Timoteo Carletti

Epidemiology from mean field to networked models

naxys
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UNIVERSITÉ DE NAMUR

Who am I?

Doctorat
Feb 2000
U Florence et Paris

PostDoct
9/2000 – 2/2001
U Padoue

A.T.E.R.
3/2001 – 8/2001
U Paris XI

PostDoct
9/2001 – 2/2003
U Florence

PostDoct
3/2003 – 3/2005
SNS Pise

9/2005 – aujourd'hui
(Full) Professeur FUNDP/UNamur
Head "Equipe systèmes dynamiques"
Directeur naXys (2010-2014)

Visiting Professor
4/2005 – 12/2005
Venise ECLT
(EU FP6 PACE)
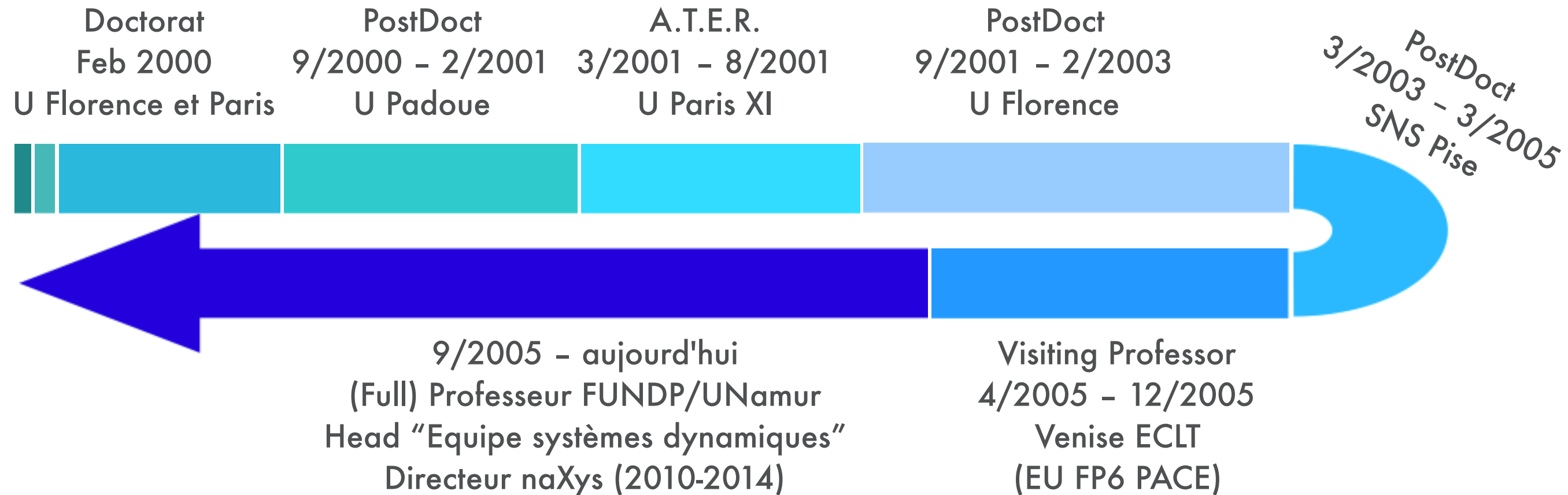
❑ PhD Thesis title:
"*Stability of orbits and Arithmetics for some discrete dynamical systems*"

❑ Most cited paper:
T.C., S. Marmi, *Linearization of Analytic and Non-Analytic Germs of Diffeomorphisms of (C,0)*, Bulletin SMF, **128**, (2000), pp. 69-85
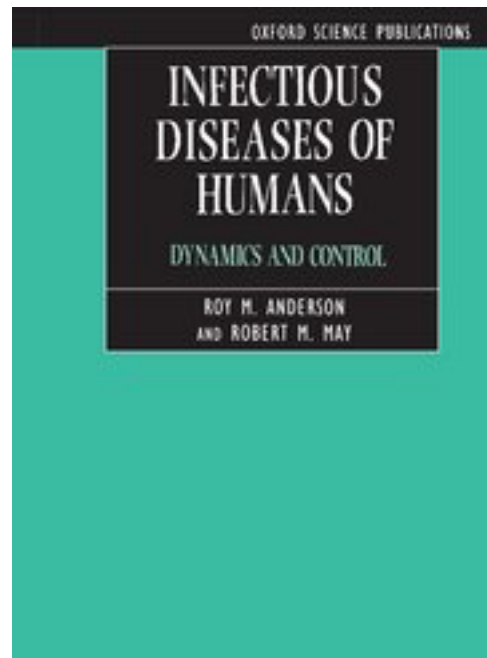
Word cloud of the titles of my papers

Outlook of the lecture

Epidemiology from mean field to networked models

OXFORD SCIENCE PUBLICATIONS

INFECTIOUS DISEASES OF HUMANS

DYNAMICS AND CONTROL

ROY M. ANDERSON AND ROBERT M. MAY
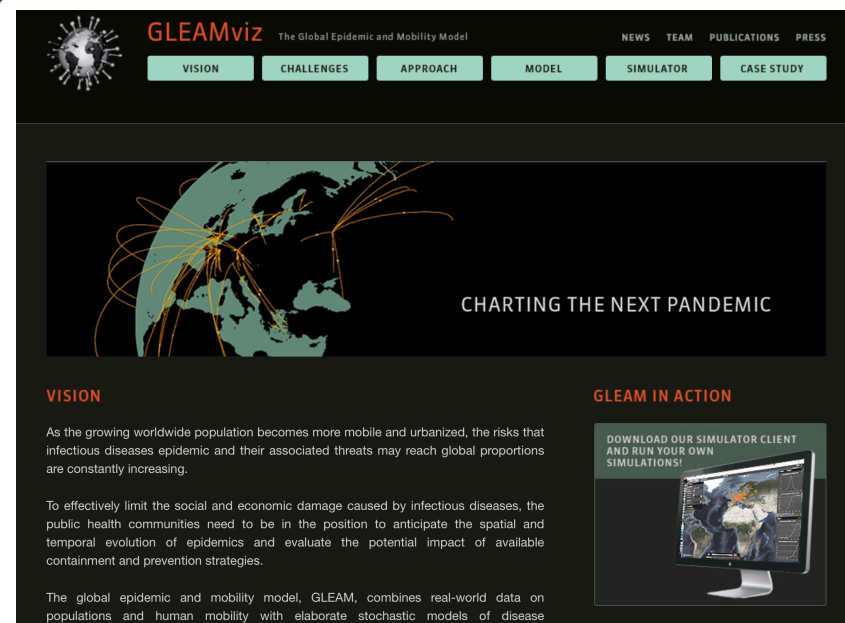
SIAM REVIEW
Vol. 42, No. 4, pp. 599–653

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The Mathematics of Infectious Diseases*

Herbert W. Hethcote[†]

Abstract. Many models for the spread of infectious diseases in populations have been analyzed mathematically and applied to specific diseases. Threshold theorems involving the basic reproduction number R_0, the contact number σ, and the replacement number R are reviewed for the classic SIR epidemic and endemic models. Similar results with new expressions for R_0 are obtained for MSEIR and SEIR endemic models with either continuous age or age groups. Values of R_0 and σ are estimated for various diseases including measles in Niger and pertussis in the United States. Previous models with age structure, heterogeneity, and spatial structure are surveyed.

Key words. thresholds, basic reproduction number, contact number, epidemiology, infectious diseases

GLEAMviz   The Global Epidemic and Mobility Model

NEWS   TEAM   PUBLICATIONS   PRESS

VISION   CHALLENGES   APPROACH   MODEL   SIMULATOR   CASE STUDY

CHARTING THE NEXT PANDEMIC

VISION

As the growing worldwide population becomes more mobile and urbanized, the risks that infectious diseases epidemic and their associated threats may reach global proportions are constantly increasing.

To effectively limit the social and economic damage caused by infectious diseases, the public health communities need to be in the position to anticipate the spatial and temporal evolution of epidemics and evaluate the potential impact of available containment and prevention strategies.

The global epidemic and mobility model, GLEAM, combines real-world data on populations and human mobility with elaborate stochastic models of disease

GLEAM IN ACTION

DOWNLOAD OUR SIMULATOR CLIENT AND RUN YOUR OWN SIMULATIONS!

EPIcx lab

RESEARCH   PEOPLE   PAPERS   NEWS   OUTREACH   MEDIA   TOOLS   CONTACTS

Emerging diseases

Sciences du 31/01/2015

Comprendre la propagation des épidémies - Corpus. Oct 2014.

Epidemies, la menace invisible - Arte. Sept 9, 2014.

Prévoir un épidémie - France 2. Jan 31, 2015.

Tout peut changer, France 3, ...

GLEaM results at Can...

Epidemie, la nouvelle menace - Encyclo. Feb 11, 2014.

Tout peut changer - France 3. Dec 9, 2013.

CTV Canada AM - Jan 30, 2007.

When does a disease turn epidemic? - Computing the epidemic threshold on time-evolving networks

Pacific Standard - "Fighting Epidemics With Math" May 7, 2015.
APS Physics highlight- "When does a disease turn Epidemic?" Apr 8, 2015.

ECONOMETRIC SOCIETY MONOGRAPHS

Complex Social Networks

Fernando Vega-Redondo

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The Structure and Dynamics of NETWORKS

MARK NEWMAN
ALBERT-LÁSZLÓ BARABÁSI
DUNCAN J. WATTS
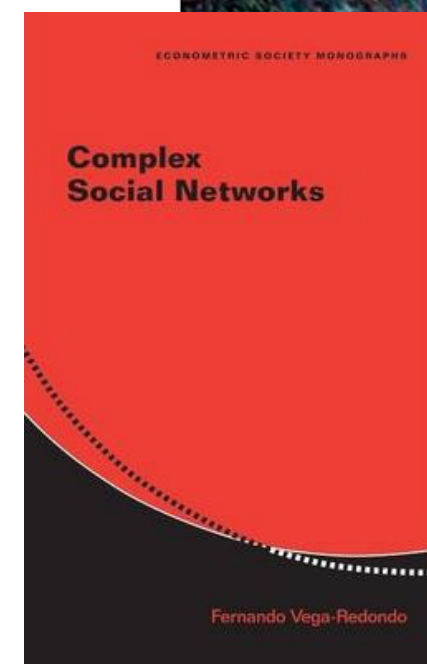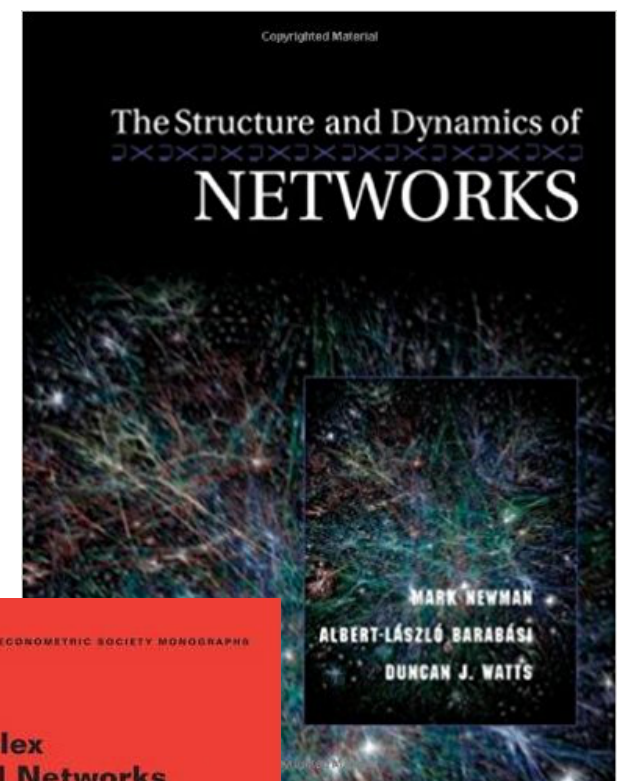
All models are wrong but some are useful

Box, George E. P. (1979), "Robustness in the strategy of scientific model building", in Launer, R. L.; Wilkinson, G. N., Robustness in Statistics, Academic Press, pp. 201–236.

What matters is the question you are interested in and the level of precision you want to achieve.

PART 1

Epidemic models

A (synthetic) experiment

Complexity Explorables

About   Explorables

Epidemonic

A simple model for contagion dynamics in a population

EXPLORABLES

Epidemonic

by Janina Schöneberger

3 October, 2017

Divide the population into **classes**, each class being characterized by one possible "state" of the illness.

All agents in the same class behave in the same way and interactions among agents belonging to different classes are proportional to their number (homogeneous mixing: mean field assumption).
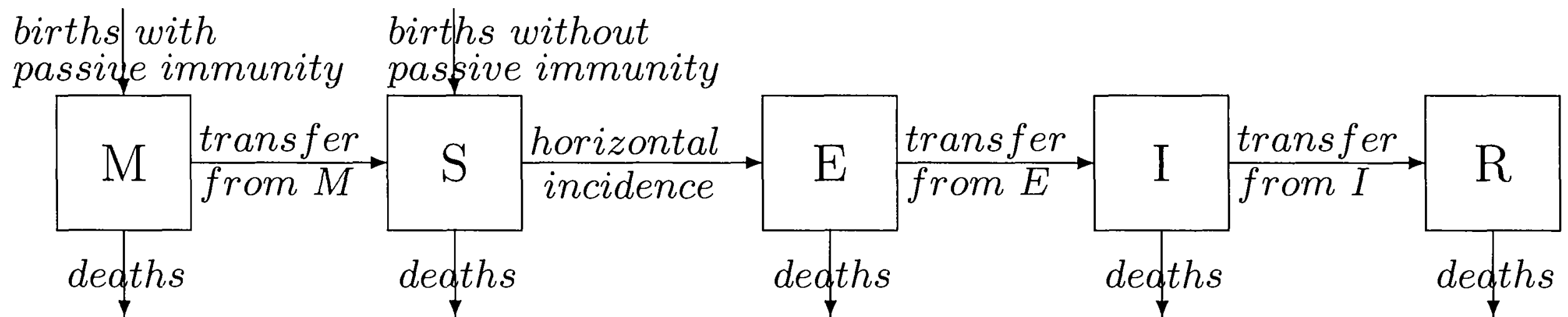
Fig. I   *The general transfer diagram for the MSEIR model with the passively immune class M, the susceptible class S, the exposed class E, the infective class I, and the recovered class R.*

Susceptible - Infected : SIS model

You get sick, then recover, but <u>without immunity</u>, e.g. the common cold.
Two classes: susceptible and infected persons.

Susceptible ⇨ Infected ⇨ Susceptible

▷ Susceptible people become <u>infected at rate a</u>

▷ Infected ones <u>recover at rate b</u>

Susceptible - Infected : SIS model

$$S \xrightarrow[b]{a} I$$
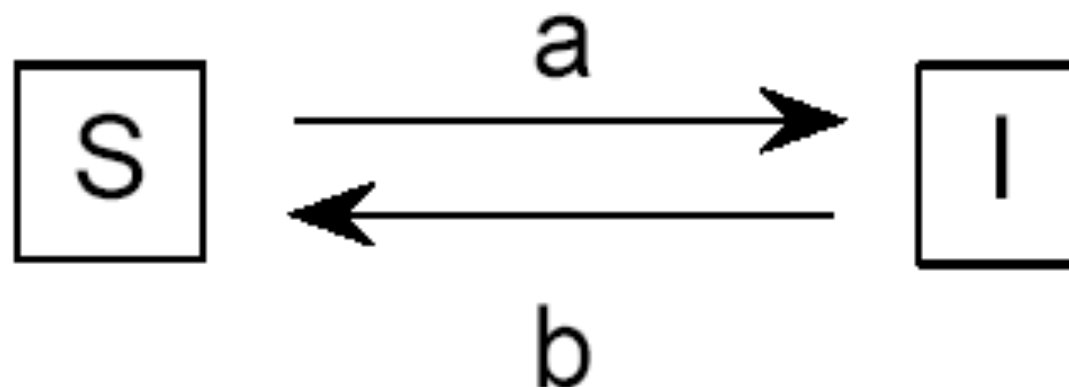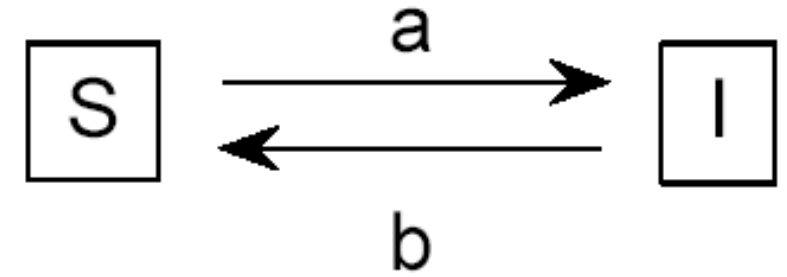
▷ S = number of susceptible persons

▷ I = number of infected persons

▷ Becoming infected depends on contact between Susceptible and Infected;

▷ Recovery is at a constant rate, proportional to number of Infected;

$$\begin{cases} \dfrac{dS}{dt} = bI - aSI \\ \dfrac{dI}{dt} = aSI - bI \end{cases}$$

Some computations and numerical simulations.

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Susceptible - Infected : SIS model

$$S \xrightarrow[\;\;b\;\;]{\;\;a\;\;} I$$
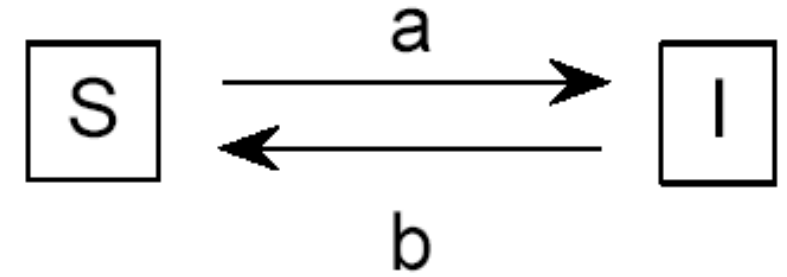
▷ S = fraction of susceptible persons

▷ I = fraction of infected persons

▷ Becoming infected depends on contact between Susceptible and Infected;

▷ Recovery is at a constant rate, proportional to number of Infected;

$$\begin{cases} \dfrac{dS}{dt} = bI - aSI \\ \dfrac{dI}{dt} = aSI - bI \end{cases}$$

Some computations and numerical simulations.

Basic Reproduction Number :
$$\mathcal{R}_0 = \frac{a}{b}$$

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Susceptible - Infected - Recovered : SIR model

You get sick, then recover, but <u>with immunity</u>. Three classes: susceptible, infected persons and recovered.

Susceptible ⇨ Infected ⇨ Recovered

▷ Susceptible people become <u>infected at rate a</u>

▷ Infected ones <u>recover at rate b</u>

Note that sometimes R stands for Removed (i.e. dead or immune, not able to diffuse the illness)

$$ \boxed{S} \xrightarrow{a} \boxed{I} \xrightarrow{b} \boxed{R} $$

Susceptible - Infected - Recovered : SIR model

▷ S = fraction of susceptible persons

$$S \xrightarrow{a} I \xrightarrow{b} R$$

▷ I = fraction of infected persons

▷ R= fraction of recovered persons

▷ Becoming infected depends on contact between Susceptible and Infected;

▷ Recovery is at a constant rate, proportional to number of Infected;

$$\begin{cases} \dfrac{dS}{dt} = -aSI \\ \dfrac{dI}{dt} = aSI - bI \\ \dfrac{dR}{dt} = bI \end{cases}$$

SIR : possible generalizations

▷ Vaccination: people can pass directly from S to R (constant rate c)
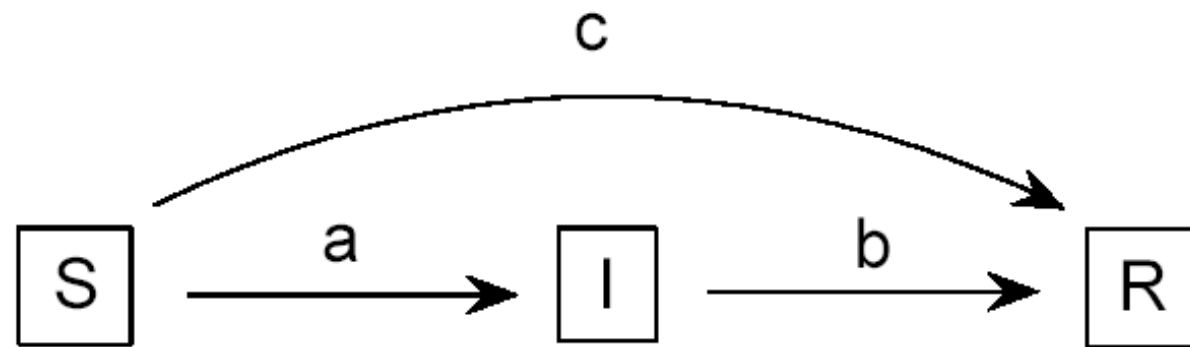
▷ Mutation: virus mutates and recovered persons become again susceptible;

▷ Time Delay: the infected persons need some time before to able to spread the infection;

▷ Consider natural births and deaths;

▷ Consider age groups

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PART 2

Network models

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Networks are everywhere

world flights map

social networks

SocioPatterns

Human Disease Network
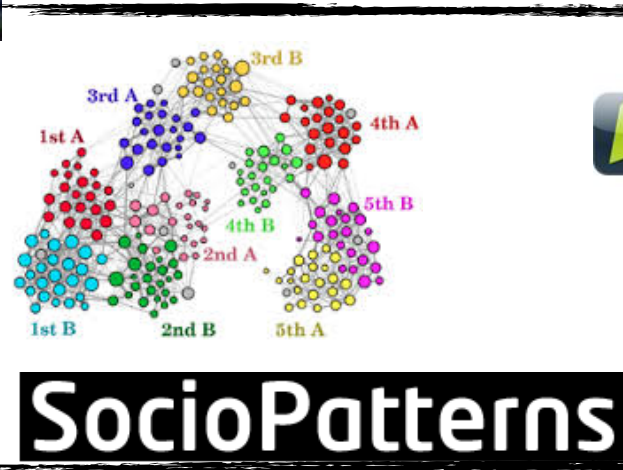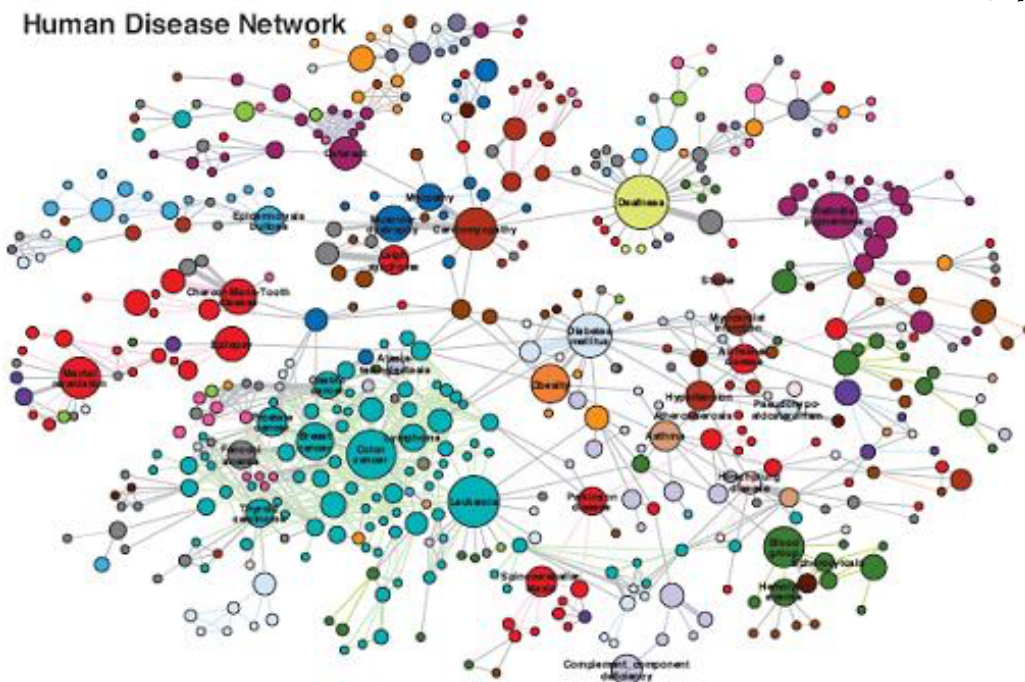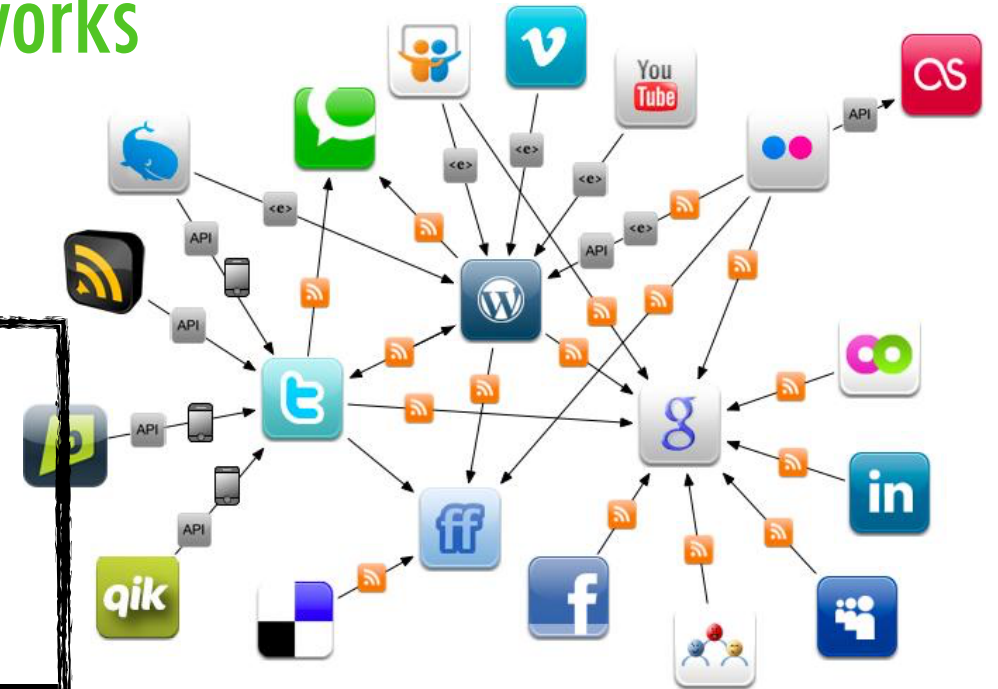
proteins networks
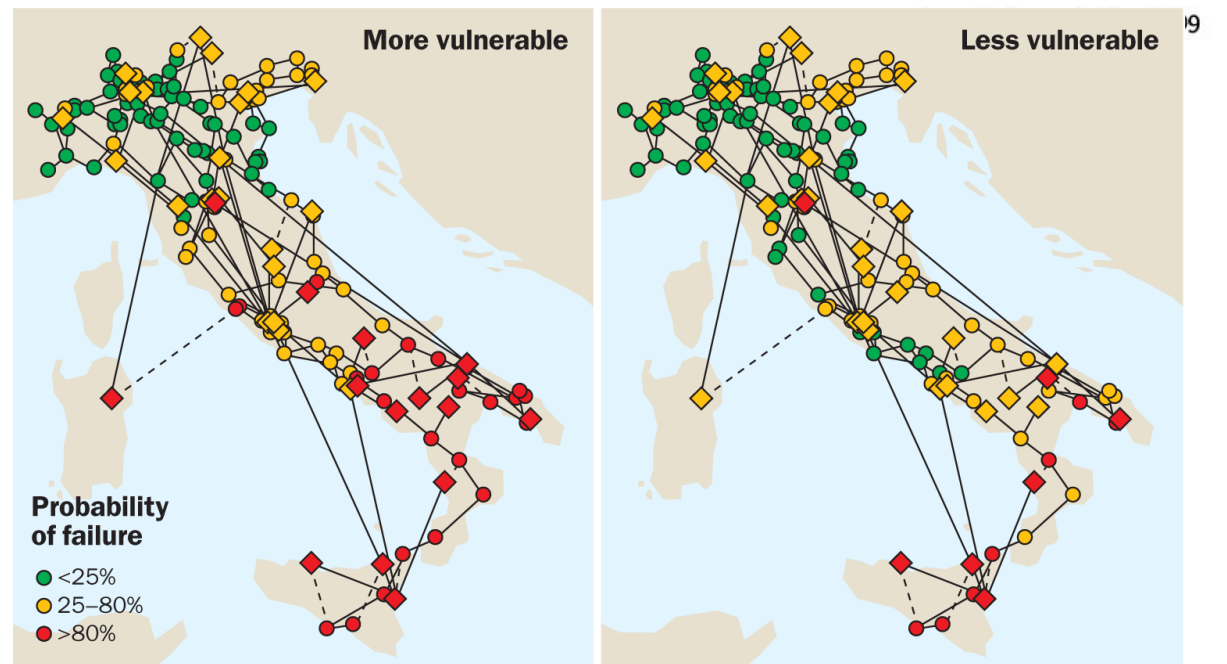
technological networks

Probability of failure
- <25%
- 25–80%
- >80%

More vulnerable

Less vulnerable

A network is a set of nodes connected by links (edges)
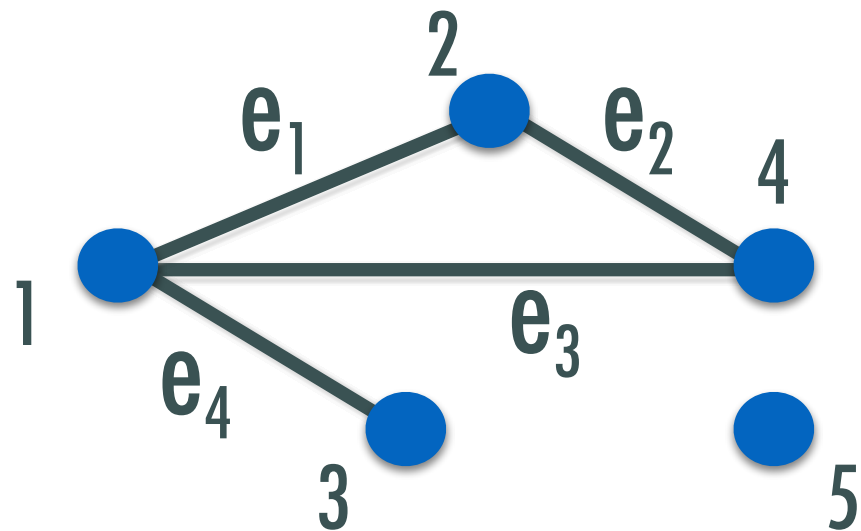
Ex.: 5 nodes and 4 edges (undirected)

Adjacency matrix

$$A_{ij} = \begin{cases} 1 & \text{if nodes } i \text{ and } j \text{ are linked} \\ 0 & \text{otherwise} \end{cases}$$

The number of links entering (going out) from each node is called <u>in-degree (out-degree)</u>

Ex.: "degree node 1" = 3
"degree nodes 2 & 4" = 2
"degree node 3" = 1
"degree node 5" = 0

A network is said to be <u>complex</u> if the <u>degree distribution is not trivial</u>, i.e. not constant (lattice) nor Poissonian (random, Erdős-Rényi)

Models of Networks

Erdős-Rényi model

Erdős, P.; Rényi, A. (1959). "On Random Graphs",
Publicationes Mathematicae, **6**, 290–297

> Given n nodes, consider all the possible couples (i,j) and with some probability p add the link ij.

The probability to have a node with degree k is given by:

$$P(\deg(v) = k) = \binom{n-1}{k} p^k (1-p)^{n-1-k}$$

The <u>average degree</u> is given by $\langle k \rangle = np$

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Watts-Strogatz (small world) model

Watts, D. J.; Strogatz, S. H. (1998). "Collective dynamics of 'small-world' networks", *Nature*, **393** (6684): 440–442.

Given n nodes arranged into a regular ring, each with 2m neighbours, consider all the possible couples (i,j) and with some probability p rewire the link ij (i.e. delete ij and make a new link ik)
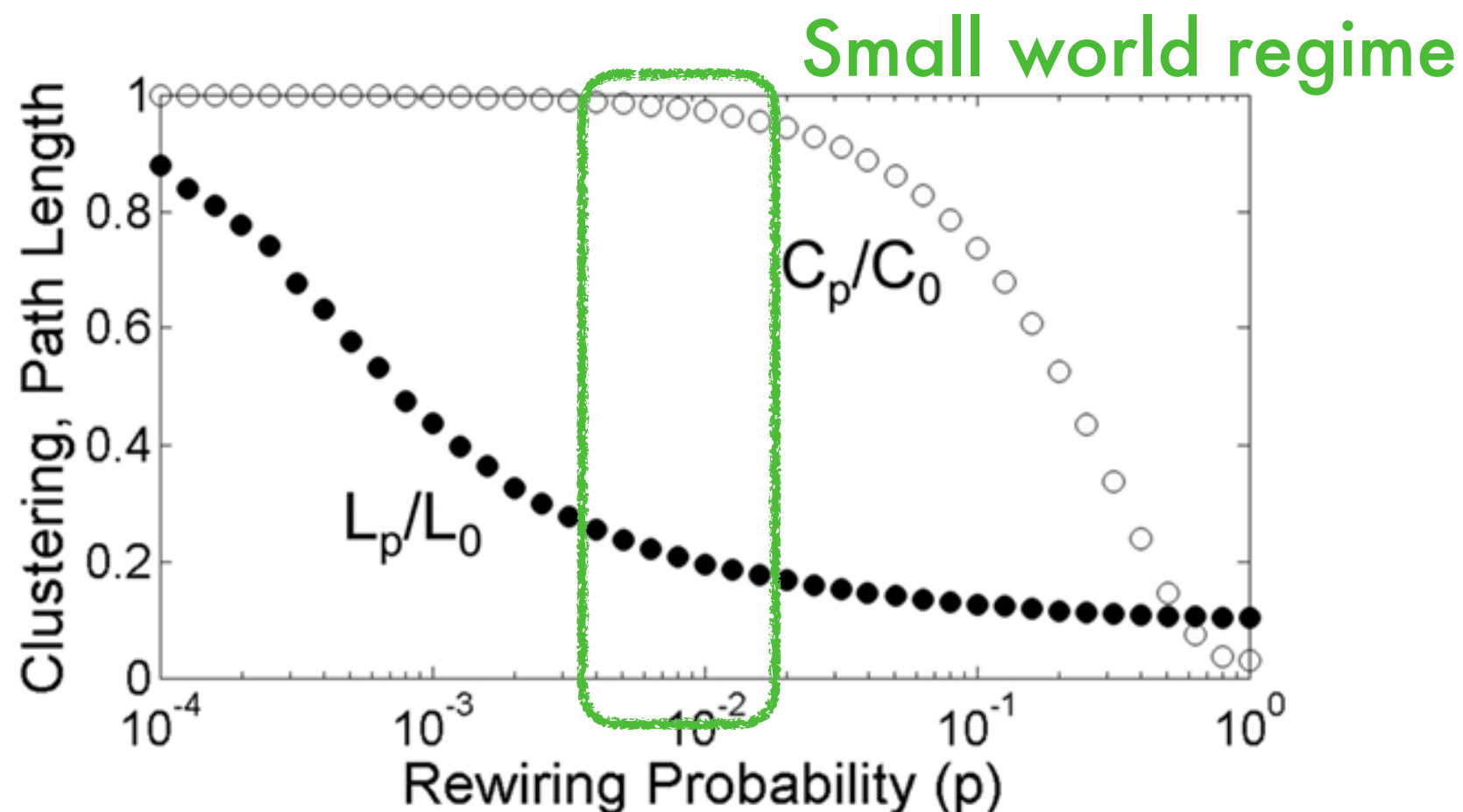

Regular   Small-world   Random

$p = 0$ → $p = 1$

Increasing randomness
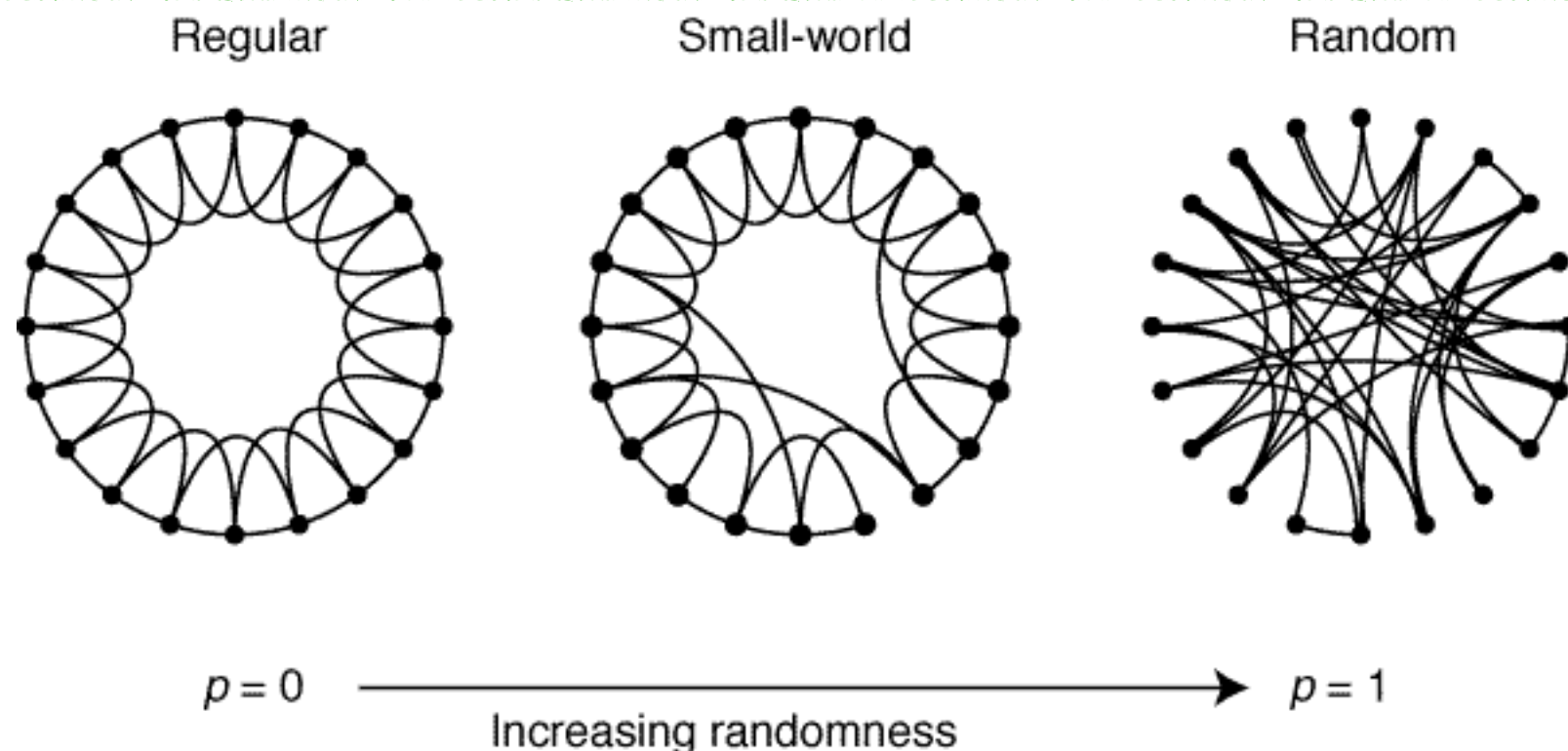
Watts-Strogatz (small world) model

Path among two nodes: minimum number of "hops" to pass from one node to the other one.

Clustering coefficient is a measure of the degree to which nodes in a graph tend to cluster together (triangles).

Small world regime

Barabási-Albert (preferential attachment) model
Barabási, A.; Albert, R. (1999), "Emergence of scaling in random networks", *Science*, **286** (5439): 509–512.

Each time step a new node enters into the system and it makes a new link to an already existing node with a probability that is proportional to the number of links that the existing nodes already have.

Preferential attachment means that the more connected a node is, the more likely it is to receive new links. Nodes with higher degree have stronger ability to grab links added to the network.

Barabási-Albert (preferential attachment) model

The probability to have a node with degree k is given by:

$$P(\deg(v) = k) \sim \frac{1}{k^3}$$
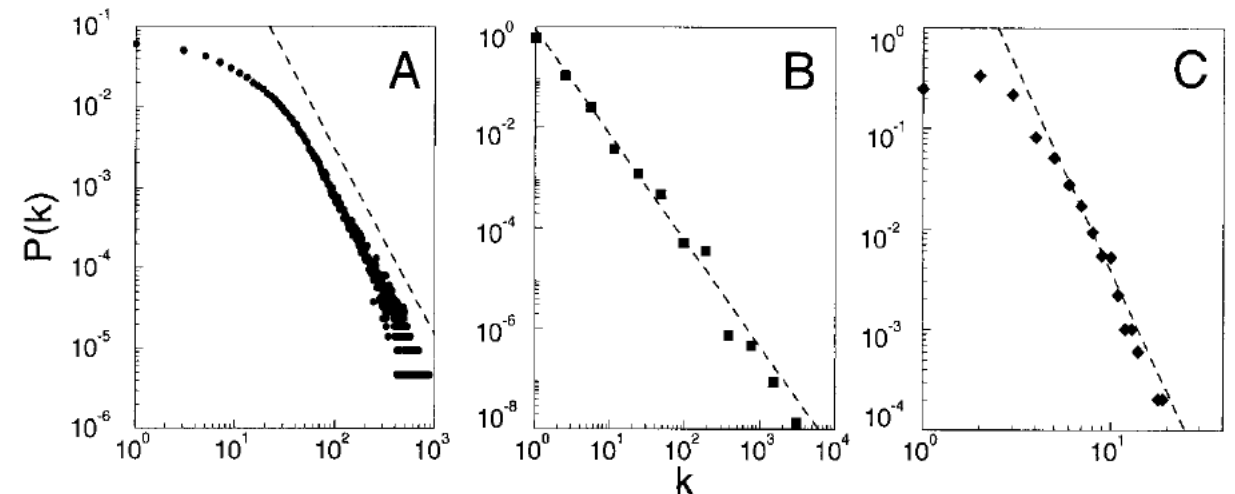
Fig. 1. The distribution function of connectivities for various large networks. **(A)** Actor collaboration graph with $N = 212,250$ vertices and average connectivity $\langle k \rangle = 28.78$. **(B)** WWW, $N = 325,729$, $\langle k \rangle = 5.46$ (6). **(C)** Power grid data, $N = 4941$, $\langle k \rangle = 2.67$. The dashed lines have slopes **(A)** $\gamma_{actor} = 2.3$, **(B)** $\gamma_{www} = 2.1$ and **(C)** $\gamma_{power} = 4$.

Scale Free networks. The degree distribution is "broad" and can be described by the functions :

$$P(\deg(v) = k) \sim \frac{1}{k^\gamma}$$

PART 3

Epidemics & networks

1 node = 1 person

1 node = 1 person

SIS model on **homogeneous network** (all nodes have - almost - the same degree, i.e. all persons have - almost - the same number of neighbors).

$$\begin{cases} \dfrac{dS}{dt} & = bI - aSI \\ \dfrac{dI}{dt} & = aSI - bI \end{cases}$$

$$\begin{cases} S & = 1 - I \\ \dfrac{dI}{dt} & = a\langle k\rangle(1 - I)I - bI \end{cases}$$

former model (no space)

two competing times scales

Epidemic & networks: homogeneous mean-field

1 node = 1 person

SIS model on **homogeneous network** (all nodes have - almost - the same degree, i.e. all persons have - almost - the same number of neighbors).

For short times (I(t) is assumed to be small):

$$\frac{dI}{dt} \sim (a\langle k \rangle - b)I$$

Epidemic threshold:

$a\langle k \rangle / b > 1$ (exp) growth

$a\langle k \rangle / b < 1$ (exp) decrease

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1 node = 1 person

SIS model on **homogeneous network** (all nodes have - almost - the same degree, i.e. all persons have - almost - the same number of neighbors).

For long times ($\frac{dI}{dt} = 0$) :

$$a\langle k\rangle(1 - I_\infty)I_\infty = bI_\infty$$

Epidemic threshold:

$$a\langle k\rangle/b > 1 \qquad I_\infty = 1 - b/(a\langle k\rangle)$$

$$a\langle k\rangle/b < 1 \qquad I_\infty = 0$$

1 node = 1 person

SIS model on **homogeneous network** (all nodes have - almost - the same degree, i.e. all persons have - almost - the same number of neighbors).

I_∞

Absorbing phase

Virus death

Active phase

Finite prevalence

$\lambda_c = \langle k \rangle^{-1}$

$\lambda = a/b$
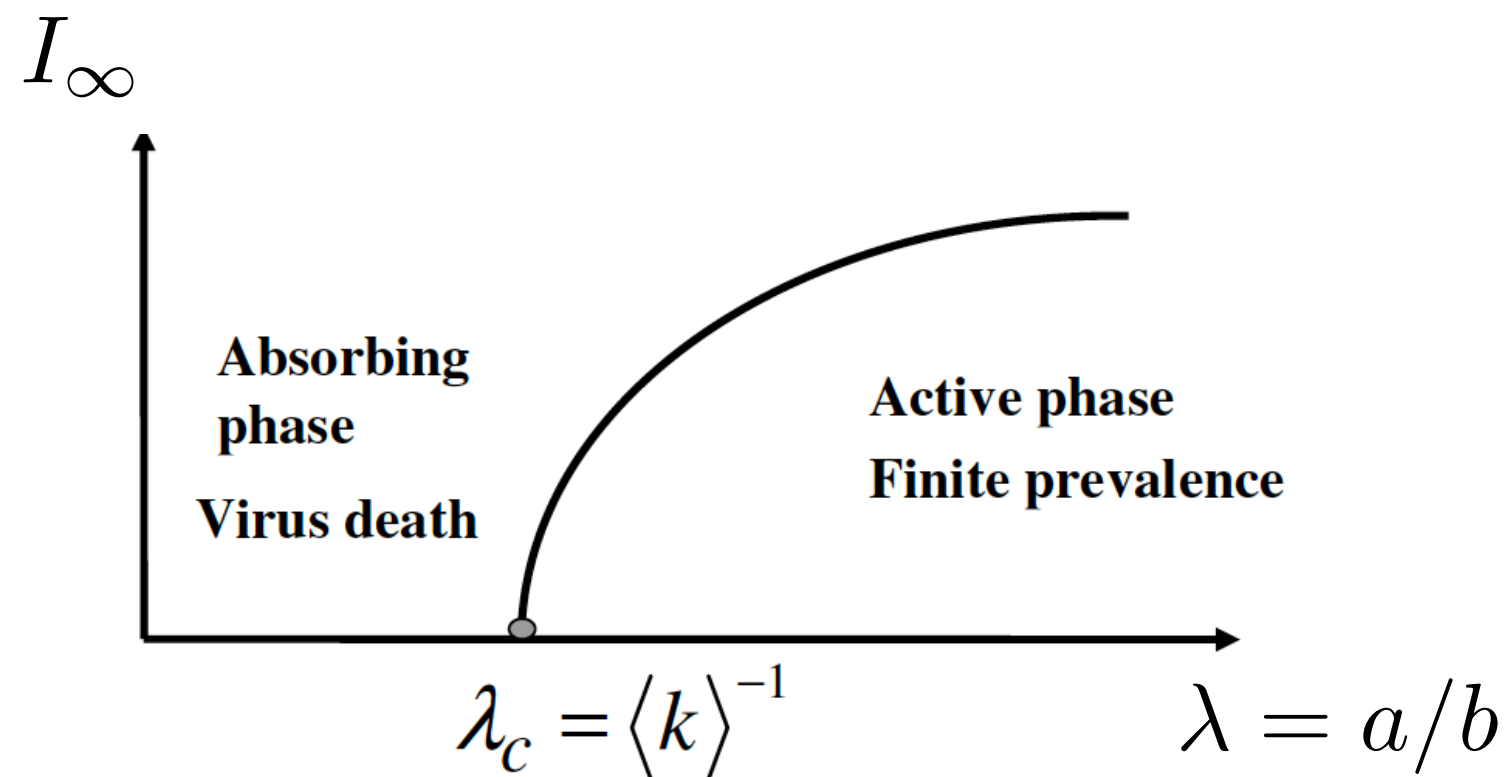
All persons with the same degree behave in the same way. Classes of state and degree.

All persons with the same degree behave in the same way. Classes of state and degree.

The number of neighbors can vary a lot $\langle k^2 \rangle \gg \langle k \rangle$

Heterogeneous mean-field

$$x_k = X_k / N_k$$

fraction of person in the state X
(*x = s, i* or *r*) having degree k

$$x = \sum_k x_k P(k)$$

total fraction of person in the state X

$P(k)$ Probability to have degree k

All persons with the same
degree behave in the same way.
Classes of state and degree.

SIS heterogeneous mean-field model

$$\frac{di_k}{dt} = ak(1 - i_k)\Theta_k - bi_k \qquad s_k = 1 - i_k$$

$$\Theta_k = \sum_{k'} P(k'|k)i_{k'}$$

Probability that a generic link (with deg k)
points to an infected node (with deg k')

$$P(k'|k)$$

Probability that a link originated in a node
with connectivity k points to a node with connectivity k'

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All persons with the same degree behave in the same way. Classes of state and degree.

SIS heterogeneous mean-field model

$$\frac{d}{dt}\Theta_k = \sum_{k'} P(k'|k)\frac{di_{k'}}{dt} = \sum_{k'} P(k'|k)\left[ak'(1 - i_{k'})\Theta_{k'} - bi_{k'}\right]$$

For short times (i_k is assumed to be small and thus $i_k\Theta_k \ll 1$)

$$\frac{d}{dt}\Theta_k \sim \sum_{k'} P(k'|k)\left[ak'\Theta_{k'} - bi_{k'}\right] = a\sum_{k'} P(k'|k)k'\Theta_{k'} - b\Theta_k$$

All persons with the same degree behave in the same way. Classes of state and degree.

SIS heterogeneous mean-field model

for uncorrelated networks $\quad P(k'|k) = \dfrac{k'}{\langle k \rangle} P(k')$

$$\frac{d}{dt}\Theta_k \sim a \sum_{k'} P(k'|k)k'\Theta_{k'} - b\Theta_k$$

$$\Theta_k = \sum_{k'} \frac{k'}{\langle k \rangle} P(k')i_{k'} \equiv \Theta$$

$$\frac{d\Theta}{dt} = \left(a\frac{\langle k^2 \rangle}{\langle k \rangle} - b \right)\Theta$$

All persons with the same degree behave in the same way. Classes of state and degree.

SIS heterogeneous mean-field model

For long times and uncorrelated networks:

$$\frac{di_k}{dt} = ak(1 - i_k)\Theta - bi_k = 0 \implies i_k^\infty = \frac{ak\Theta^\infty}{b + ak\Theta^\infty}$$

$$\Theta^\infty = \sum_{k'} \frac{k'}{\langle k \rangle} P(k') i_{k'}^\infty$$

Implicit equation for Θ^∞

$$\Theta^\infty = \frac{1}{\langle k \rangle} \sum_k \frac{ak^2\Theta^\infty}{ak\Theta^\infty + b} P(k)$$

All persons with the same degree behave in the same way. Classes of state and degree.

SIS heterogeneous mean-field model

Epidemic threshold (uncorrelated networks)

$$\frac{a}{b} > \frac{\langle k \rangle}{\langle k^2 \rangle}$$

For scale free networks

$$\langle k^2 \rangle \gg 1$$
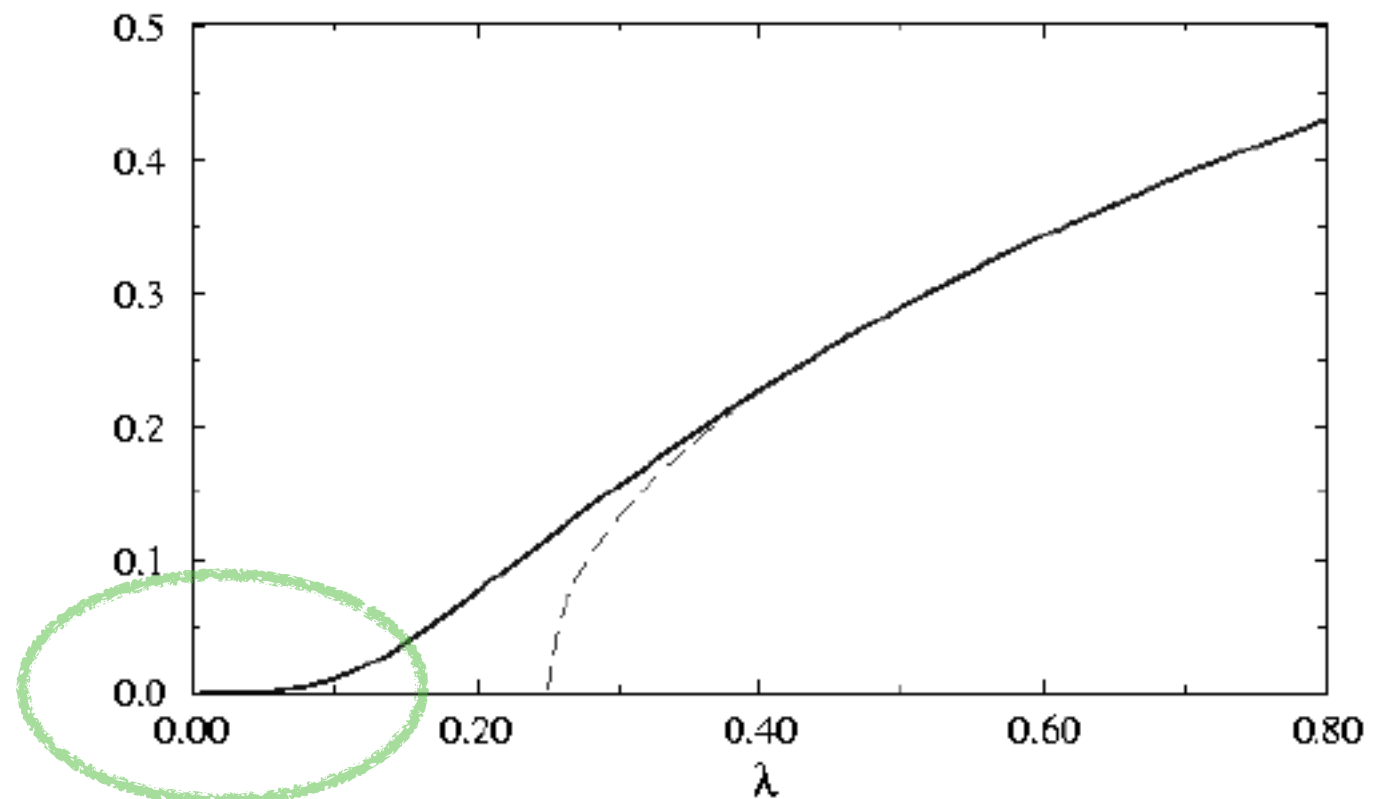
Epidemic & networks

1 node = 1 city/country

metapopulation model
-- macro scale

-- micro scale
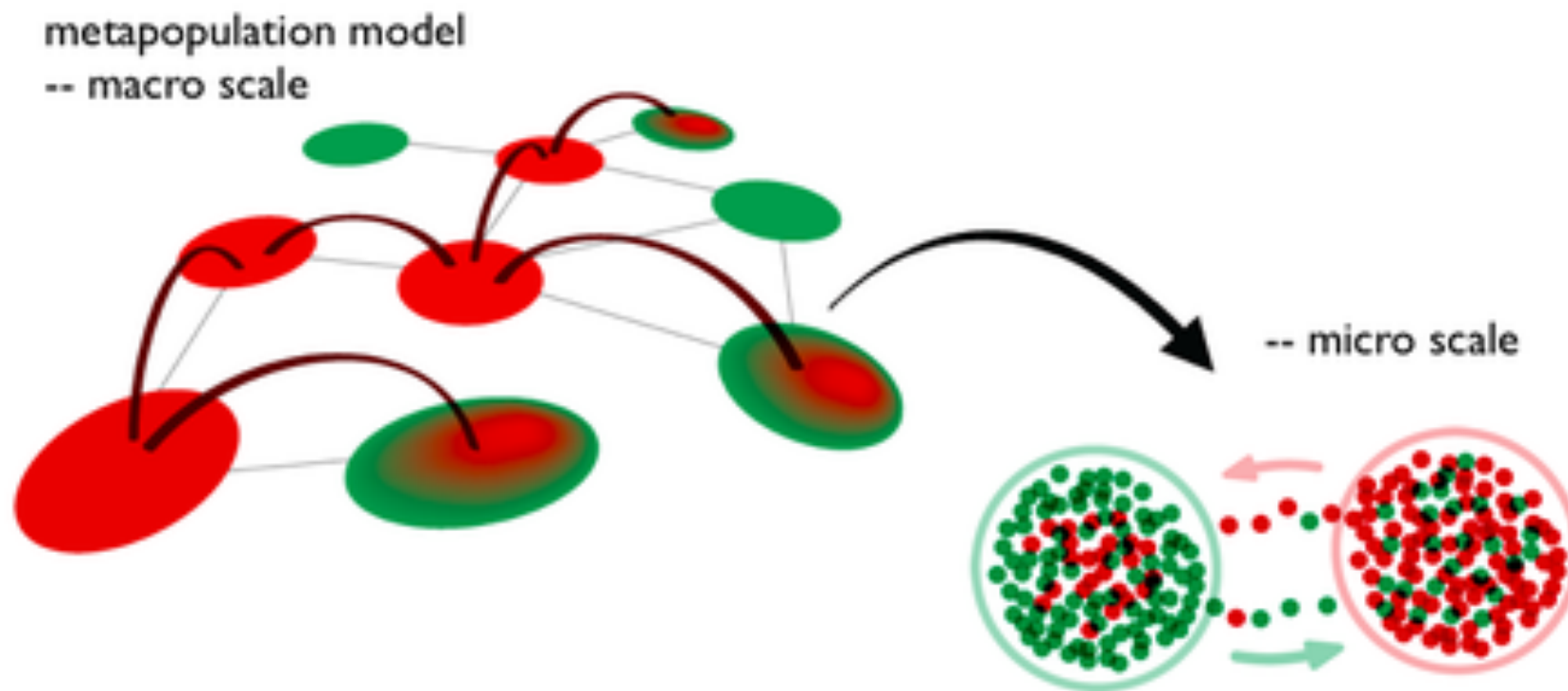
Metapopulation models
e.g. in the framework of ecology:
May R., *Will a large complex system be stable?*
Nature, 238, pp. 413, (1972)

Interactions occur at each node. Diffusion occurs across edges.

interaction term:

$$\begin{cases} \dfrac{dS_j}{dt} & = -aS_jI_j \\ \dfrac{dI_j}{dt} & = aS_jI_j - bI_j \\ \dfrac{dR_j}{dt} & = bI_j \end{cases}$$

At each node j=1,...,n, "species" S, I and R <u>interact</u> through the SIR model depending on the <u>quantities available at the j-th node</u> (metapopulation assumption)

We assume the parameters to be the same for all nodes.

Diffusion term:

Diffusive transport of species into a certain node i is given by the sum of <u>incoming fluxes</u> to node <u>i</u> from other <u>connected nodes j</u>, fluxes are proportional to the concentration difference between the nodes (Fick's law).
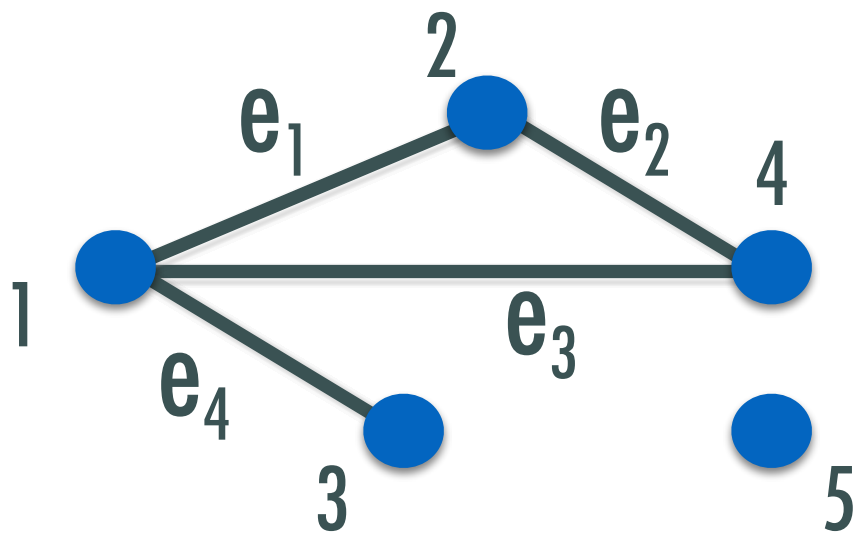
Ex.: consider the amount of u in node 1,
u can enter from 2, 3 and 4
u can leave 1 to go to 2, 3 and 4

$$u_2 + u_3 + u_4 - 3u_1 = \sum_j A_{1j} u_j - k_1 u_1 = \sum_j \left(A_{1j} - \delta_{1j} k_j \right) u_j := \sum_j L_{1j} u_j$$

## L is called Laplacian matrix of the network

The model:

$$
\begin{cases}
\frac{dS_j}{dt} & = -aS_j I_j + D_s \sum_{k=1}^n L_{jk} S_k \\
\frac{dI_j}{dt} & = aS_j I_j - bI_j + D_i \sum_{k=1}^n L_{jk} I_k \\
\frac{dR_j}{dt} & = bI_j + D_r \sum_{k=1}^n L_{jk} R_k
\end{cases}
$$

D_s, D_i and D_r are the diffusion coefficients of S, I and R, i.e. capability to move

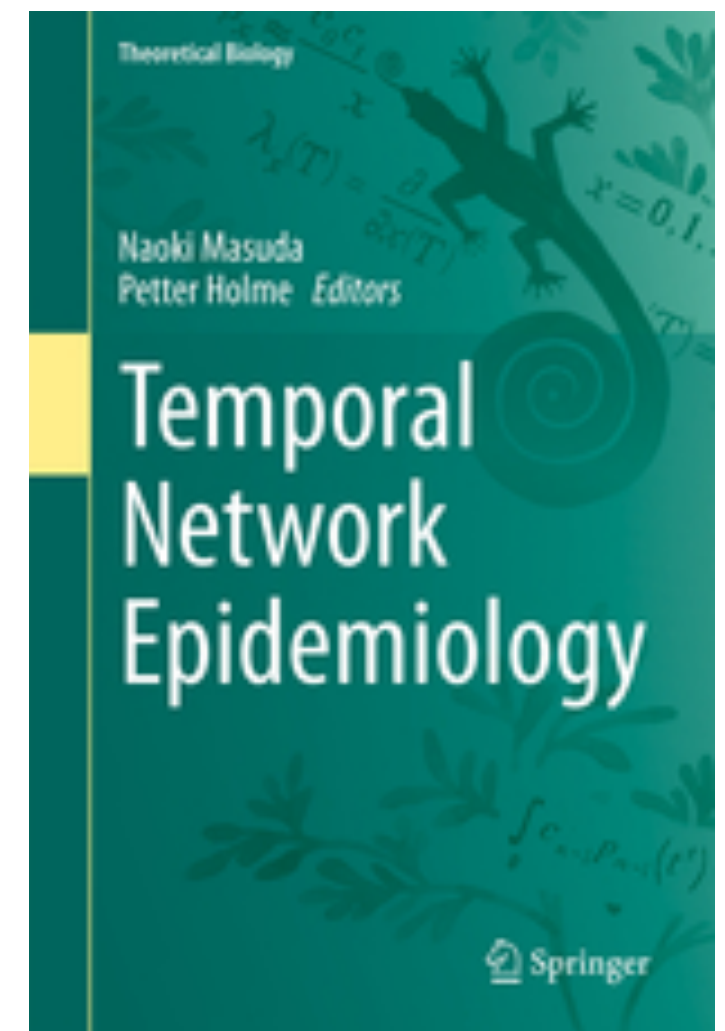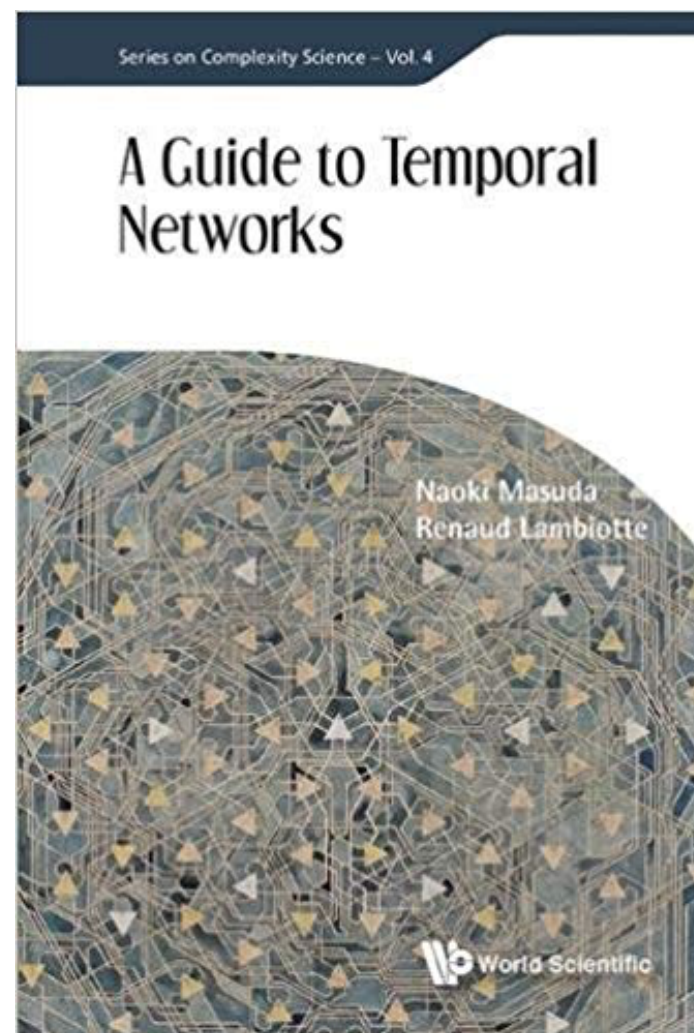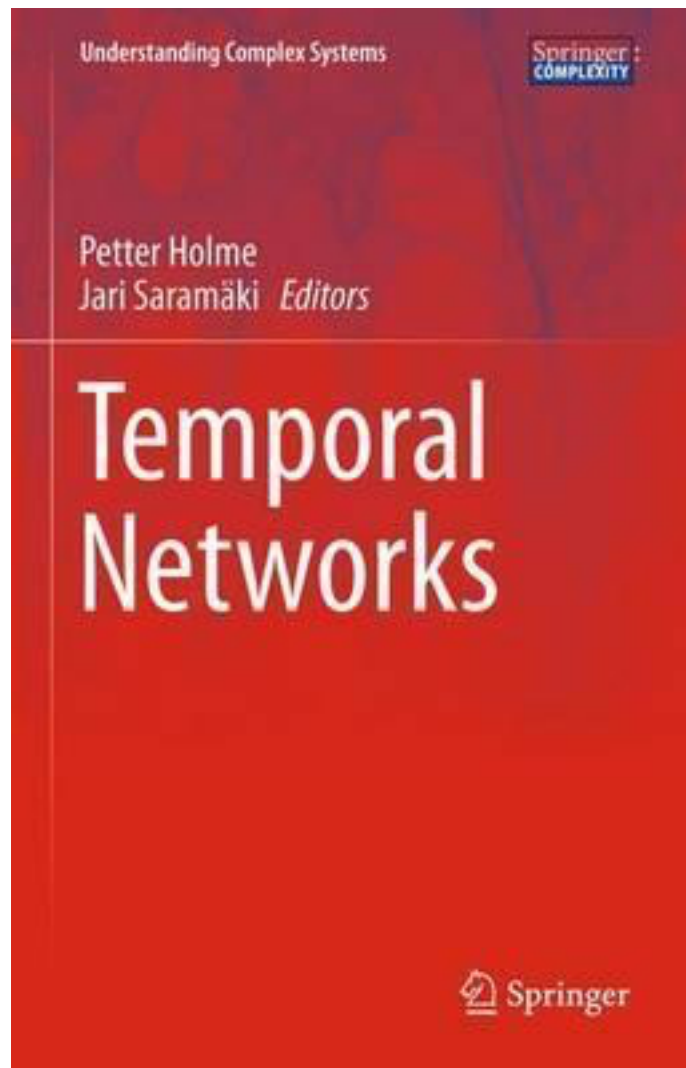# Systems composed by layers of networks: **Multiplexes**

Social networks

layers=different social networks

nodes=same agent in each SN

(c)

(a)

(b)

Bus

Metro

Rail
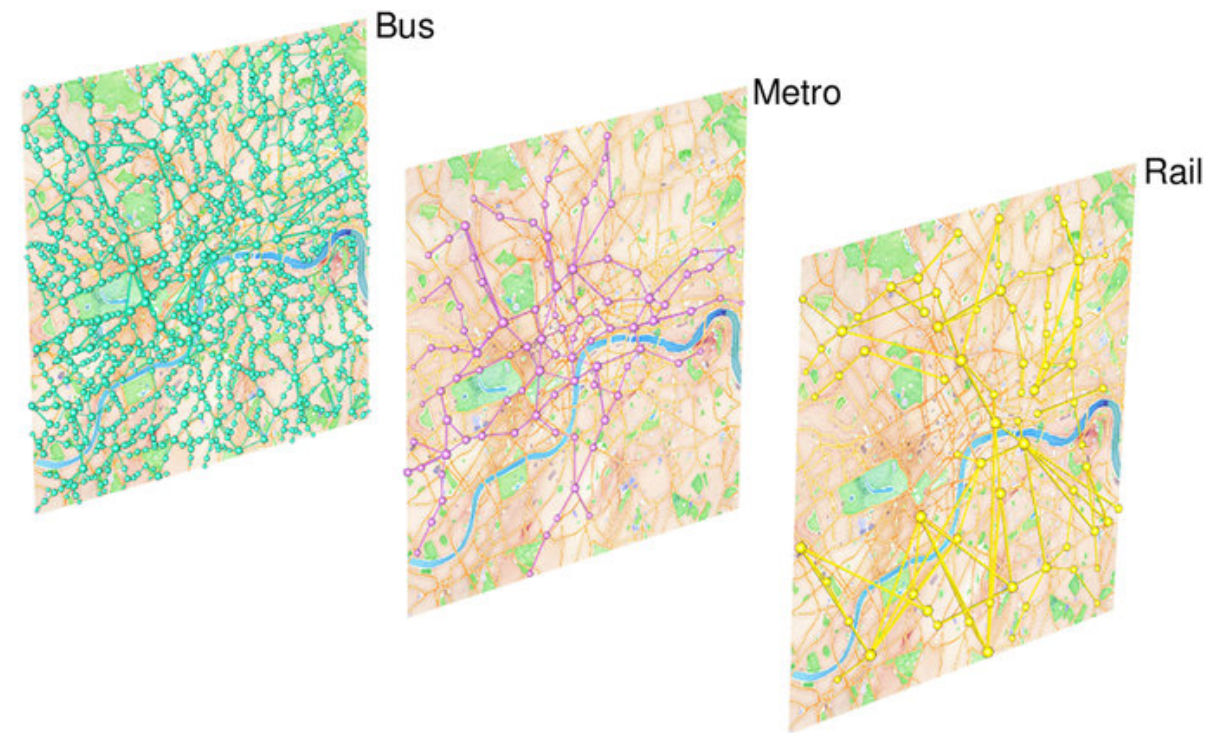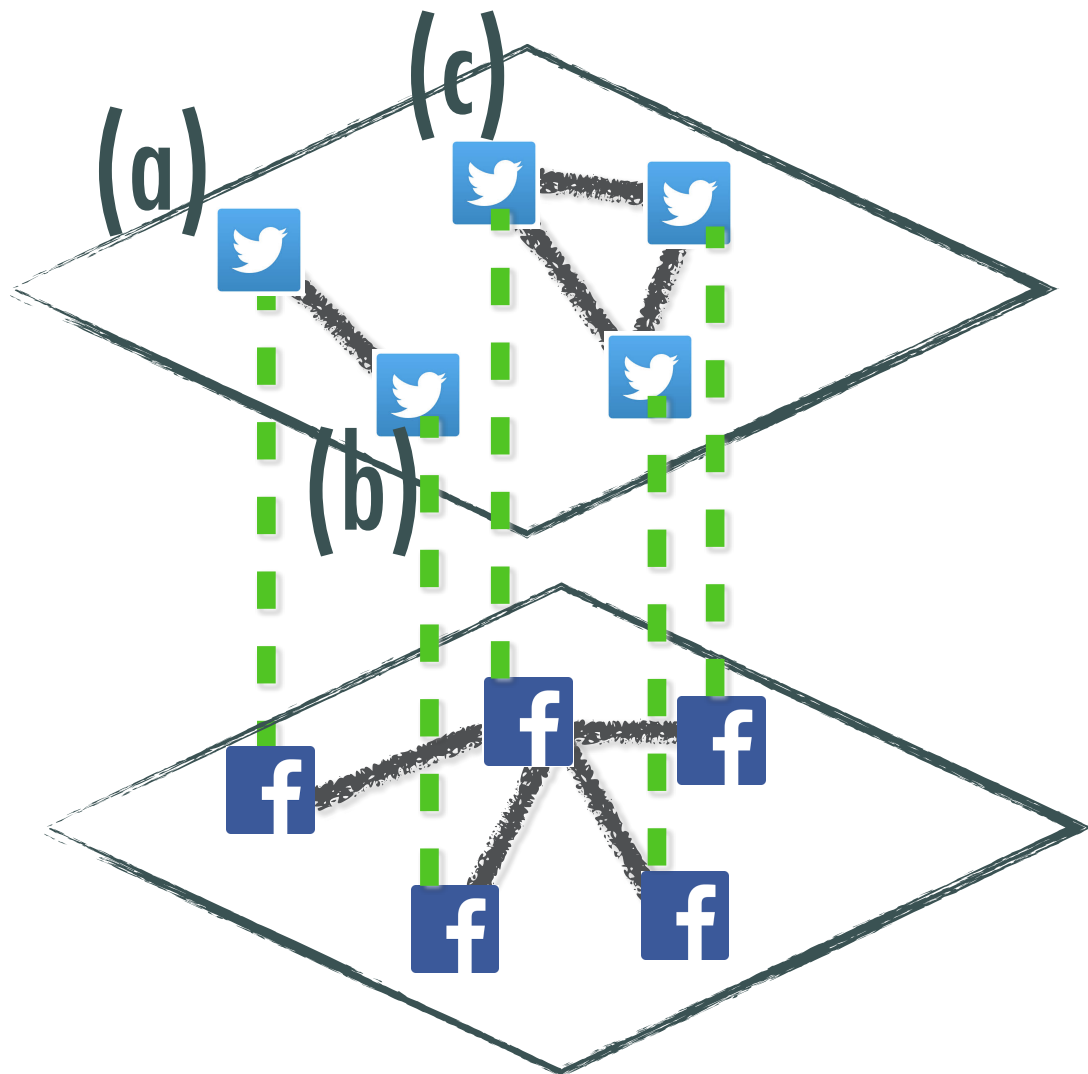
Transportation networks

layers=different modalities

nodes=same spatial location

Time varying networks (temporal networks)
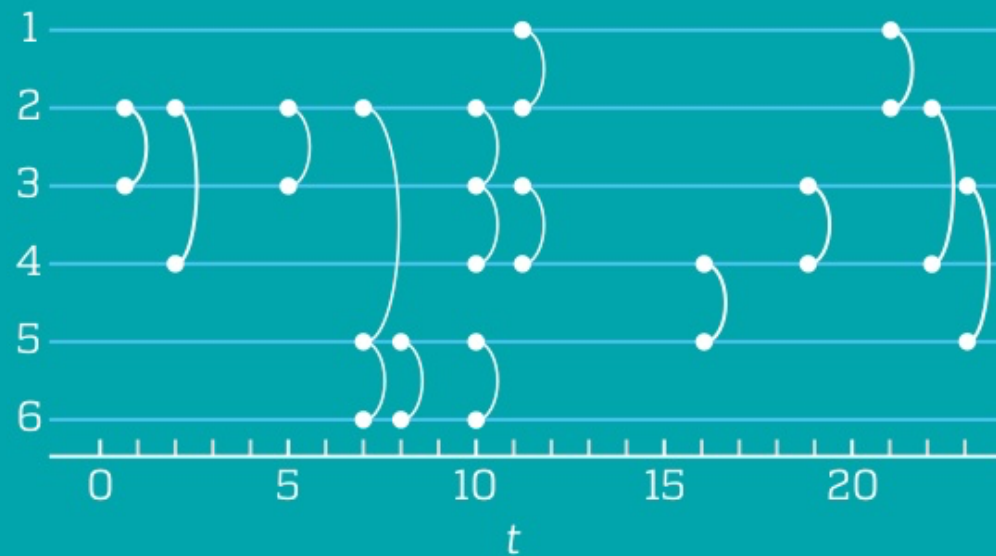
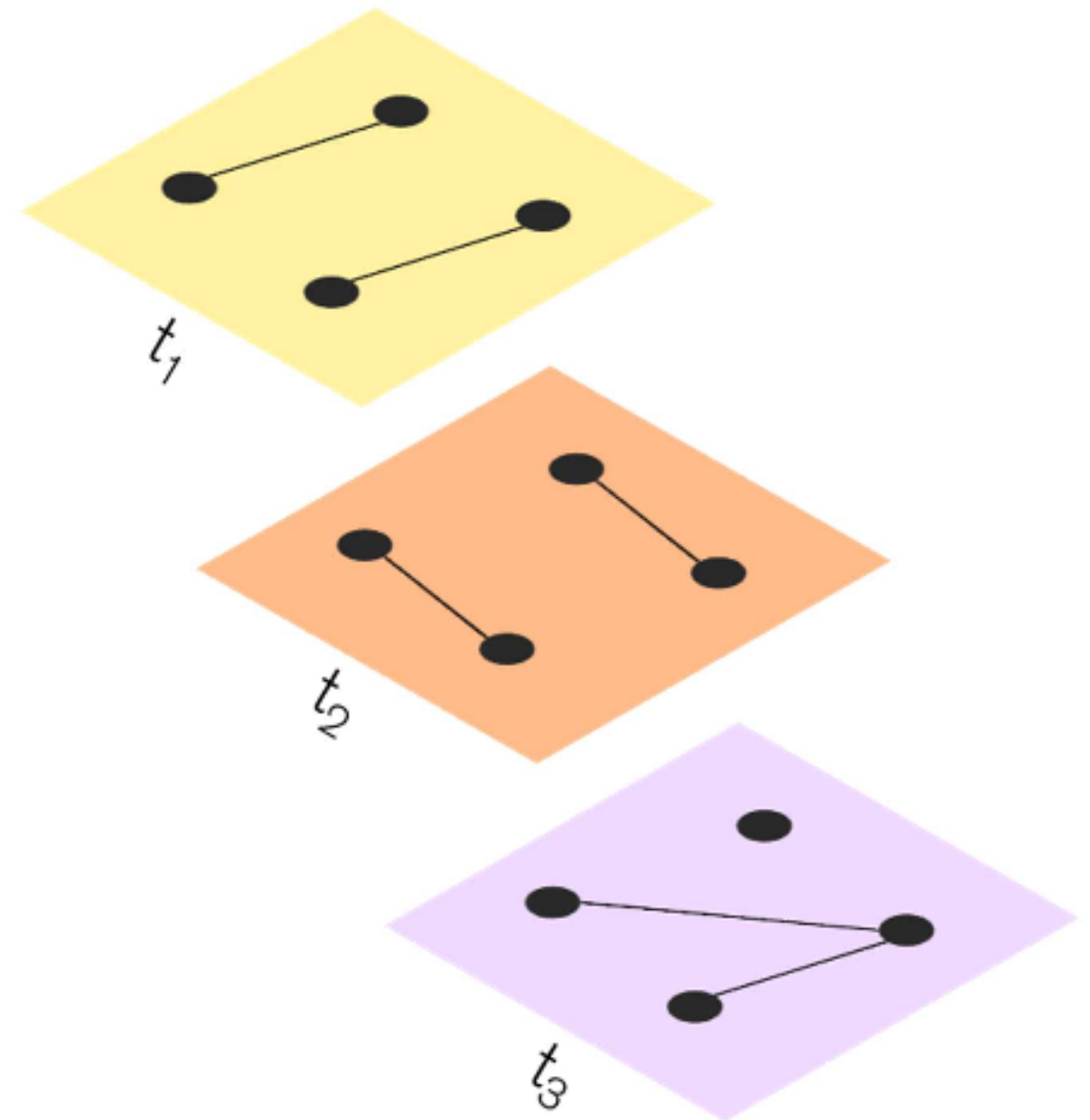# Epidemics on contact (social) networks

Theory of Turing Patterns on Time Varying Networks

Julien Petit,[1,2] Ben Lauwens,[2] Duccio Fanelli,[3,4] and Timoteo Carletti[1,*]

[1]naXys, Namur Institute for Complex Systems, University of Namur, B5000 Namur, Belgium
[2]Department of Mathematics, Royal Military Academy, B1000 Brussels, Belgium
[3]Dipartimento di Fisica e Astronomia and CSDC, Università degli Studi di Firenze, 50019 Sesto Fiorentino, Italy
[4]INFN Sezione di Firenze, 50019 Sesto Fiorentino, Italy
(Received 22 May 2017; published 4 October 2017)

The process of pattern formation for a multispecies model anchored on a time varying network is studied. A nonhomogeneous perturbation superposed to an homogeneous stable fixed point can be amplified following the Turing mechanism of instability, solely instigated by the network dynamics. By properly tuning the frequency of the imposed network evolution, one can make the examined system behave as its averaged counterpart, over a finite time window. This is the key observation to derive a closed analytical prediction for the onset of the instability in the time dependent framework. Continuously and piecewise constant periodic time varying networks are analyzed, setting the framework for the proposed approach. The extension to nonperiodic settings is also discussed.

Concurrency-Induced Transitions in Epidemic Dynamics on Temporal Networks

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Social contact networks underlying epidemic processes in humans and animals are highly dynamic. The spreading of infections on such temporal networks can differ dramatically from spreading on static networks. We theoretically investigate the effects of concurrency, the number of neighbors that a node has at a given time point, on the epidemic threshold in the stochastic susceptible-infected-susceptible dynamics on temporal network models. We show that network dynamics can suppress epidemics (i.e., yield a higher epidemic threshold) when the node's concurrency is low, but can also enhance epidemics when the concurrency is high. We analytically determine different phases of this concurrency-induced transition, and confirm our results with numerical simulations.

Contact-based model for epidemic spreading on temporal networks

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We present a contact-based model to study the spreading of epidemics by means of extending the dynamic message passing approach to temporal networks. The shift in perspective from node- to edge-centric quantities allows to accurately model Markovian susceptible-infected-recovered outbreaks on time-varying trees, i.e., temporal networks with a loop-free underlying topology. On arbitrary graphs, the proposed contact-based model incorporates potential structural and temporal heterogeneity of the underlying contact network and improves analytic estimations with respect to the individual-based (node-centric) approach at a low computational and conceptual cost. Within this new framework, we derive an analytical expression for the epidemic threshold on temporal networks and demonstrate the feasibility on empirical data.