Networks and hypernetworks 2

Dynamics on networks

Rui Vilela Mendes
http://label2.ist.utl.pt/vilela/

Networks in epidemiology: The SIS Model

$S+I=1$

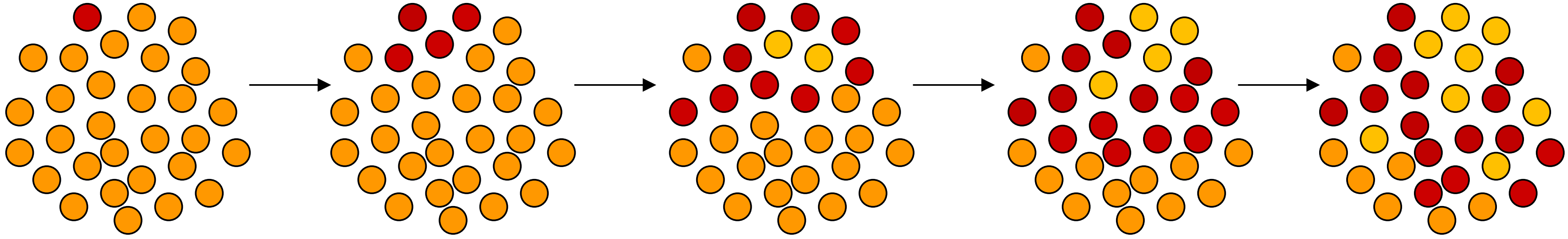
$$ds/dt = -asi+bi$$
$$di/dt = asi-bi$$

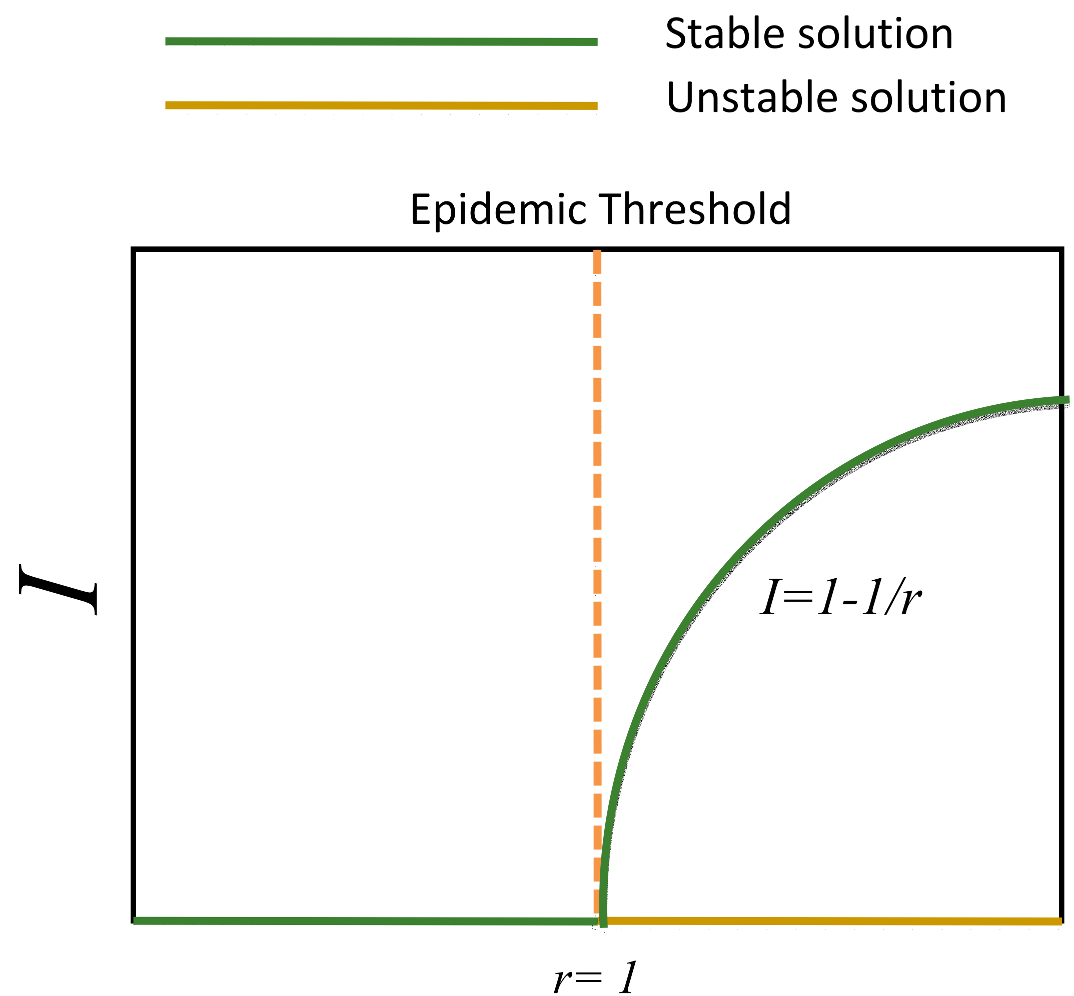
$r=a/b$

$$ds/dt = -rsi+i$$
$$di/dt = rsi-i$$

$$di/dt = r(1-i)i-i$$
$$di/dt = ri-ri^2-i$$
$$di/dt=i(r-ri-1)$$

$$\boxed{di/dt=0 \to i=1-1/r}$$





Stable solution
Unstable solution
Epidemic Threshold
I
I=1-1/r
r= 1

In SF networks we have many groups corresponding to nodes with different degree, s_k, i_k

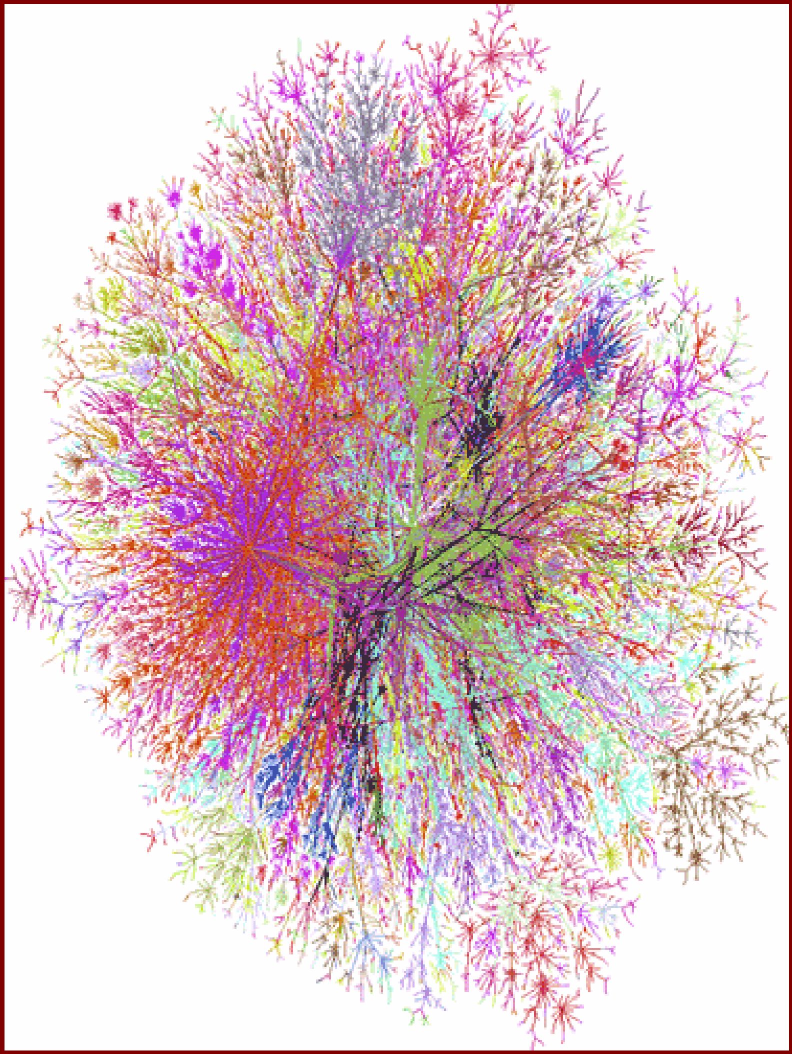
$$di_k/dt = -i_k + rk(1-i_k)\sum i_{k'} P(k,k')$$
$$di_k/dt = -i_k + rk(1-i_k)\theta$$

$$i_k = rk\theta/(1+rk\theta) \qquad (1)$$

$$\theta = \langle k\rangle^{-1} \sum i_k k P(k) \qquad (2)$$

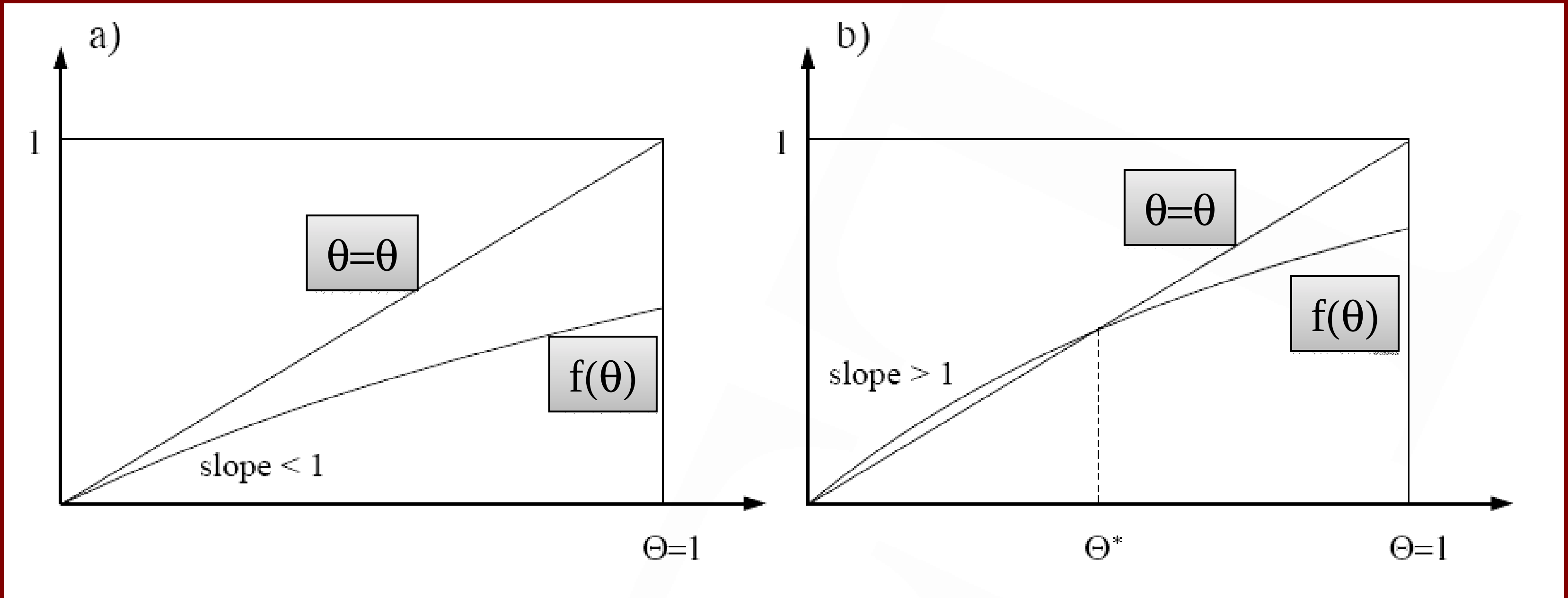
$(1) \rightarrow (2)$

$$\theta = \langle k\rangle^{-1} \sum kP(k)\, rk\theta/(1+rk\theta)$$



R. Pastor-Satorras and A. Vespignani.
Epidemic spreading in scale-free networks.
Physical Review Letters **86**, 3200-3203 (2001).

$\theta = \langle k\rangle^{-1} \Sigma kP(k)\ rk\theta/(1+rk\theta) = f(\theta)$



$df/d\theta|_{\theta=0} \geq 1 \quad -> \quad r\langle k^2\rangle/\langle k\rangle \geq 1$

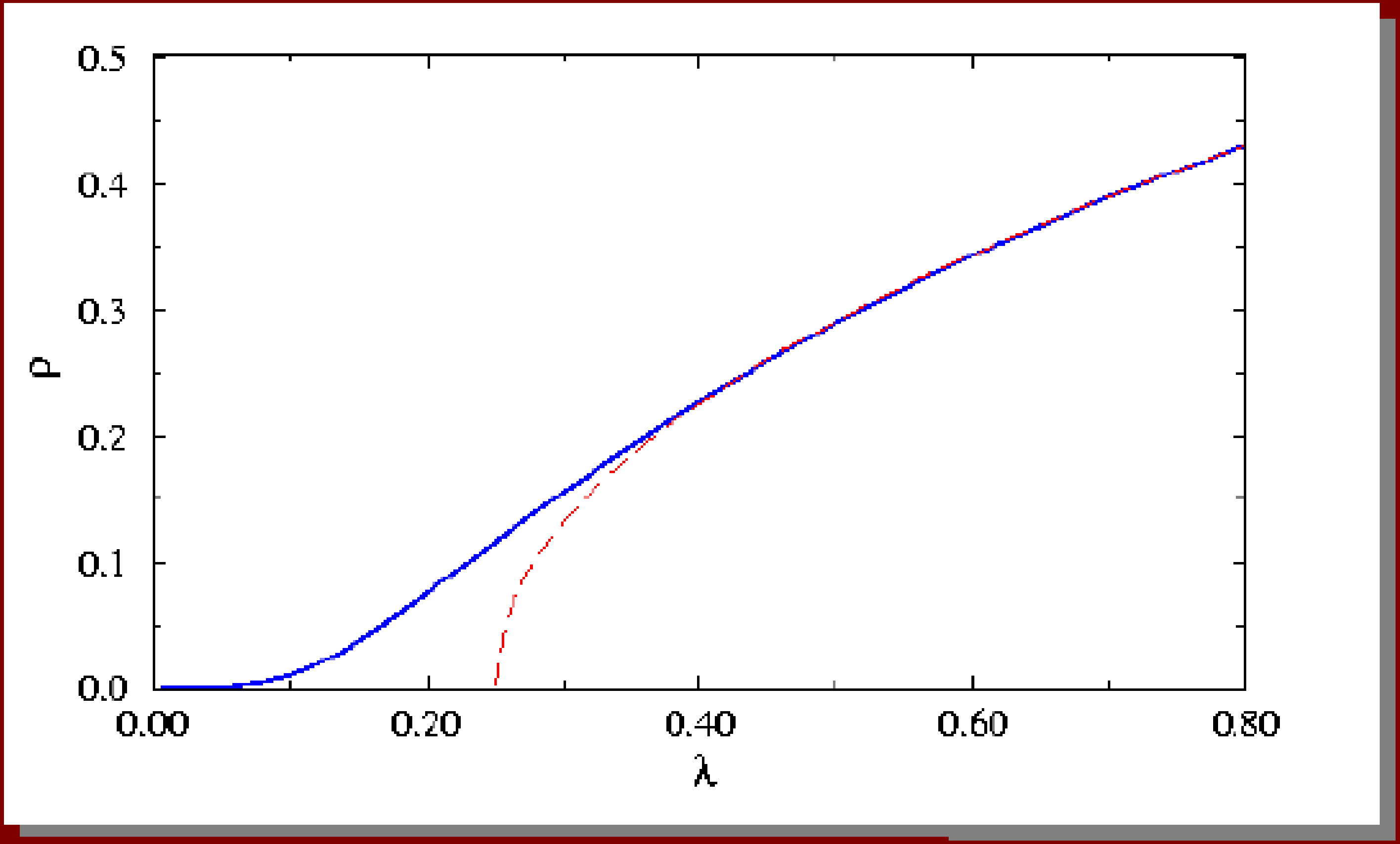
$r \geq \langle k\rangle/\langle k^2\rangle$

$P(k) \sim k^{-a} \quad r_c = 0 \quad \textit{for } a =< 3$

R. Pastor-Satorras and A. Vespignani.
Epidemic spreading in scale-free networks.
Physical Review Letters **86**, 3200-3203 (2001).

No epidemic threshold in SF

(Threshold in exp. net. (WS))

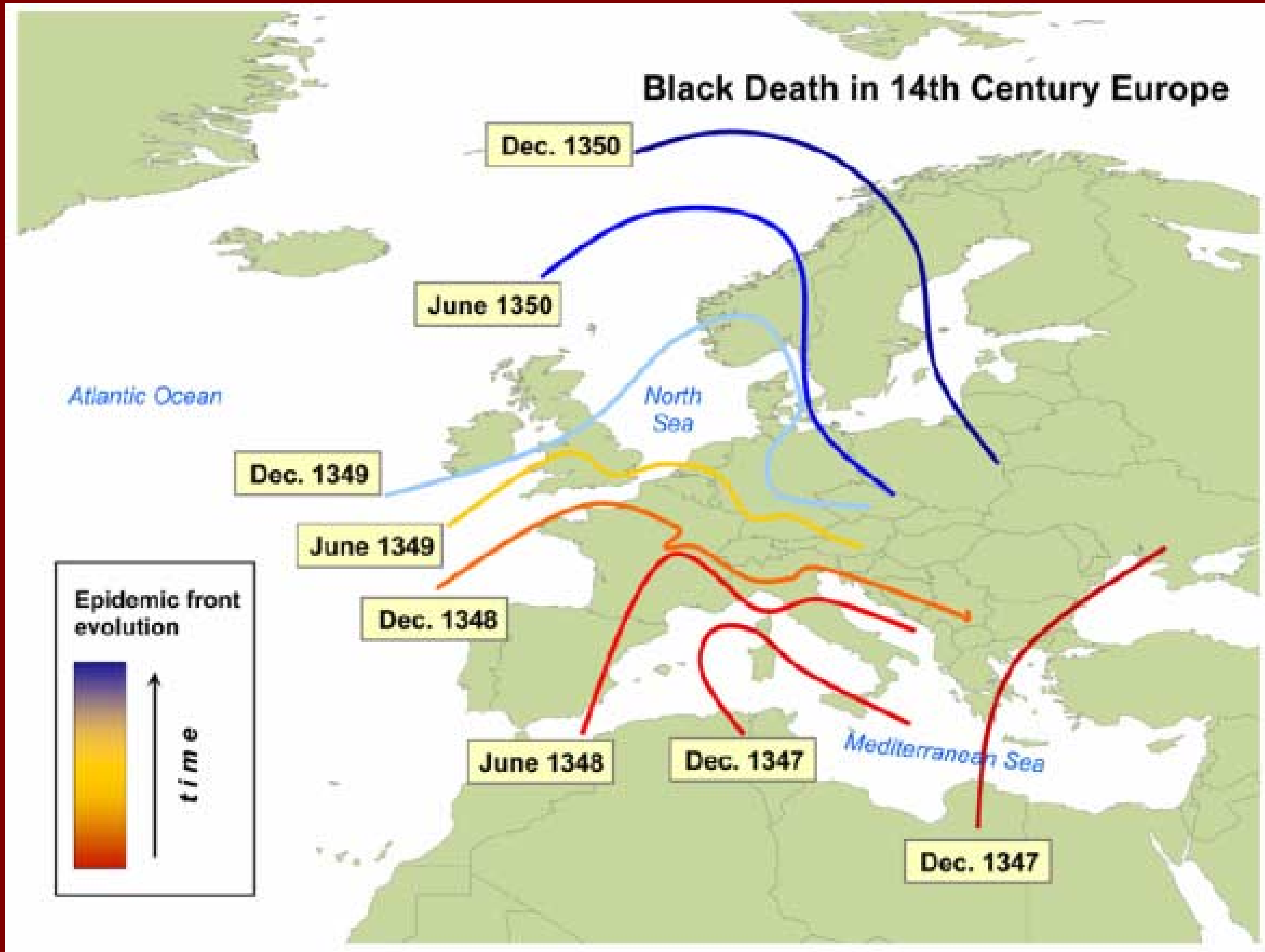


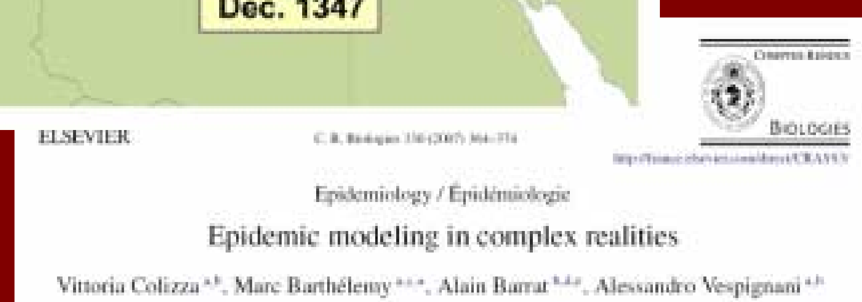
Infected

r_c

R. Pastor-Satorras and A. Vespignani.
Epidemic spreading in scale-free networks.
Physical Review Letters **86**, 3200-3203 (2001).

Scale free networks have a vanishing epidemic threshold




ELSEVIER

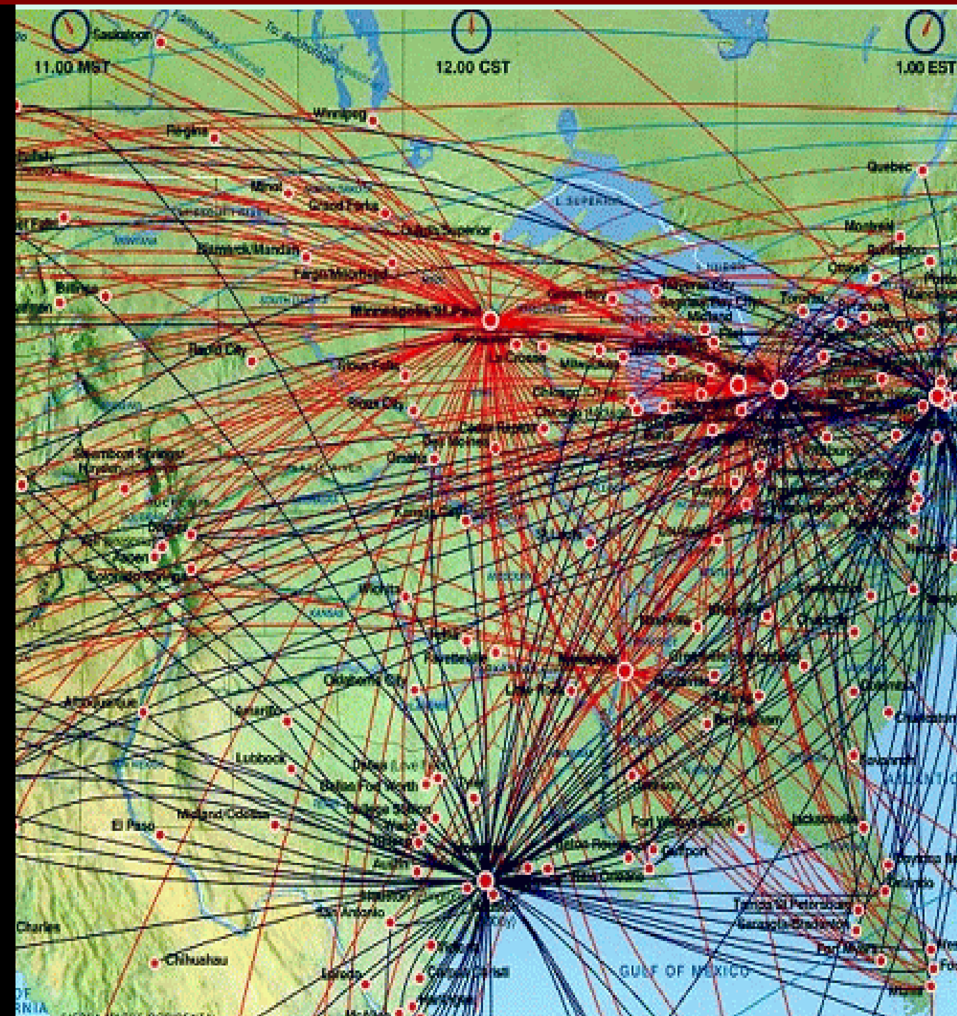
BIOLOGIES

Epidemiology / Épidémiologie

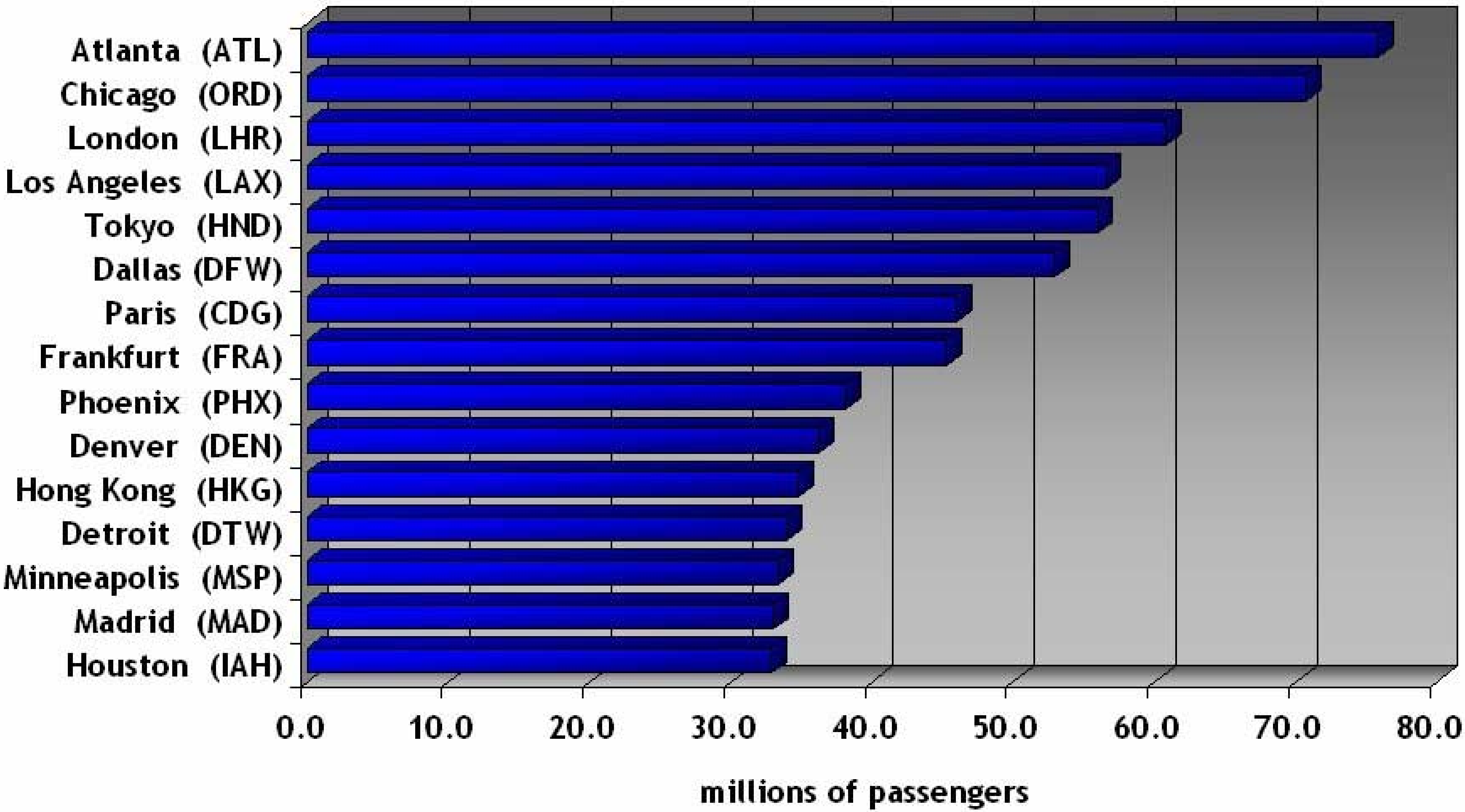
Epidemic modeling in complex realities

Vittoria Colizza, Marc Barthélemy, Alain Barrat, Alessandro Vespignani

The modern long-range links


11.00 MST
12.00 CST
1.00 EST
Saskatoon
Winnipeg
Regina
Quebec
Minneapolis/St.Paul
Memphis
Lubbock
El Paso
San Antonio
Chihuahua
GULF OF MEXICO

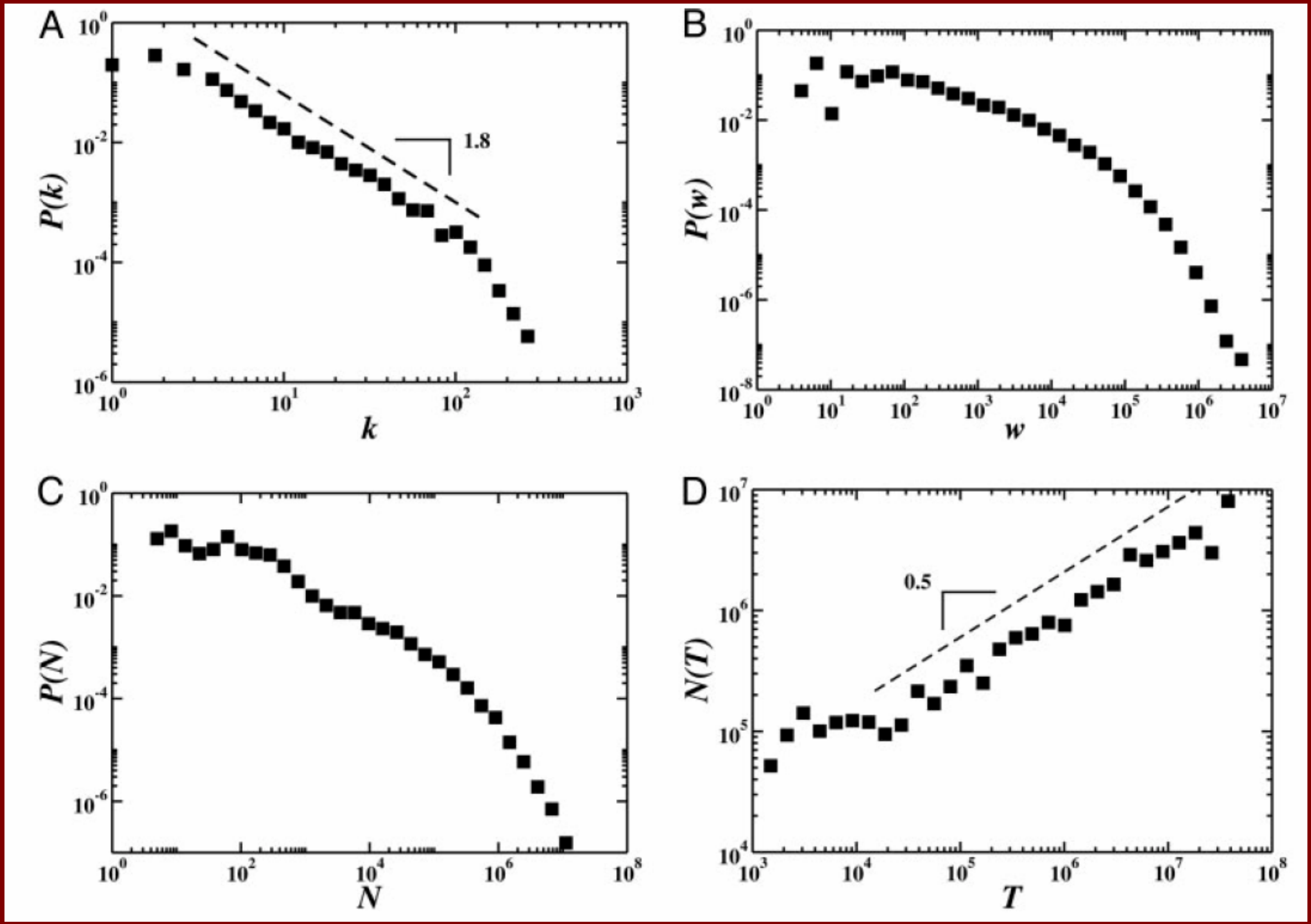
ANNUAL TRAFFIC *(IATA 2002)*



The role of the airline transportation network in the prediction and predictability of global epidemics

Vittoria Colizza*, Alain Barrat†, Marc Barthélemy*‡, and Alessandro Vespignani*§

PNAS



Oppinion dynamics

(Viral memes)

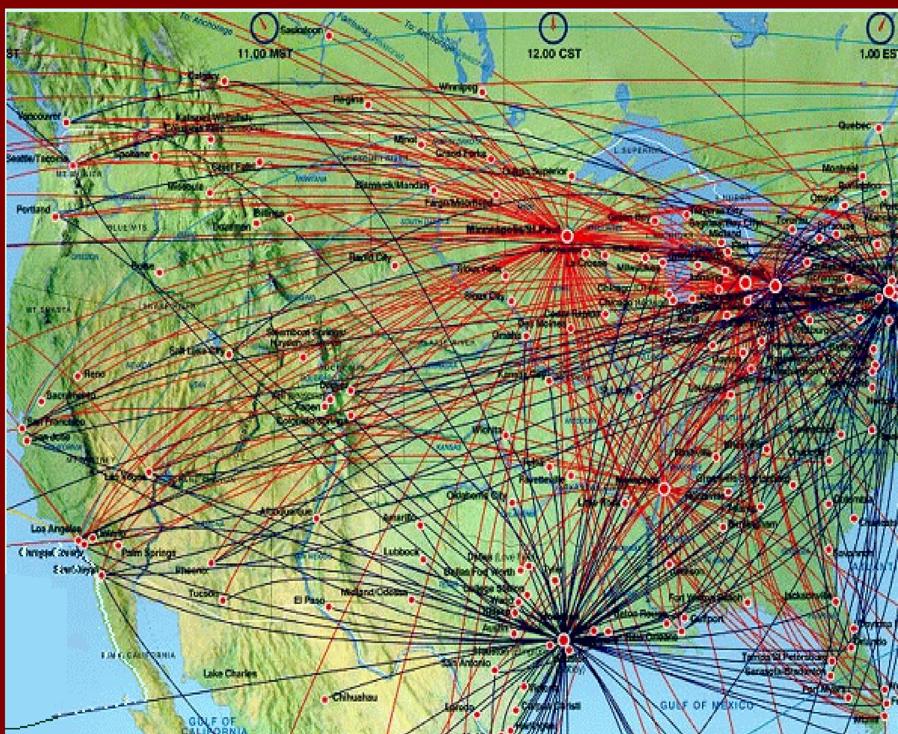
PNAS

Structure and tie strengths in mobile communication networks

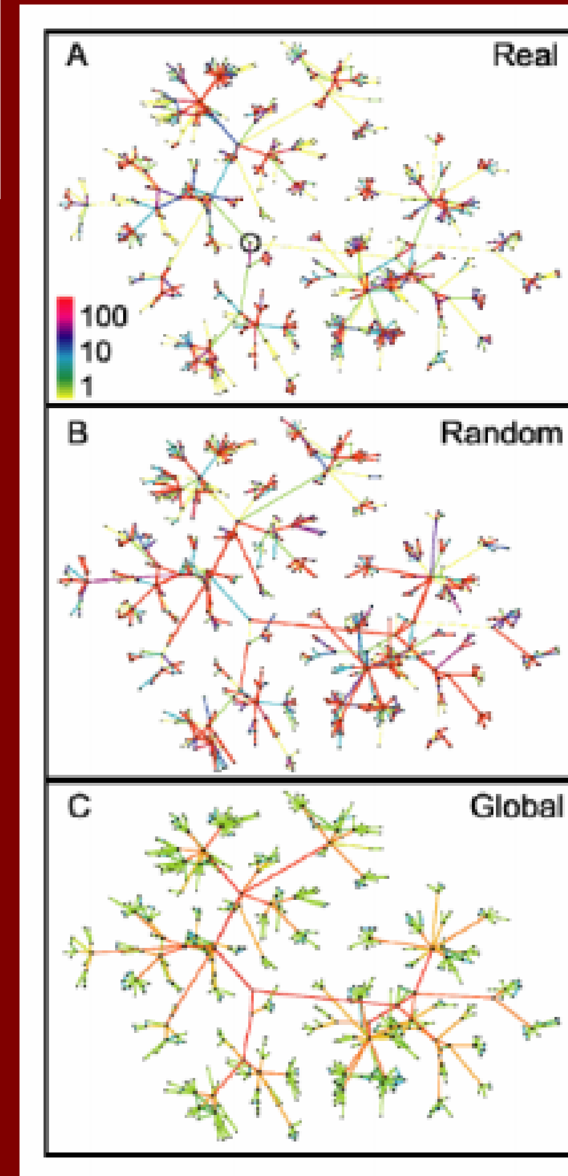
J.-P. Onnela*[†‡], J. Saramäki*, J. Hyvönen*, G. Szabó[§¶], D. Lazer[‖], K. Kaski*, J. Kertész*[,**], and A.-L. Barabási[§¶]

*Laboratory of Computational Engineering, Helsinki University of Technology, P.O. Box 9203, FI-02015 TKK, Helsinki, Finland; [†]Physics Department, Clarendon Laboratory, Oxford University, Oxford OX1 3PU, United Kingdom; [§]Department of Physics and Center for Complex Networks Research, University of Notre Dame, South Bend, IN 46556; [¶]Center for Cancer Systems Biology, Dana–Farber Cancer Institute, Harvard University, Boston, MA 02115; [‖]John F. Kennedy School of Government, Harvard University, Cambridge, MA 02138; and **Department of Theoretical Physics, Budapest University of Technology and Economics, H1111, Budapest, Hungary

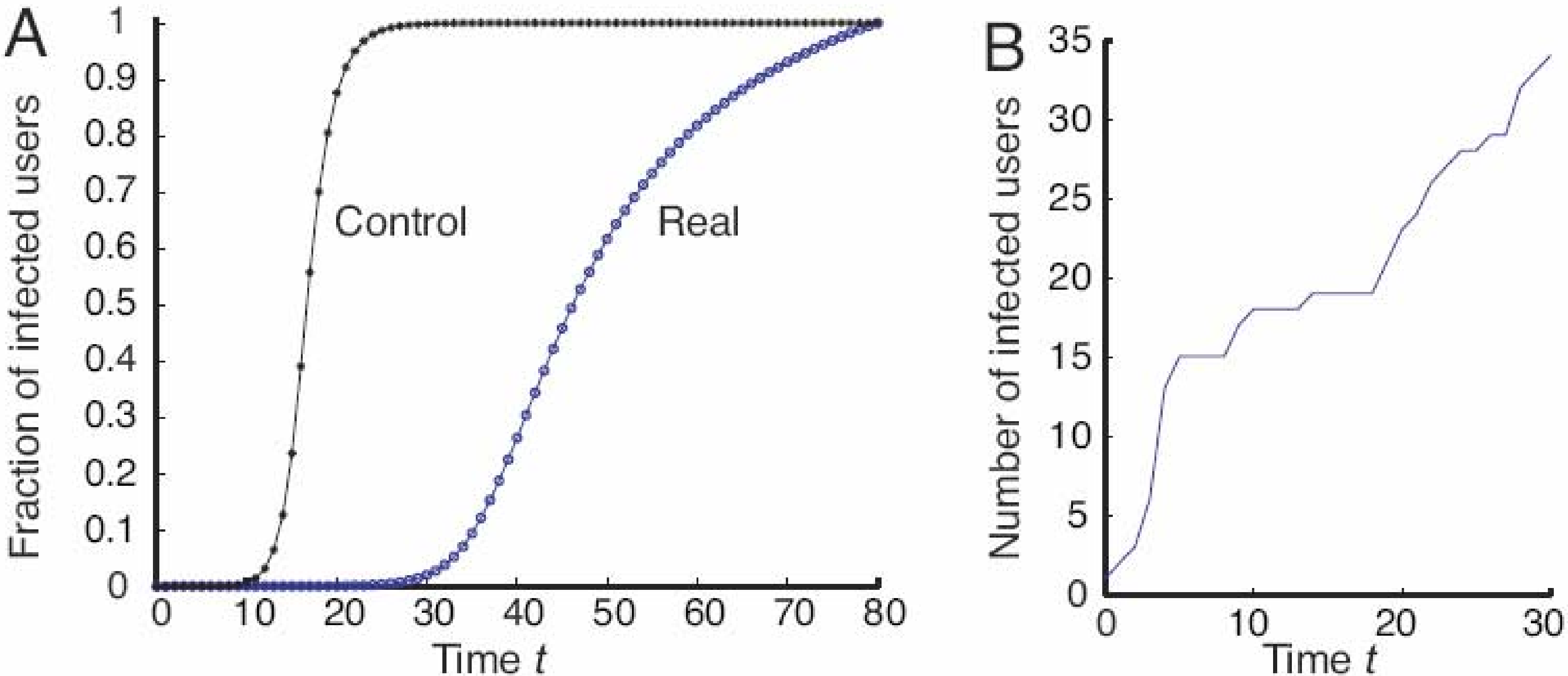
Edited by H. Eugene Stanley, Boston University, Boston, MA, and approved January 27, 2007 (received for review November 18, 2006)



High weight – High Betweenness



Low weight – High Betweenness



A
Fraction of infected users
1
0.9
0.8
0.7
0.6
0.5
0.4
0.3
0.2
0.1
0
Control
Real
0
10
20
30
40
50
60
70
80
Time t
B
Number of infected users
35
30
25
20
15
10
5
0
0
10
20
30
Time t

Characterizing dynamics by ergodic parameters

- *Invariant measures and ergodic parameters*

$$I(\mu) = \lim_{T\to\infty} \frac{1}{T} \sum_{n=1}^{T} F\left(f^n x_0\right)$$

- *Lyapunov and conditional exponents*

From the k x k and (m-k)x(m-k) blocks of the Jacobian , obtain the conditional exponents as the eigenvalues of the limits

$$\lim_{n\to\infty} \left(D_k f^{n*}(x) D_k f^n(x)\right)^{\frac{1}{2n}}$$

$$\lim_{n\to\infty} \left(D_{m-k} f^{n*}(x) D_k f^n(x)\right)^{\frac{1}{2n}}$$

or

$$\lim_{n\to\infty} \frac{1}{n} \log \|D_k f^n(x) u\| = \xi_i^{(k)}$$

with $0 \neq u \in E_x^i / E_x^{i+1}$, E_x^i being the subspace of R^k spanned by eigenstates corresponding to eigenvalues $\leq \exp(\xi_i^{(k)})$.

Existence of the conditional exponents

- First proposed by Pecora and Carroll to study the phenomenology of synchronization of chaotic systems
  PRL 64 (1990) 821 ; PRA 44 (1991) 2374

- *Theorem: The existence of the conditional exponents is guaranteed under the same conditions as for the Lyapunov exponents*

  Existence of a measurable map from the dynamical space V to m x m matrices

  $$T : V \to M_m$$

  and $$\int \mu(dx) \log^+ \left\| T(x) \right\| < \infty$$

  The proof follows the same steps as for the Oseledec's theorem
  PLA 248 (1998) 167

- Regular functionals of the exponents will also be well-defined ergodic parameters

Structures and self-organization

- Structure index

$$S = \frac{1}{N}\sum_{i=1}^{N_+}\left(\frac{\lambda_0}{\lambda_i} - 1\right)$$

diverges whenever a Lyapunov exponent approaches zero from above (points where long time correlations develop)

- Self-organization (partitions $\Sigma_k = \mathrm{R}^k \times \mathrm{R}^{m-k}$)

$$I(S,\Sigma,\mu) = \sum_{k=1}^{N}\{h_k(\mu) + h_{m-k}(\mu) - h(\mu)\}$$

$$h_k(\mu) = \sum_{\xi_i^{(k)}>0}\xi_i^{(k)} \; ; \; h_{m-k}(\mu) = \sum_{\xi_i^{(m-k)}>0}\xi_i^{(m-k)} \; ; \; h(\mu) = \sum_{\lambda_i>0}\lambda_i$$

(Physica A276 (2000) 550-571)

- Self-organization concerns the dynamical relation of the whole to its parts. Therefore, $I_\Sigma(\mu)$ is a measure of dynamical self-organization
- It is a measure of apparent dynamical freedom (or apparent rate of information production), that each agent may infer from the local dynamics
- Self-organization occurs when local information is very different from global behavior
- These global parameters, besides providing information on structure formation and self-organization may also be used to characterize the topology of the interactions (network connectivity)

Examples :

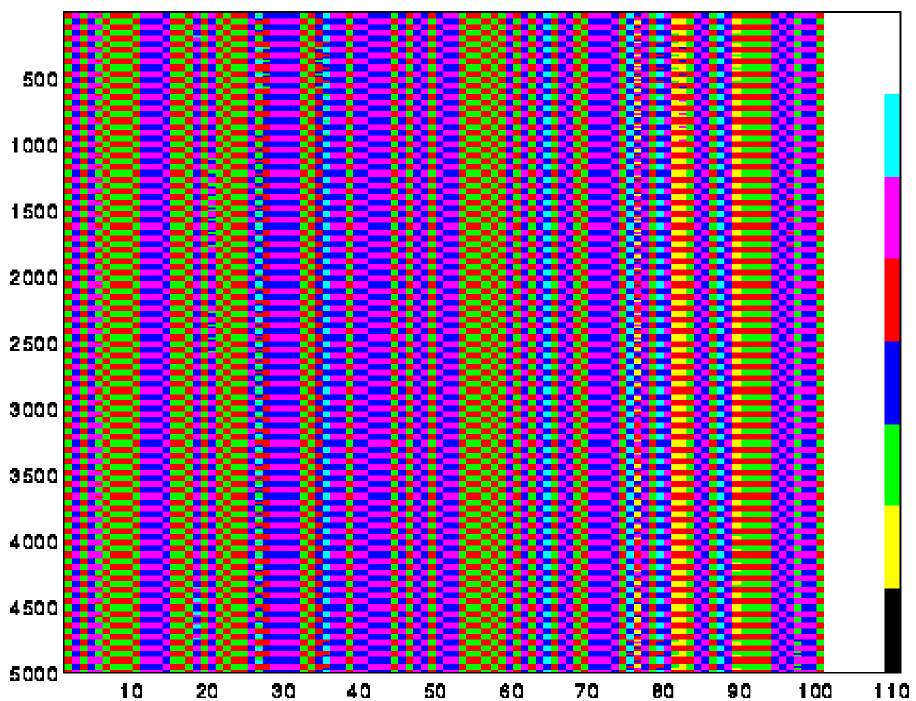
- Fully coupled system

$$x_i(t+1) = (1-c)\, f(x_i(t)) + (c/(N-1)) \sum_{k\neq i} f(x_k(t))$$

$$f(x)=2x \pmod 1$$

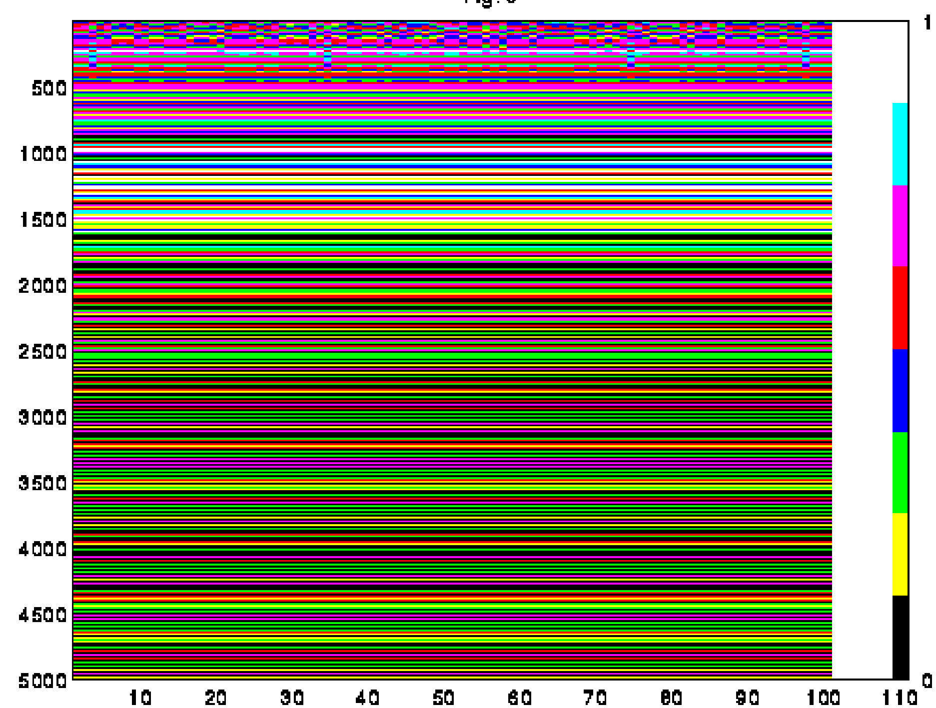
c = 0.495

Fig. 2

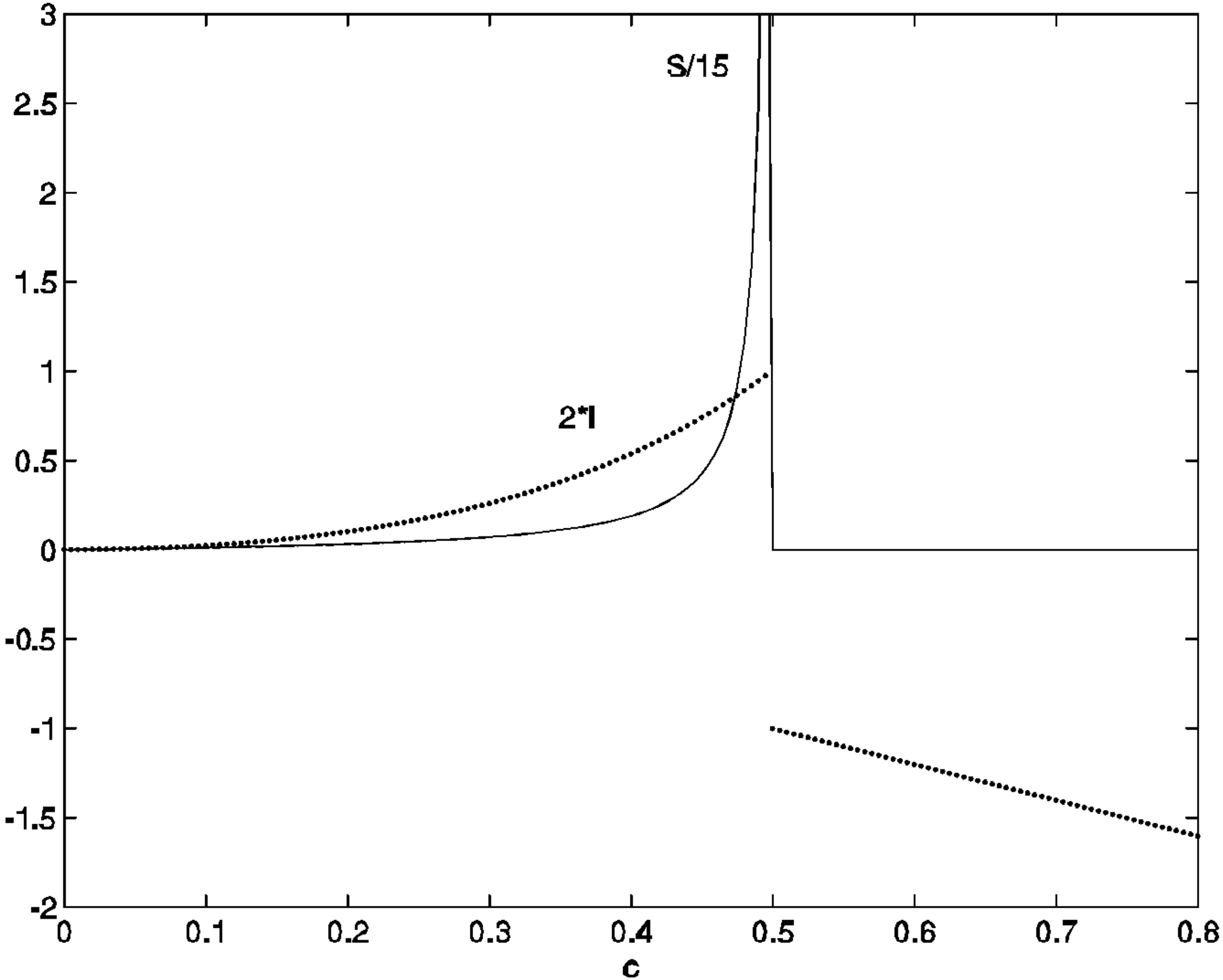


c = 0.51

Fig. 3

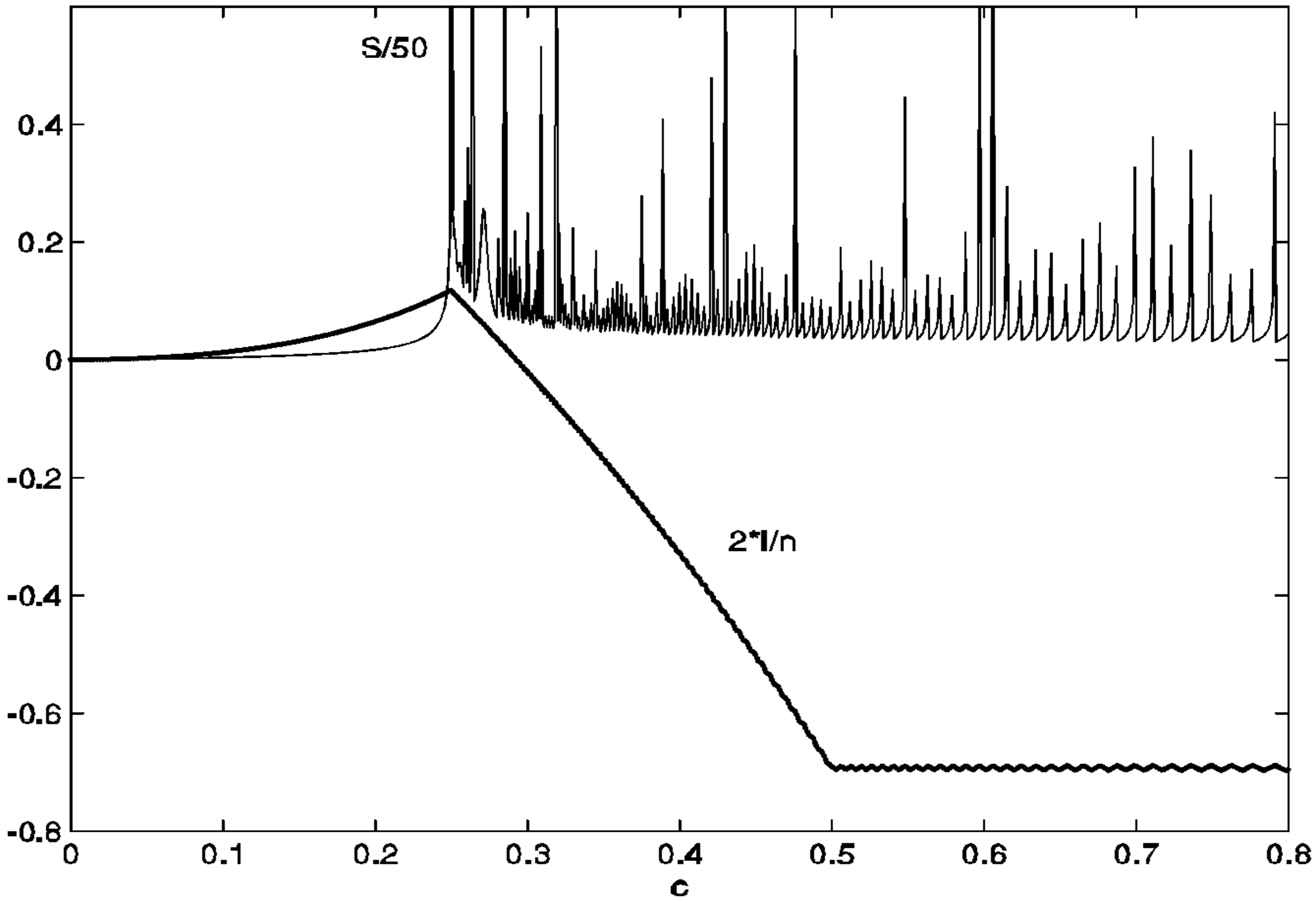


Fully coupled system. Structure and self-organization index



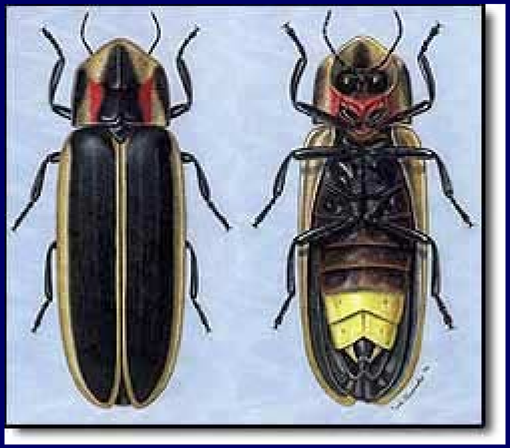
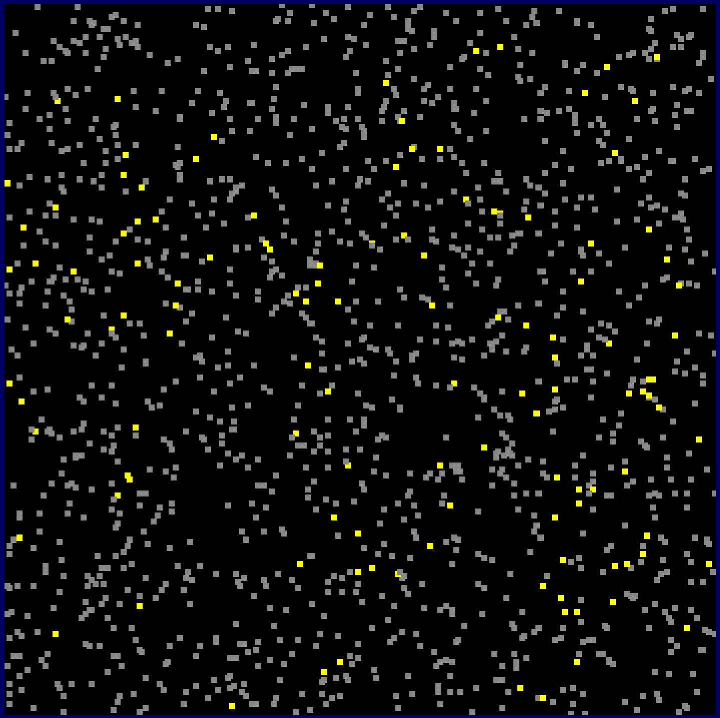
Nearest-neighbor coupling

- $x_i(t+1) = (1-c)\, f(x_i(t)) + (c/2)\,(\, f(x_{i+1}(t)) + f(x_{i-1}(t))\,)$



Synchronization and beyond

- Synchronous flashing of fireflies, cells, fads, ...


Synchronization and beyond

- Synchronization
  (Classical mathematical example: the Kuramoto model)
  A similar, discrete-time oscillators model :

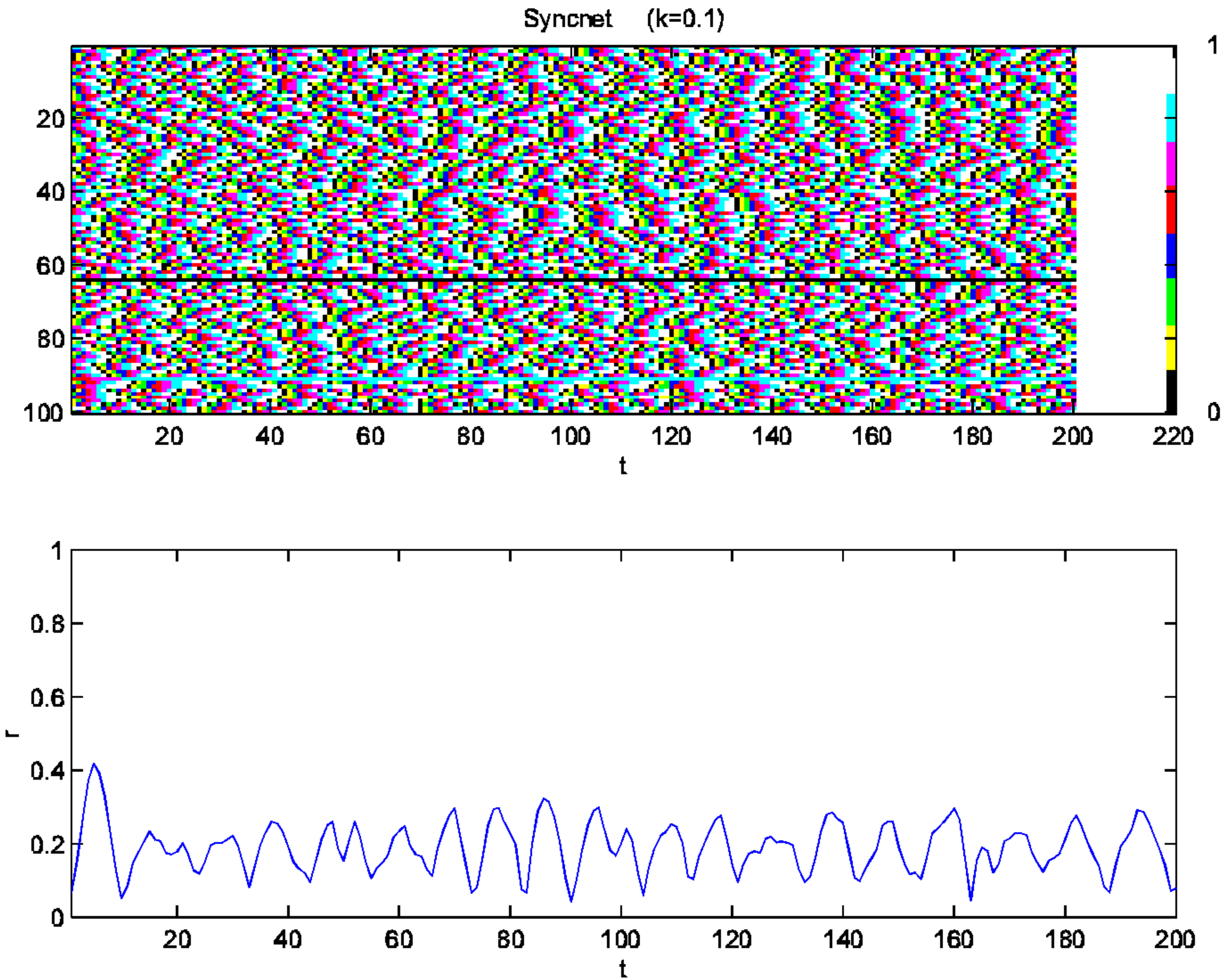
$$x_i(t+1) = x_i(t) + \omega_i + \frac{k}{N-1}\sum_{j=1}^{N} f_\alpha(x_j - x_i)$$

$$p(\omega) = \frac{\gamma}{\pi\left[\gamma^2 + (\omega - \omega_0)^2\right]}$$

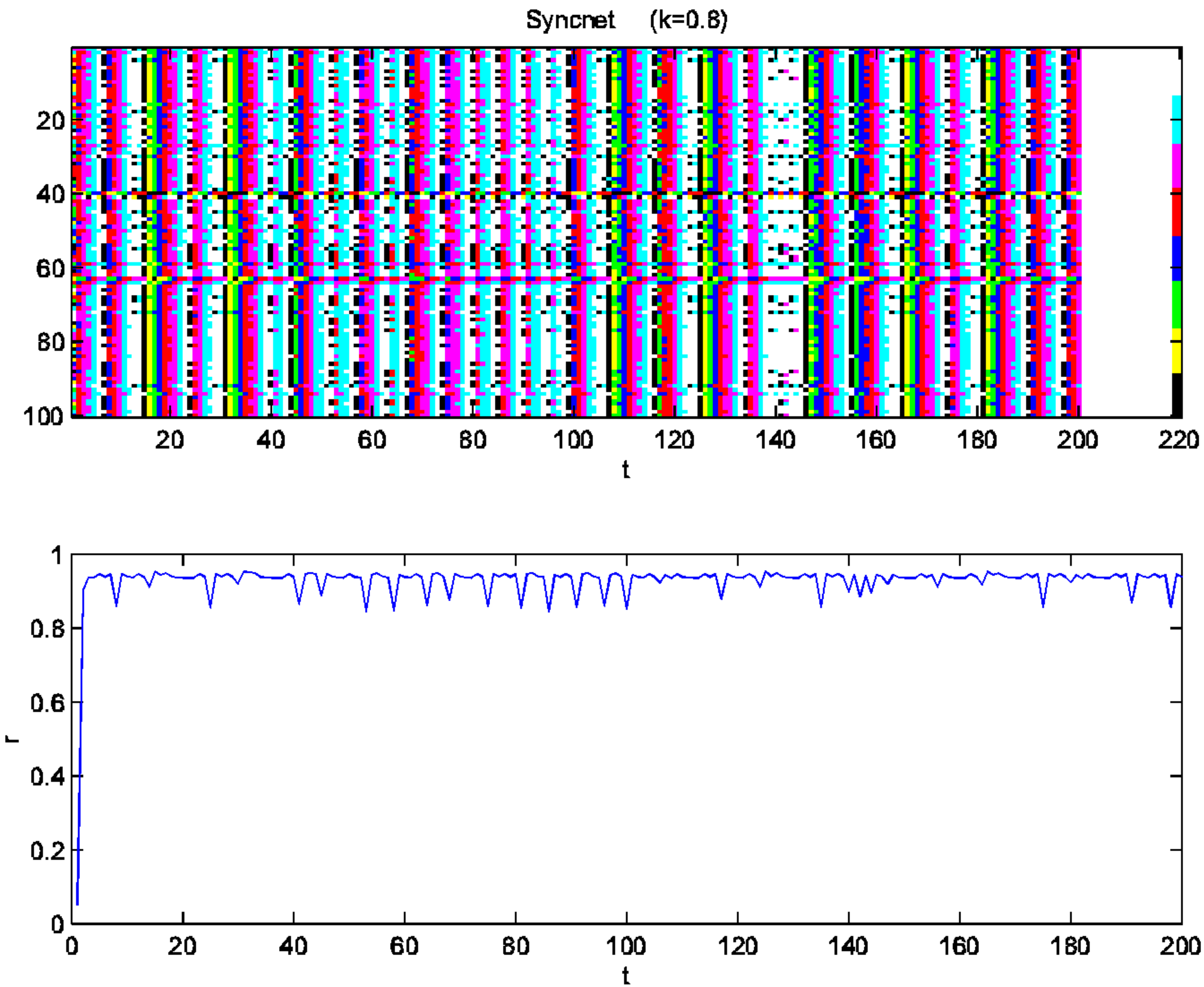
$$f(x) = 2x \pmod{1}$$

- Order parameter

$$r(t) = \left|\frac{1}{N}\sum_{j=1}^{N} e^{i\theta_j(t)}\right|$$



Syncnet (k=0.1)
20
40
60
80
100
20
40
60
80
100
120
140
160
180
200
220
t
1
0
r
0
0.2
0.4
0.6
0.8
1
20
40
60
80
100
120
140
160
180
200
t



Syncnet (k=0.8)
t
r
t

- The Lyapunov spectrum controls the dynamical self-organization of the system.

- In this case

  $\lambda_1=0$ and

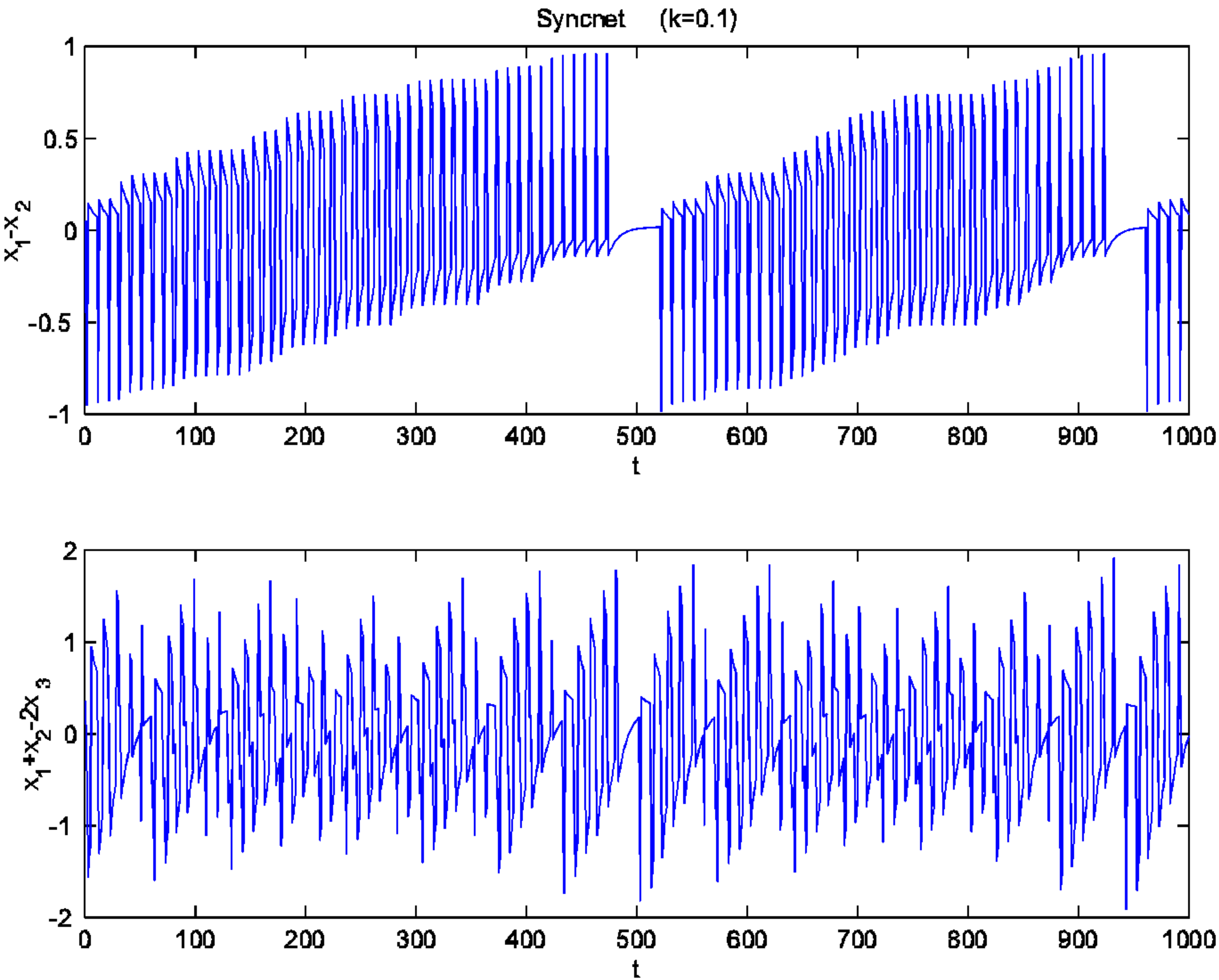
  $\lambda_i=\log(1-\alpha\lambda k(N/N-1))$ (N-1) times

  N-1 contracting directions for $k\neq 0$

  "One-dimensional" system !

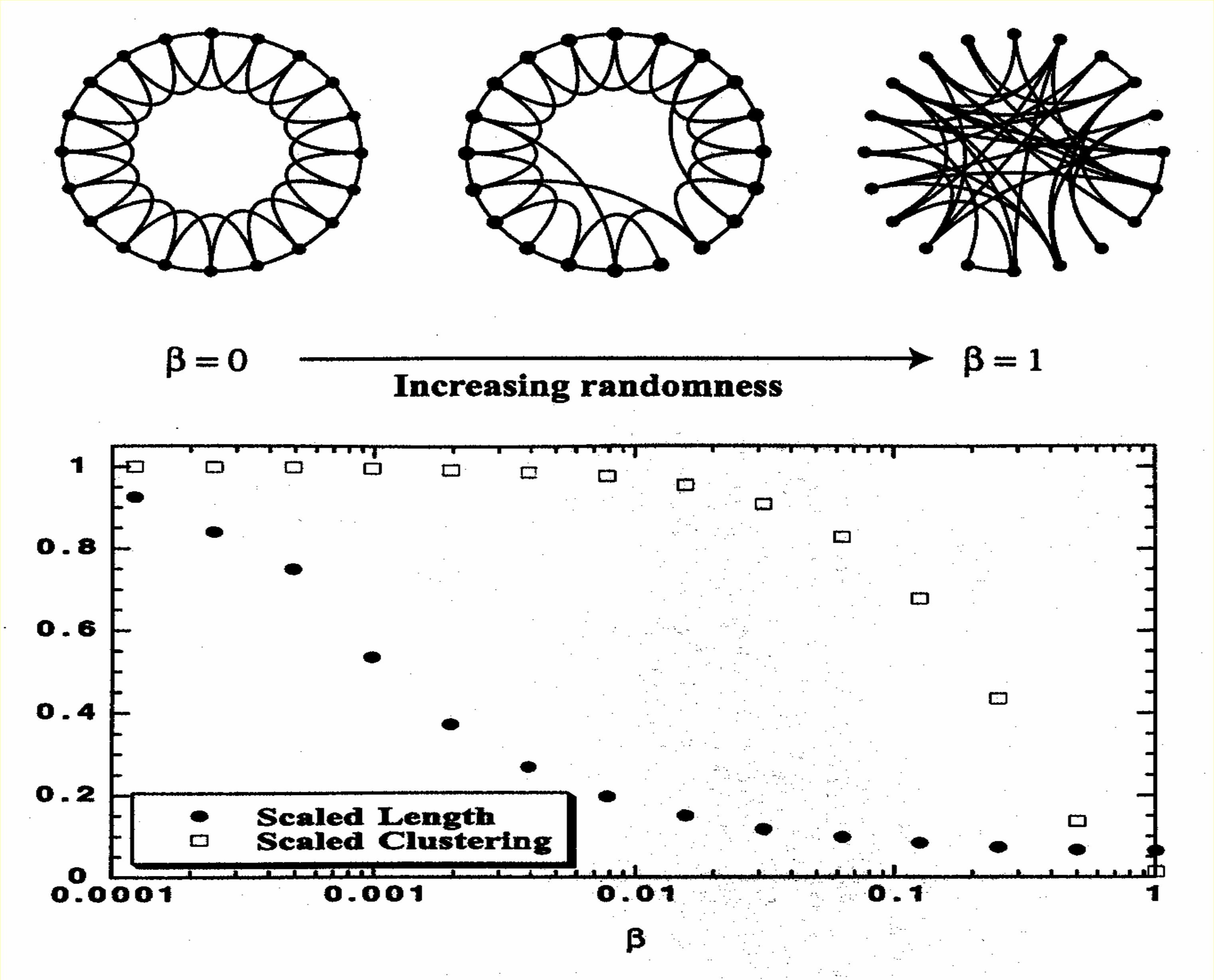
- $\Rightarrow$ strong dynamical correlations even before synchronization

  (*Int. J. Bifurcation and Chaos, 15 (2005) 1185-1213*)



Syncnet (k=0.1)
x1-x2
1
0.5
0
-0.5
-1
0 100 200 300 400 500 600 700 800 900 1000
t
x1+x2-2x3
2
1
0
-1
-2
0 100 200 300 400 500 600 700 800 900 1000
t

Network structure and dynamics.The small world phase



Define a dynamical system on the network nodes

- $x_i(t+1) = \Sigma_{k=1}^{N} W_{ik} f(x_k(t))$

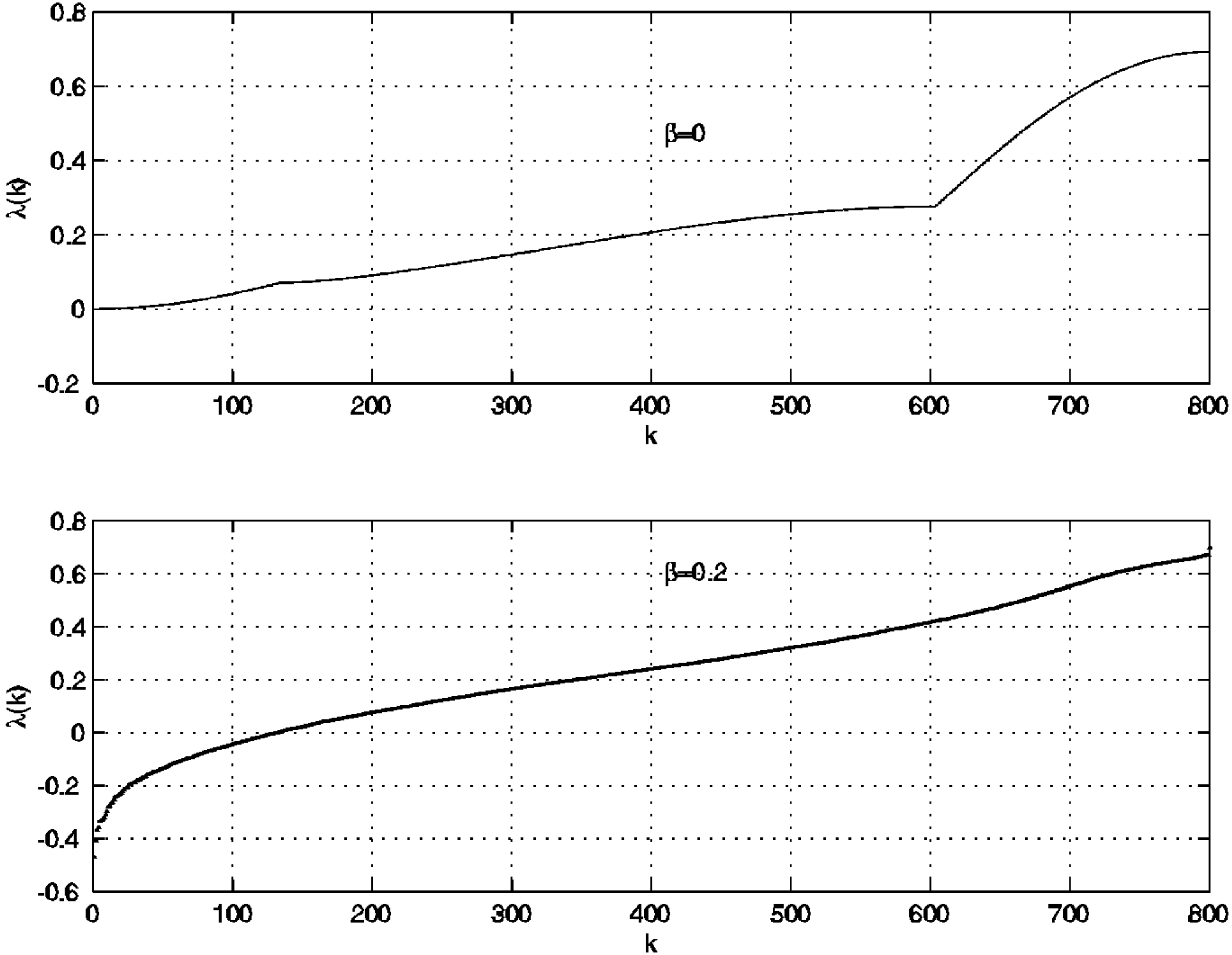
  $f(x)=\alpha x \pmod 1$

$$W_{ik} = \begin{cases} 1-\dfrac{n_v(i)}{2v}c & \text{if } i = k \\ \dfrac{c}{2v} & \text{if } i \neq k \text{ and } k \in n_v(i) \\ 0 & \text{0 otherwise} \end{cases}$$

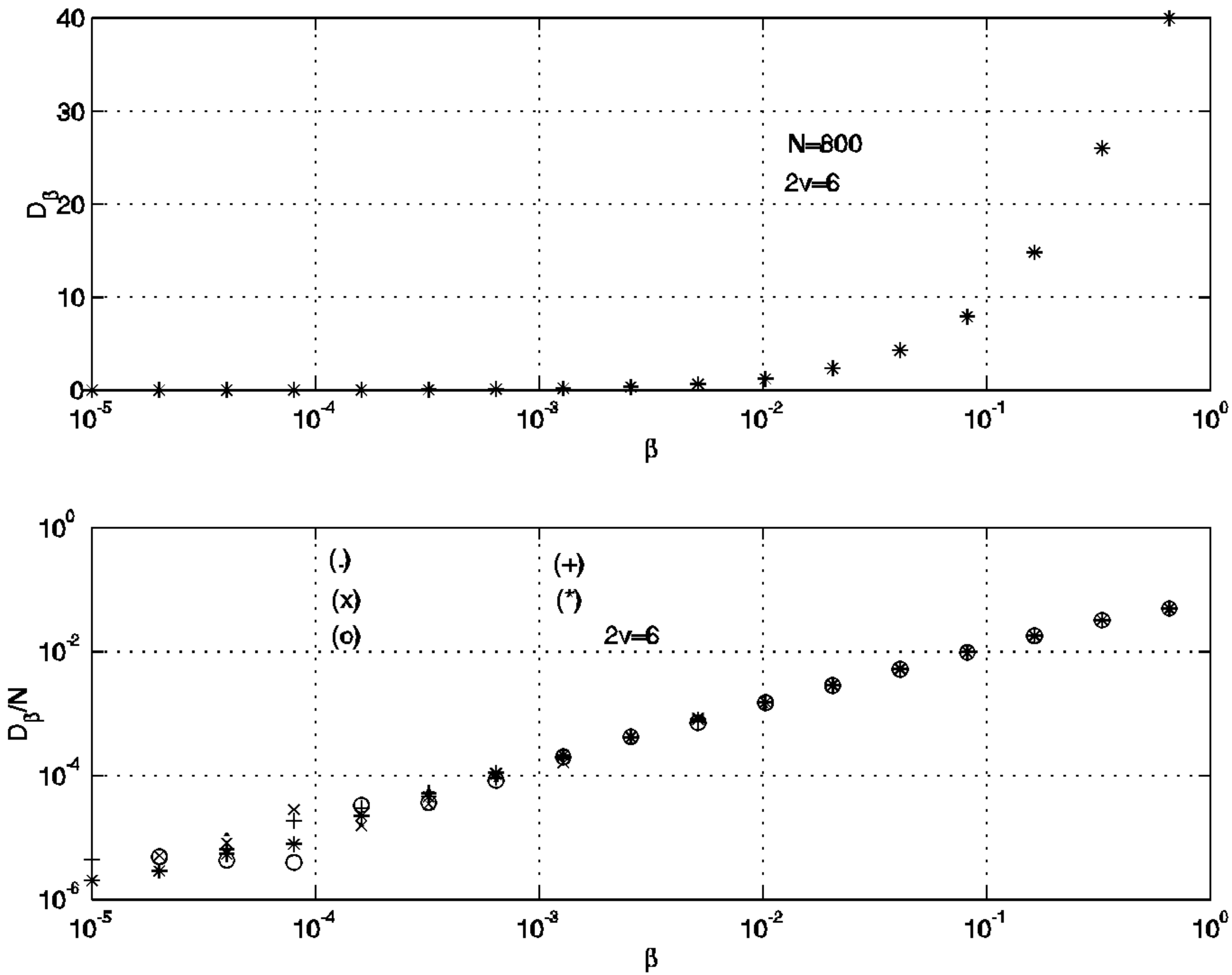
- $D_\beta = - \Sigma_{\lambda_i<0} \lambda_i$

  $D_\beta = c N (\beta - \beta_{c1})^{\eta}$ $\quad \beta_{c1} < 10^{-5}$ $\quad \eta = 1.01 \pm 0.06$

(*T. Araújo, J. Seixas, RVM, Phys. Lett. A319 (2003) 285-289*)



β=0
λ(k)
k
β=0.2
λ(k)
k



40
30
20
10
0
D_β
N=800
2v=6
10^{-5} 10^{-4} 10^{-3} 10^{-2} 10^{-1} 10^{0}
β
10^{0}
10^{-2}
10^{-4}
10^{-6}
D_β/N
(.)
(x)
(o)
(+)
(*)
2v=6
10^{-5} 10^{-4} 10^{-3} 10^{-2} 10^{-1} 10^{0}
β

Define a dynamical system on the network nodes

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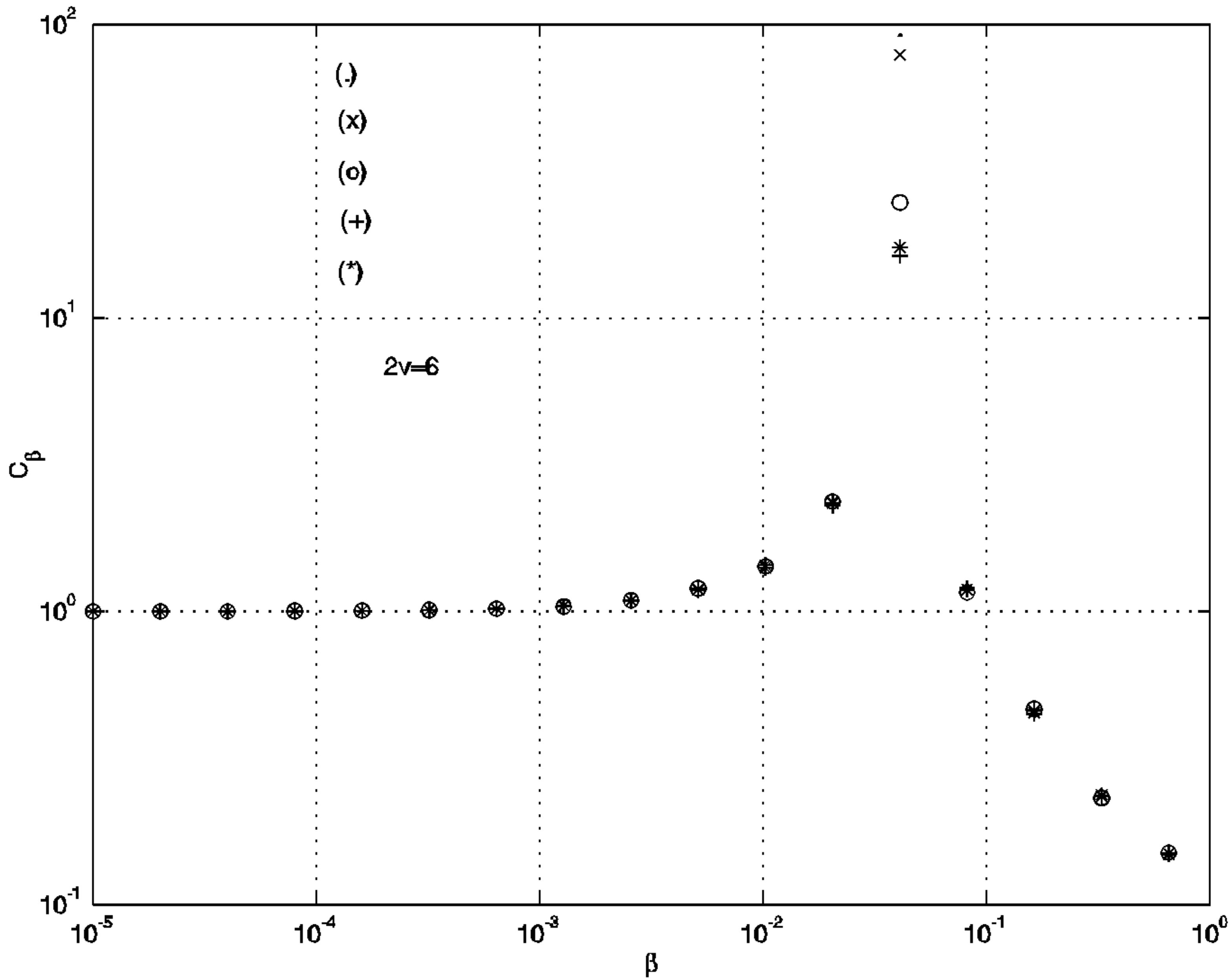
- $D_\beta = - \Sigma_{\lambda_i<0} \lambda_i$

  $D_\beta = c N (\beta - \beta_{c1})^\eta$ $\beta_{c1} < 10^{-5}$ $\eta = 1.01 \pm 0.06$

- $C_\beta = \left| \frac{h_0^* - h_0}{h_\beta^* - h_\beta} \right|$; $h_\beta^* = \sum_{i=1}^{N} \left(\frac{1}{d_i} \sum_{\lambda_\beta^*>0} \lambda_\beta^*(j) \right)$; $h_\beta = \sum_{\lambda_\beta>0} \lambda_\beta(j)$

  $\beta_{c2} = 0.04$ $C_\beta \sim |\beta - \beta_{c2}|^{-\delta}$ $\delta_1 = 1.14$ $\delta_2 = 0.93$

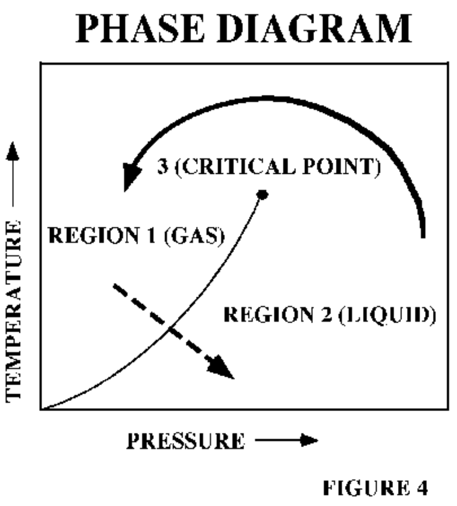


(.)
(x)
(o)
(+)
(*)
2v=6
C_β
β
10^2
10^1
10^0
10^{-1}
10^{-5}
10^{-4}
10^{-3}
10^{-2}
10^{-1}
10^0

Scaling laws and self-organized criticality (SOC)

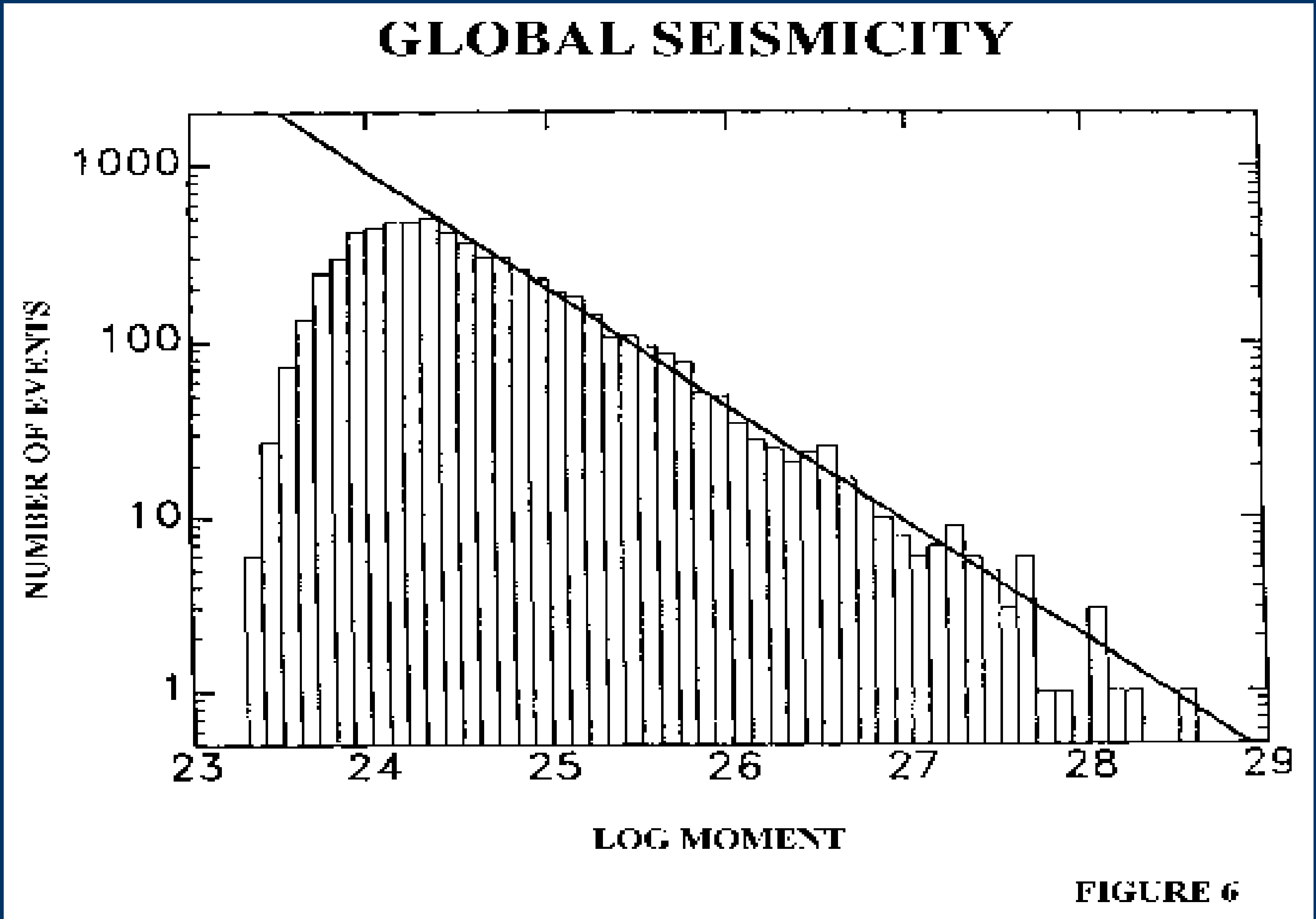
- A qualitative definition :
  SOC = *mechanism of slow energy accumulation and fast energy redistribution (avalanches) driving the system towards a critical state, where the distribution of avalanche sizes is a power law obtained without fine tuning, that is, there is no tunable parameter in the model*.
- Power law → no natural scale, excitations at all scales
- No tunable parameter ≠ usual critical points in phase transitions
- A critical point as an attractor ?
- Ubiquity of SOC (geophysics, cosmology evolutionary biology, ecology, economics sociology, solar physics, …)

- Question: Can SOC be characterized by ergodic parameters ?



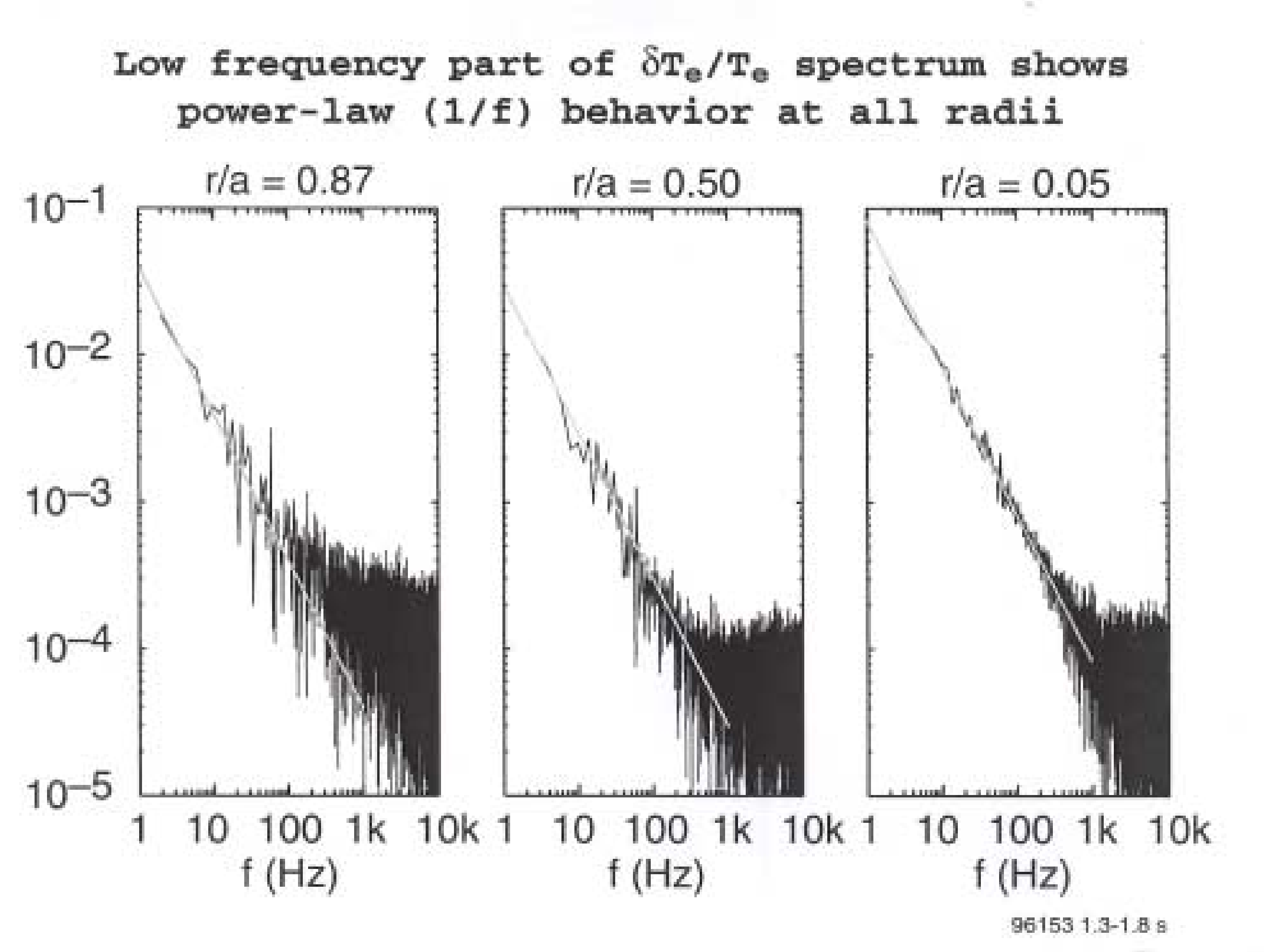
Real world manifestations

- *The Gutenberg-Richter law*
  Data from 1977-1995



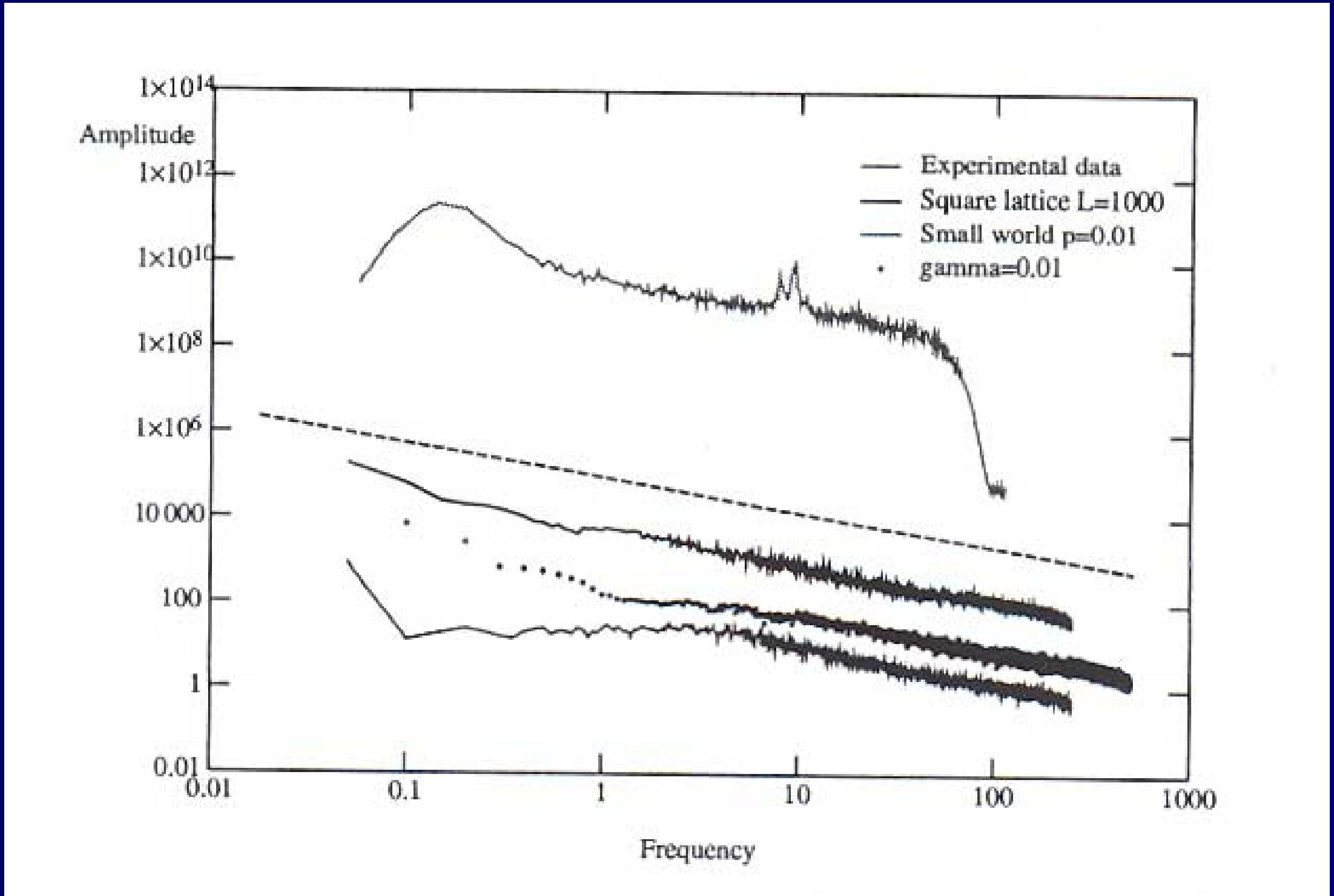
Real world manifestations

- *Electron temperature fluctuations in a magnetically confined plasma (ECE diagnostic)*
  (Politzer, PRL 84 (2000) 1192)



Real world manifestations

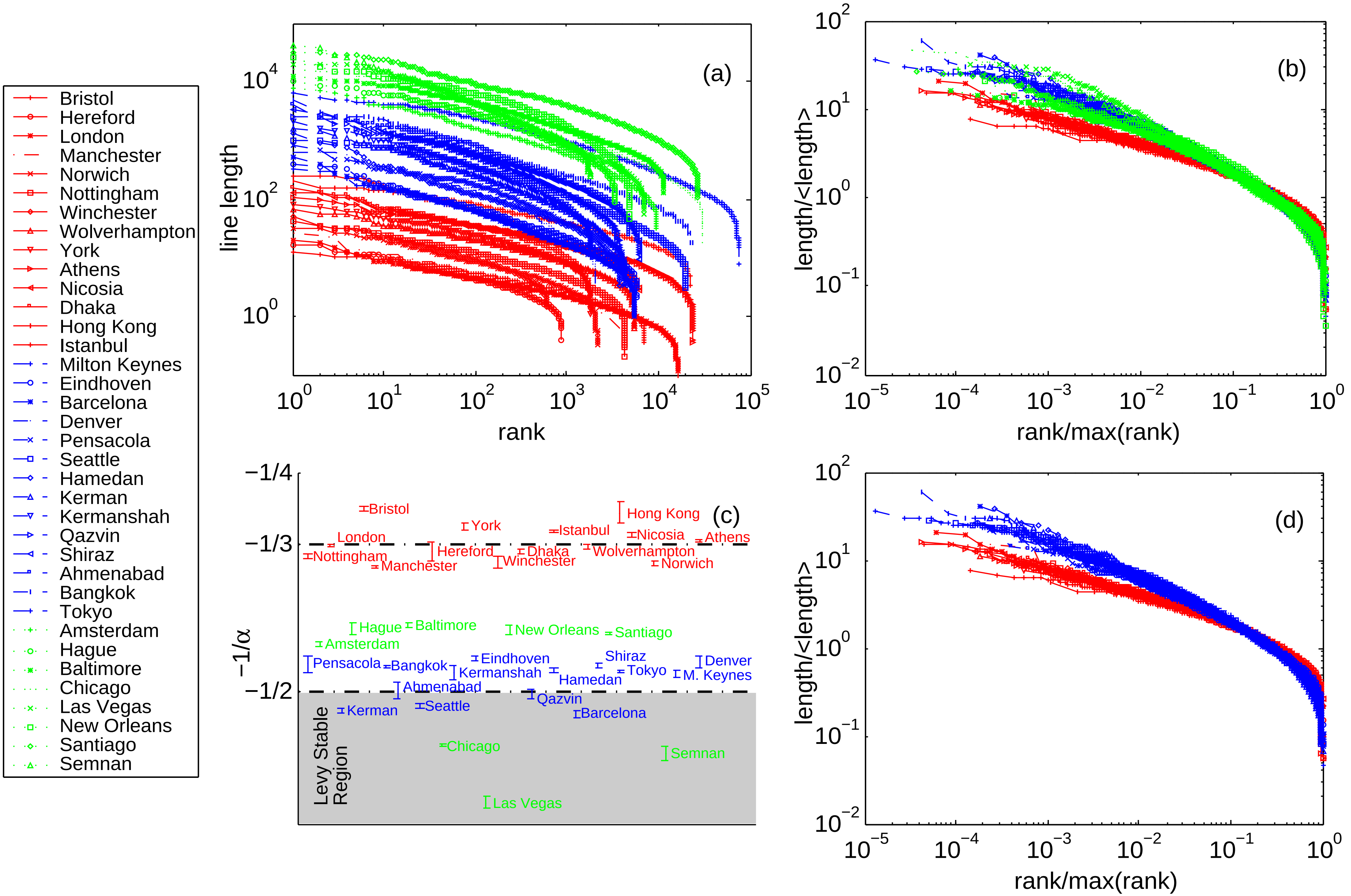
- *Avalanches in living neurons*
  Magnetoencephalography data compared with models
  (de Arcangelis et al. PRL 96 (2006) 028107)



Real world manifestations

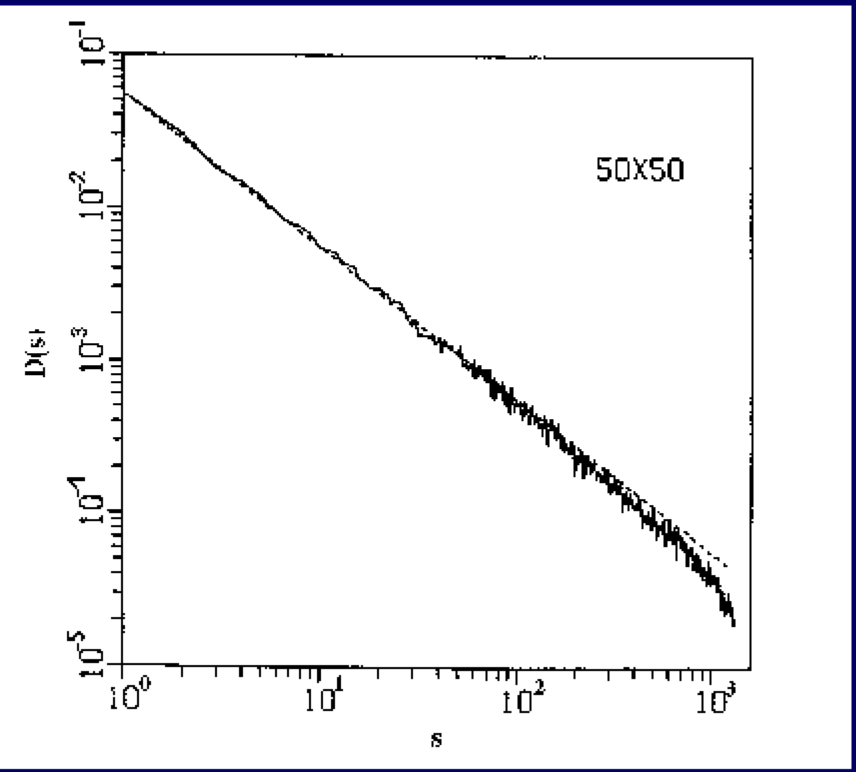
- *Distribution of lengths of open spaces in urban environments*
  (Carvalho and Penn, Physica A 332 (2004) 539)

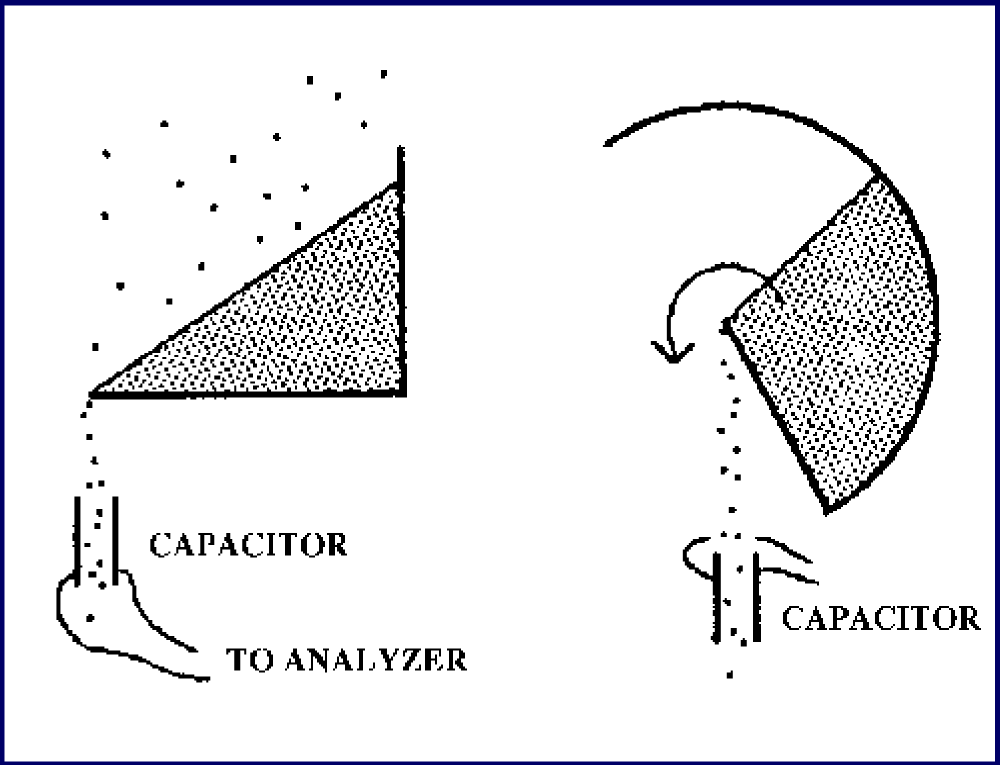
Real world manifestations



Toy models

- Sand piles on the computer and on the lab

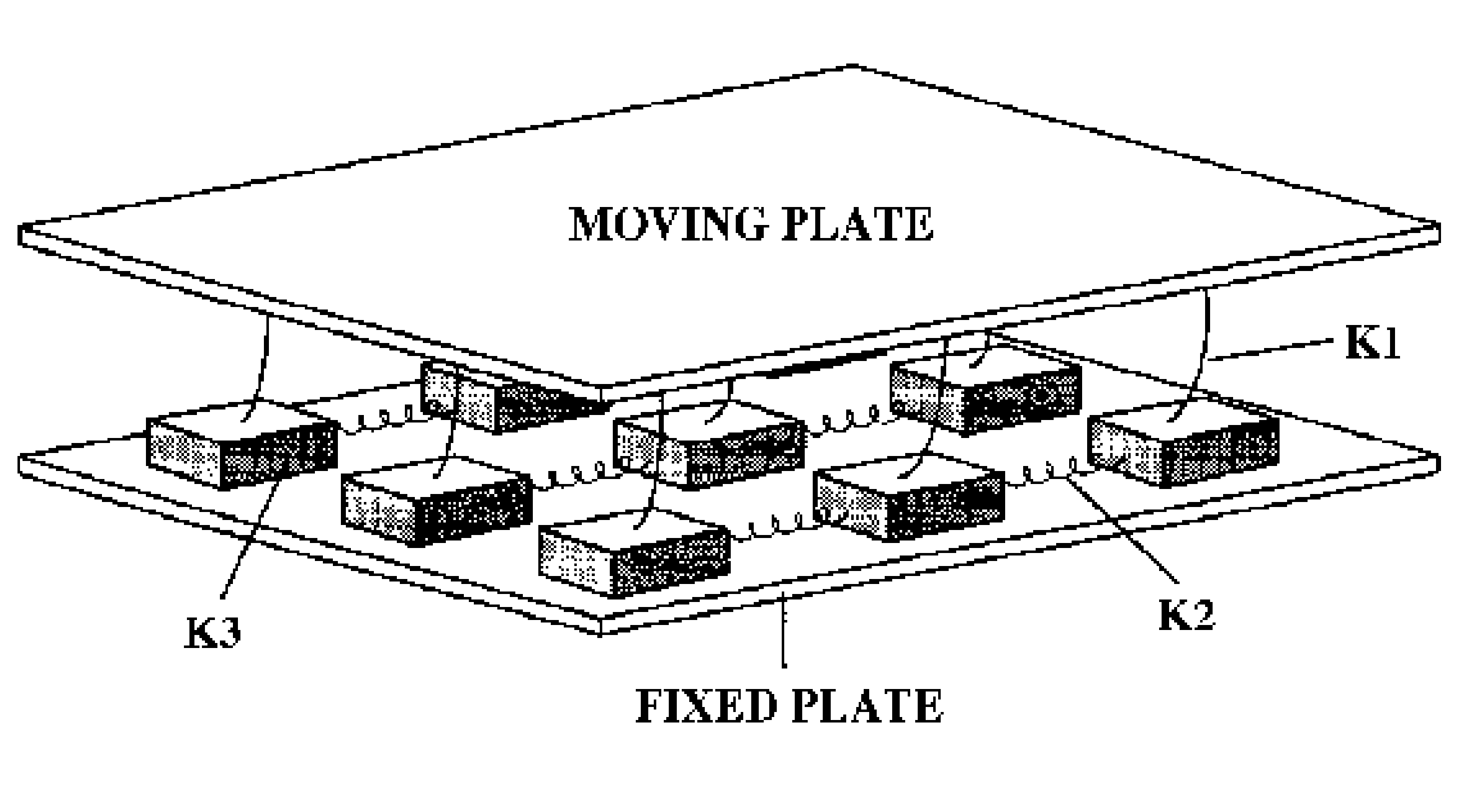




- However, the emergence of scaling laws on lab sand piles depend on grain size and shape

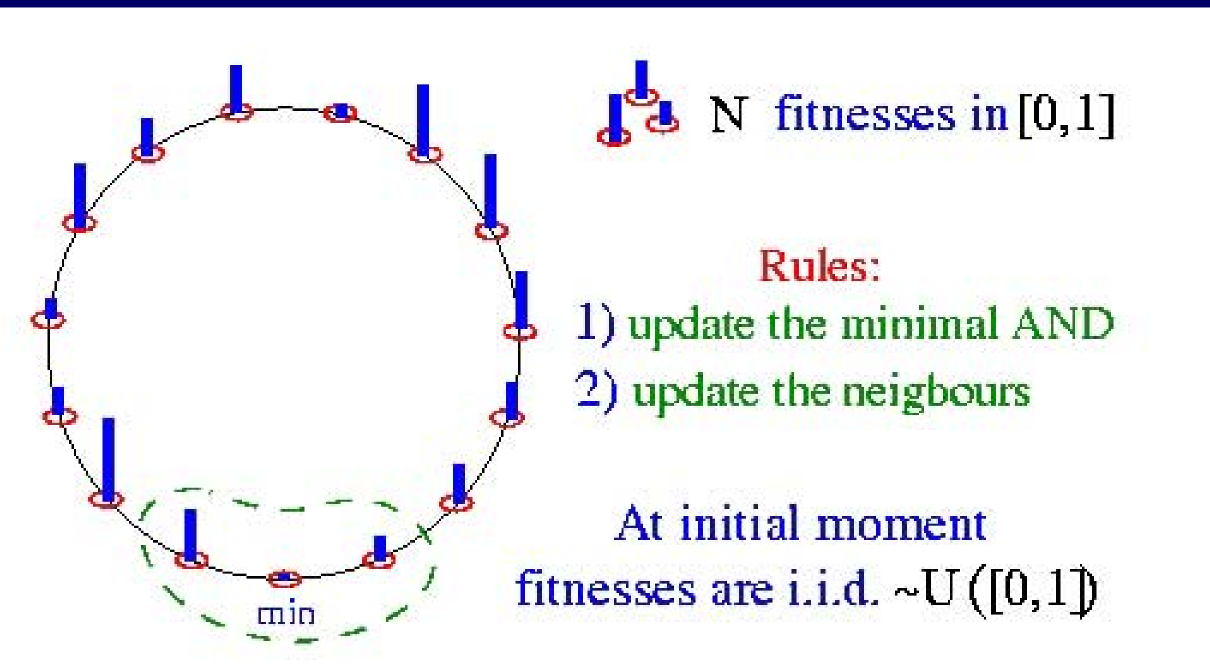
Toy models

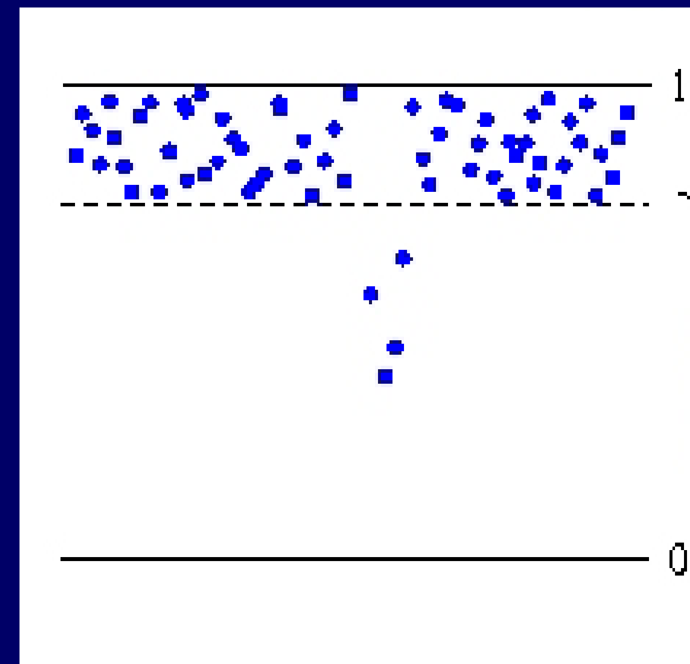
- Springer – slider block mode
  (friction of the blocks on the fixed plate)



A mathematical model: Bak-Sneppen (BS)

- *Toy model for the evolution of species*





- After a short transitory period the system self-organizes with most species having fitness above 0.667
- Avalanches show power-law behavior

Two features of most models and a mathematical result

- *Most SOC models display :*
  - Unstable behavior of the local dynamics
  - Extremal dynamics

- *Theorem If, in a N-agent model :*
  - The single-agent dynamics has positive Lyapunov exponents and
  - The global dynamics is extremal with finite range
  then, in the $N \rightarrow \infty$, the Lyapunov spectrum converges to 0^+

- In the $T \rightarrow \infty$ limit, used to compute the Lyapunov spectrum, the tangent maps have only a nontrivial finite size block during an average time of order $(2r+1)T/N$
- With the Lyapunov spectrum converging to 0^+ there are no dynamical scales. Thus, in the $N \rightarrow \infty$, the system is "tuned" to SOC

(Physica D 214 (2006) 182-186)

- The SOC state has zero measure, but its finite-dimensional projections have full measure.
- It is not an attractor, nor a repeller (not invariant)
- “Ghost weak repeller”
- The invariant measure is like a cloud around the SOC state.

Beyond the classical ergodic parameters

- Lyapunov and conditional exponents and derived quantities depend on the actual (or expected) ***average*** rates of expansion
- ***Fluctuations*** of the expansion rates along the trajectories

Generalized Lyapunov exponents

$$\Lambda(\beta) = \lim_{N\to\infty} \frac{1}{\beta N} \log \int d\mu(x_0) \exp\left[\beta \sum_{n=0}^{N-1} \log\left| f^{'}(x_n) \right| \right]$$

Dynamical Rényi entropies

$$K(\alpha) = \lim_{N\to\infty} \frac{1}{1-\alpha} \frac{1}{N} \log \sum_{i_0 \ldots i_{N-1}} (p(i_0 \ldots i_{N-1}))^{\alpha} \qquad \Lambda(\beta) = K(1-\beta)$$

Cumulants of the Lyapunov spectrum

$$K(\alpha) \cong \sum_{s=1}^{\infty} c_s \frac{(1-\alpha)^{s-1}}{s!}$$

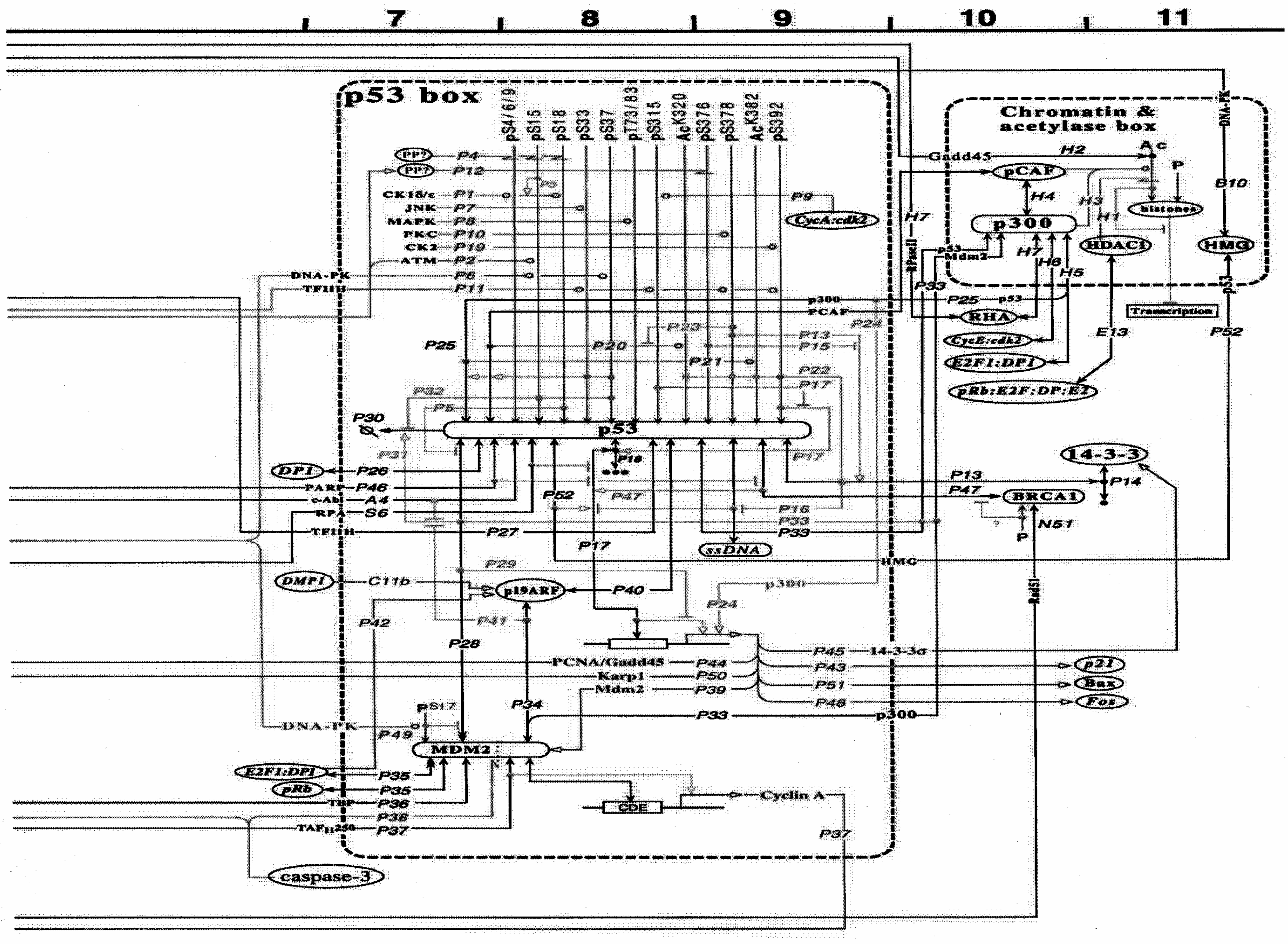
Traces of Hessian powers

$$\frac{1}{2} H_N = \delta_{\alpha,\beta} \delta_{j,k} - (1-\delta_{k,N}) \delta_{k,j-1} \frac{\partial f^{\alpha}(x_k)}{\partial x_k^{\beta}} - (1-\delta_{j,N}) \delta_{j,k-1} \frac{\partial f^{\beta}(x_j)}{\partial x_j^{\alpha}} + (1-\delta_{j,N}) \delta_{j,k} \frac{\partial f^{\gamma}(x_j)}{\partial x_j^{\beta}} \frac{\partial f^{\gamma}(x_j)}{\partial x_j^{\alpha}}$$

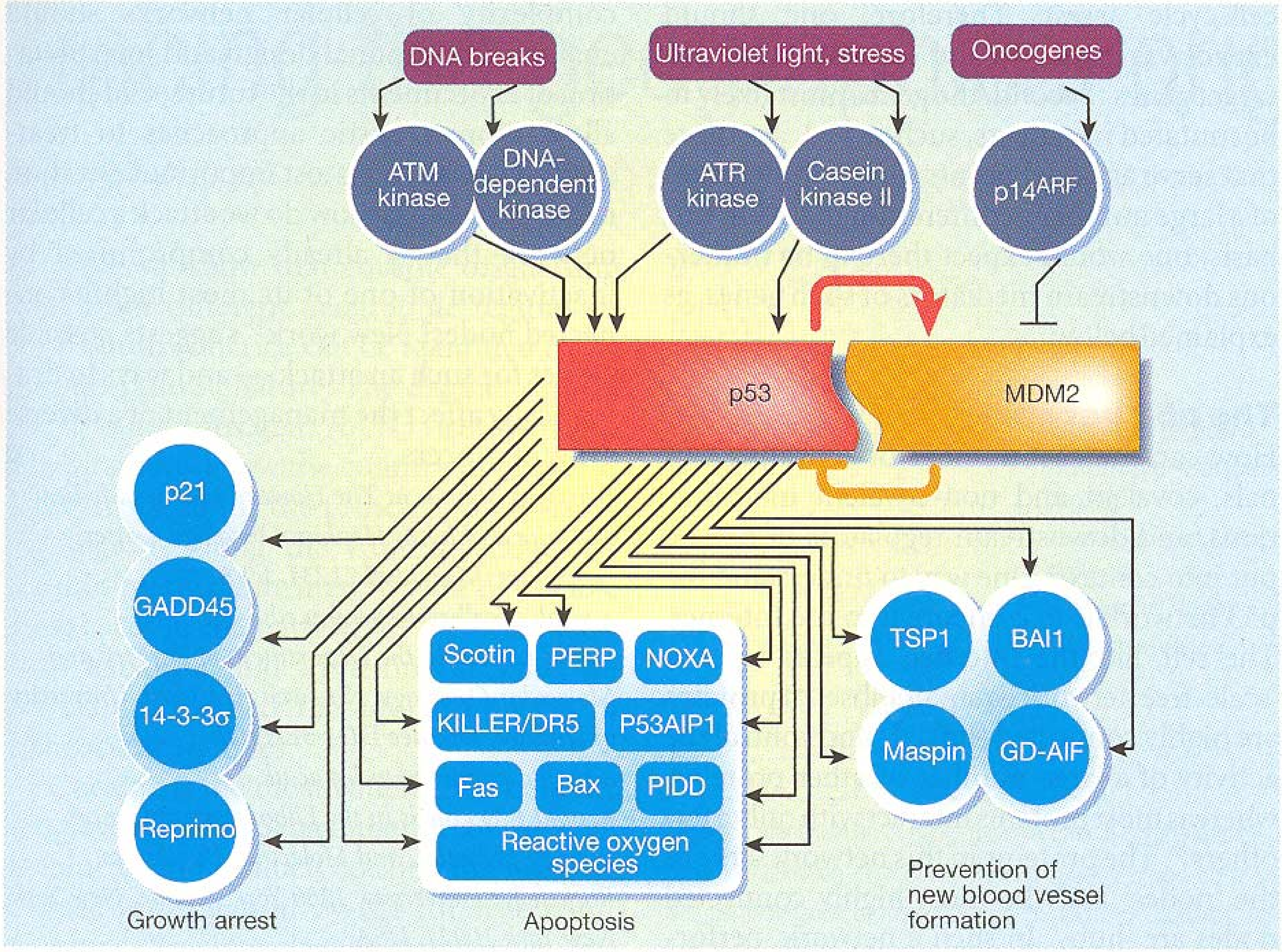
The p53 network

- p53 is a tumor suppressor gene. Its protein acts by :
  - Inhibition of progress through the cell cycle
  - Apoptosis
  - Inhibition of blood-vessel formation (angiogenic phase)
  - Modulating the balance between respiration and glycolysis
- Is activated by :
  - DNA damage (ATM, Chk2)
  - Aberrant growth signals ($p14^{ARF}$)
  - Cell stress (ATR, Casein II, …)
- Is “off” in normal circumstances. Produced at some rate but degraded by ubiquitin labelling (MDM2, ...)
- Is activated through inhibition of degradation
- It activates its own controller
- In many tumors (~ 50%) it is found to be mutated
- In a number of cases normal p53 cannot achieve control

- A very complex network of interactions (an highly connected module in the mammalian cell cycle control and DNA repair system)



7
8
9
10
11
p53 box
pS4/6/9
pS15
pS18
pS33
pS37
pT73/83
pS315
AcK320
pS376
pS378
AcK382
pS392
Chromatin & acetylase box
Gadd45
H2
pCAF
H4
H3
histones
p300
H1
H7
HDAC1
HMG
B10
CK1δ/ε
P1
JNK
P7
MAPK
P8
PKC
P10
CK2
P19
ATM
P2
DNA-PK
P6
TFIIH
P11
P4
P12
P9
CycA:cdk2
P25
P20
P23
P13
P15
P21
P22
P17
P32
P5
P30
p53
P31
P18
DP1
P26
PARP
P46
c-Abl
A4
RPA
S6
P27
P52
P47
P16
P33
ssDNA
HMG
RHA
CycE:cdk2
E2F1:DP1
pRb:E2F:DP:E2
E13
14-3-3
P14
BRCA1
N51
Transcription
DMP1
C11b
p19ARF
P40
P29
P42
P41
P28
P24
PCNA/Gadd45
P44
Karp1
P50
Mdm2
P39
P45
14-3-3σ
P43
p21
P51
Bax
P48
Fos
P34
P33
p300
pS17
P49
MDM2
E2F1:DP1
P35
pRb
TBP
P36
P38
TAFII250
P37
CDE
Cyclin A
caspase-3
Rad51

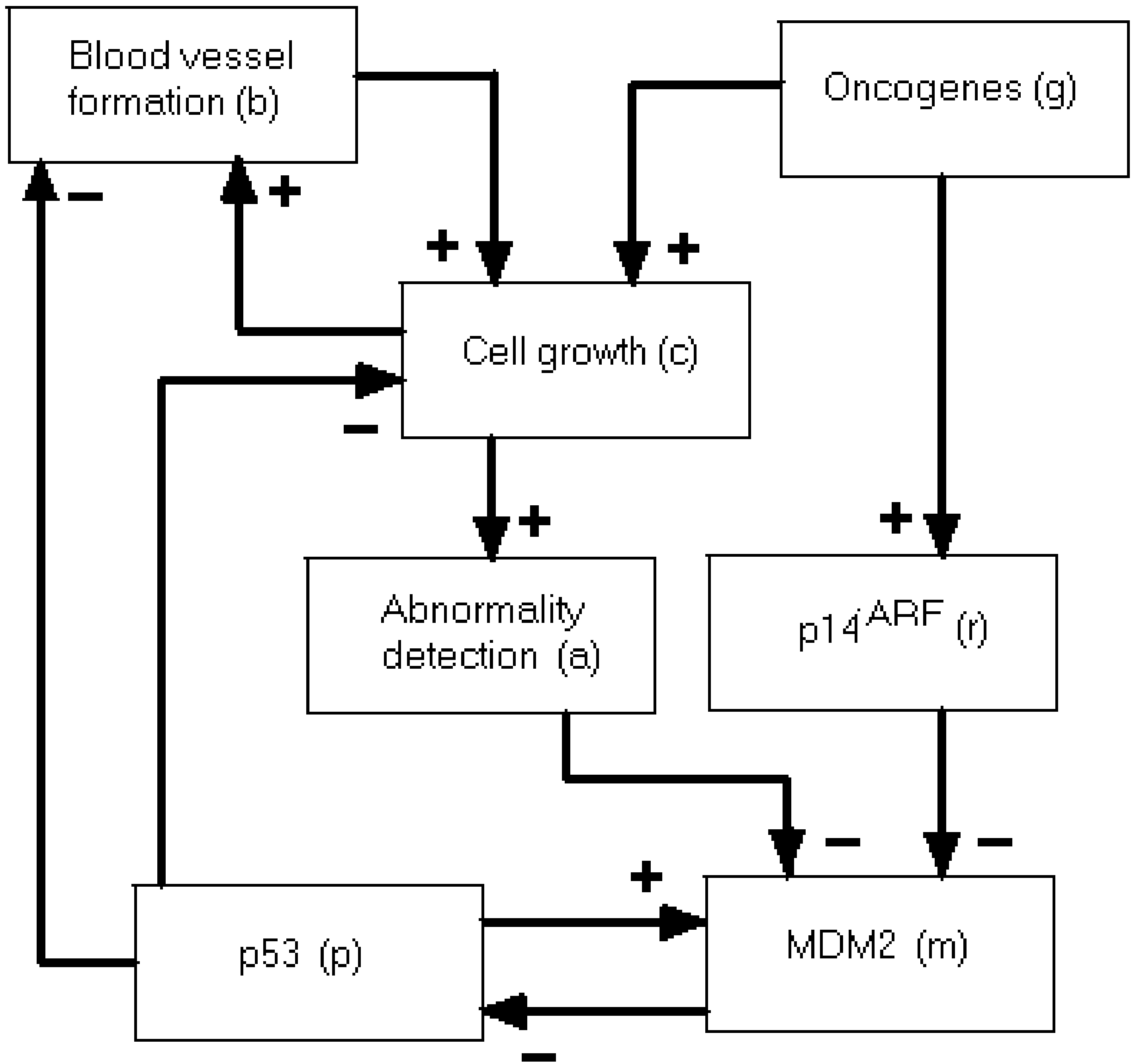


DNA breaks
Ultraviolet light, stress
Oncogenes
ATM kinase
DNA-dependent kinase
ATR kinase
Casein kinase II
p14ARF
p53
MDM2
p21
GADD45
14-3-3σ
Reprimo
Growth arrest
Scotin
PERP
NOXA
KILLER/DR5
P53AIP1
Fas
Bax
PIDD
Reactive oxygen species
Apoptosis
TSP1
BAI1
Maspin
GD-AIF
Prevention of new blood vessel formation

- Our motivation : The non mutated 50%.
- Malfunction of wild type p53

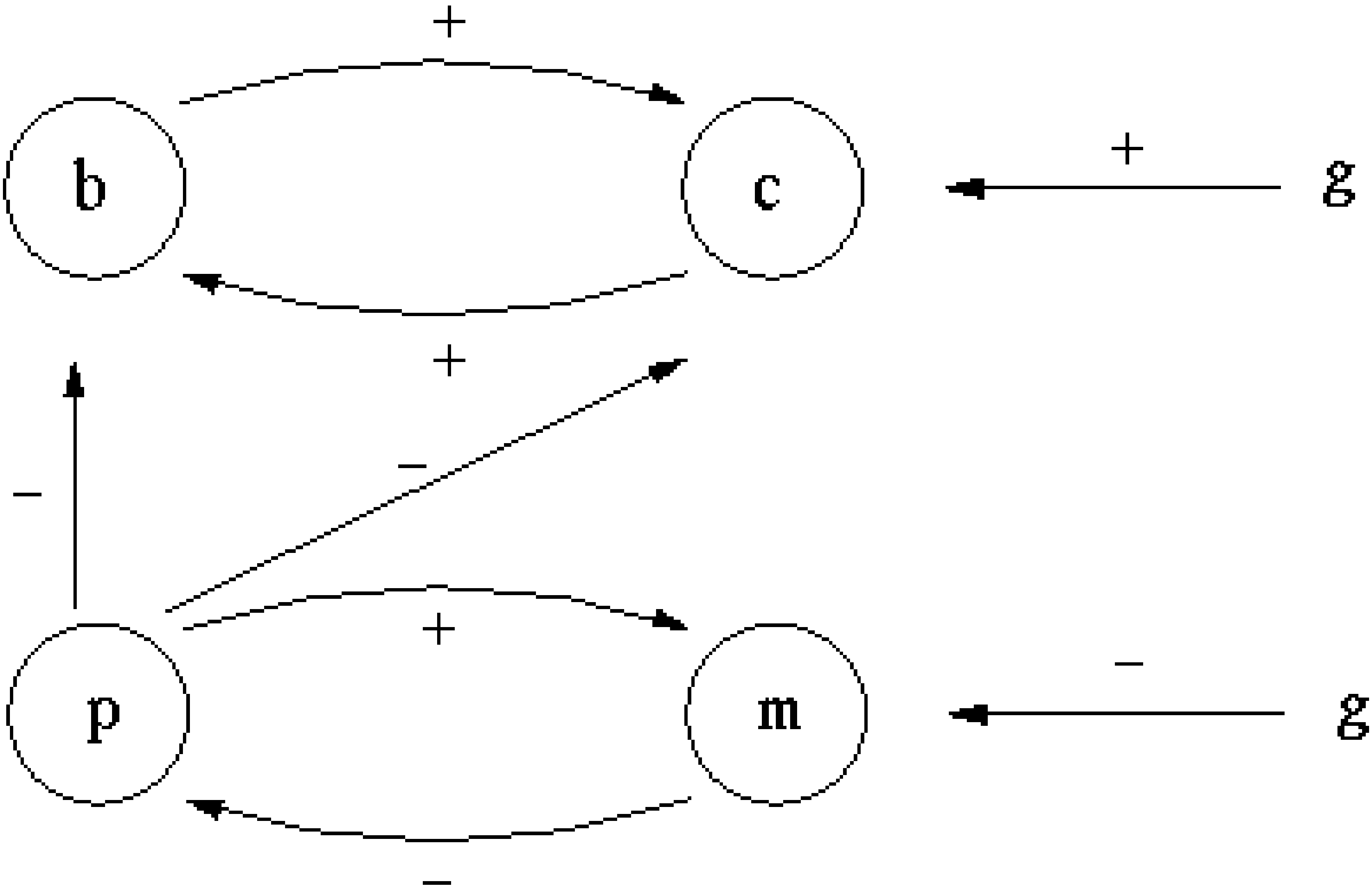
Mechanism of inactivating p53	Typical tumours	Effect of inactivation
Amino-acid-changing mutation in the DNA-binding domain	Colon, breast, lung, bladder, brain, pancreas, stomach, oesophagus and many others	Prevents p53 from binding to specific DNA sequences and activating the adjacent genes
Deletion of the carboxy-terminal domain	Occasional tumours at many different sites	Prevents the formation of tetramers of p53
Multiplication of the MDM2 gene in the genome	Sarcomas, brain	Extra MDM2 stimulates the degradation of p53
Viral infection	Cervix, liver, lymphomas	Products of viral oncogenes bind to and inactivate p53 in the cell, in some cases stimulating p53 degradation
Deletion of the $p14^{ARF}$ gene	Breast, brain, lung and others, expecially when p53 itself is not mutated	Failure to inhibit MDM2 and keep p53 degradation under control
Mislocalization of p53 to the cytoplasm, outside the nucleus	Breast, neuroblastomas	Lack of p53 function (p53 functions only in the nucleus)

- Is there also a dynamical (malfunction) mechanism in the network ?



Blood vessel formation (b)
Oncogenes (g)
Cell growth (c)
Abnormality detection (a)
p14^{ARF} (r)
p53 (p)
MDM2 (m)
+
−

A positive and a negative cycle in interaction



A positive and a negative cycle in interaction

- $p(t+1) = a_p\, p(t) + W_{pm}\, H(T_m - m(t))$
  $m(t+1) = a_m\, m(t) + W_{mp}\, H(p(t) - T_p) + W_{mg}\, H(T_g - g)$
  $c(t+1) = a_c\, c(t) + W_{cb}\, H(b(t) - T_b) + W_{cp}\, H(T_p - p(t)) + W_{cg}\, H(g - T_g)$
  $b(t+1) = a_b\, b(t) + W_{bc}\, H(c(t) - T_c) + W_{bp}\, H(T_p - p(t))$

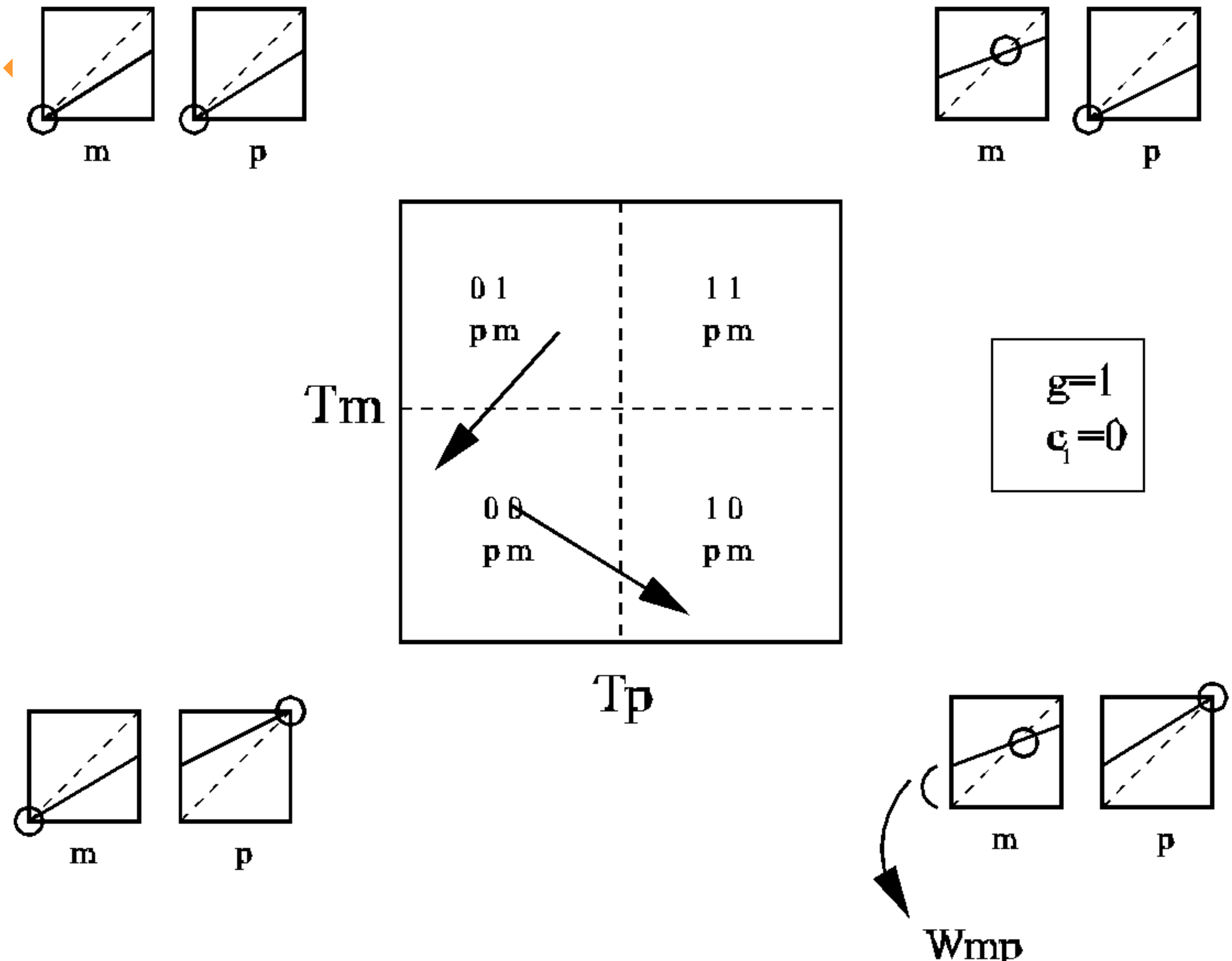
  $a_i + \sum_k W_{ik} = 1$
- ***Piecewise linear dissipative dynamics with thresholds***
- **"Extended" modular approach**

- **Important quantities :**
  $fmg = W_{mg} / (1 - a_m)$; $fmp = 1 - fmg$

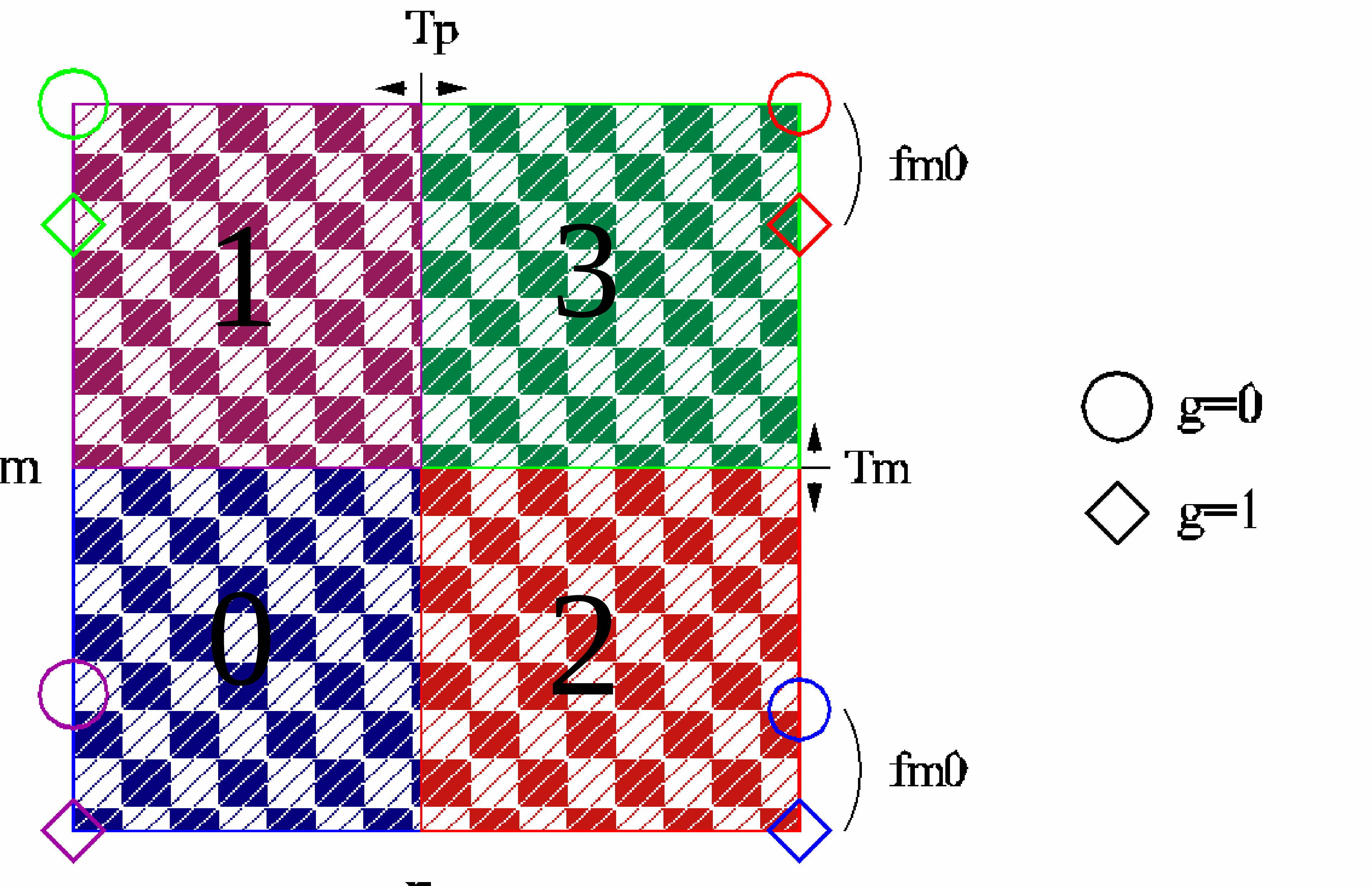
  $fcp = W_{cp} / (1 - a_c)$; $fcb = W_{cb} / (1 - a_c)$; $fcg = 1 - fcp - fcb$

  $fbc = W_{bc} / (1 - a_b)$; $fbp = 1 – fbc$

(*C. Aguirre, J. Martins, RVM, Int. J. Bifurcation and Chaos 16 (2006) 393-394*)



m
p
m
p
0 1
p m
1 1
p m
Tm
g=1
$c_i=0$
0 0
p m
1 0
p m
Tp
m
p
m
p
Wmp



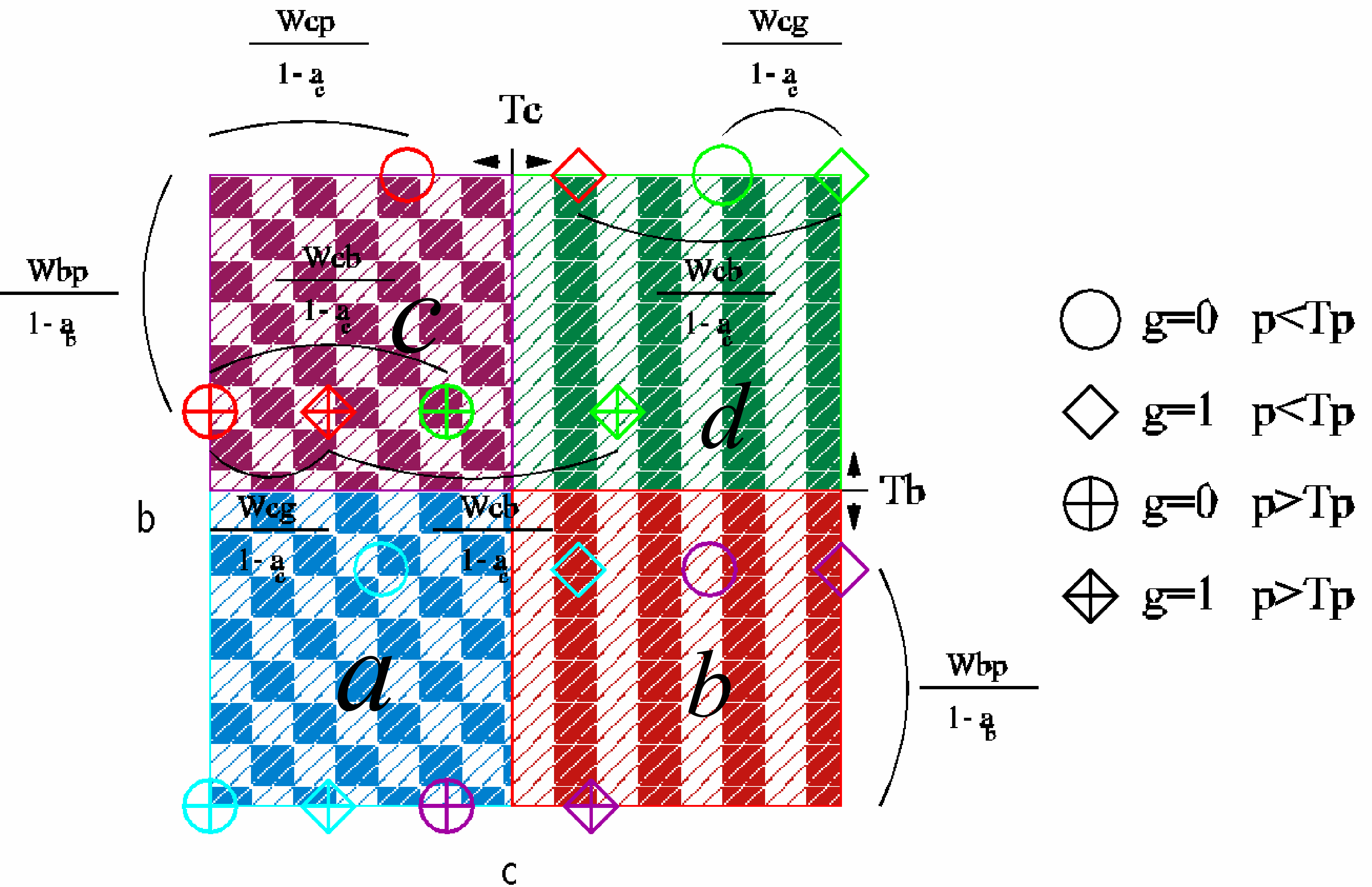
Tp
fm0
1
3
m
Tm
0
2
fm0
g=0
g=1

- **Complete characterization of asymptotic states and coding of trajectories**

- **The qualitative nature of the dynamics only depends on the 4 quantities (fmg, fcp, fcb, fbc) and on the position of the thresholds.**
  The periods (and delays)depend on the specific values of the parameters

The p - m system

	g	Asympt. behavior	Coding
$T_m < fmg$ $T_m > fmp$	$g < T_g$	$p \to 0; m \to fmg$	$\begin{array}{ccccc} 0 & \to & 3 & \to & 2 \\ & & \uparrow & & \\ & & 1 & & \end{array}$
	$g > T_g$	$p \to 1; m \to fmp$	$\begin{array}{ccccc} 2 & \to & 0 & \to & 1 \\ & & \uparrow & & \\ & & 3 & & \end{array}$
$T_m < fmg$ $T_m < fmp$	$g < T_g$	$p \to 0; m \to fmg$	$\begin{array}{ccccc} 0 & \to & 3 & \to & 2 \\ & & \uparrow & & \\ & & 1 & & \end{array}$
	$g > T_g$	oscillation	$\begin{array}{ccc} 0 & \to & 1 \\ \uparrow & & \downarrow \\ 2 & \leftarrow & 3 \end{array}$
$T_m > fmg$ $T_m > fmp$	$g < T_g$	oscillation	$\begin{array}{ccc} 0 & \to & 1 \\ \uparrow & & \downarrow \\ 2 & \leftarrow & 3 \end{array}$
	$g > T_g$	$p \to 1; m \to fmp$	$\begin{array}{ccccc} 2 & \to & 0 & \to & 1 \\ & & \uparrow & & \\ & & 3 & & \end{array}$
$T_m > fmg$ $T_m < fmp$	$g < T_g$	oscillation	$\begin{array}{ccc} 0 & \to & 1 \\ \uparrow & & \downarrow \\ 2 & \leftarrow & 3 \end{array}$
	$g > T_g$	oscillation	$\begin{array}{ccc} 0 & \to & 1 \\ \uparrow & & \downarrow \\ 2 & \leftarrow & 3 \end{array}$


Wcp
1- a c
Wcg
1- a c
Tc
Wbp
1- a b
Wcb
1- a c
c
Wcb
1- a c
d
b
Wcg
1- a c
Wcb
1- a c
Tb
a
b
Wbp
1- a b
c
g=0 p<Tp
g=1 p<Tp
g=0 p>Tp
g=1 p>Tp

The c - b system

TABLE 2A. (Case $g < T_g$ and $p < T_p$)

$g < T_g$, $p < T_p$	Asympt. behavior	Coding
$T_c <$ f cp $T_b <$ f bp	b → 1 c → f cp + f cb	a→d←b ↑ c
$T_c <$ f cp $T_b >$ f bp	b → 1 c → f cp + f cb	a→b→d ↑ c
f cp + f cb > $T_c >$ f cp $T_b <$ f bp	b → 1 c → f cp + f cb	a→c→d ↑ b
f cp + f cb > $T_c >$ f cp $T_b >$ f bp	b → 1; c → f cp + f cb or b → f bp; c → f cp or oscillation	d [a] a [a] b ¿ c
$T_c >$ f cp + f cb $T_b <$ f bp	b → f bp c → f cp + f cb	a→c←b ↑ d
$T_c >$ f cp + f cb $T_b >$ f bp	b → f bp c → f cp	b→c→a ↑ d

TABLE 2B. (Case $g < T_g$ and $p > T_p$)

$g < T_g$, $p > T_p$	Asympt. behavior	Coding
$T_c <$ f cb $T_b <$ f bc	$b \to 0$; $c \to 0$ or $b \to$ f bc; $c \to$ f cb or oscillation	a [a] d [a] b ¿ c
$T_c <$ f cb $T_b >$ f bc	$b \to 0$ $c \to 0$	$\begin{array}{ccccc} c & \to & b & \to & a \\ & & \uparrow & & \\ & & d & & \end{array}$
$T_c >$ f cb $T_b <$ f bc	$b \to 0$ $c \to 0$	$\begin{array}{ccccc} b & \to & c & \to & a \\ & & \uparrow & & \\ & & d & & \end{array}$
$T_c >$ f cb $T_b >$ f bc	$b \to 0$ $c \to 0$	$\begin{array}{ccccc} c & \to & a & \leftarrow & b \\ & & \uparrow & & \\ & & d & & \end{array}$

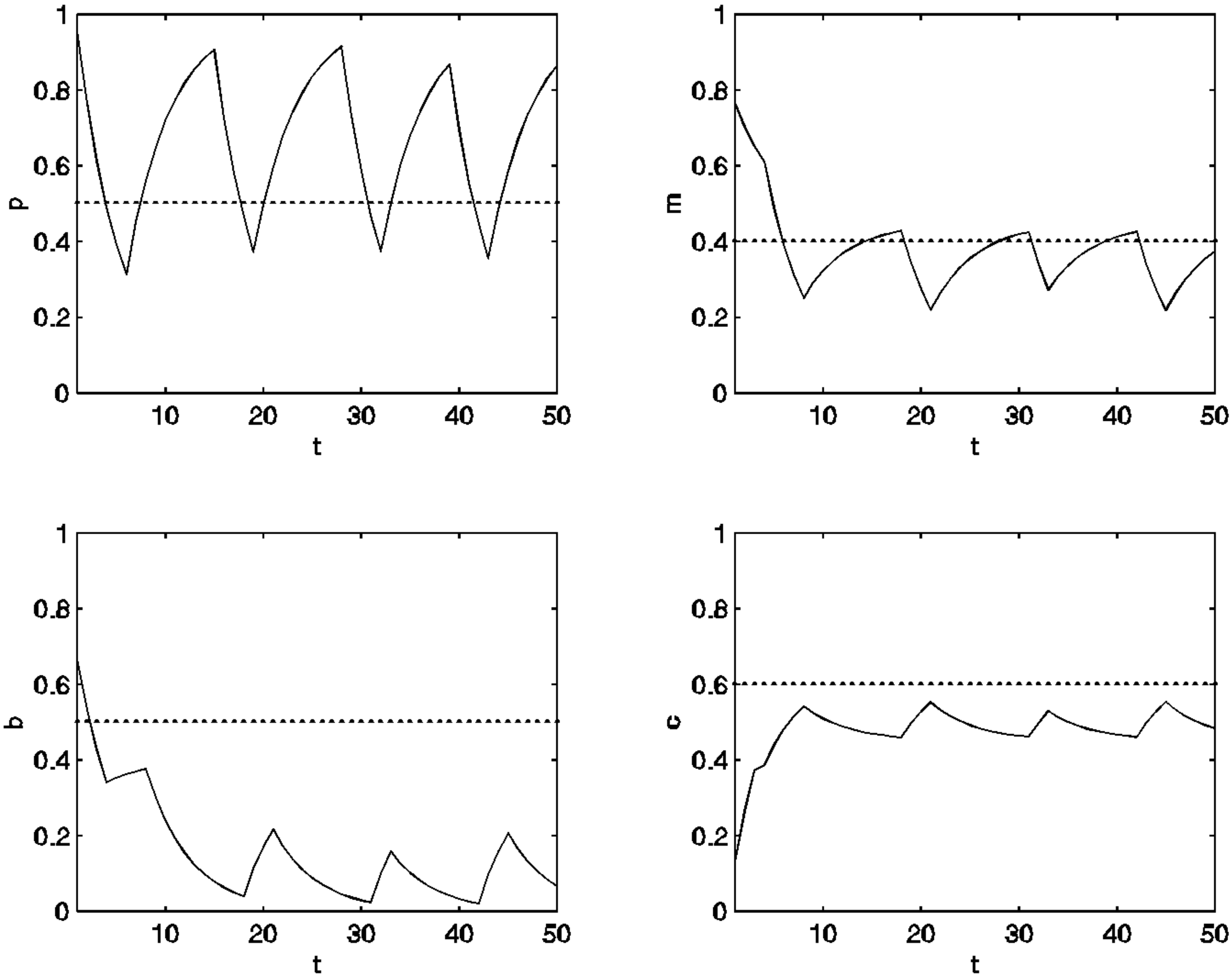
TABLE 2C. (Case $g > T_g$ and $p < T_p$)

$g > T_g$, $p < T_p$	Asympt. behavior	Coding
$T_c <$ f cg + f cp $T_b <$ f bp	$b \to 1$ $c \to 1$	$c \to d \leftarrow b$ $\uparrow$ a
$T_c <$ f cg + f cp $T_b >$ f bp	$b \to 1$ $c \to 1$	$a \to b \to d$ $\uparrow$ c
$T_c >$ f cg + f cp $T_b <$ f bp	$b \to 1$ $c \to 1$	$a \to c \to d$ $\uparrow$ b
$T_c >$ f cg + f cp $T_b >$ f bp	$b \to 1$; $c \to 1$ or $b \to$ f bp; $c \to$ f cg + f cp or oscillation	d [a] a [a] b ¿ c

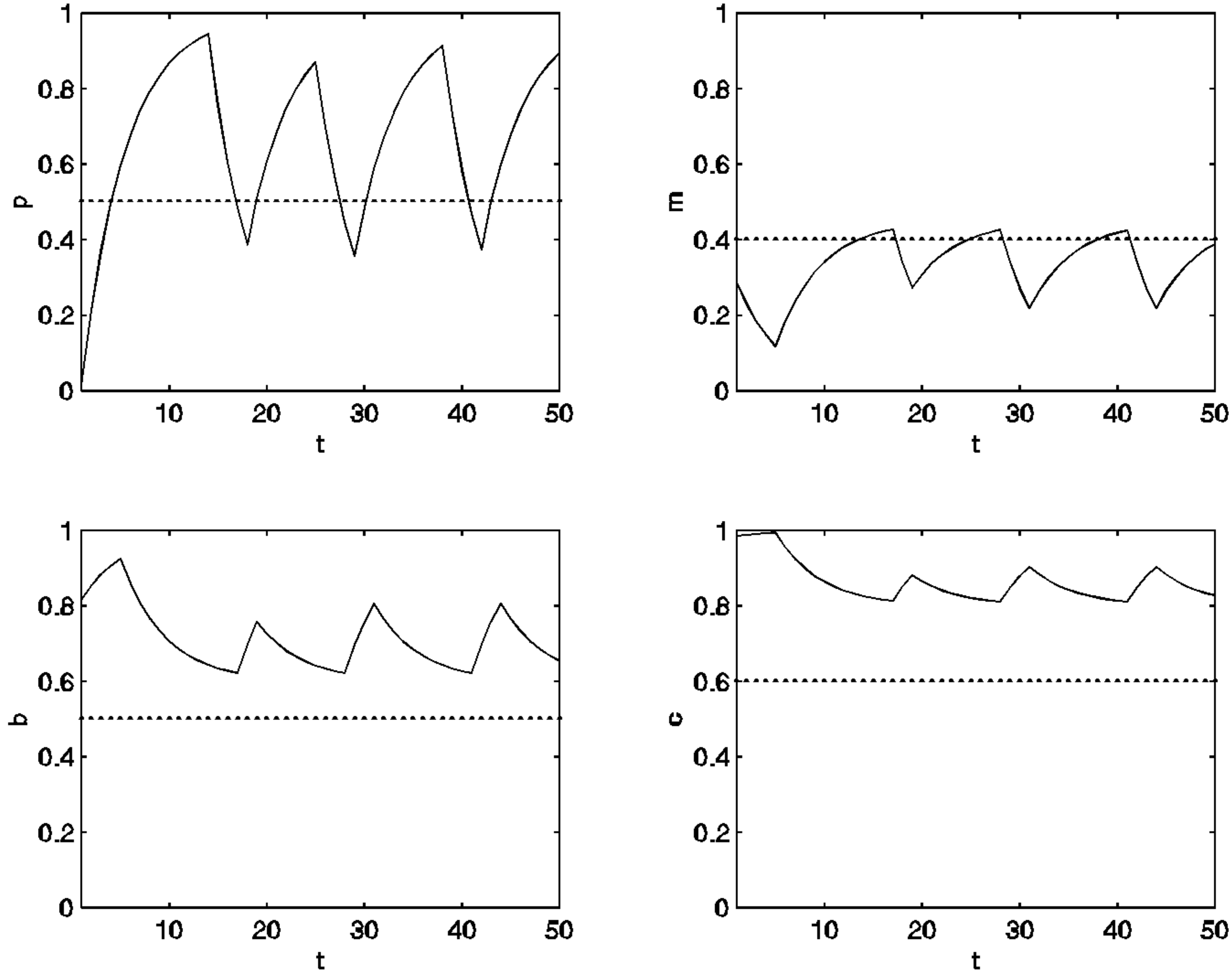
TABLE 2D. (Case $g > T_g$ and $p > T_p$)

$g > T_g$, $p > T_p$	Asympt. behavior	Coding
$T_c < f\ cg$ $T_b < f\ bc$	$b \to f\ bc$ $c \to f\ cg + f\ cb$	$a \to b \to d$ $\uparrow$ c
$T_c < f\ cg$ $T_b > f\ bc$	$b \to f\ bc$ $c \to f\ cg$	$a \to b \leftarrow d$ $\uparrow$ c
$f\ cg + f\ cb > T_c > f\ cg$ $T_b < f\ bc$	$b \to f\ bc$; $c \to f\ cg + f\ cb$ or $b \to 0$; $c \to f\ cg$ or oscillation	d [a] a [a] b ¿ c
$f\ cg + f\ cb > T_c > f\ cg$ $T_b > f\ bc$	$b \to 0$ $c \to f\ cg$	$c \to b \to a$ $\uparrow$ d
$T_c > f\ cg + f\ cb$ $T_b < f\ bc$	$b \to 0$ $c \to f\ cg$	$b \to c \to a$ $\uparrow$ d
$T_c > f\ cg + f\ cb$ $T_b > f\ bc$	$b \to 0$ $c \to f\ cg$	$c \to a \leftarrow b$ $\uparrow$ d

- Complete characterization of asymptotic states and coding of trajectories.
- Biological realism imposes some conditions on the threshold values :
  $T_m <$ fmg (for g< T_g p53 is at a low level)
  $T_c >$ fcp + fcb ; $T_b <$ fbp (for g< T_g cell growth should not be explosive)
- Most interesting case : Expressed p53 ($p>T_p$) with oncogenes ($g>T_g$)
  - $T_c >$ fcg+fcb $\Rightarrow$ effective control of cell growth
  - $T_c <$ fcg and $T_b <$ fbc $\Rightarrow$ no control
  - fcg < $T_c <$ fcg+fcb ; $T_b <$ fbc $\Rightarrow$ depends on initial conditions (a matter of chance)
- fmg = 0.55 ; fbp = 0.4 ; fcb = 0.35 ; fcp = 0.2 ; a_i =0.8



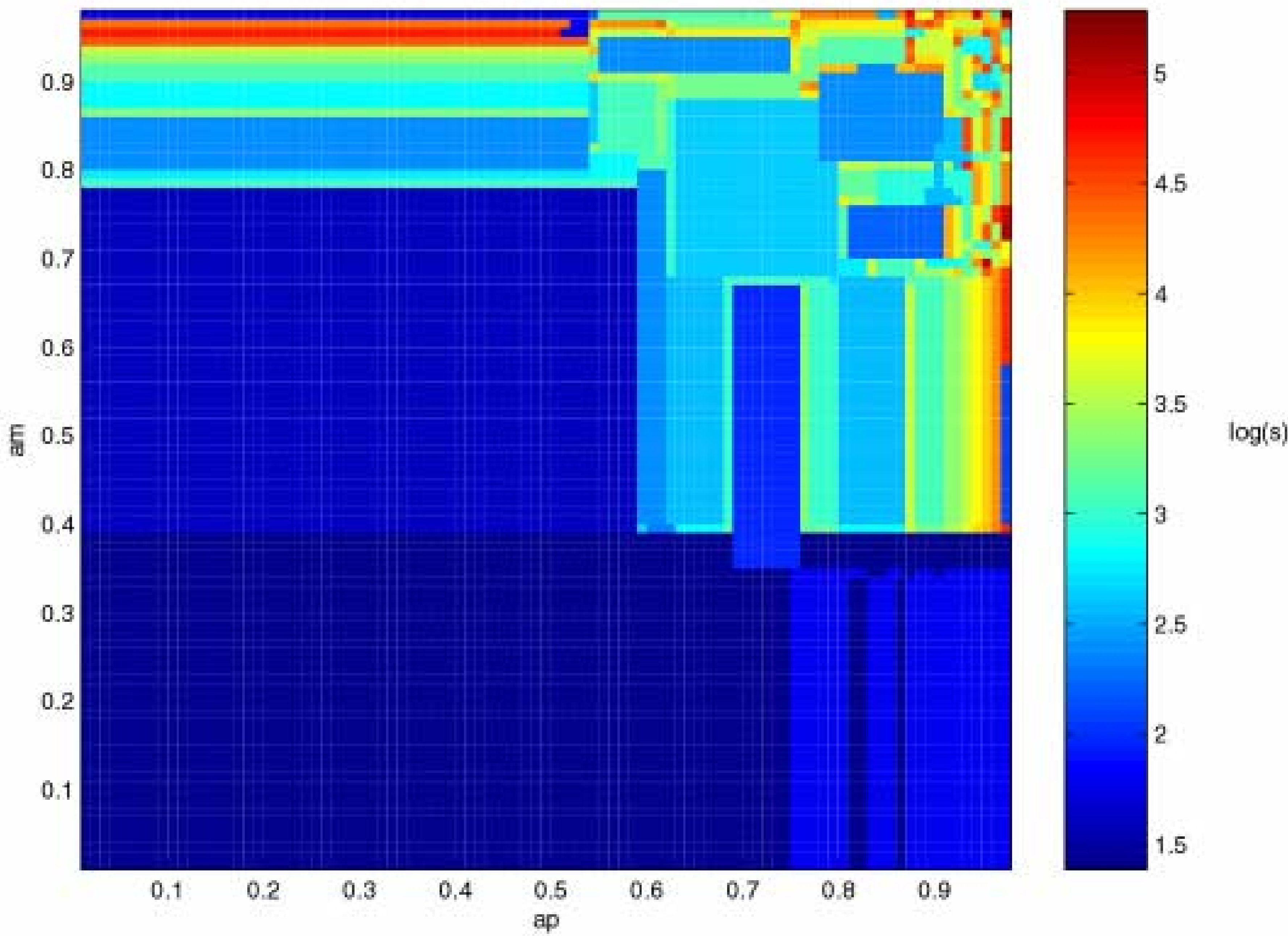
p
m
b
c
t
t
t
t



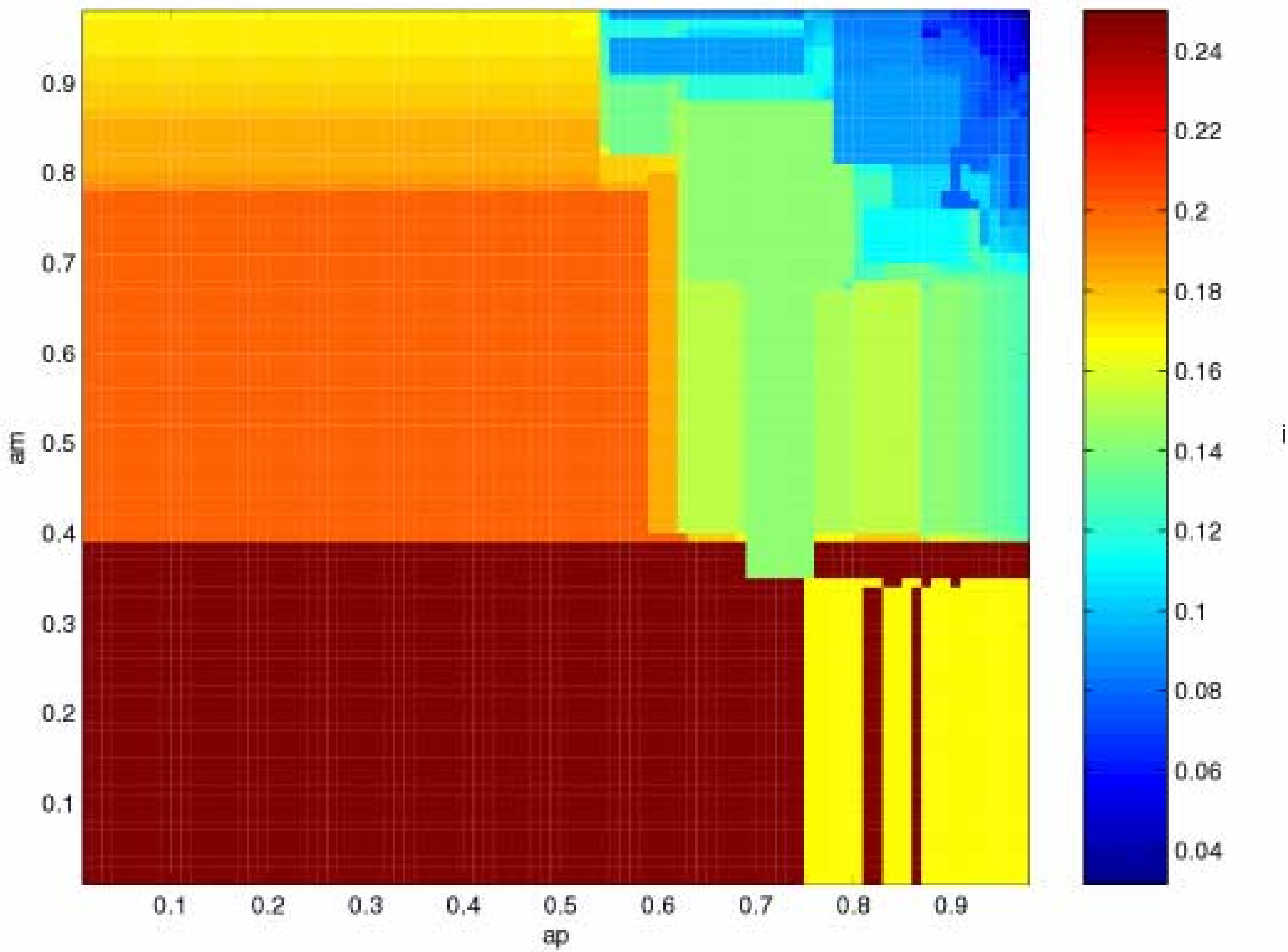
p
m
b
c
t
0
0.2
0.4
0.6
0.8
1
10
20
30
40
50

Periods and rotation numbers

- $g > T_g$; $T_p = 0.5$; $T_m = 0.4$; $fmg = 0.5$



0.1
0.2
0.3
0.4
0.5
0.6
0.7
0.8
0.9
ap
am
1.5
2
2.5
3
3.5
4
4.5
5
log(s)



0.9
0.8
0.7
0.6
0.5
0.4
0.3
0.2
0.1
am
0.1
0.2
0.3
0.4
0.5
0.6
0.7
0.8
0.9
ap
0.24
0.22
0.2
0.18
0.16
0.14
0.12
0.1
0.08
0.06
0.04
i

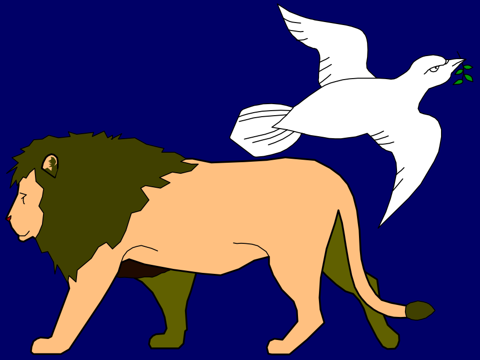
Networks in evolutionary sociology. Strong reciprocity

- Nash equilibrium and experimental games
- Homo Oeconomicus rejected in all cases
- The player's behavior is strongly correlated with existing social norms in their societies and market structure
- Human decision problems involve a mixture of self-interest and a background of (internalized) social norms
- Exits Homo Oeconomicus
- Enters Homo Reciprocans (Samuel Bowles, Herbert Gintis) Strong reciprocity

(*Advances in Complex Syst. 7 (2004) 357-368*)

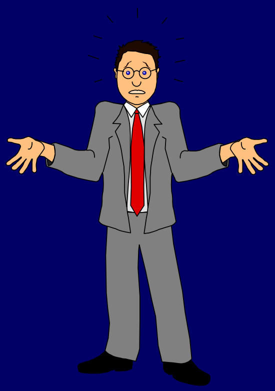
Homo reciprocans

- Homo reciprocans comes to new social situations with a propensity to cooperate and share, responds to cooperative behavior by maintaining or increasing the level of cooperation and responds to selfish free-riding behavior on the part of others by retaliating, even at a cost to himself and even when he could not expect future personal gains
- Strong reciprocity is a form of altruism in that it benefits others at the expense of the individual that exhibits this trait.



Homo reciprocans

- Monitoring and punishing selfish agents or norm violators is a costly (and dangerous) activity without immediate direct benefit to the agent that performs it
- It seems that the strong reciprocity trait could not invade a population of self-interested agents, nor be maintained in a stable population equilibrium
- Not evolutionary stable ?



Homo reciprocans. The Bowles-Gintis model

- Small hunter-gatherer bands of the late Pleistocene
- Population of size N with two species of agents:
- Reciprocators (R-agents)
- Self-interested (S-agents)
- Public goods activity: each agent can produce a maximum amount of goods q at cost b
- The benefit that an S-agent takes from shirking is the cost of effort $b(\sigma)$, σ being the fraction of shirking time
- $b(0)=b$ $b(1)=0$ $b'(\sigma)<0$ $b''(\sigma)>0$ $q(1-\sigma)>b(\sigma)$
- At every level of effort, working helps the group more than it hurts the worker

Homo reciprocans. The Bowles-Gintis model

- R-agents never shirk and punish free-riders at cost $c\sigma$, the cost being shared by all R-agents
- f = fraction of R-agents
- For an S-agent the estimated cost of being punished is $s\sigma$. He chooses σ^* to minimize the function

$$B(\sigma) = b(\sigma) + s\, f\, \sigma + q(1-\sigma)/N$$

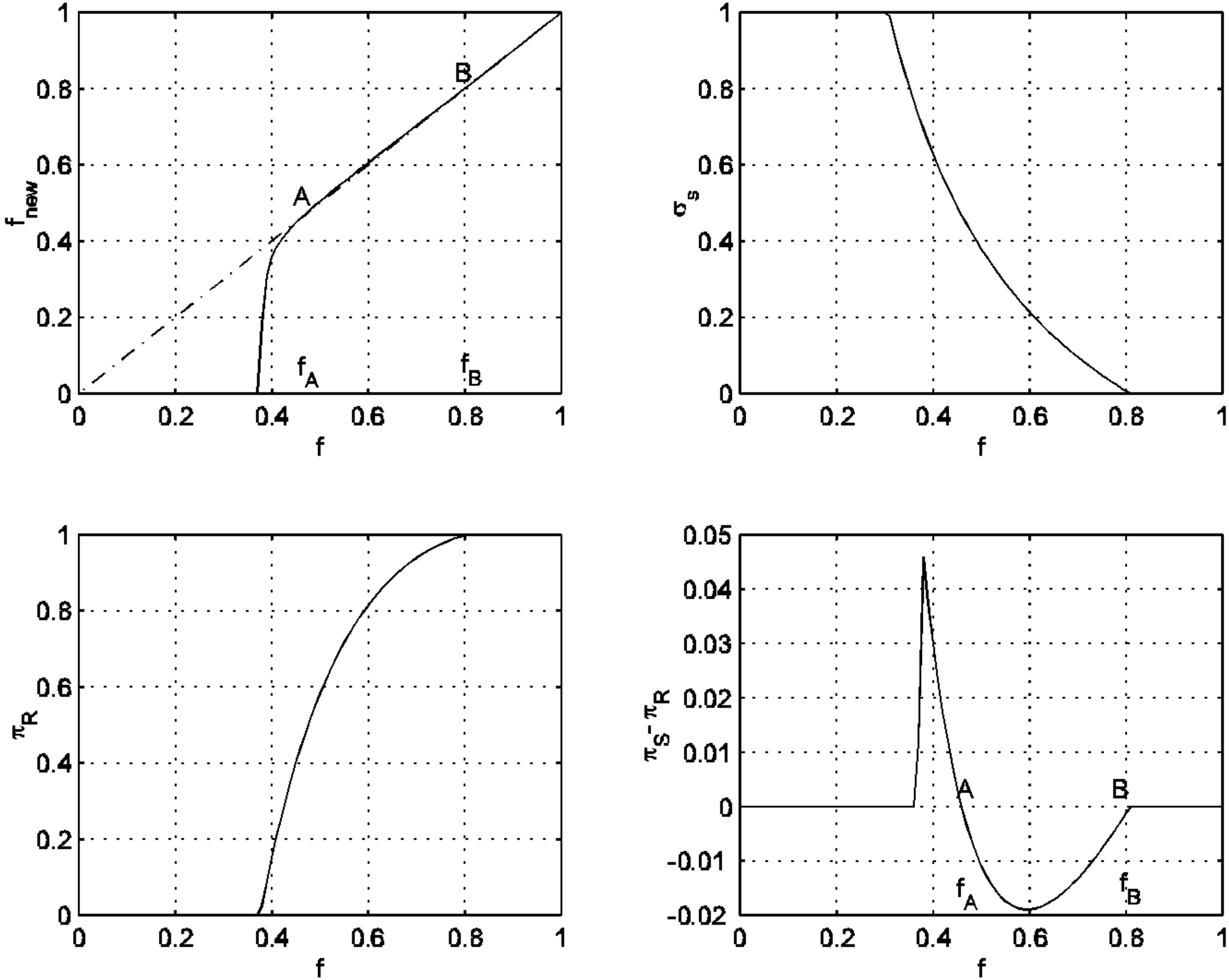
- Fitness of each species :

$$\pi_S = \max(\, q(1-(1-f)\,\sigma^*) - b(\sigma^*) - \gamma\, f\, \sigma^*\,,\, 0)$$

$$\pi_R = \max(\, q(1-(1-f)\,\sigma^*) - b - c(1-f)N\sigma/(Nf)\,,\, 0)$$

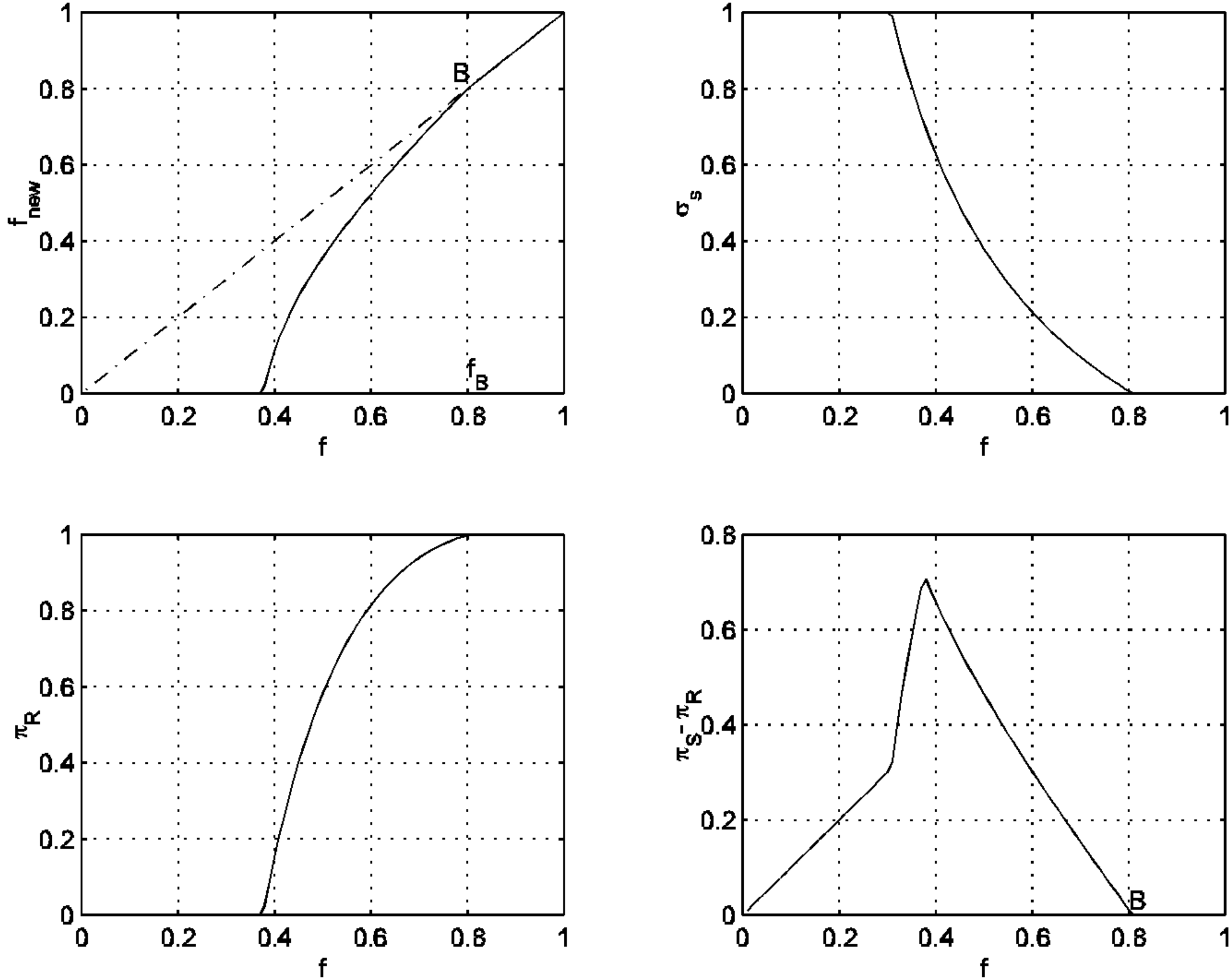
- Replicator dynamics

$$f_{new} = f\,\frac{\Pi_R(f)}{(1-f)\,\Pi_S + f\,\Pi_R(f)}$$



q=2, b=1, s=2, c=0.1, γ=3.5, N=1000

Fig.1



q=2, b=1, s=2, c=0.1, γ=1, N=1000

Fig.2

Homo reciprocans. The Bowles-Gintis model

- If γ is large enough, the map has an unstable fixed point (A) and a left-stable one (B)
- Between B and f = 1 there is a continuum of marginally stable fixed points
- For smaller γ the region between A and B disappears and only the marginally stable fixed points remain
- The asymptotic behavior corresponds either to f = 0 ($\sigma^*=1$) or to f between 0 and 1 but $\sigma^*=0$
- When $f \neq 0$, reciprocators and shirkers remain in the population but shirkers choose not to shirk
- For initial f smaller than f_A the fraction of reciprocators falls very rapidly to zero

Homo reciprocans. The Bowles-Gintis model

- *Intragroup dynamics* :
  either reciprocators are eliminated from the population or they remain in equilibrium with a large number of shirkers (which do not shirk for fear of being punished)
- Intragroup dynamics cannot explain how strong reciprocity might have become a dominant trait.
- *Intergroup dynamics* :
  Only groups that contain at the start $f>f_A$ will have in the end a nonzero fitness. All others suffer a "tragedy of the commons" with final zero fitness.
  Groups with reciprocators tend to dominate and impose an above average predominance of the reciprocator trait.
- For initial f smaller than f_A the fraction of reciprocators falls very rapidly to zero

Network dependence of strong reciprocity

- What happens when, later on, the Pleistocene reciprocators and their fellow shirkers become imbedded into a larger society?
- Monitoring and punishment of shirkers by reciprocators necessarily looses its global collective nature.
- It becomes the business of the neighbors of the shirker
- Monitoring and (or) punishing free-riders requires force to insure the effectiveness of the punishment and to make the punisher safe from direct retaliation from the violator.
- Central authorities play a role in the control of serious offenses, but not so much on the day to day monitoring of public goods work

Network dependence of strong reciprocity

- Punishing a norm-violator requires a minimal social power and consensus. Punishment occurs only if at least two neighbors agree to do so.
- R-agents and (1-f) S-agents placed at random in a network where, on average, each agent is connected to k other agents, rewired with probability β
- Each reciprocator, on detecting an S-agent, looks for another reciprocator in his own neighborhood also connected to S-agent. If he finds one, he punishes by an amount proportional to the fraction of shirking.
- The amount of work an S-agent does is inversely proportional to the number of reciprocators in his neighborhood.

Network dependence of strong reciprocity

- Wk( ) = work vector
- Pu( ) = punishment vector
- Cpu( ) = cost of punishment vector
- f = fraction of reciprocators
- q = maximum amount of goods produced by each agent
- b = cost of work
- c = cost to punish
- γ = cost to be punished

Network dependence of strong reciprocity

- **Average fitness of R-agents and S-agents**

$$\pi'_R = \frac{q}{N}\sum_{all} Wk\left(i\right) - \frac{b}{fN}\sum_{i\in R} Wk\left(i\right) - \frac{c}{fN}\sum_{i\in R} Cpu\left(i\right)$$

$$\pi'_S = \frac{q}{N}\sum_{all} Wk\left(i\right) - \frac{b}{\left(1-f\right)N}\sum_{i\in S} Wk\left(i\right) - \frac{\gamma}{\left(1-f\right)N}\sum_{i\in S} Pu\left(i\right)$$

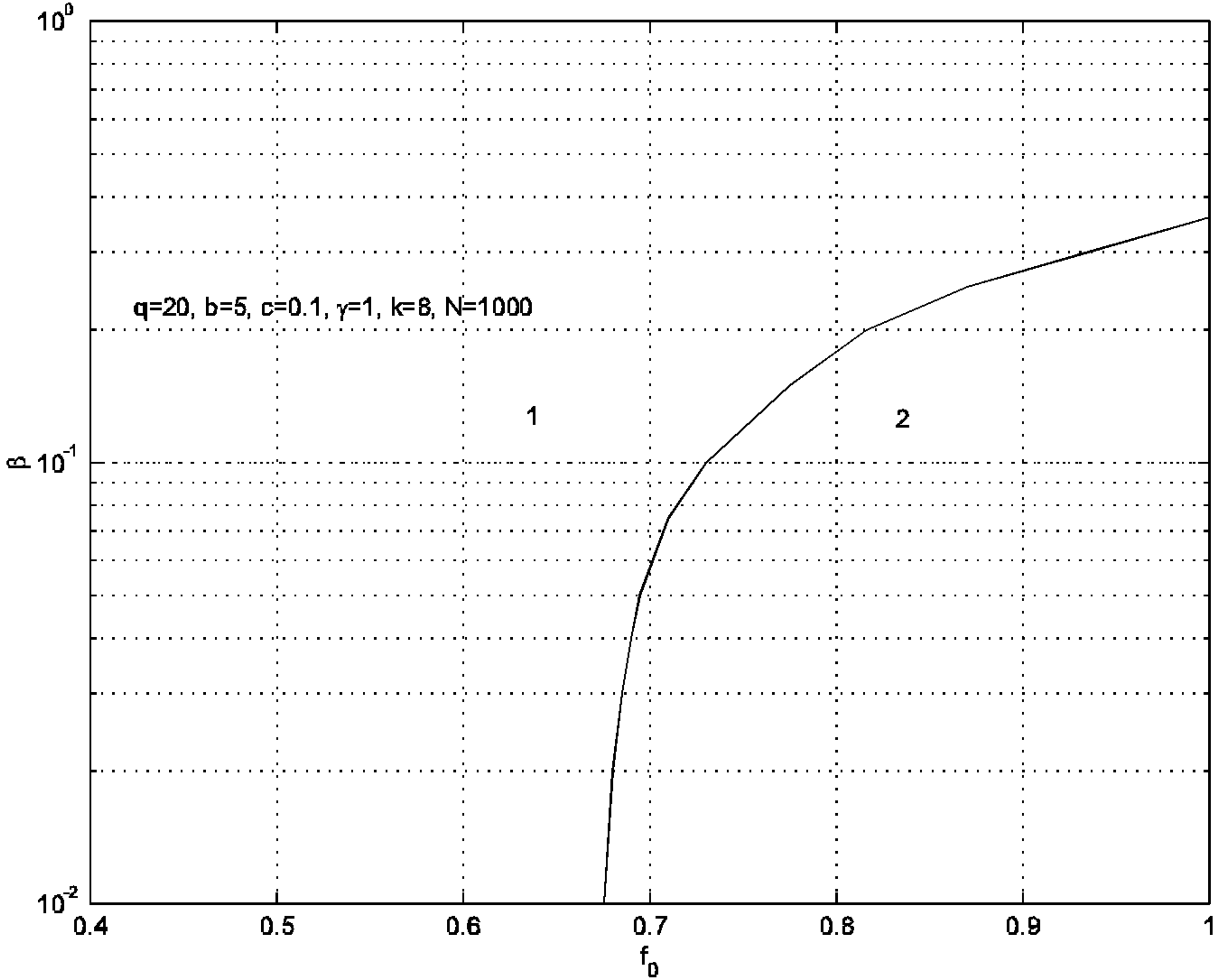


Fig.3

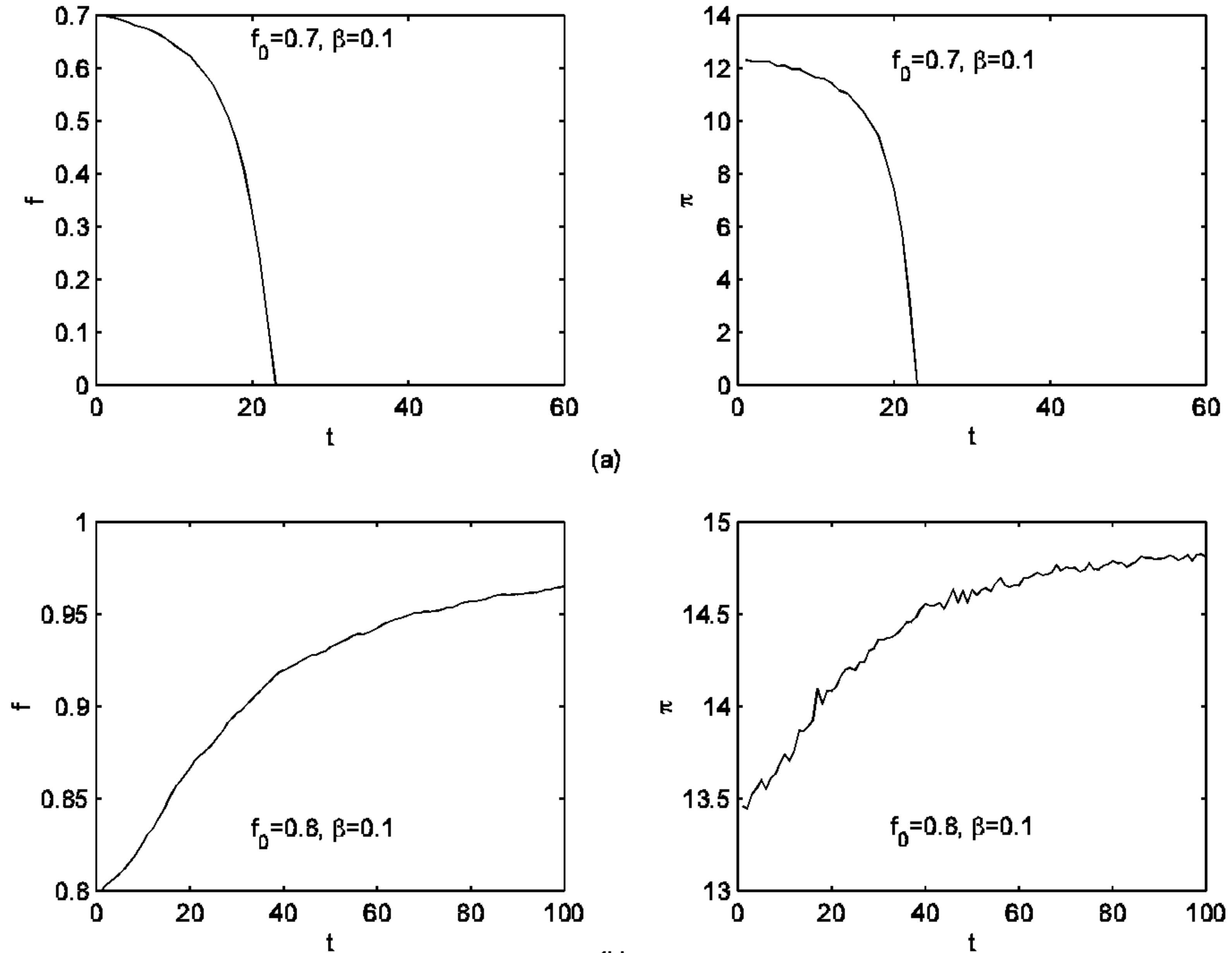

f_0=0.7, β=0.1
f
t
0.7
0.6
0.5
0.4
0.3
0.2
0.1
0
0
20
40
60
f_0=0.7, β=0.1
π
t
14
12
10
8
6
4
2
0
0
20
40
60
(a)
f_0=0.8, β=0.1
f
t
1
0.95
0.9
0.85
0.8
0
20
40
60
80
100
f_0=0.8, β=0.1
π
t
15
14.5
14
13.5
13
0
20
40
60
80
100
(b)

Fig.4

Conclusions

- 1 - In small groups with collective monitoring, the interplay of intra- an intergroup dynamics makes the emergence of the strong reciprocity trait a likely event.
- 2 - Self-interested (S-agents) are not completely invaded. If the social structure changes, they may be a source of instability and invade the population.
- 3 - In a large population, monitoring of the public goods behavior cannot be a fully collective activity and punishment of free-riders requires a certain amount of local consensus among reciprocators.
- 4 - The clustering nature of the society plays an important role in the maintenance and evolution of the reciprocator trait.

Conclusions

- Modern societies are "small worlds" in the sense of short path lengths but not necessarily in the sense of also maintaining a high degree of clustering.
- Therefore if the reciprocator trait has a high cultural component, it may very well happen that, eventually, we will see homo oeconomicus leaving the benches of economy classes for a life on the streets.