = \mu i(t)$$



- Fraction of infected nodes peaks
- Fraction of removed nodes saturates
- Fraction of susceptible nodes not necessarily saturates to 0

Epidemic spreading models

Basic properties

	SI	SIS	SIR
Early behaviour Exponential growth of i(t)	$i(t) = \frac{i_0 e^{\beta t}}{1 - i_0 + i_0 e^{\beta t}}$	$i(t) = \left(1 - \frac{\mu}{\beta}\right)\frac{Ce^{(\beta-\mu)t}}{1+Ce^{(\beta-\mu)t}}$	$i(t) = \left(1 - \frac{\mu}{\beta}\right)\frac{Ce^{(\beta-\mu)t}}{1+Ce^{(\beta-\mu)t}}$
Late behaviour	$i(t) \to 1$	$i(t) \to 1 - \frac{\mu}{\beta}$	$i(t) \to 0$
Epidemic threshold $\lambda = \beta/\mu$	$\beta = 0$	$\lambda_c = 1$	$\lambda_c = 1$

Spreading processes on networks

Epidemic spreading on networks

Homogeneous mixing (on networks)

- Each node has k number of neighbours
- Each infected node infects its susceptible neighbours with probability βdt in a unite time
- A susceptible node with degree k will be infected with probability $\beta k i(t) dt$ in a unite time

SI model

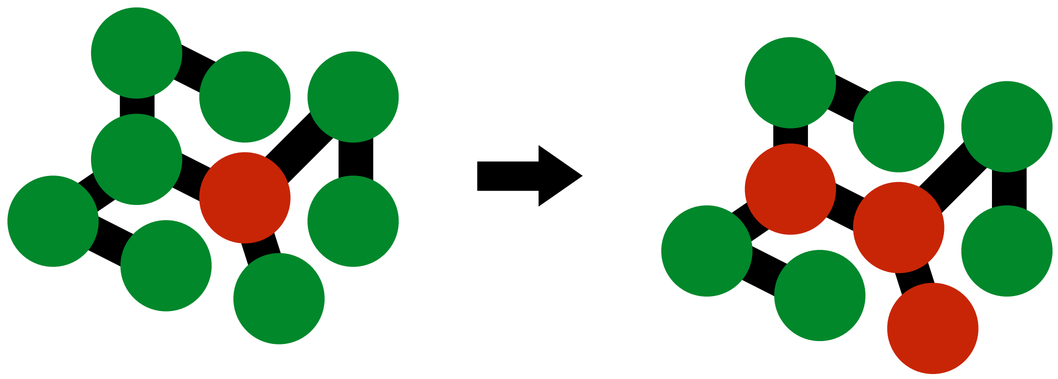
$$\frac{di(t)}{dt} = \beta \langle k \rangle i(t) [1 - i(t)]$$

SIS model

$$\frac{di(t)}{dt} = -\mu i(t) + \beta \langle k \rangle i(t) [1 - i(t)]$$

First approximation (on homogeneous networks)

- $k \simeq \langle k \rangle$: The network has homogeneous degree distribution
- E.g. random networks



SIR model

$$\frac{ds(t)}{dt} = -\beta \langle k \rangle i(t) [1 - r(t) - i(t)]$$

$$\frac{di(t)}{dt} = -\mu i(t) + \beta \langle k \rangle i(t) [1 - r(t) - i(t)]$$

$$\frac{dr(t)}{dt} = \mu i(t).$$

Epidemic spreading on heterogeneous networks

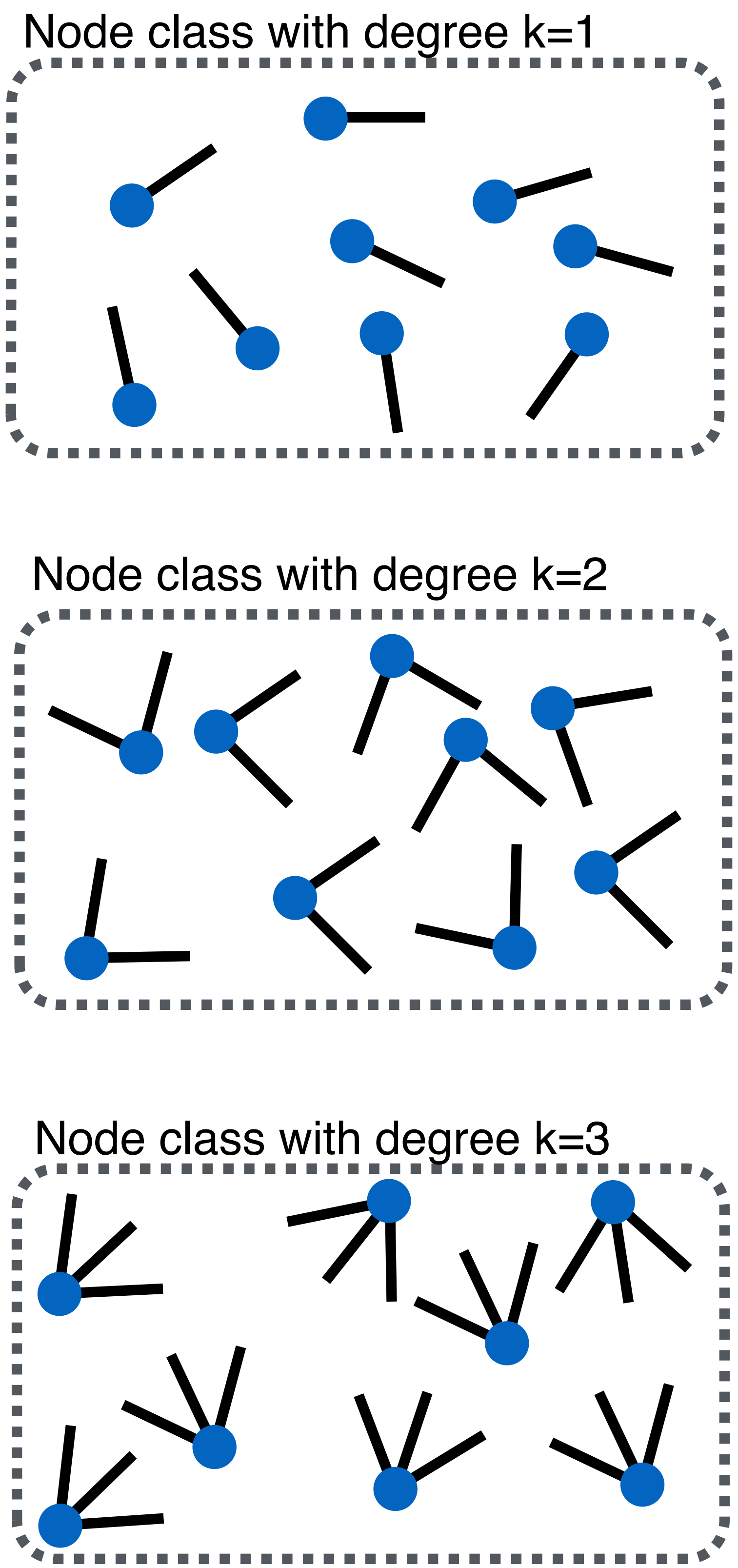
- In degree heterogeneous networks the $k \simeq \langle k \rangle$ approximation does not hold

- Solution: Degree Block Approximation
  - Assumption: all nodes with the same degree are statistically equivalent
  - Look for infection/susceptible node densities in the degree groups

$$i_k = \frac{I_k}{N_k} \qquad s_k = \frac{S_k}{N_k}$$

  - Calculate the global average by a sum considering the degree distribution

$$i = \sum_k P(k) i_k \qquad s = \sum_k P(k) s_k$$



SI process on heterogeneous networks

- SI process: all nodes are infected in the end - no epidemic threshold
- Question: how degree heterogeneities influence the speed of spreading

$$\frac{di_k(t)}{dt} = \beta\,[1 - i_k(t)]\,k\,\Theta_k(t)$$

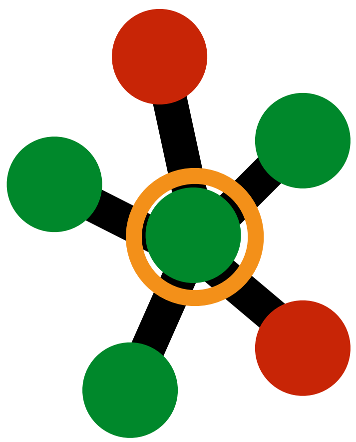
Spreading rate

Probability that a node with degree k is not infected

Degree

Density of infected neighbours of a node with degree k

- $\Theta_k(t)$: Probability that any neighbour of a node with degree k is infected



- I am a node with degree k=5 and I am susceptible
- $\Theta_k(t)$=2/5 fraction of my neighbours are infected actually
- In case of homogeneous networks: $\Theta_k(t)=i(t)$

Barrat, Barthelemy, Vespignani book

SI process on heterogeneous networks

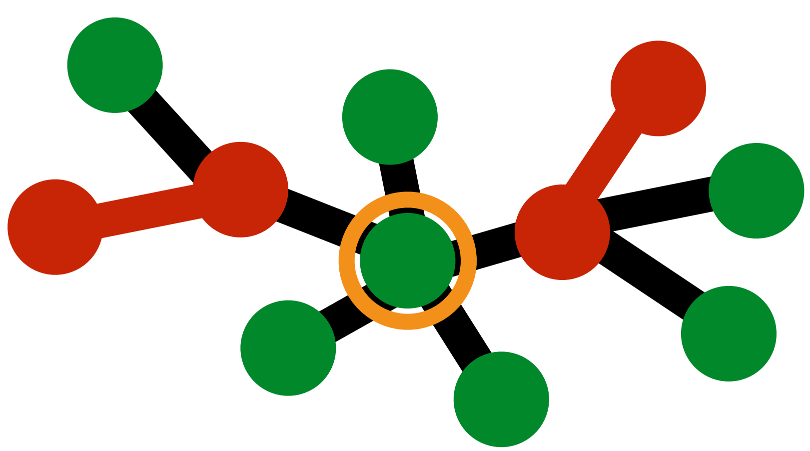
- Assume: no degree-degree correlations in the network
  - Probability that a node with degree k connects to a node with k'

a node can connect in k' ways to a node with degree k'

$$P(k'|k) = \frac{k'P(k')}{\sum_{k'} k'P(k')} = \frac{k'P(k')}{\langle k \rangle}$$

- Using this $\Theta_k(t)$ can be written as

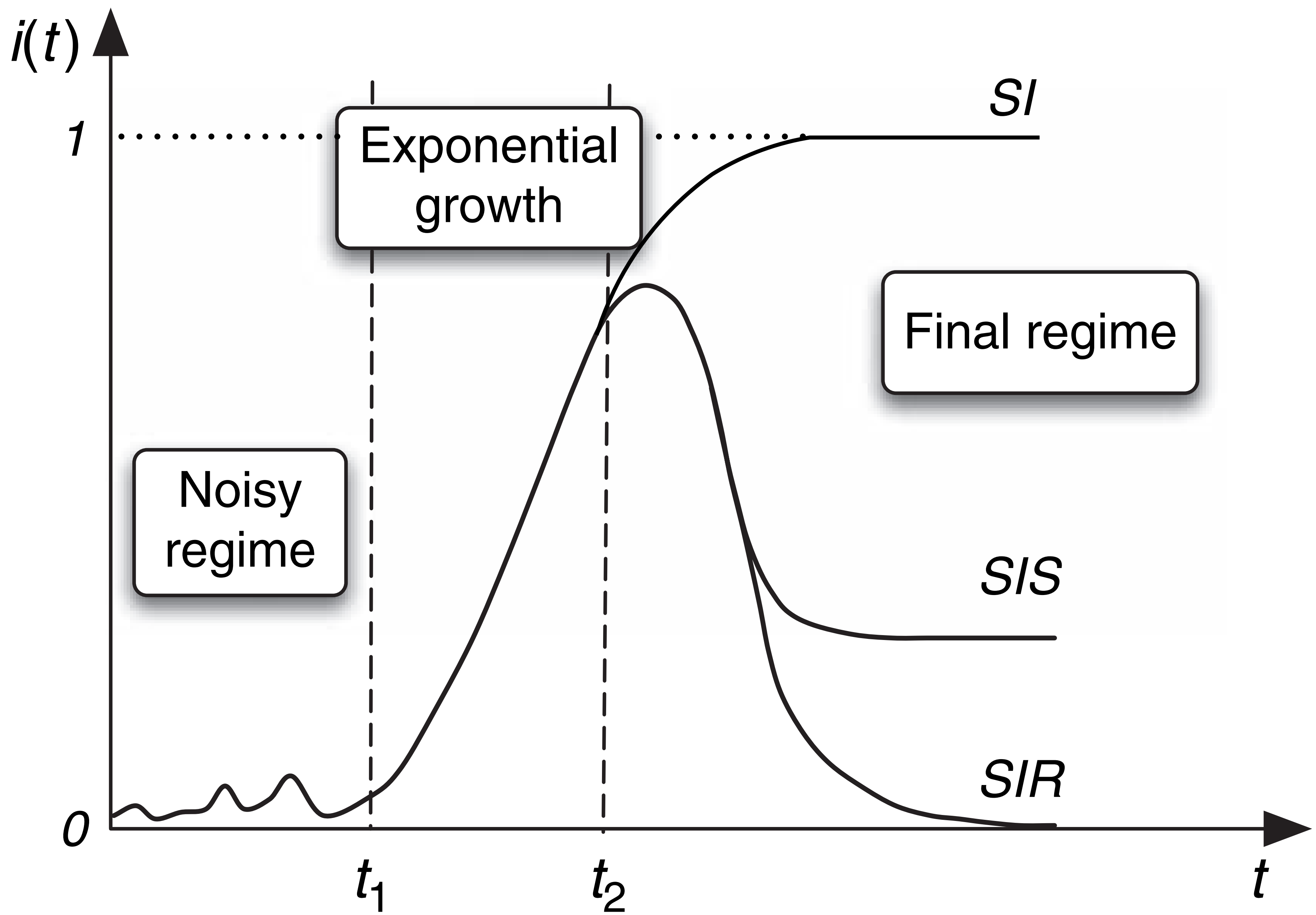
$$\Theta_k(t) = \Theta(t) = \frac{\sum_{k'} (k'-1)P(k')i_{k'}(t)}{\langle k \rangle}$$



k'-1 because the linked infected node surely has an infected neighbour, thus the central node can connect to it only in k'-1 ways

Barrat, Barthelemy, Vespignani book

The spreading curve regimes



Barrat, Barthelemy, Vespignani book

Early time behaviour of epidemic spreading

- Approximation: assume that $i(t) << 1$ (early time behaviour)

Why is it important?

- Vaccination, cures, and medical interventions take years to develop
- Their application is the most effective during the outbreak of a disease
- The best way to stop epidemics
  - early quarantine
  - early vaccination
- Epidemics spreading shows exponential grows in the beginning of the process
- Vanishing epidemic thresholds

Early time spreading behaviour

SI process on heterogeneous networks

- Approximation: assume that *i(t)<<1* (early time behaviour)

$$\frac{\mathrm{d}i_k(t)}{\mathrm{d}t} = \beta\,[1 - i_k(t)]\,k\Theta_k(t) \approx \beta k \Theta_k(t)$$

- Differentiation of $\Theta_k(t)$ by t gives

$$\frac{d\Theta(t)}{dt} = \frac{\sum_{k'}(k'-1)P(k')}{\langle k\rangle}\frac{di_{k'}(t)}{dt} = \frac{\sum_{k'}(k'-1)P(k')}{\langle k\rangle}\beta k'\Theta_{k'}(t) = \beta\frac{\sum_{k'}(k'^2-k')P(k')}{\langle k\rangle}\Theta(t)$$

$$\frac{\mathrm{d}\Theta(t)}{\mathrm{d}t} = \beta\left(\frac{\langle k^2\rangle}{\langle k\rangle} - 1\right)\Theta(t)$$

Let's denote $\tau = \frac{\langle k\rangle}{\beta\left(\langle k^2\rangle - \langle k\rangle\right)}$ time-scale parameter

$$\frac{d\Theta(t)}{\Theta(t)} = \frac{dt}{\tau} \Rightarrow \Theta(t) = Ce^{t/\tau} \Rightarrow \Theta(t=0) = C = i_0\frac{\langle k\rangle - 1}{\langle k\rangle}$$

$$\Rightarrow \Theta(t) = i_0\frac{\langle k\rangle - 1}{\langle k\rangle}e^{t/\tau}$$

Barrat, Barthelemy, Vespignani book

SI process on heterogeneous networks

$$\frac{di_k(t)}{dt} \approx \beta k \Theta_k(t) \qquad \Theta(t) = i_0 \frac{\langle k \rangle - 1}{\langle k \rangle} e^{t/\tau} \qquad \tau = \frac{\langle k \rangle}{\beta\left(\langle k^2 \rangle - \langle k \rangle\right)}$$

$$\frac{di_k(t)}{dt} \approx \beta k i_0 \frac{\langle k \rangle - 1}{\langle k \rangle} e^{t/\tau}$$

valid if *i(t)<<1* (early time behaviour)

$$i_k(t) = \int_0^t \frac{di_k(t)}{dt} dt \approx \beta k i_0 \frac{\langle k \rangle - 1}{\langle k \rangle} \int_0^t e^{t/\tau} dt = \beta k i_0 \frac{\langle k \rangle - 1}{\langle k \rangle} \tau e^{t/\tau}$$

$$i_k(t) - i_0 \approx \beta k i_0 \frac{\langle k \rangle - 1}{\langle k \rangle} \tau (e^{t/\tau} - 1) = \beta k i_0 \frac{\langle k \rangle - 1}{\langle k \rangle} \frac{\langle k \rangle}{\beta(\langle k^2 \rangle - \langle k \rangle)} (e^{t/\tau} - 1) =$$

$$= k i_0 \frac{\langle k \rangle - 1}{\langle k^2 \rangle - \langle k \rangle} (e^{t/\tau} - 1)$$

$$i_k(t) = k i_0 \frac{\langle k \rangle - 1}{\langle k^2 \rangle - \langle k \rangle} (e^{t/\tau} - 1) + i_0 = i_0 \left(\frac{k(\langle k \rangle - 1)}{\langle k^2 \rangle - \langle k \rangle} (e^{t/\tau} - 1) + 1 \right)$$

Barrat, Barthelemy, Vespignani book

SI process on heterogeneous networks

$$i = \sum_k P(k) i_k \qquad i_k(t) = i_0 \left(\frac{k(\langle k \rangle - 1)}{\langle k^2 \rangle - \langle k \rangle} (e^{t/\tau} - 1) + 1 \right)$$

$$i(t) = \int i_k(t) P(k) dk = i_0 \left(1 + \frac{\langle k \rangle^2 - \langle k \rangle}{\langle k^2 \rangle - \langle k \rangle} (e^{t/\tau} - 1) \right)$$

M. Barthélemy et al., *PRL* **92**, 178701 (2004)

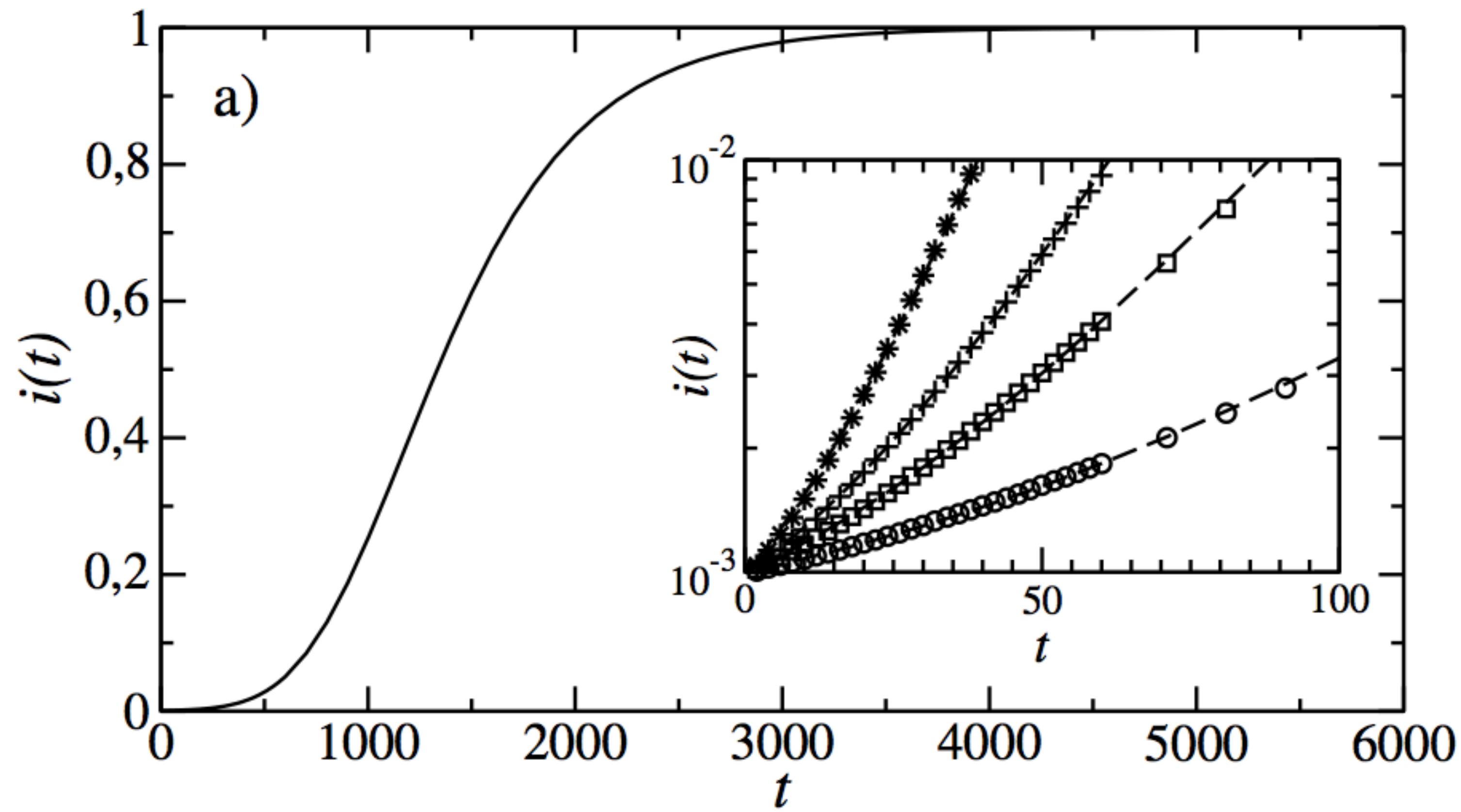
- In degree heterogeneous networks, where

  $$\langle k^2 \rangle \to \infty$$

  the SI spreading is critically faster than in degree homogeneous networks where

  $$\langle k^2 \rangle$$

  is finite



Inset: BA networks with m=4,8,12,20 (from bottom to top)

Barrat, Barthelemy, Vespignani book

SIR process on heterogeneous networks

Epidemic threshold

- To obtain an epidemic threshold we need to have $\tau>0$ (otherwise the epidemic spreads instantaneously)

SIR on ER network

$$\tau_{ER} = \frac{1}{\beta\langle k\rangle - \mu} > 0 \rightarrow \lambda \equiv \frac{\beta}{\mu} > \frac{1}{\langle k\rangle}$$

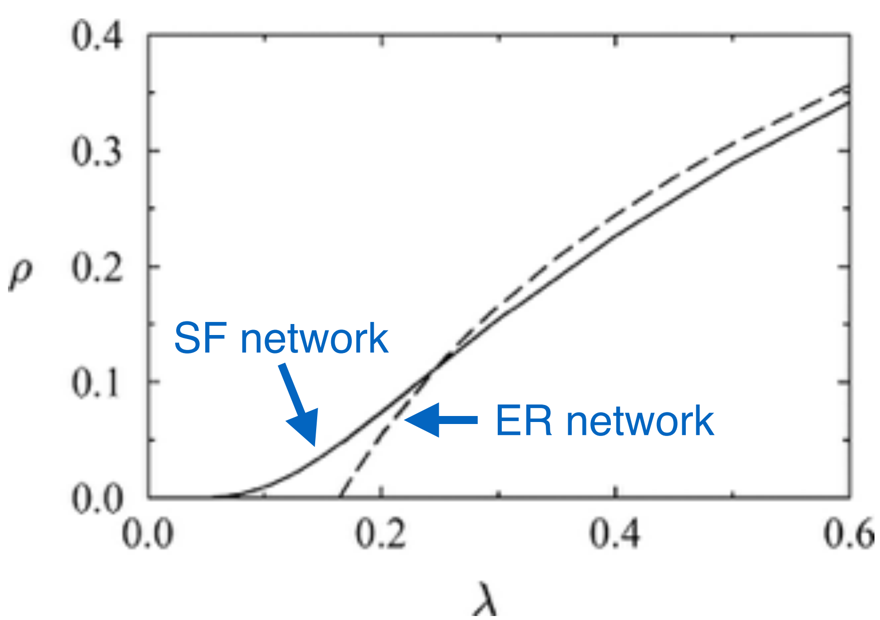
Threshold: $$\lambda_c = \frac{1}{\langle k\rangle}$$

SIR on SF network

$$\tau_{SF} = \frac{\langle k\rangle}{\beta\langle k^2\rangle - (\mu+\beta)\langle k\rangle} > 0 \rightarrow \lambda \equiv \frac{\beta}{\mu} > \lambda_c$$

Threshold: $$\lambda_c = \frac{1}{\frac{\langle k^2\rangle}{\langle k\rangle} - 1}$$

Barrat, Barthelemy, Vespignani book



- Threshold is vanishing for heterogeneous networks
- Epidemics spread in any case

Pastor-Satorras &Vespignani, PRL 86, 3200(2001)

SI and SIR processes on heterogeneous networks

SI: $$\tau = \frac{\langle k \rangle}{\beta(\langle k^2 \rangle - \langle k \rangle)}$$

- Time scale assigns the speed of infection spreading
- The smaller τ the faster the process evolving

SIR: $$\tau = \frac{\langle k \rangle}{\beta \langle k^2 \rangle - (\mu + \beta)\langle k \rangle}$$

- ER network

$$\langle k^2 \rangle = \langle k \rangle (\langle k \rangle - 1)$$

SI: $$\tau_{ER} = \frac{1}{\beta \langle k \rangle}$$

SIR: $$\tau_{ER} = \frac{1}{\beta \langle k \rangle - \mu}$$

- The more connected the network is, the faster the epidemic evolves

- SF network
  - If $\gamma < 3$ and $N \to \infty$

    then $\langle k^2 \rangle \to \infty$ and $\tau \longrightarrow 0$

  - For heterogeneous networks the characteristic time vanishes
  - The epidemics becomes instantaneous
  - It is due to hubs who get infected first and disseminate the epidemics to many other nodes

Barrat, Barthelemy, Vespignani book

Late time spreading behaviour

SIS process on heterogeneous networks

- Approximation: assume that $t \to \infty$ (asymptotic large t behaviour)

SIS model

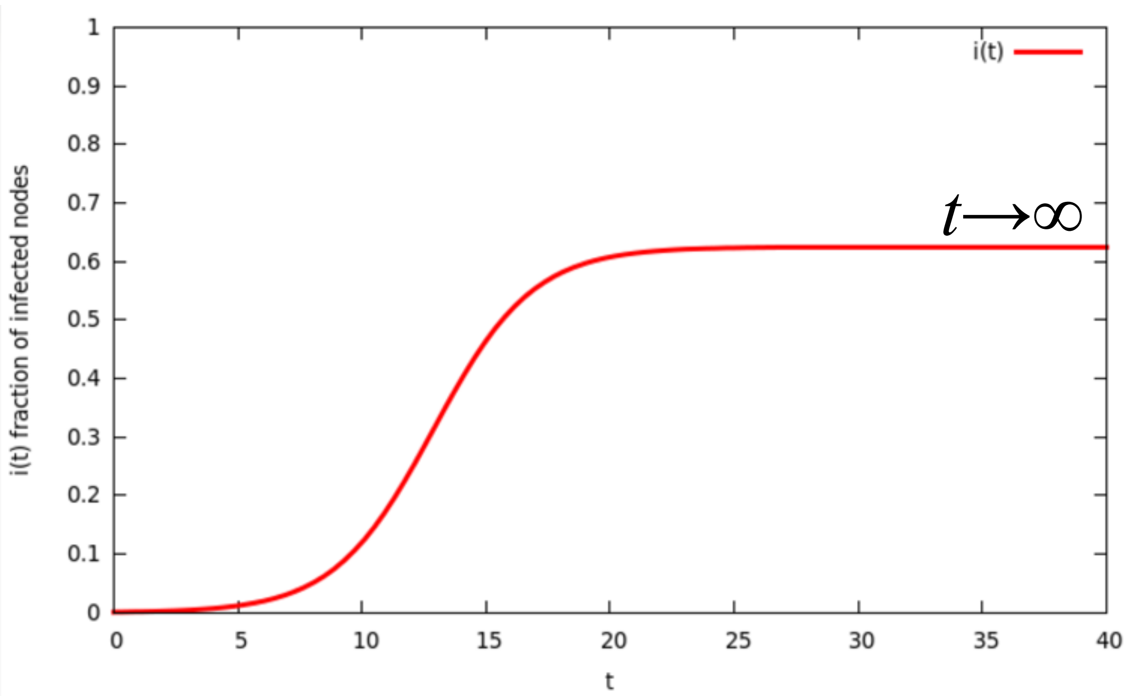
$$\frac{\mathrm{d}i_k(t)}{\mathrm{d}t} = -\mu i_k(t) + \beta k \left[1 - i_k(t)\right] \Theta_k(t)$$

Stationary state:

- $i_k(t)$ saturates

$$\frac{di_k(t)}{dt} = 0$$

$$0 = \beta(1 - i_k(t))k\Theta(t) - \mu i_k(t)$$



$$i_k = \frac{k\beta\Theta}{\mu + k\beta\Theta}$$

Stationary state: number of newly infected nodes is equal to the number of recovering nodes per unite time

Barrat, Barthelemy, Vespignani book

SIS process on heterogeneous networks

$$i_k = \frac{k\beta\Theta}{\mu + k\beta\Theta}$$

We assume no degree-degree correlations in the network

$$\Theta \approx \frac{\sum_{k'} k' P(k') i_{k'}}{\langle k \rangle}$$

$$\Theta = \frac{1}{\langle k \rangle} \sum_k k P(k) \frac{\beta k \Theta}{\mu + \beta k \Theta}$$

Self-consistent equation

Graphical solution

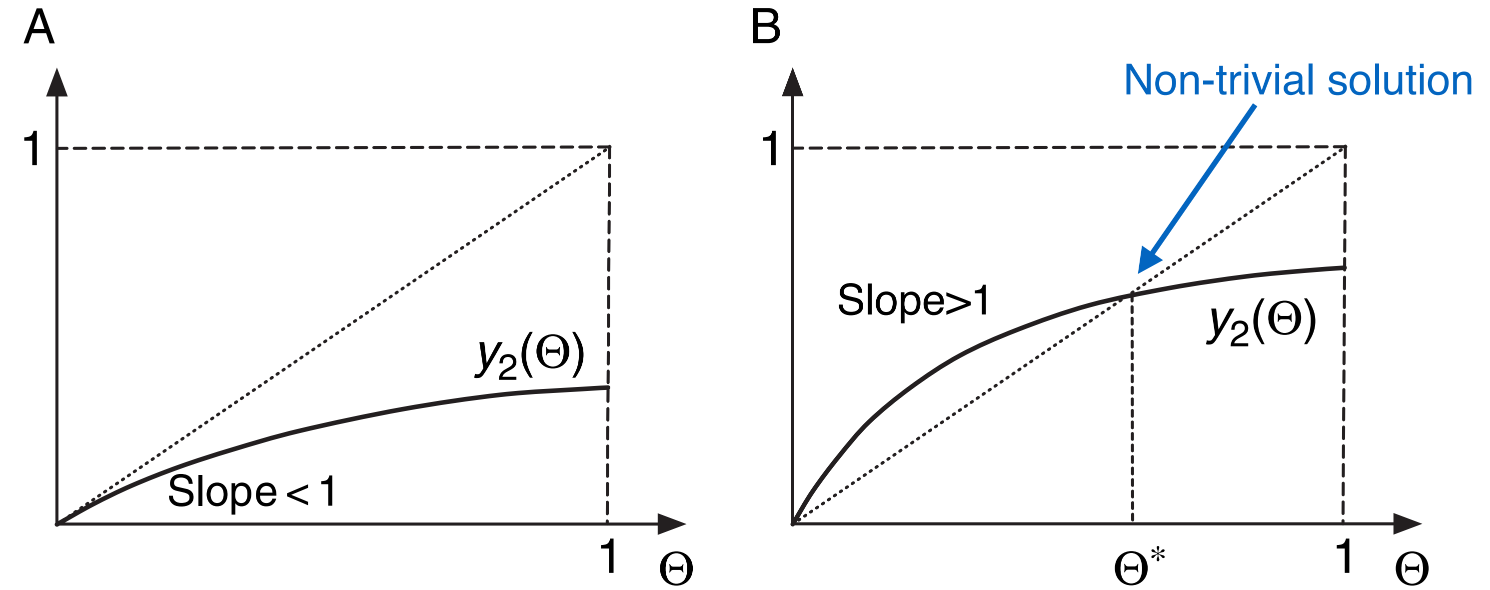
$$y_1(\Theta) = \Theta$$

- Linear function

$$y_2(\Theta) = \frac{1}{\langle k \rangle} \sum_k k P(k) \frac{\beta k \Theta}{\mu + \beta k \Theta}$$

- Monotonously increasing function between $y_2(0)=0$ and $y_2(1)<1$

- The slope of $y_2(\theta)$ at $\theta=0$ what matters
- We have to match the derivative of $y_1(\theta)$ and $y_2(\theta)$



Barrat, Barthelemy, Vespignani book

SIS process on heterogeneous networks

$$\frac{dy_1(\Theta)}{d\Theta} = 1$$

$$\frac{dy_2(\Theta)}{d\Theta} = \frac{d}{d\Theta}\left(\frac{1}{\langle k\rangle}\sum_k kP(k)\frac{\beta k\Theta}{\mu+\beta k\Theta}\right)_{\Theta=0} = \ldots =$$

$$= \frac{1}{\langle k\rangle}\sum_k kP(k)\beta k\left(\frac{\mu+\beta k\Theta-\beta k\Theta}{(\mu+\beta k\Theta)^2}\right)_{\Theta=0} = \frac{1}{\langle k\rangle}\sum_k kP(k)\beta k\frac{\mu}{\mu^2} =$$

$$= \frac{\beta}{\mu}\frac{1}{\langle k\rangle}\langle k^2\rangle = \lambda\frac{\langle k^2\rangle}{\langle k\rangle}$$

At the critical point the derivatives are equal: $\lambda\frac{\langle k^2\rangle}{\langle k\rangle} = 1$

$$\lambda_c = \frac{\langle k\rangle}{\langle k^2\rangle}$$

- Topological fluctuations lower the threshold
- If $\langle k^2\rangle \to \infty$ then $\lambda_c \to 0$

Pastor-Satorras &Vespignani, PRL 86, 3200(2001)

Barrat, Barthelemy, Vespignani book

SIS process - scale free exponent

$$P(k) = (\gamma - 1)K_{min}^{\gamma-1}k^{-\gamma}$$

Normalised degree distribution

$2 < \gamma < 3$

$\lambda_c = 0$

$i(\lambda) \approx \lambda^{1/(3-\lambda)}$

There is non-zero prevalence at the stationary state for any value of λ

$\gamma = 3$

$\lambda_c = 0$

$i(\lambda) \approx 2e^{-1/K_{min}\lambda}$

Prevalence approaches to 0 in a continuous way

$3 < \gamma < 4$

$\lambda_c > 0$

$$i(\lambda) \approx \left(\lambda - \frac{\gamma - 3}{K_{min}(\gamma - 2)}\right)^{1/(\gamma-3)}$$

$4 < \gamma$

$\lambda_c > 0$

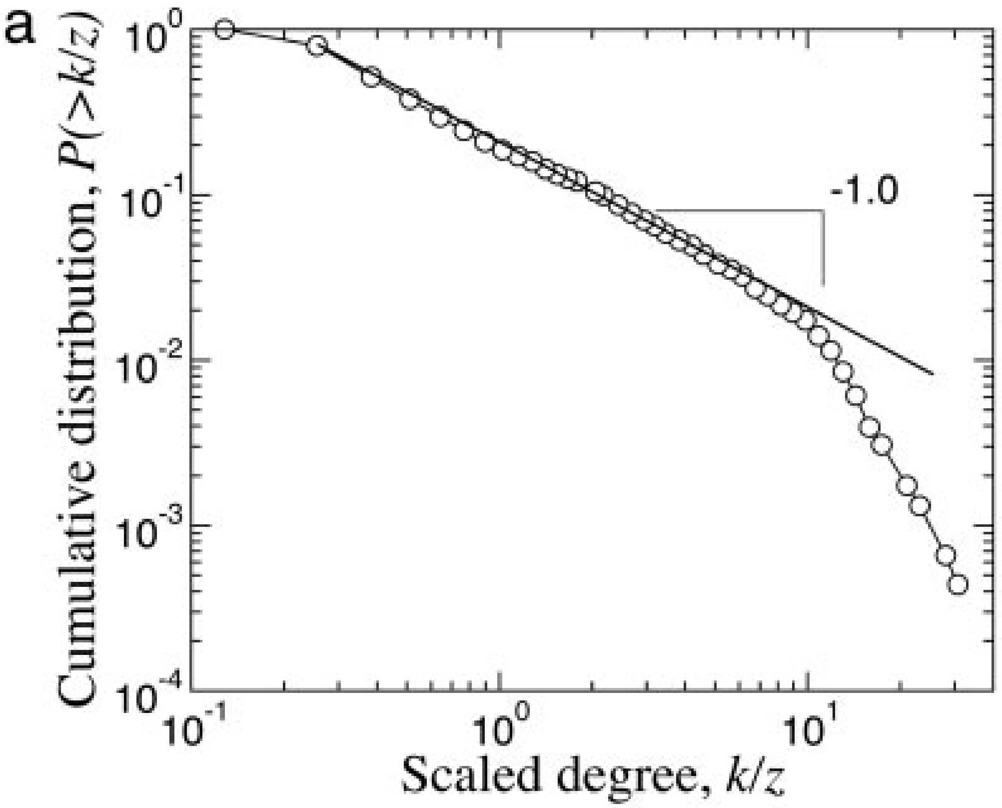
$$i(\lambda) \approx \lambda - \frac{\gamma - 3}{K_{min}(\gamma - 2)}$$

Homogeneous mixing model is recovered

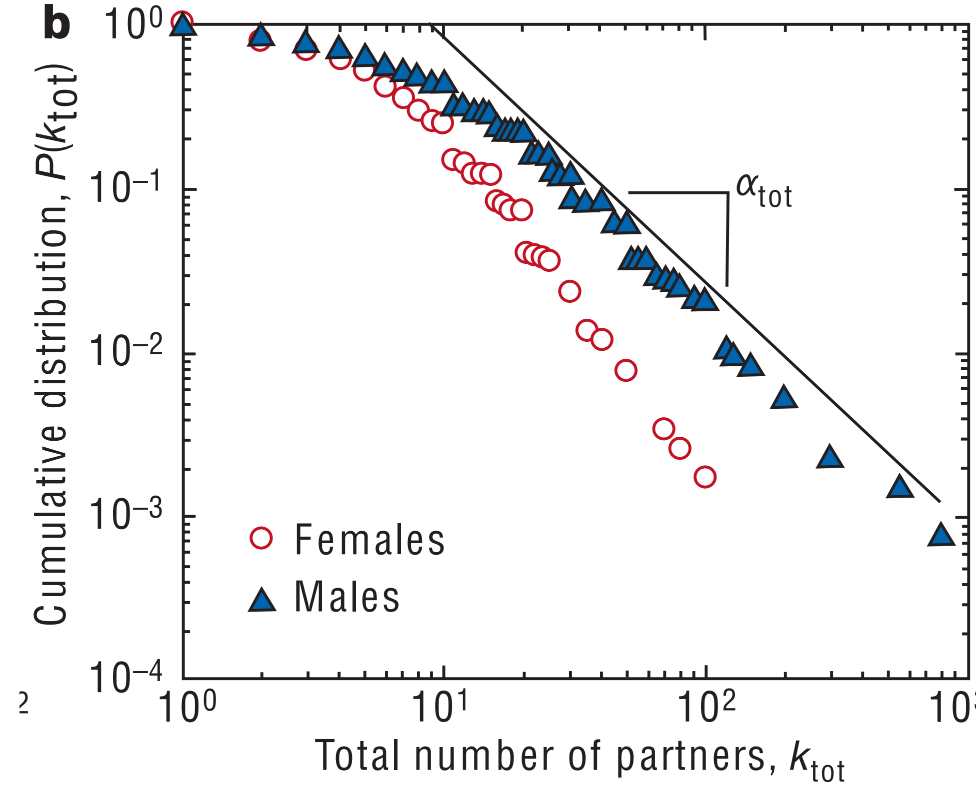
Barrat, Barthelemy, Vespignani book

Real networks

Guimera et.al. (2004) - airplane network



Liljeros et.al. (2001) - human sexual networks



$$\lambda_c = \frac{\langle k \rangle}{\langle k^2 \rangle - \langle k \rangle}$$

Many real networks have broad degree distribution with small exponent and vanishing epidemic threshold

Barrat, Barthelemy, Vespignani book

Immunisation and control

How to control epidemics?

- Transmission reduced intervention
  - face masks
  - gloves, hand washing
- Contact reducing interventions
  - quarantaines
  - closing schools
  - reduce travels and mobility
- Vaccination: remove nodes from the network
  - Question: who should we vaccinate?

These strategies may reduce the transmission rate if applied for the majority of the population

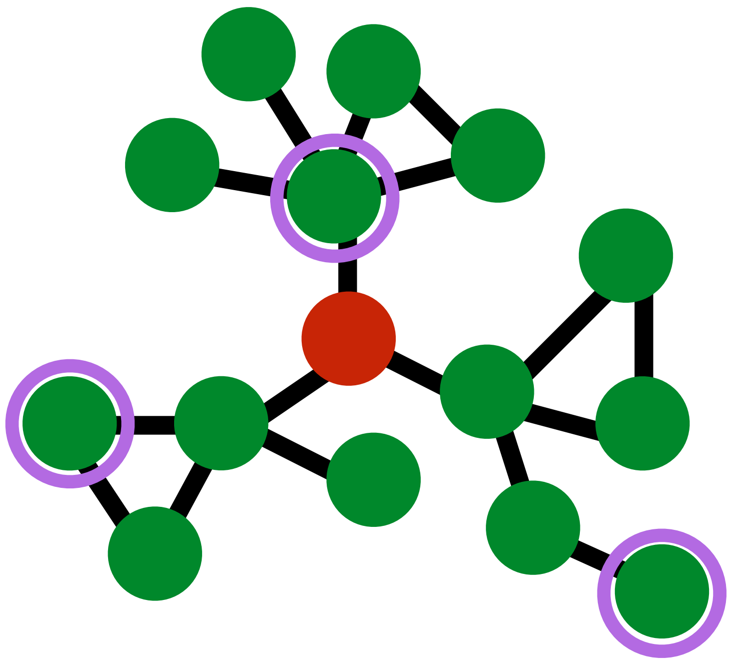
These strategies make the networks sparser and may increase the critical transmission rate (and they are very expensive)

These strategies suppress the population below the epidemic threshold

Barrat, Barthelemy, Vespignani book

Immunisation strategies

Random immunisation



- Immunise g fraction of nodes randomly selected from the population
- This strategy does not make difference between nodes of different degrees
- It is rescaling the spreading rate

$$\beta \rightarrow \beta(1-g)$$

- A critical immunised population size can be defined

$$\frac{\beta}{\mu}(1-g_c) = \frac{\langle k \rangle}{\langle k^2 \rangle}$$ where g_c must be $g_c = 1$

- If not as $\langle k^2 \rangle \rightarrow \infty$ the threshold still goes $\lambda_c \rightarrow 0$, only the epidemics will be slower
- Random immunisation cannot prevent the outbreak

Barrat, Barthelemy, Vespignani book

Immunisation strategies

Targeted immunisation

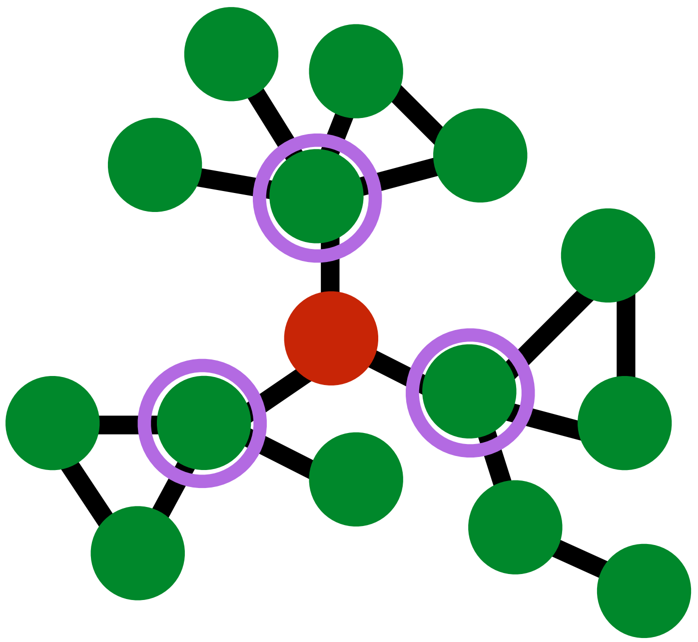
- Rank nodes by their degrees and introduce a g fraction with the highest degree
- By removing a g fraction of highest degrees the degree distribution will change - fluctuations of $\langle k^2 \rangle$ will be reduced
- There will be a critical g_c fraction which will stop the epidemics as threshold will became larger than zero

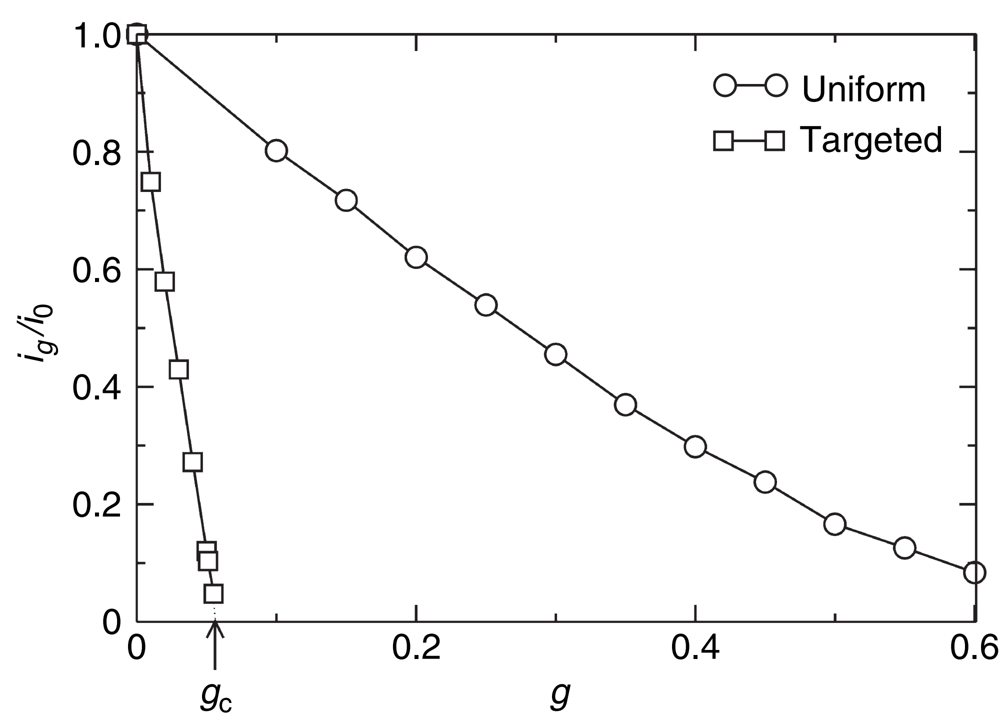
$$\frac{\langle k \rangle_{g_c}}{\langle k^2 \rangle_{g_c}} = \frac{\beta}{\mu}$$

- The leading term of the critical fraction (for a BA network with $\gamma=3$):

$$g_c \sim \exp(-2\mu/m\beta)$$

- This is the point where the network becomes "disconnected" regarding the diffusion of epidemics
- Problem: this immunisation strategy requires complete knowledge about the network (what we usually do not have!)





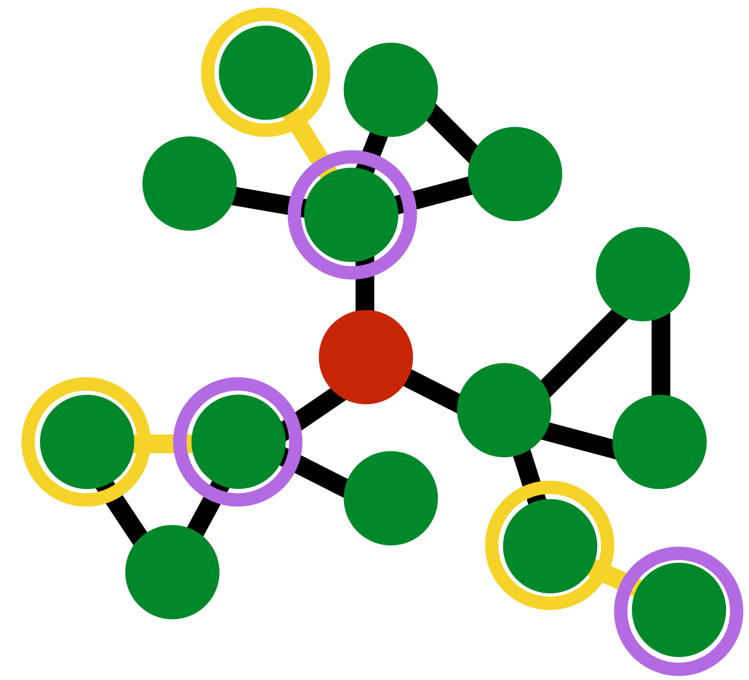
Pastor-Satorras &Vespignani, (2002)

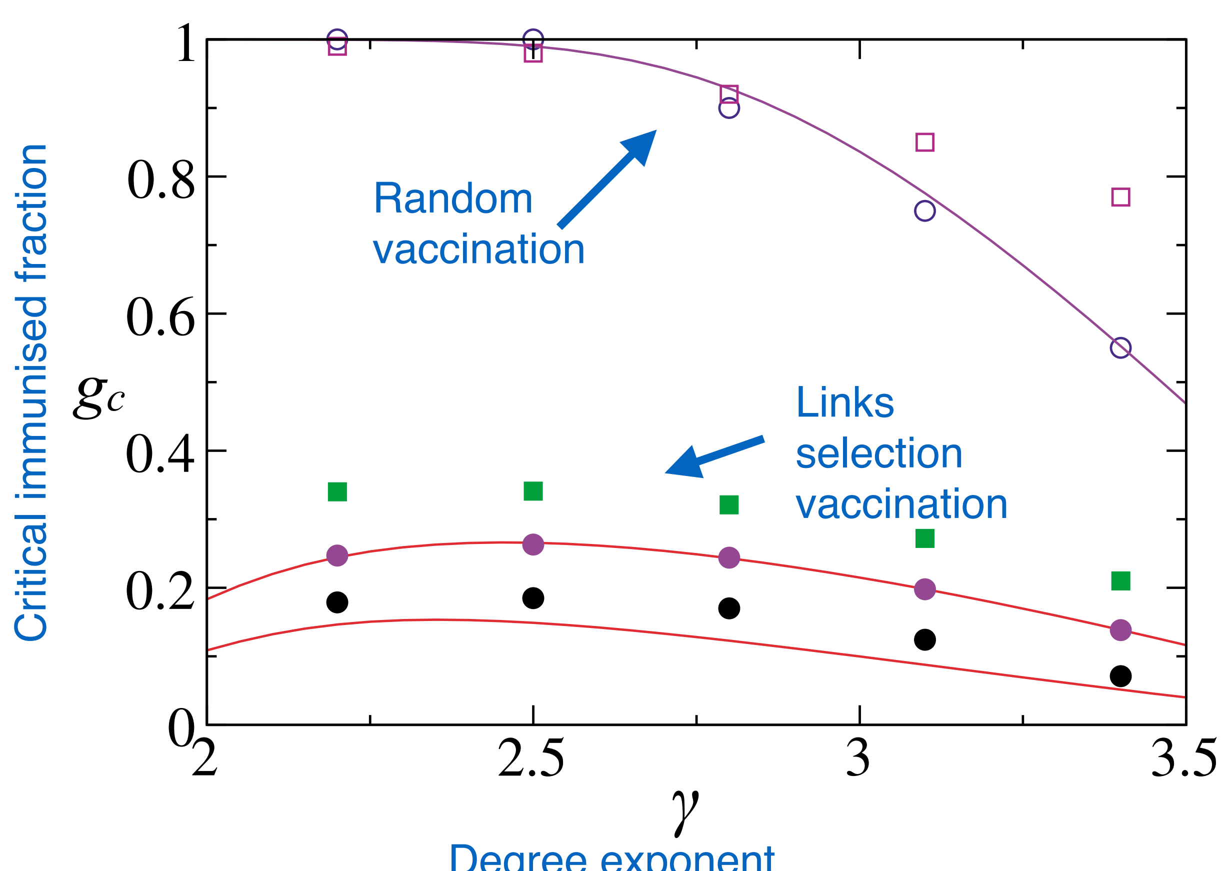
Barrat, Barthelemy, Vespignani book

Immunisation strategies

Immunisation without global knowledge

- Exploit degree heterogeneity and that a randomly selected link connects to a large degree node with higher probability
- Method:
  - Select a random node (~P(k))
  - Select a random link of the randomly selected node and immunise (~kP(k))





- Considerably out-performs the random strategy
- It performs worse compared to the targeted strategy
- Does not require global knowledge about the network structure

R. Cohen et al, Phys. Rev. Lett 91, 247901 (2003)

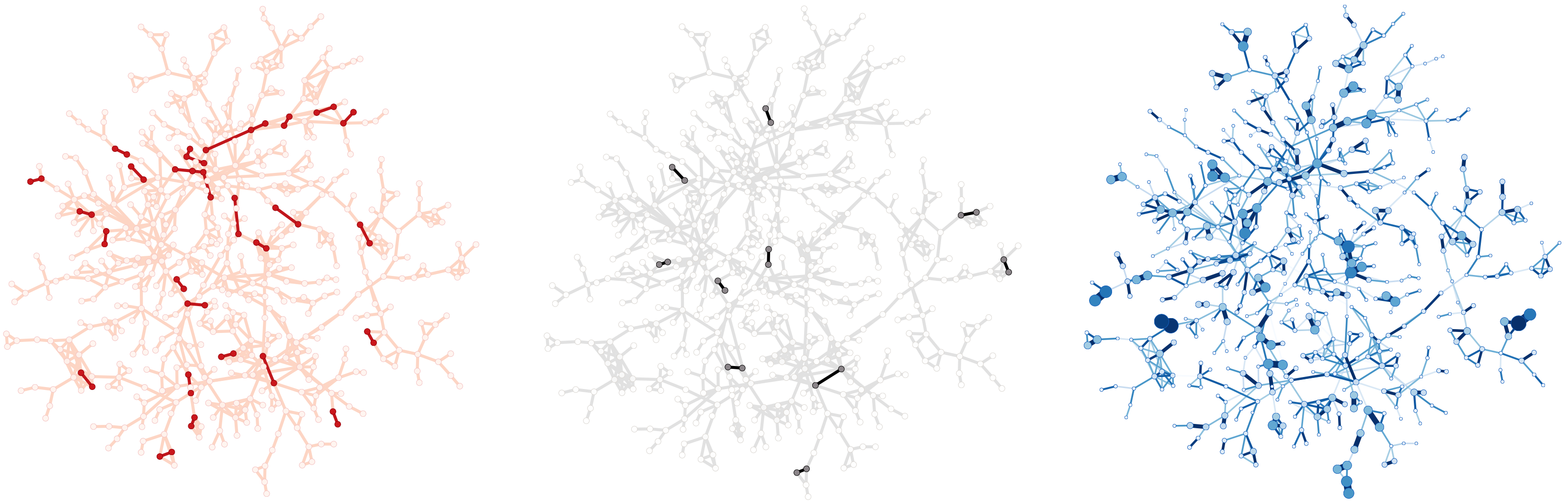
Barrat, Barthelemy, Vespignani book

Spreading processes on temporal networks

Temporal networks

- Interactions between nodes are very fast and repetitive on established links
  - Description of networks on the level of single nodes and events
  - Challenge: Understand which microscopic correlations are responsible for the emergence of global structure
- Structural properties: measures must be re-defined with time considered
  - Microscopic level: temporal centrality and time respecting paths
  - Mezoscopic level: temporal and recurrent motifs
  - Macroscopic level: circadian fluctuations, …
- Dynamical processes
  - co-evolving with the network: processes and interactions are evolving on the same time-scales
  - Effect of spatial, topological and temporal correlations are amplified

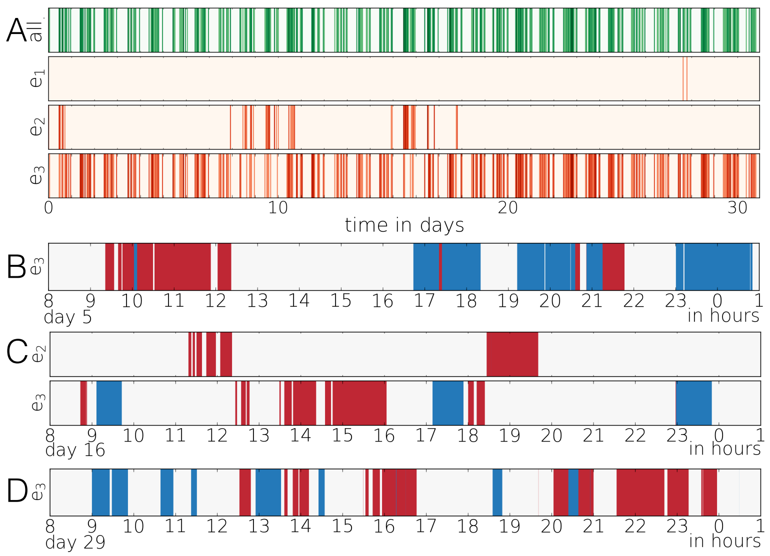
Challenge: completely new concept which need to build on novel methodology



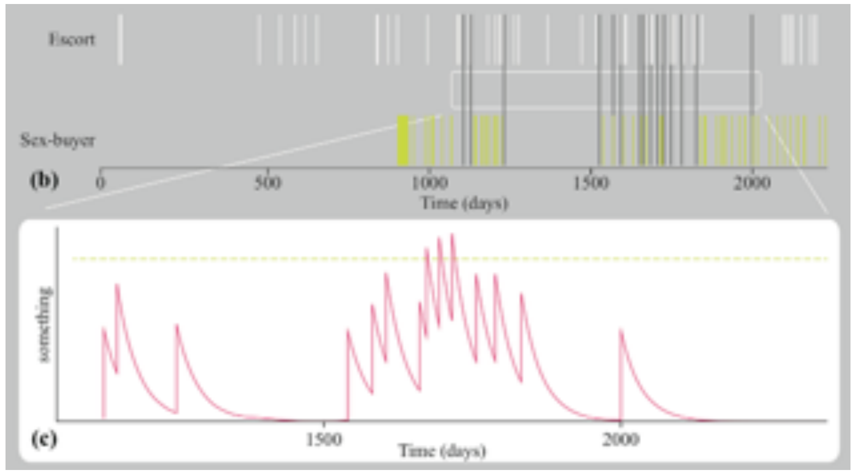
Karsai et.al. (2013)

Examples for temporal networks

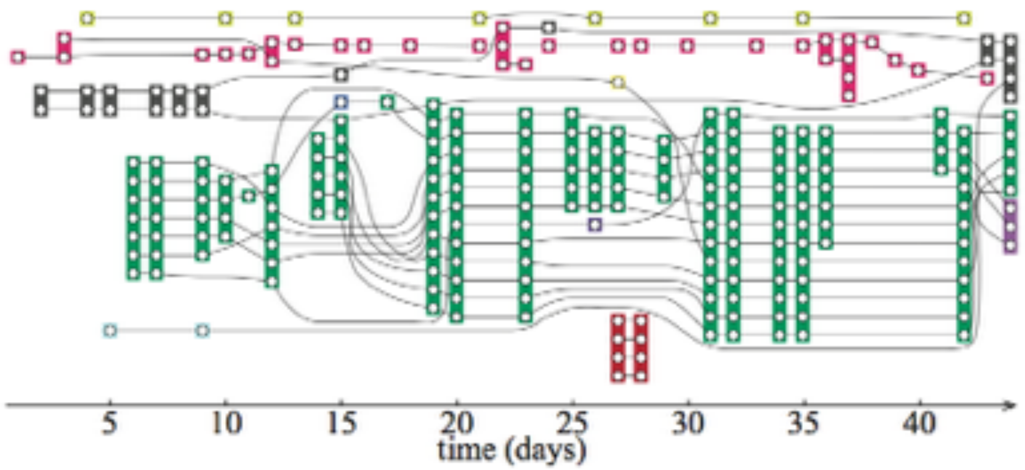
- Person-to-person communication
  - Mobile-phone calls
  - Email communication
  - Face-to-face interactions
- One-to-many information dissemination
  - Information broadcasting
  - Microblogging
- Distributed computing
  - Communication between computational units
- Infrastructural systems
  - Transportation
- Cell biology
  - Protein-protein interactions
  - Gene regulatory networks
- …



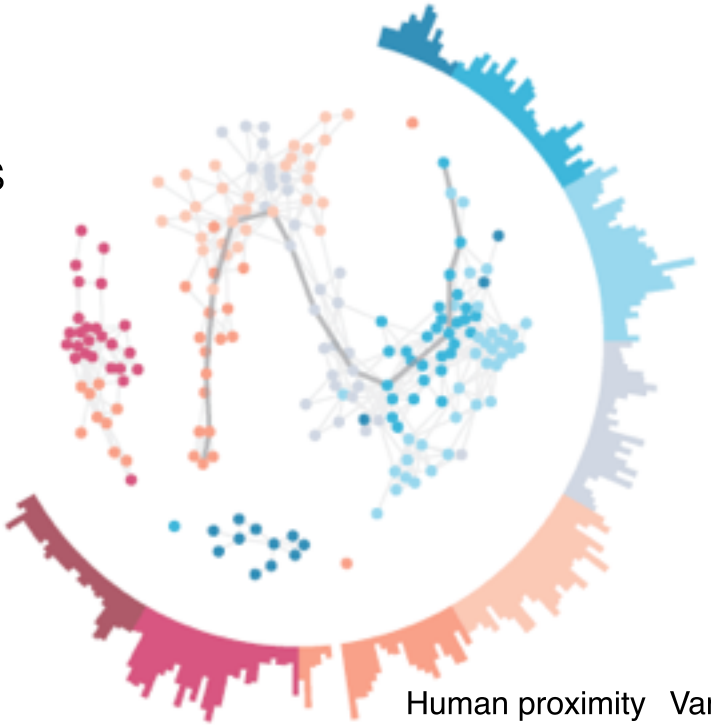
Temporal networks of mobile call communication
M. Karsai, et.al. (2012)



Temporal network of sex buyers and sellers
L. E. C. Rocha, et.al. (2010



Temporal network of zebras C. Tantipathananandh, et.al. (2007)



Human proximity Van der Broeck et.al. (2011)

Information spreading on temporal networks

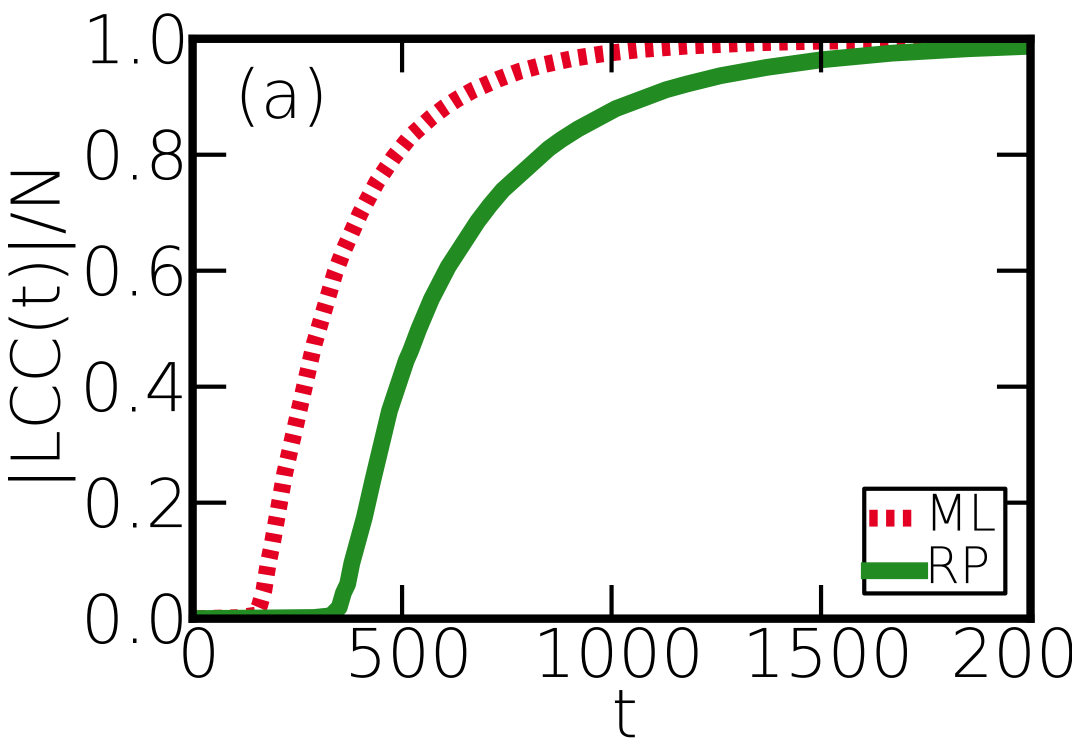
Earlier assumptions:

- The network structure is static
  - Every node and link are present in the network from the very beginning
- Dynamical process evolving on the top of the static structure
  - It is only effected by the (heterogeneous) topology of the network

Observation: information spreading can be very slow on temporal networks

Role of evolving structure

- The slow evolution of the network structure effects the dynamics of any collective phenomena
- Nothing can spread on the network globally without the emergence of a large connected component
- Speed of the LCC evolution sets up an upper limit for the speed of spreading phenomena



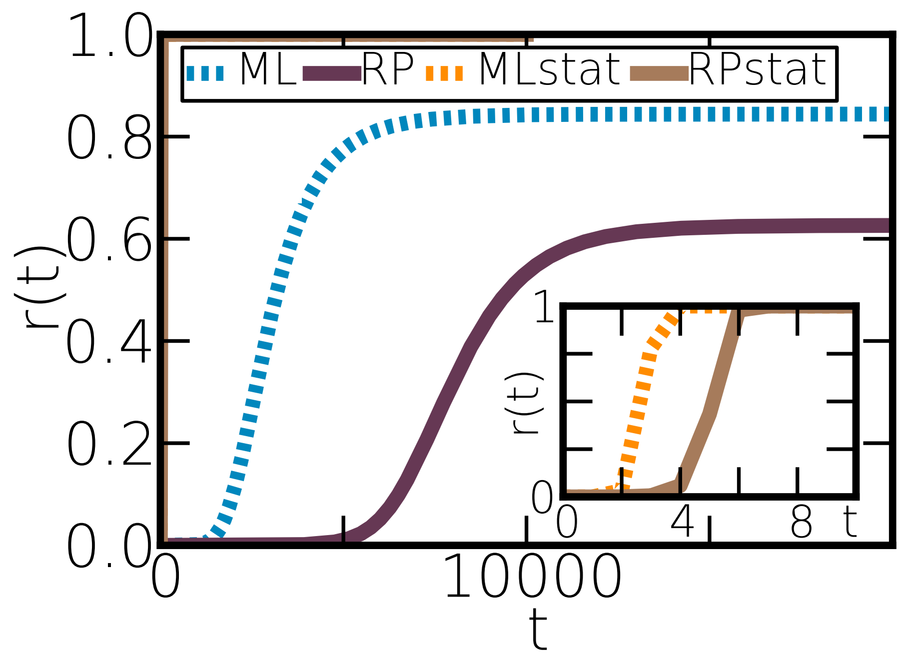
Spreading on temporal networks

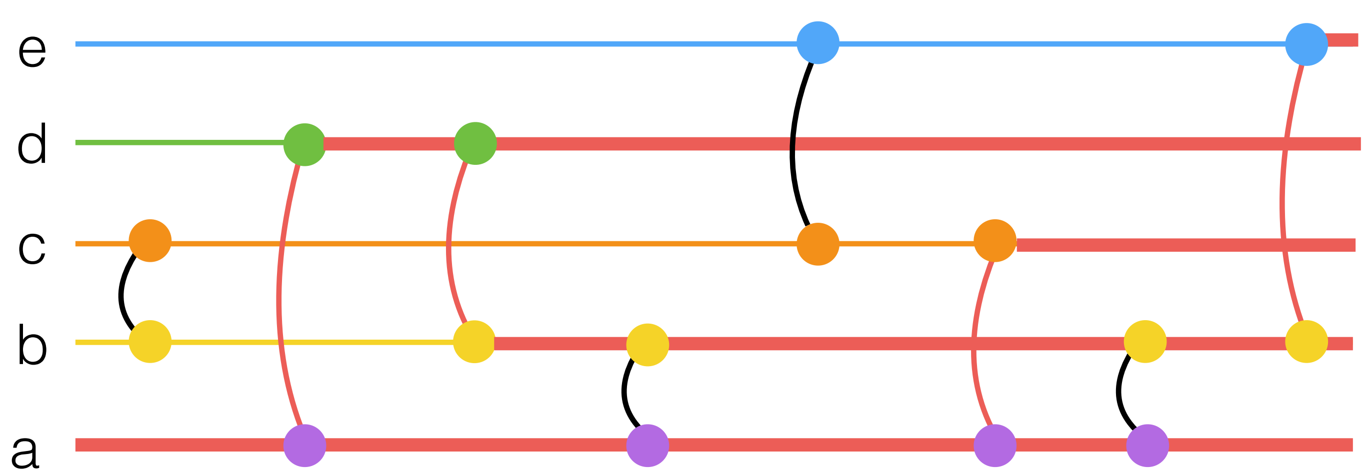
Earlier assumptions:

- Because every link is presented always they allow to spread information at any time

Role of temporal interactions

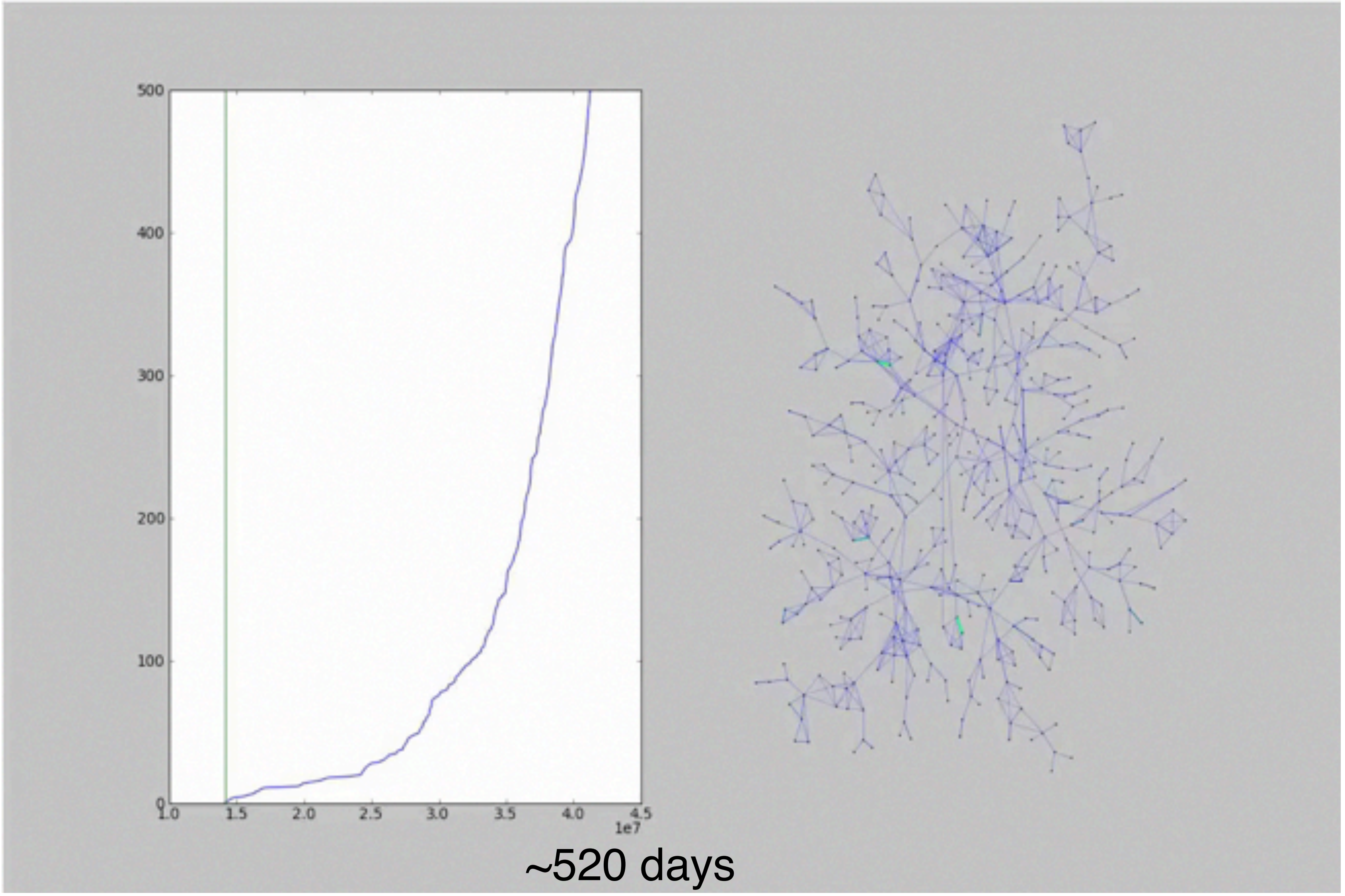
- In reality information can spread between nodes only at the time of their interactions
- The order of interactions determine the possible time respecting paths which along the information can be transmitted





Spreading on temporal networks

Role of temporal interactions



- Network: snow-ball sample of a mobile call networks
- Interactions: mobile calls
- Process: SI process initiated from a single seed
- Information spread between interacting individuals with β=1

Temporal network models

Temporal network models are still very rare and their introduction is an actual challenge…

Randomised reference models

- Take an empirical network
- Remove some characteristic correlations by random shuffling
- Static networks: Configuration model and its variances
- Temporal networks: …

Contact network models

- Take a set of nodes
- Define a dynamics which drives their (temporal) interactions
- Evolving networks: Barabási-Albert model
- Temporal networks: …

Spreading on temporal networks

Data-driven model experiment

- Take the sequence of mobile communication events
- Initiate an SI process from a randomly selected nodes at a randomly selected time
- Allow spreading with $\beta=1$ only between interacting individuals at the time of their interactions (independent of the direction of the communication)
- Once you reached the last event apply a periodic temporal boundary condition (jump to the beginning of the sequence)
- Perform the process until everyone is reached in the network by the information
- Repeat the experiment on randomised reference model with certain correlations removed

Why SI?

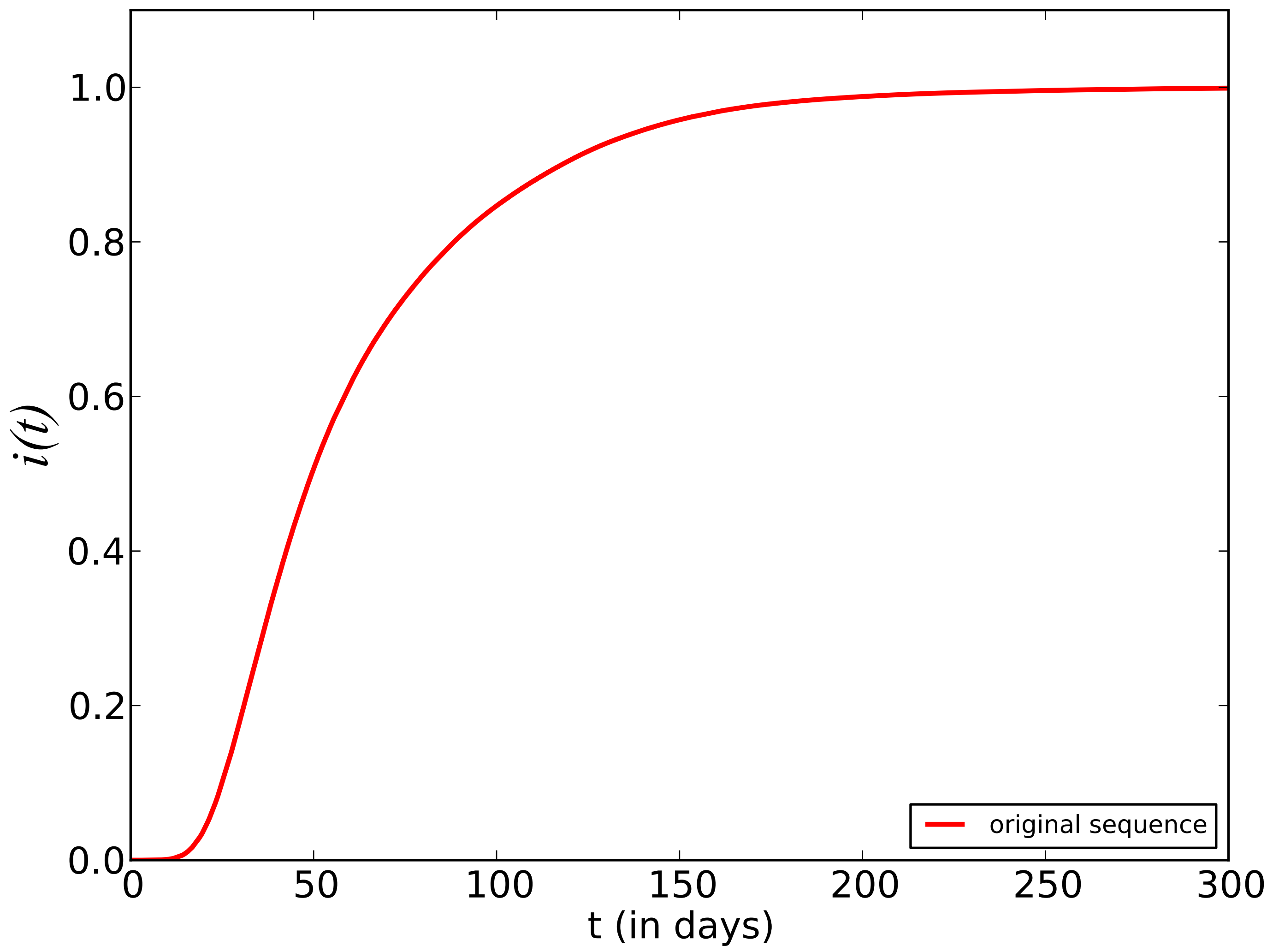
- This is the simplest model of information spreading
- No critical threshold: the process always reach 100% prevalence
- Since $\beta=1$ it gives the fastest possible scenario of information spreading

Original event sequence

- Time ordered sequence of original call events
- It contains all possible correlations which take place in the system
- Measure

$$i(t) = \frac{I(t)}{N}$$

	WT	BD	LL	CS	25%m
Original	✓	✓	✓	✓	33,7



Randomised reference model

Community structure

- Densely connected subgroups

Weight-topology correlations

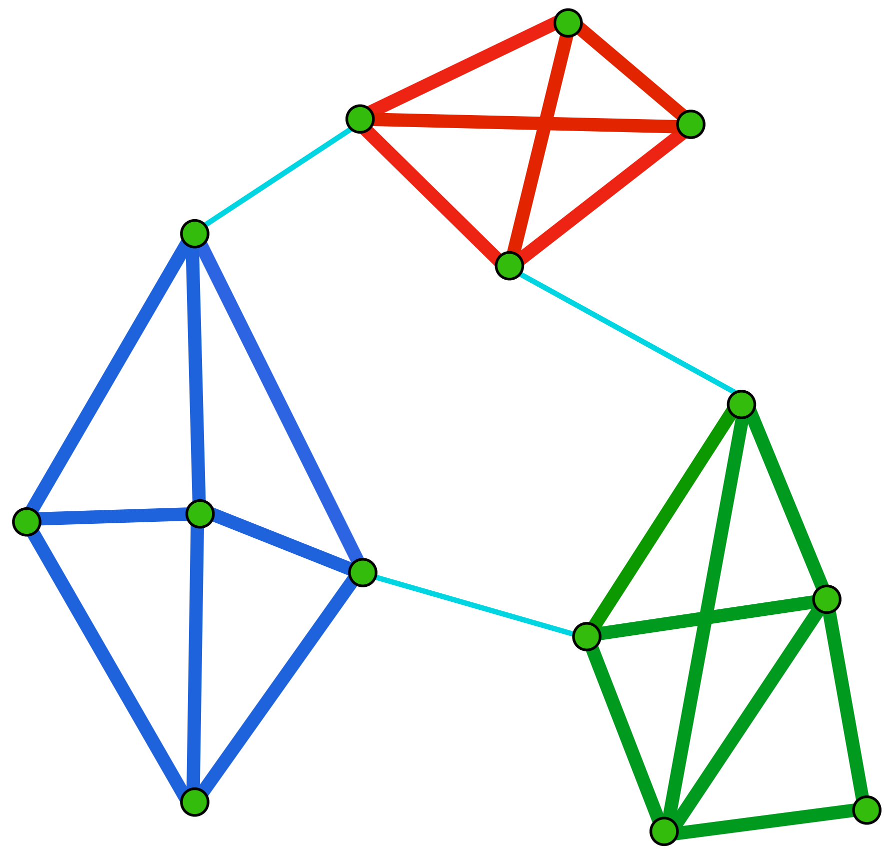
- Granovetter's Theorem (PNAS 104, 7332 (2007))

Bursty dynamical behavior

- Events are clustered in time (J.Phys.A 41,224015(2008))

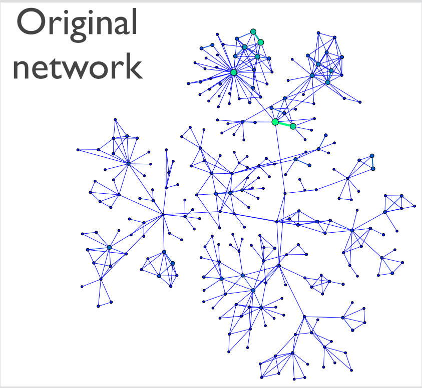
Link-link correlations

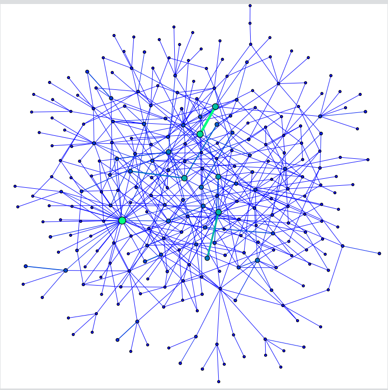
- Causality between consecutive calls



Shuffling

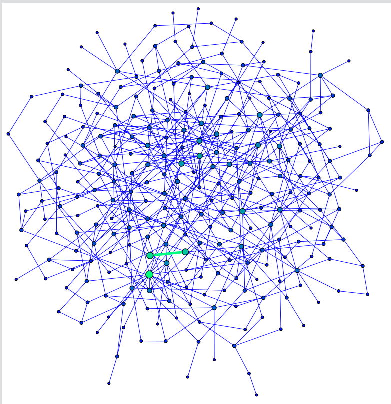
- Configuration model
- Random network





Configuration model

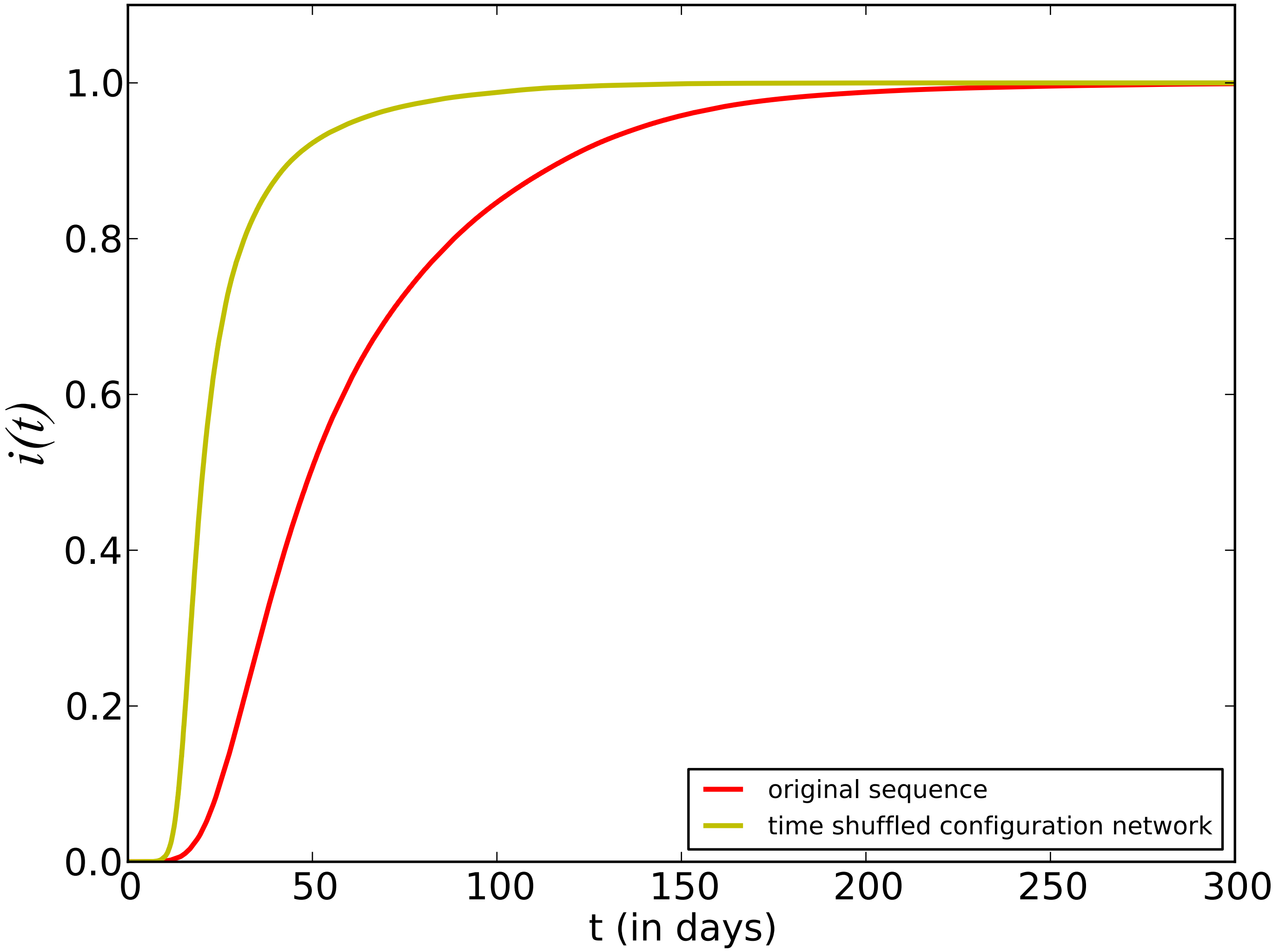
Random network



Time shuffled configuration network

- Using configuration model to **destroy community structure**, but keep N, IEI and the network connected
- Shuffle the event times to **destroy bursty dynamics**
- No correlation takes place in the system

	WT	BD	LL	CS	25%m
Original	✓	✓	✓	✓	33,7
TimeConf	✕	✕	✕	✕	16,4



Randomised reference model

Community structure

- Densely connected subgroups

Weight-topology correlations

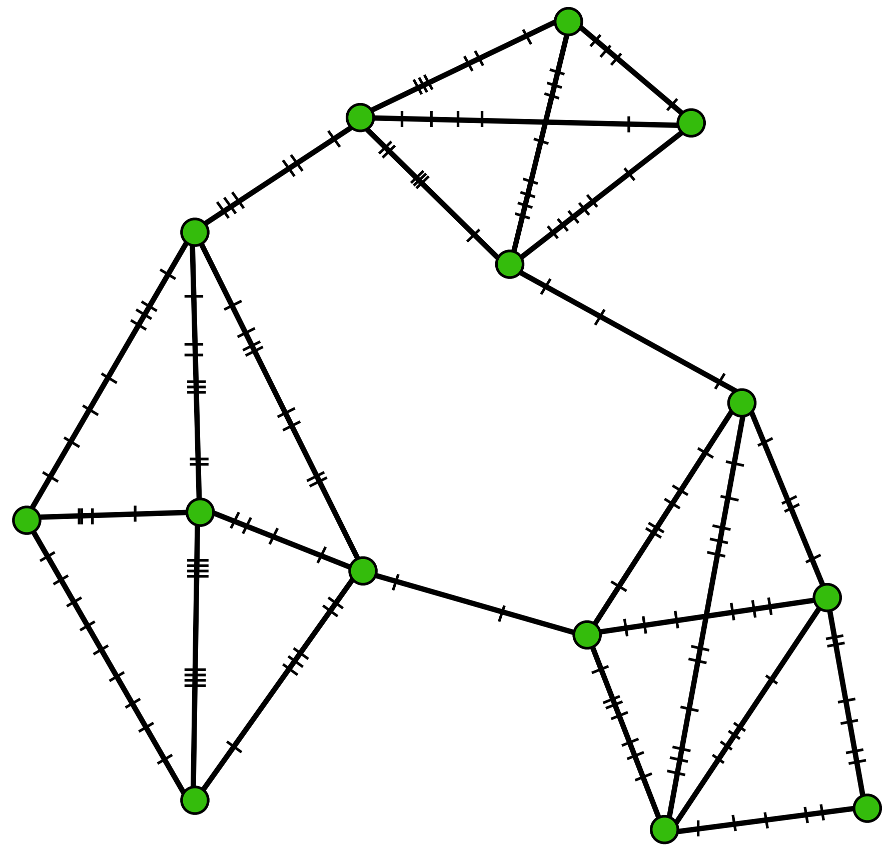
- Granovetter's Theorem (PNAS 104, 7332 (2007))

Bursty dynamical behavior

- Events are clustered in time
  (J.Phys.A 41,224015(2008))

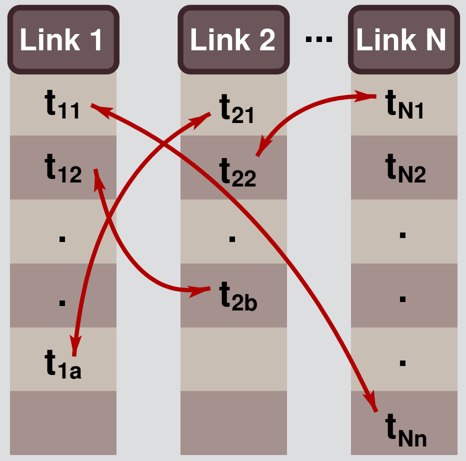
Link-link correlations

- Causality between consecutive calls



Shuffling

- Shuffle the event times of calls and destroy temporal heterogeneties
- keep P(w), P(k), P(s), w-top correlations
- destroy P(t_{ie}), link-link correlations

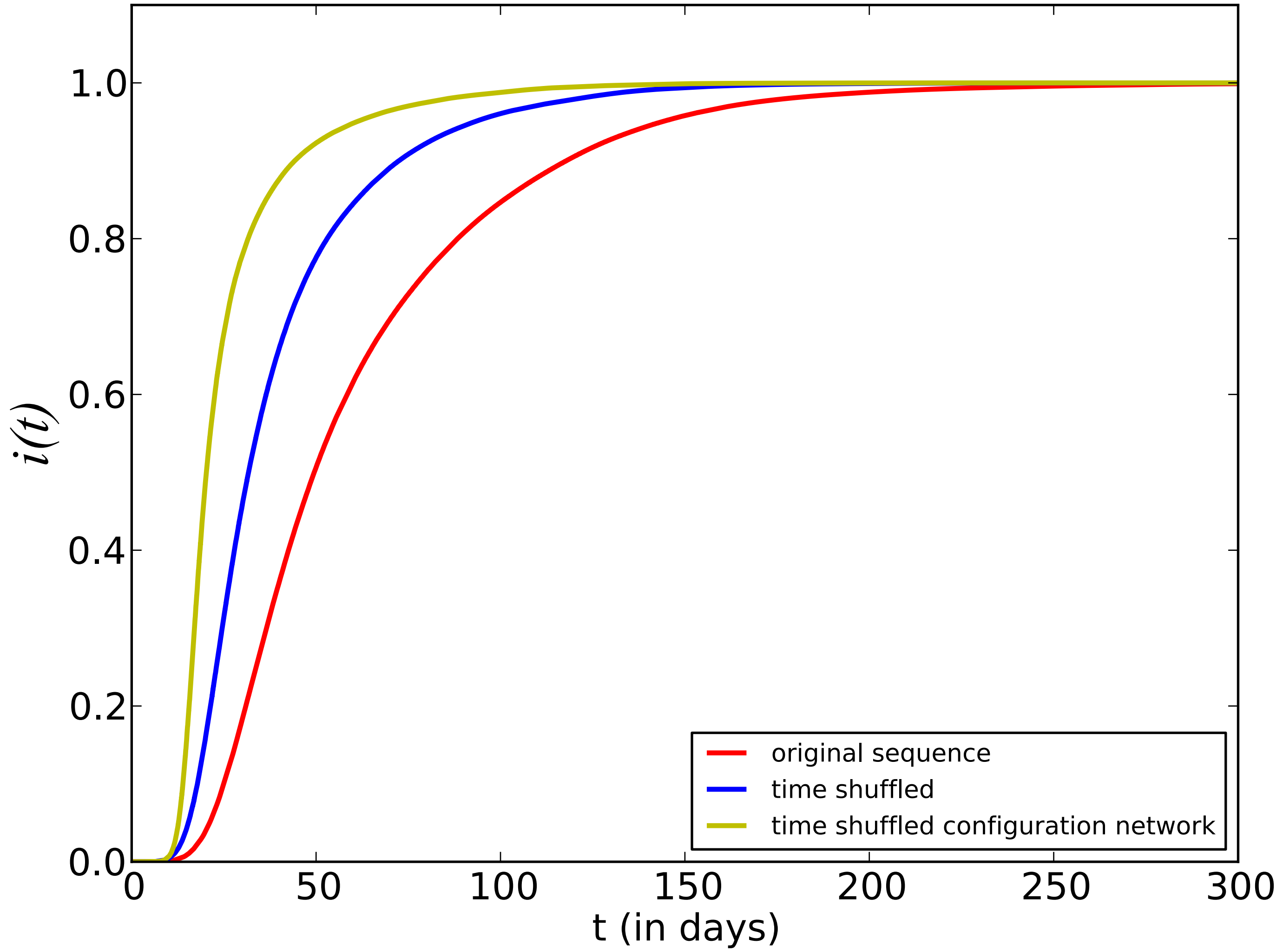


Time shuffling

Time shuffled network

- Using the original links and network
- **Bursty dynamical behaviour** and **link-link correlations** are destroyed
- The infection speed is slowed down by bursty dynamics

	WT	BD	LL	CS	25%m
Original	✓	✓	✓	✓	33,7
TimeConf	×	×	×	×	16,4
Time	✓	×	×	✓	22,9



Randomised reference model

Community structure

- Densely connected subgroups

Weight-topology correlations

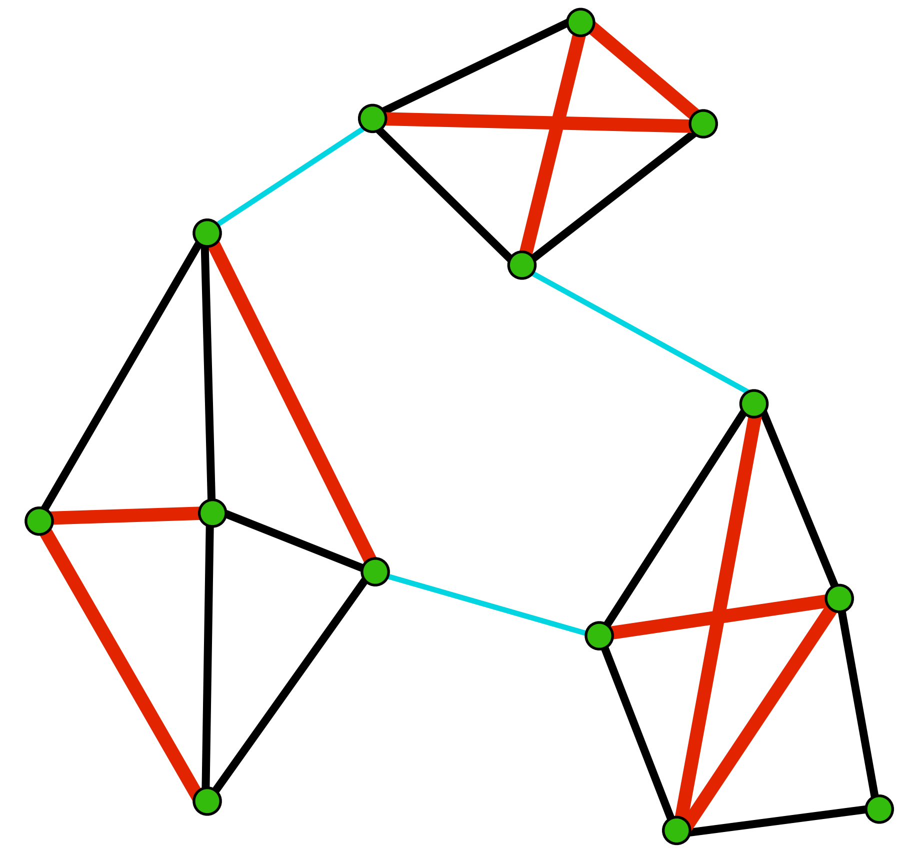
- Granovetter's Theorem (PNAS 104, 7332 (2007))

Bursty dynamical behavior

- Events are clustered in time
(J.Phys.A 41,224015(2008))

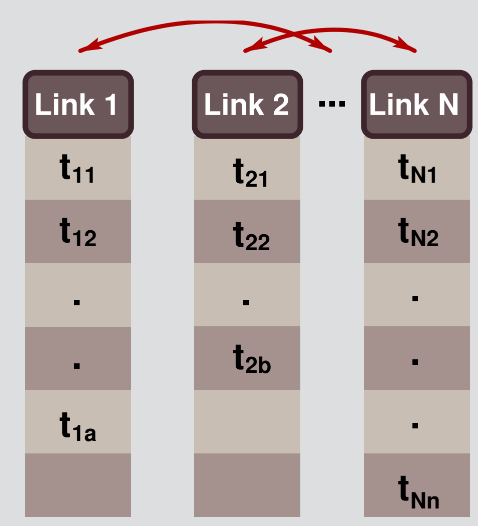
Link-link correlations

- Causality between consecutive calls



Shuffling

- Change complete call sequences of individuals regardless of their edge weight
- keep P(w), P(k), P(t_{ie})
- destroy P(s), link-link correlations, w-top correlations

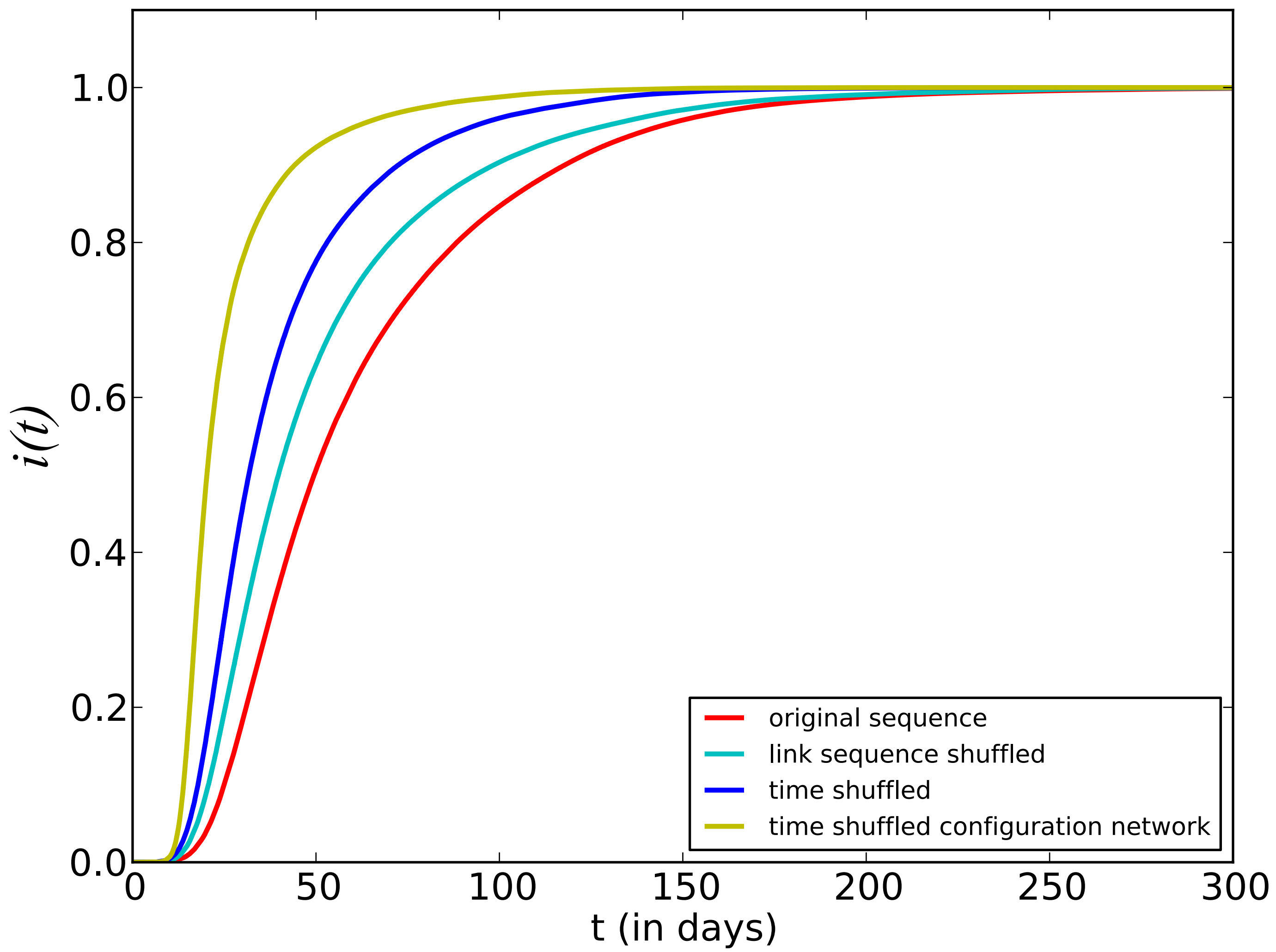


Link sequence shuffling

Link sequence shuffled event sequence

- Shuffle link call sequences between randomly chosen links
- **Link-link** and **weight-topology** correlations are switched off
- Weight-topology correlations also slow down the dynamics

	WT	BD	LL	CS	25%m
Original	✓	✓	✓	✓	33,7
TimeConf	✕	✕	✕	✕	16,4
Time	✓	✕	✕	✓	22,9
Link	✕	✓	✕	✓	27,5



Randomised reference model

Community structure

- Densely connected subgroups

Weight-topology correlations

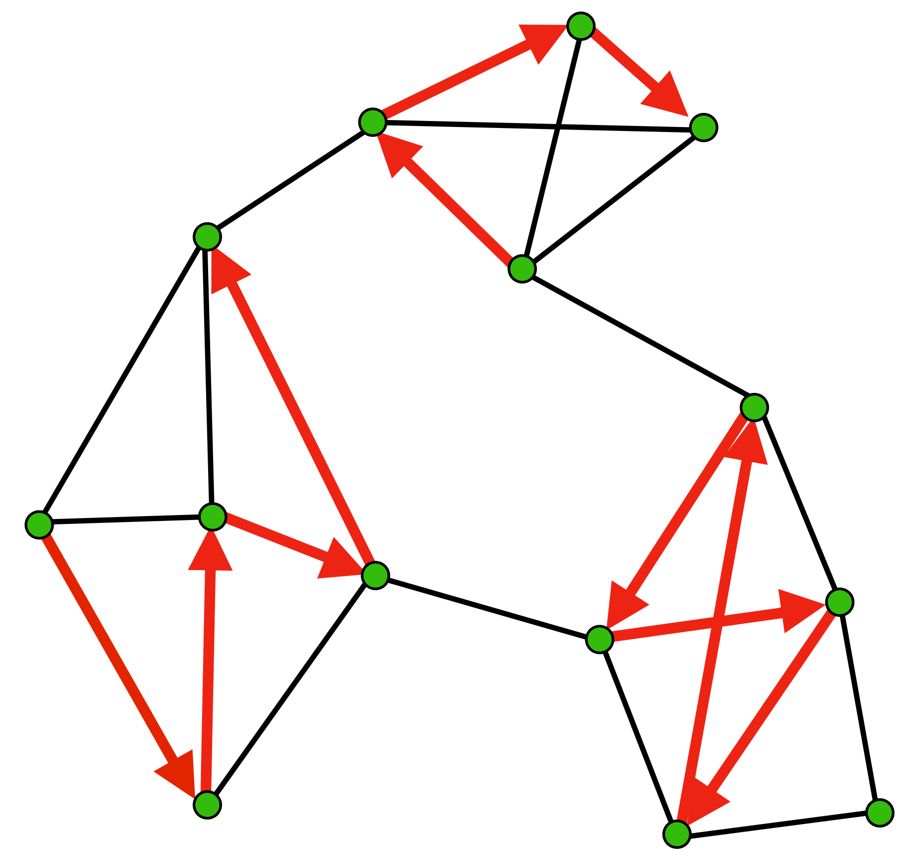
- Granovetter's Theorem (PNAS 104, 7332 (2007))

Bursty dynamical behavior

- Events are clustered in time (J.Phys.A 41,224015(2008))

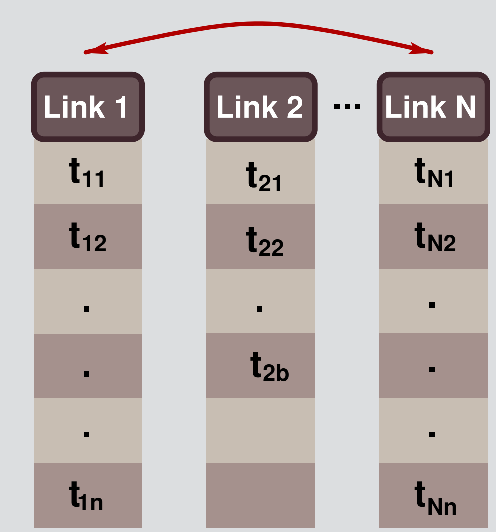
Link-link correlations

- Causality between consecutive calls



Shuffling

- Change complete call sequences of individuals
- keep P(w), P(k), P(s), P(t_{ie}), w-top correlations
- destroy link-link correlations

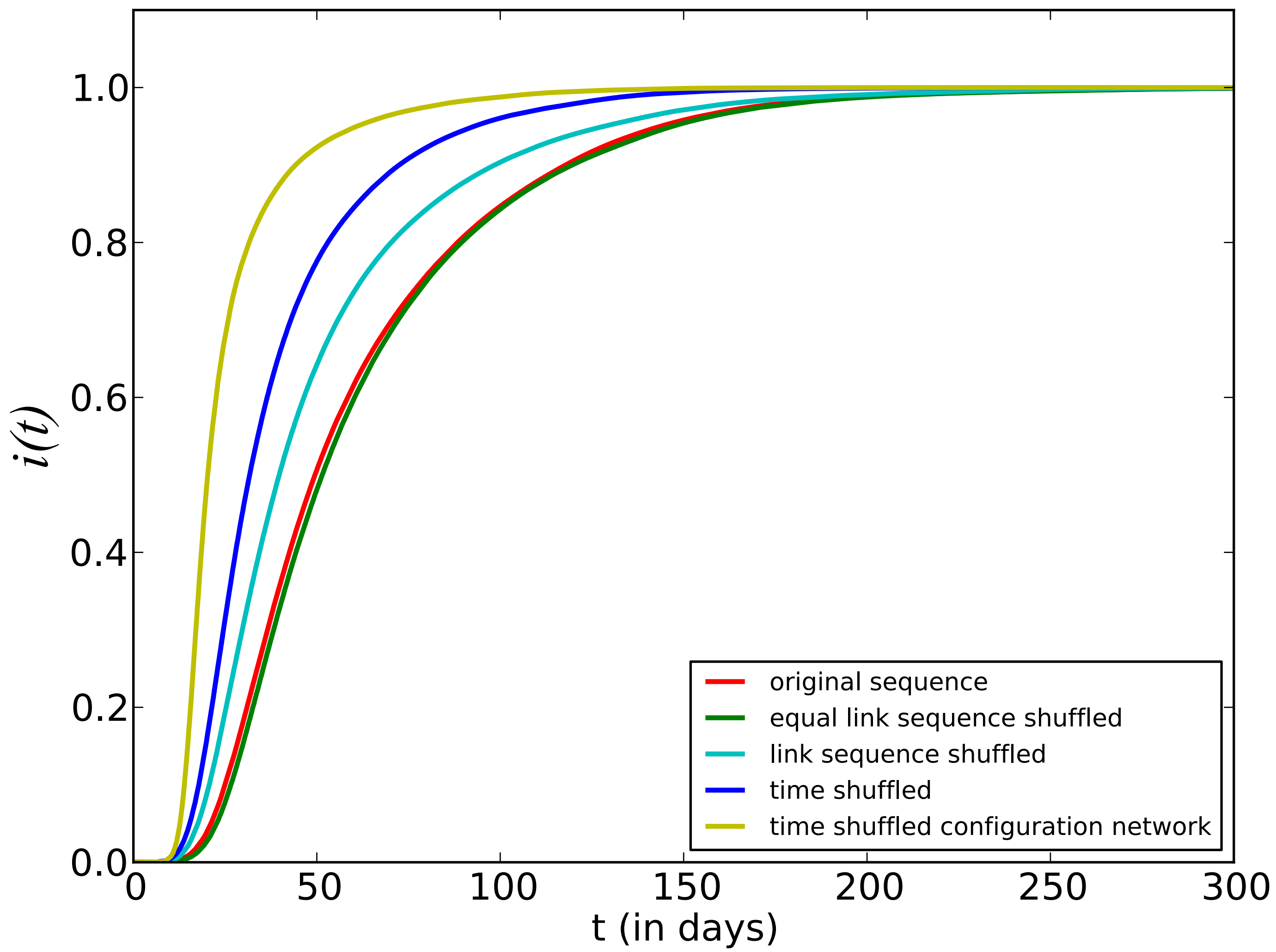


Eq.-w. link-seq. shuffling

Equal link sequence shuffled event sequence

- Shuffle call sequences between links having the same weight
- Only **link-link correlations** are destroyed
- Multilink correlations accelerate the spreading process

	WT	BD	LL	CS	25%m
Original	✓	✓	✓	✓	33,7
TimeConf	✕	✕	✕	✕	16,4
Time	✓	✕	✕	✓	22,9
Link	✕	✓	✕	✓	27,5
Equal link	✓	✓	✕	✓	35,3

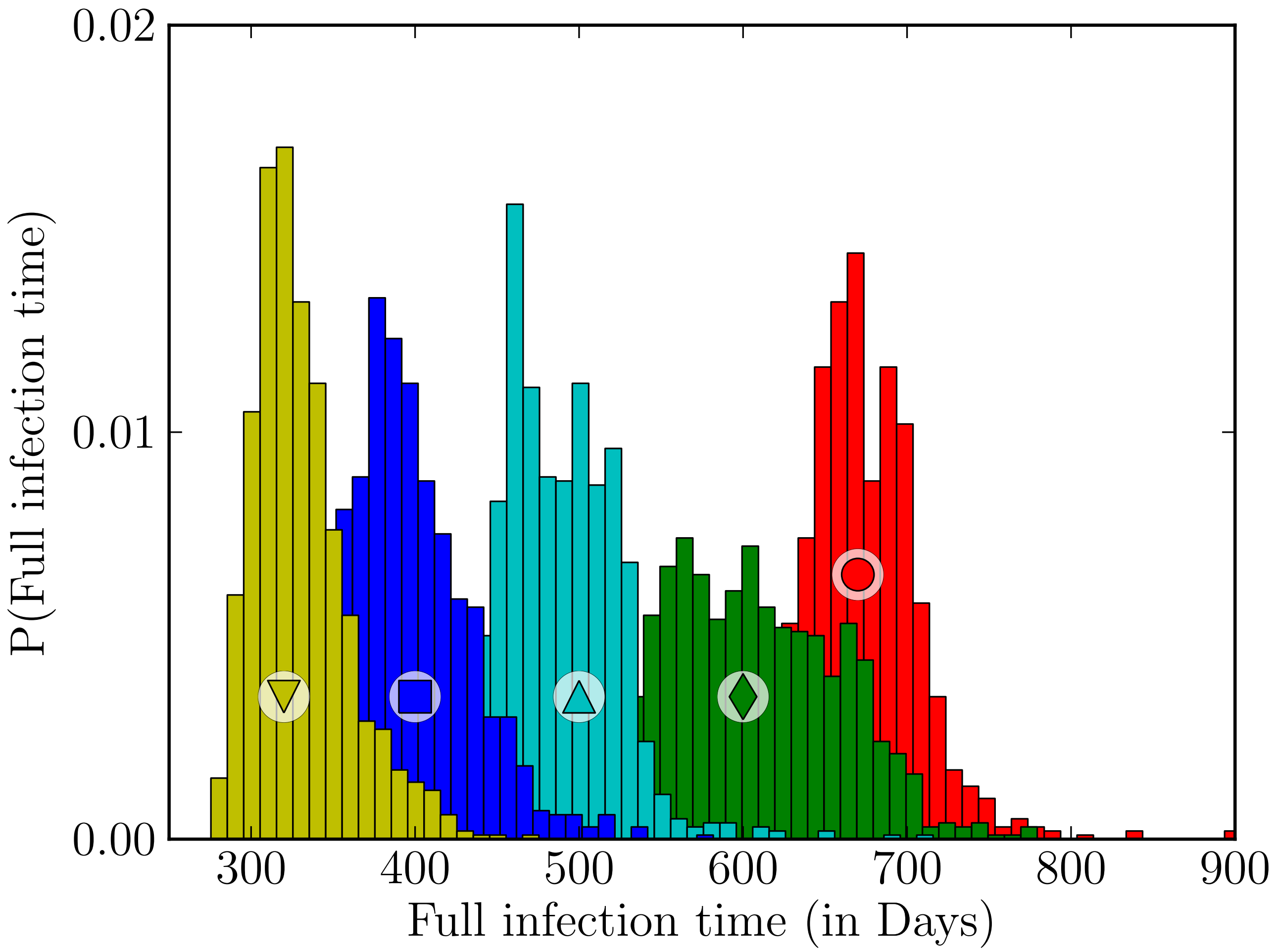


Long temporal behaviour

- Measure the distribution of complete infection time

- Clear evidence for influence of different correlations at late time stage

- Multilink correlations play a contrary role than at early time stage



Weight-topology correlations and bursty temporal behaviour are responsible mostly for the slow spreading

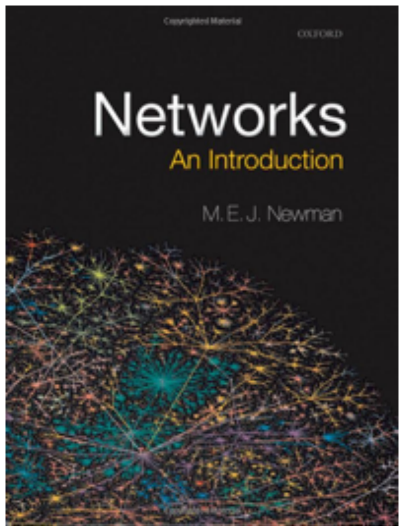
M Karsai, M Kivelä, RK Pan, K Kaski, J Kertész, AL Barabási, J Saramäki, PRE(R) 83, 056125 (2011)

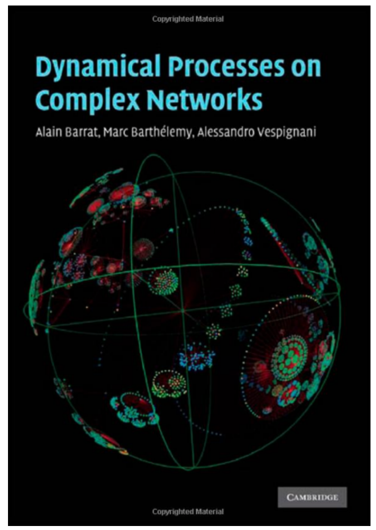
M Kivelä, RK Pan, K Kaski, J Kertész, J Saramäki, M Karsai, J. Stat. Mech. P03005 (2012)

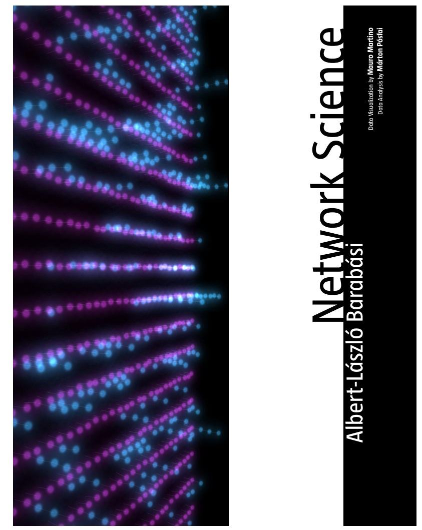
<u>Similar results with SIR:</u> G Miritello, E Moro and R Lara, PRE(R) 83 045102 (2011)

Materials

Lecture books







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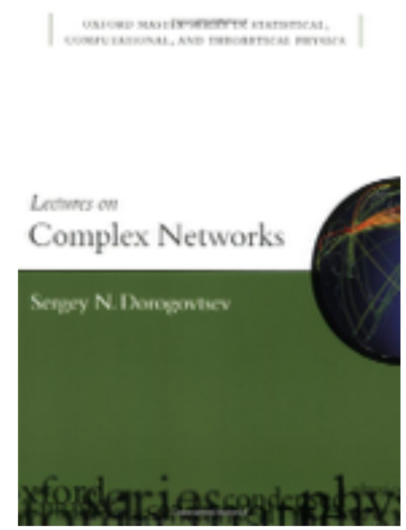

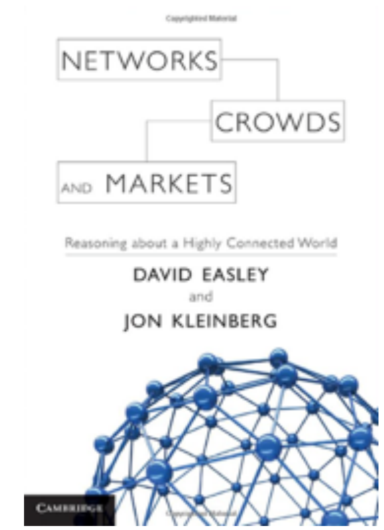

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Reviews

SIAM Review
Vol. 45, No. 2, pp. 167–256

The Structure and Function of Complex Networks*

M. E. J. Newman†

REVIEWS OF MODERN PHYSICS, VOLUME 74, JANUARY 2002

Statistical mechanics of complex networks

Réka Albert* and Albert-László Barabási
Department of Physics, University of Notre Dame, Notre Dame, Indiana 46556

Physics Reports 486 (2010) 75–174
Contents lists available at ScienceDirect
Physics Reports
journal homepage: www.elsevier.com/locate/physrep
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Community detection in graphs

Santo Fortunato*
Complex Networks and Systems Lagrange Laboratory, ISI Foundation, Viale S. Severo 65, 10133, Torino, I, Italy

Physics Reports 519 (2012) 97–125
Contents lists available at SciVerse ScienceDirect
Physics Reports
journal homepage: www.elsevier.com/locate/physrep
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Temporal networks

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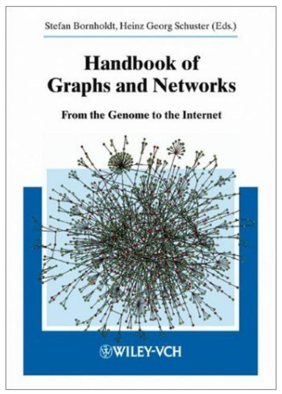
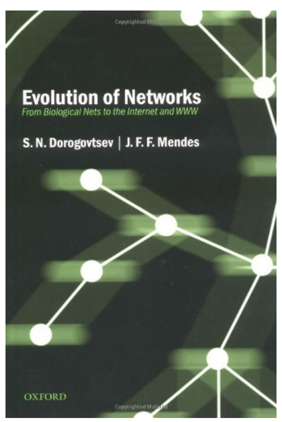
[b] *Department of Energy Science, Sungkyunkwan University, Suwon 440–746, Republic of Korea*
[c] *Department of Sociology, Stockholm University, 106 91 Stockholm, Sweden*
[d] *Department of Biomedical Engineering and Computational Science, School of Science, Aalto University, 00076 Aalto, Espoo, Finland*

Materials

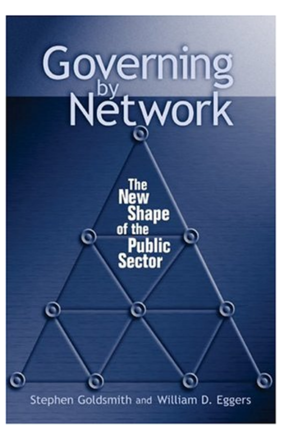
Related books



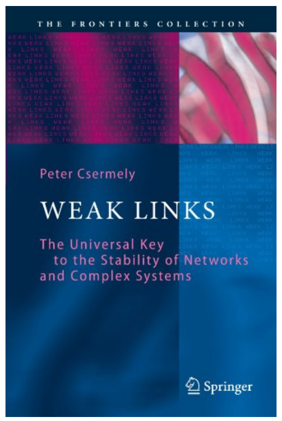
Handbook of Graphs and Networks: From the Genome to the Internet (Wiley-VCH, 2003).



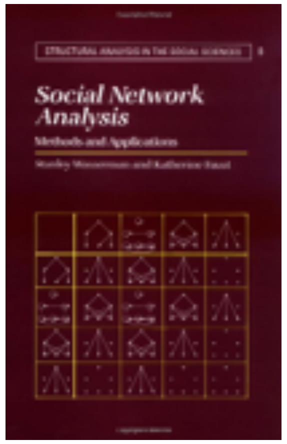
S. N. Dorogovtsev and J. F. F. Mendes, Evolution of Networks: From Biological Nets to the Internet and WWW (Oxford University Press, 2003).



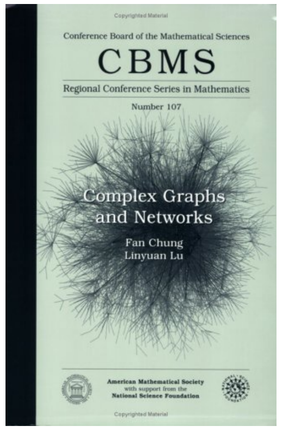
S. Goldsmith, W. D. Eggers, Governing by Network: The New Shape of the Public Sector (Brookings Institution Press, 2004).



P. Csermely, Weak Links: The Universal Key to the Stability of Networks and Complex Systems (The Frontiers Collection) (Springer, 2006), rst edn.



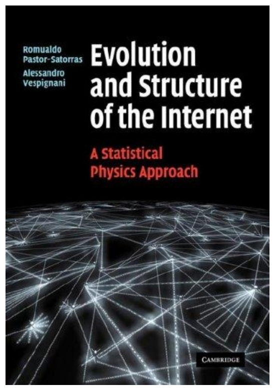
S. Wasserman and K. Faust
Social Network Analysis (Methods and Applications)
Cambridge University Press (1994)



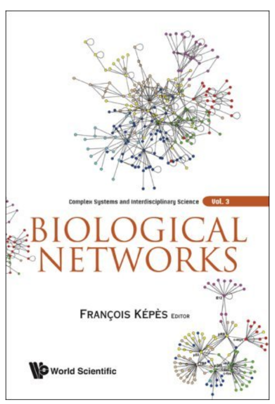
L. L. F. Chung, Complex Graphs and Networks (CBMS Regional Conference Series in Mathematics) (American Mathematical Society, 2006).

Materials

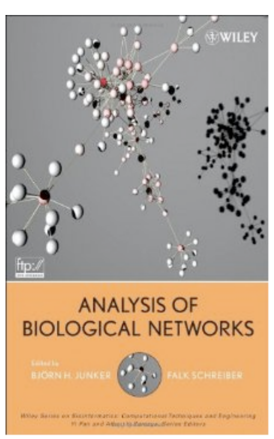
Related books



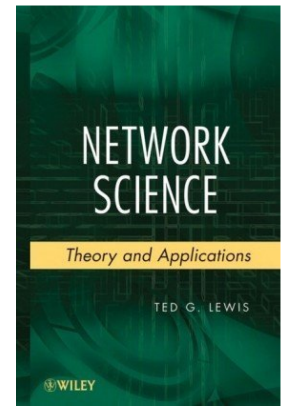
R. Pastor-Satorras, A. Vespignani, Evolution and Structure of the Internet: A Statistical Physics Approach (Cambridge University Press, 2007), rst edn.



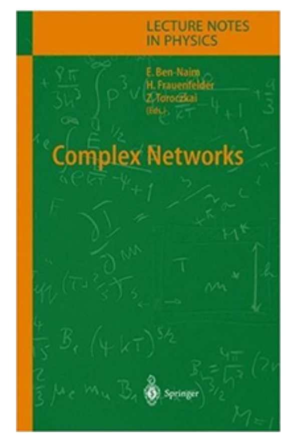
F. Kopos, Biological Networks (Complex Systems and Interdisciplinary Science) (World Scientific Publishing Company, 2007), rst edn.



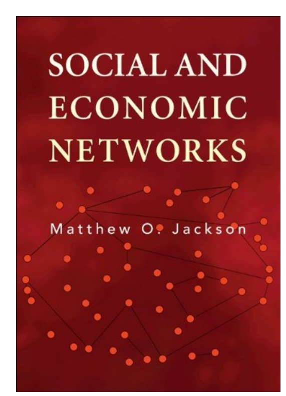
B. H. Junker, F. Schreiber, Analysis of Biological Networks (Wiley Series in Bioinformatics) (Wiley-Interscience, 2008).



T. G. Lewis, Network Science: Theory and Applications (Wiley, 2009).



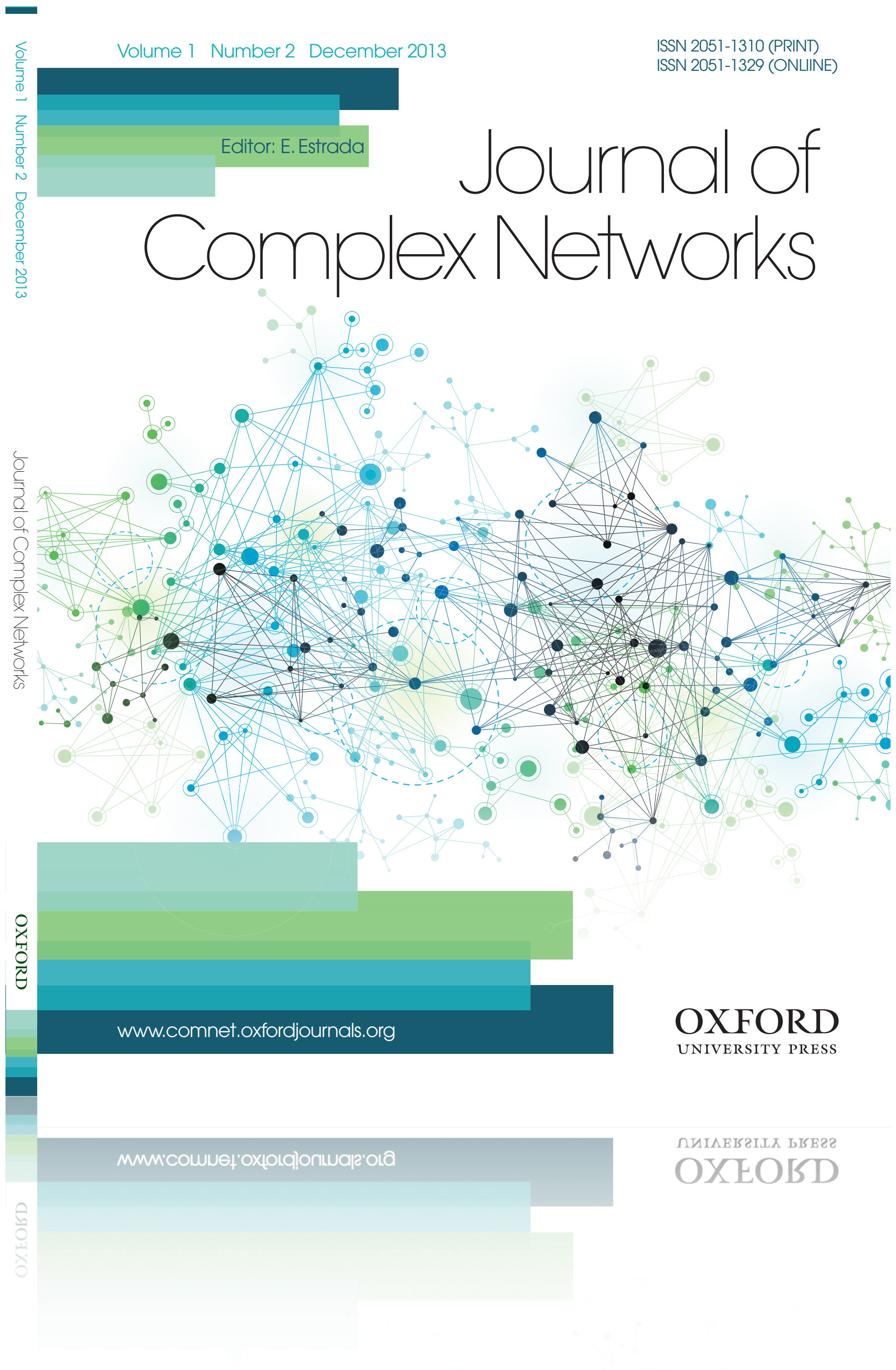
E. Ben Naim, H. Frauenfelder, Z.Torotzai, Complex Networks (Lecture Notes in Physics) (Springer, 2010), rst edn.



M. O. Jackson, Social and Economic Networks (Princeton University Press, 2010).

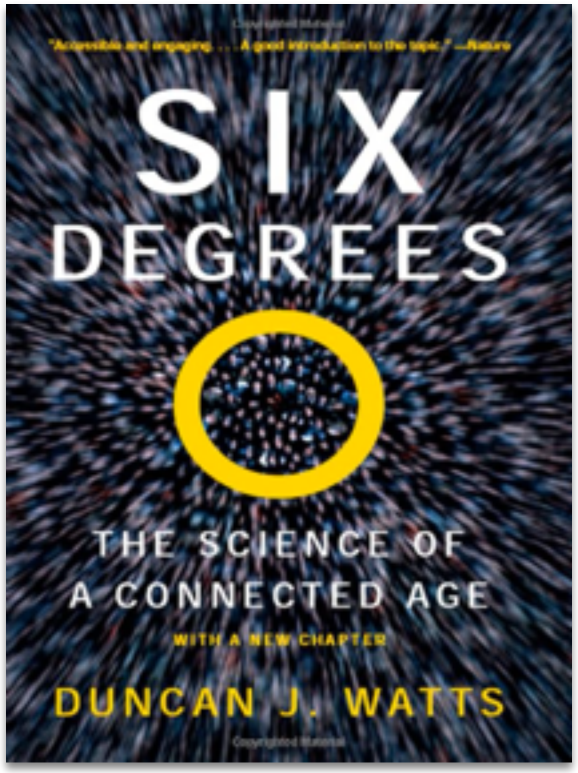
Materials

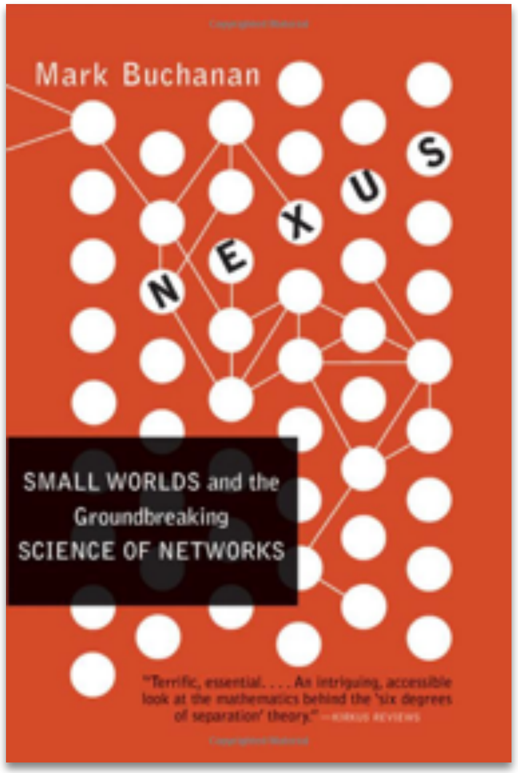
Journals

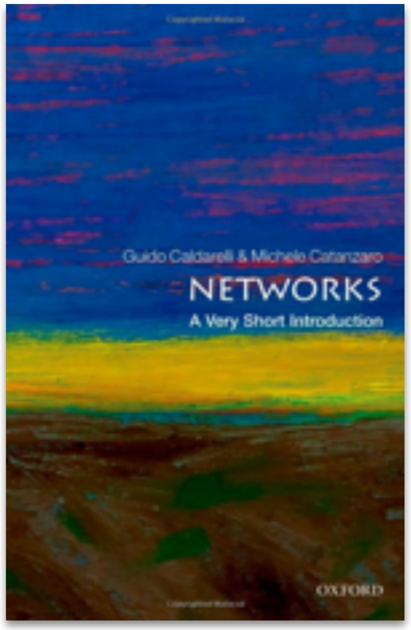


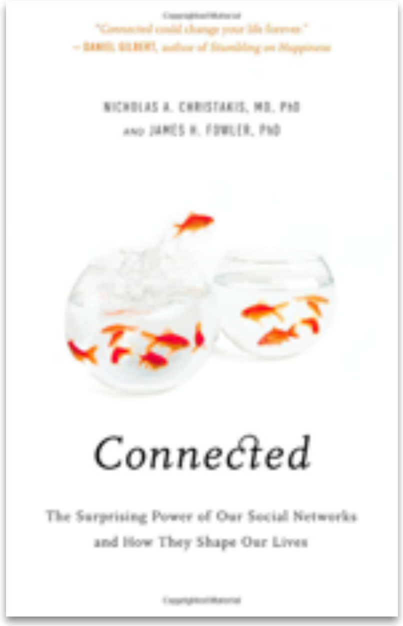
Materials

Pop-science books









Thank you
for you attention!