# Slow dynamics of the contact processes on complex networks

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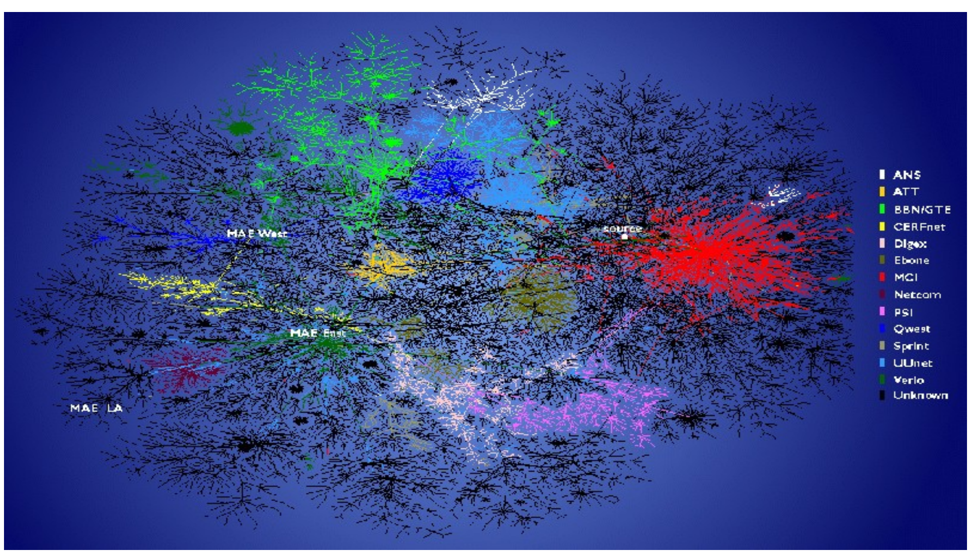
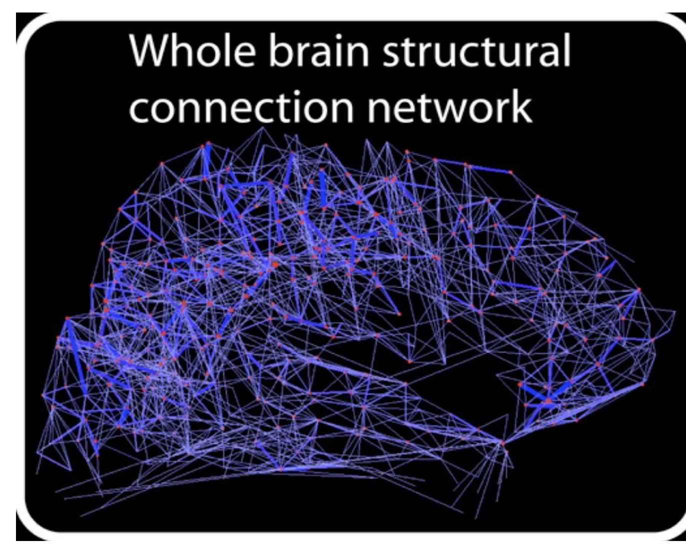
***RESEARCH INSTITUTE FOR NATURAL SCIENCES (MFA) BUDAPEST***

- *Exploration of complex networks is flourishing since ~2000 (Barabási & Albert)*
- *Dynamical systems living on networks are of current interest*
- *Origin of slow (dynamic) scaling behavior in internet, brain, quantum systems,… etc.*
- *Open question : Complex networks + quenched disorder ?*

*Internet*



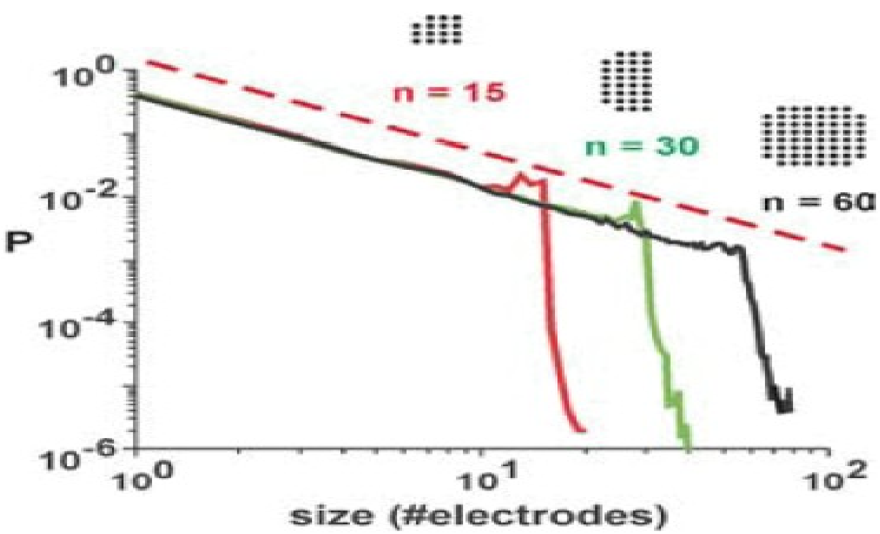




*Diffusion spectrum imaging*

# Observed slow dynamics in networks

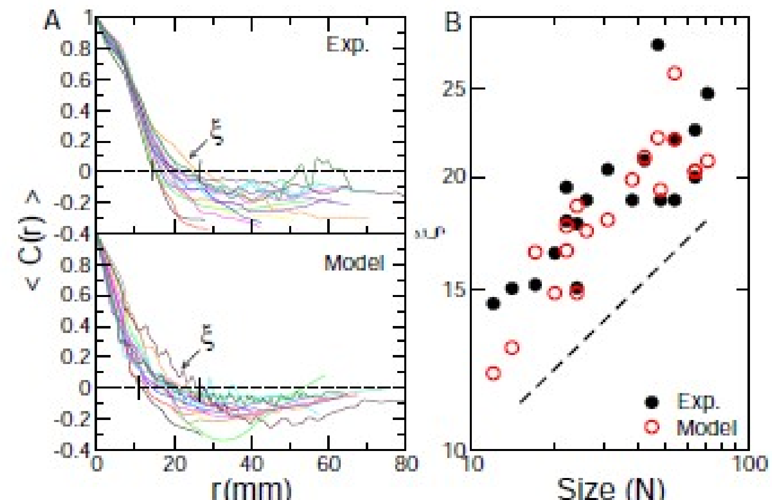
- Brain : Size distribution of neural avalanches

  *G. Werner : Biosystems, 90 (2007) 496,*

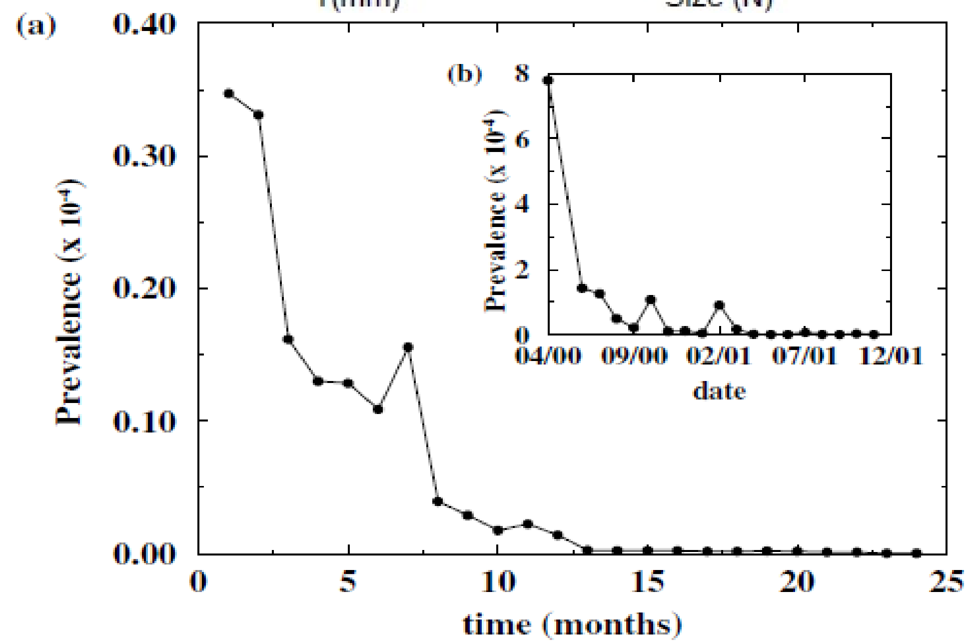



Correlation length ($\xi$) diverges

*Tagliazucchi & Chialvo (2012) : Brain complexity born out of criticality.*




- Internet: worm recovery time is slow:



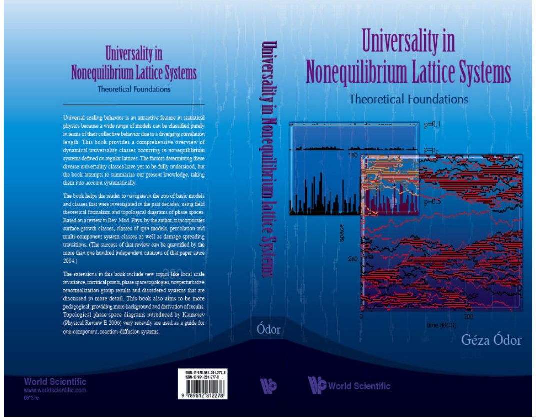

Small world networks → fast dynamics

**What is the cause ?**

# Slow dynamics, scaling in nonequilibrium

Scaling and universality classes appear in complex system due to : $\xi \to \infty$
i.e: near critical points

Basic models classified by universal scaling behavior
*G. Ódor: Universality in nonequilibrium system*
*(World Scientific 2008), Rev. Mod. Phys. 2004*

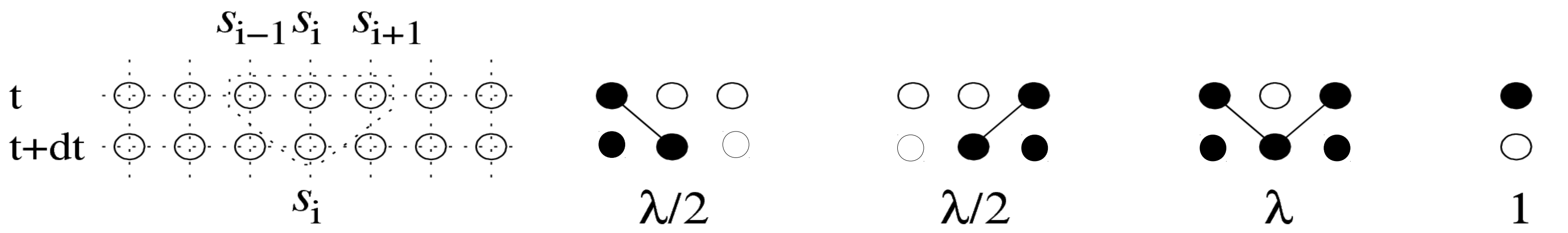



- **Why don't we see universal behavior in networks ?**
- **Tuning to critical point is needed !**

**I'll show a possible way to understand this**

# Modelling dynamics on fundamental (nonequilibrium) models

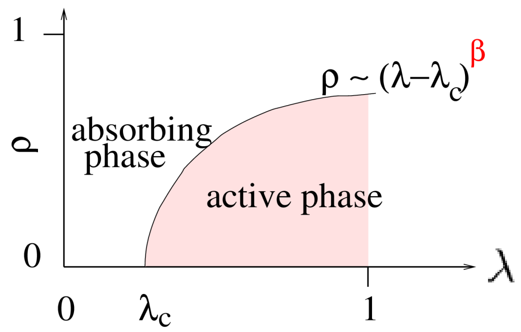
Prototype: **C**ontact **P**rocess describing “epidemic/info” propagation (1d) :




- In regular, Euclidean lattices:
  order parameter: $\rho$ the density of active sites
  phase transition between active and inactive (absorbing)
  Critical point : $\lambda_c > 0$

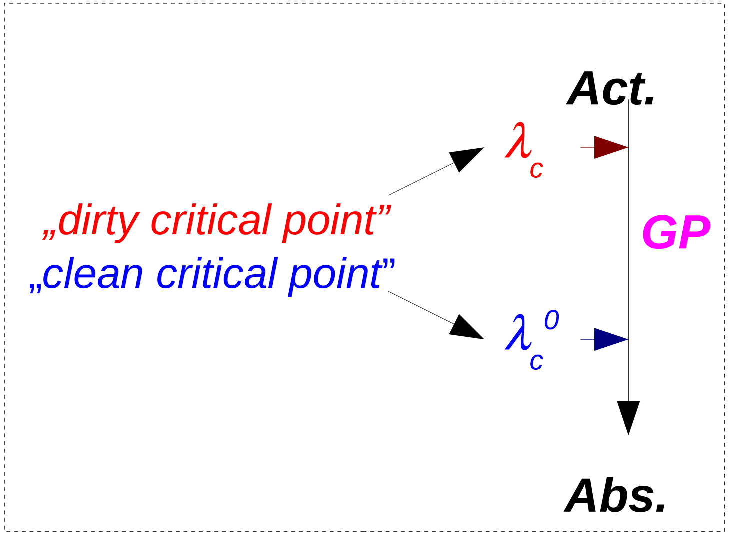
- Exhibits scaling behavior belonging to the DP universality class, still rarely observed in nature

- Sensitivity to spatially/temporal (quenched) disorder → The scaling behavior is **slow, non-universal**



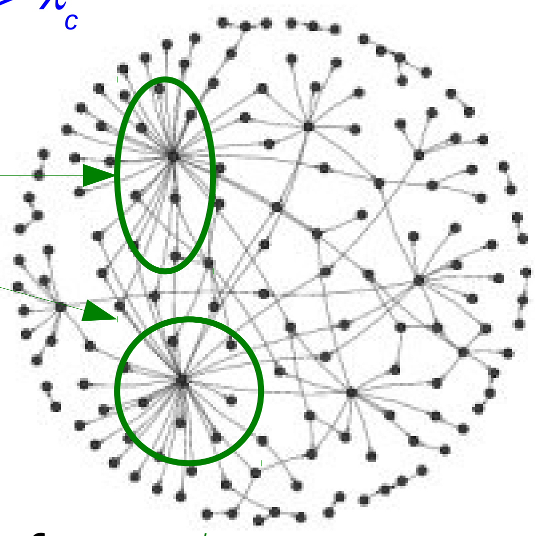
# Rare Region argument for **Q-disordered** **CP**

- Fixed (quenched) disorder/impurity
  ***changes* the local birth rate** $\Rightarrow \lambda_c > \lambda_c^0$




- **Locally active**, arbitrarily large
  Rare Regions

  in the inactive phase
  due to the *inhomogeneities*



- Probability or RR of size $L_R$:
  $w(L_R) \sim exp(-c\, L_R^d)$
  Contribute to the density: $\rho(t) \sim \int dL_R \; L_R^d \; w(L_R) \; exp[-t / \tau (L_R^d)]$

- **For** $\lambda < \lambda_c^0$: conventional (exponentially fast) decay

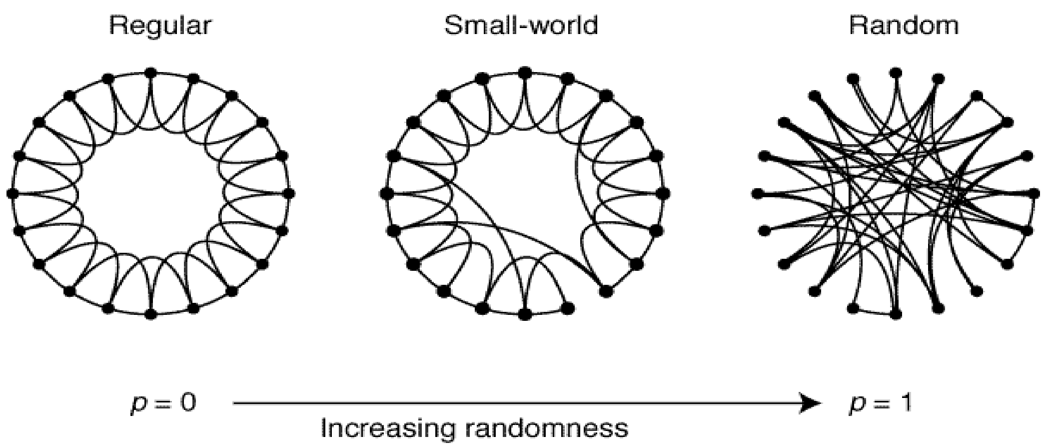
- **At** $\lambda_c^0$ the characteristic time scales as: $\tau(L_R) \sim L_R^{-Z}$ $\Rightarrow$ saddle point analysis:
  $$\ln \rho(t) \sim t^{d/(d+Z)}$$ *(stretched exponential)*

- **For** $\lambda_c^0 < \lambda < \lambda_c$ : $\tau(L_R) \sim exp(b\, L_R)$: **Griffiths Phase**
  $\Rightarrow$ saddle point analysis: $\rho(t) \sim t^{-c/b}$ *(continuously changing exponents)*

- **At** $\lambda_c$ Ultra slow time dependences : $\rho(t) \sim \ln(t)^{-\alpha}$

# Basic network models

**From regular to random networks:**




**Erdős-Rényi** ($p = 1$)

Degree ($k$) distribution in $N \rightarrow \infty$ node limit:

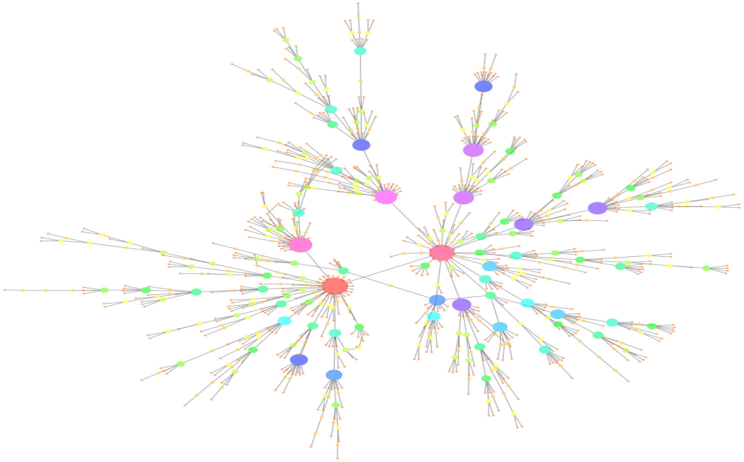
$$P(k) = e^{-<k>} <k>^{k} / k!$$

*Topological dimension: $N(r) \sim r^{d}$*

*Above perc. thresh.: $d = \infty$*

*Below percolation $d = 0$*

**Scale free networks:**



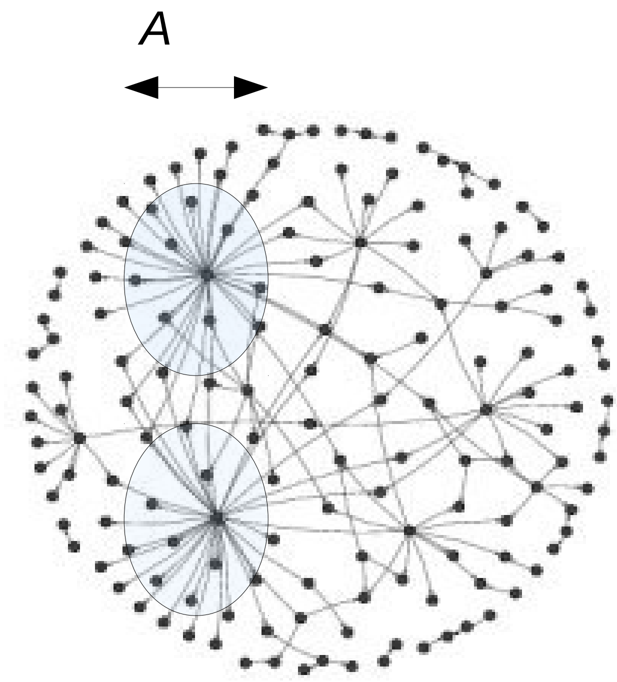
Degree distribution:

$$P(k) = k^{-\gamma} \; (2 < \gamma < 3)$$

*Topological dimension: $d = \infty$*

Example: **Barabási-Albert lin. prefetential attachment**

**Focus on dynamical systems living on networks: Fast dynamics is expected**




**Networks**: fast dynamics, mean-field behavior expected

Effect of disorder:

Rare active regions in the absorbing phase: $\tau(A) \sim e^{A}$

→ slow dynamics (Griffiths Phase) ?

*M. A. Munoz, R. Juhász, C. Castellano and G. Ódor, PRL 105, 128701 (2010)*

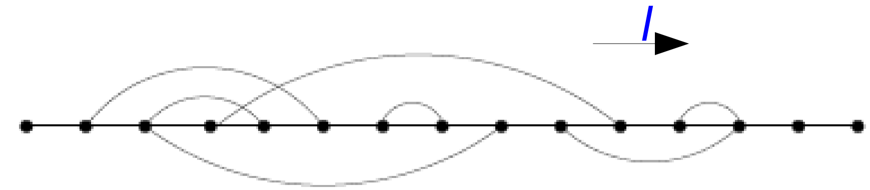
1. Inherent disorder in couplings
2. Disorder induced by topology

Optimal fluctiation theory + simulations:

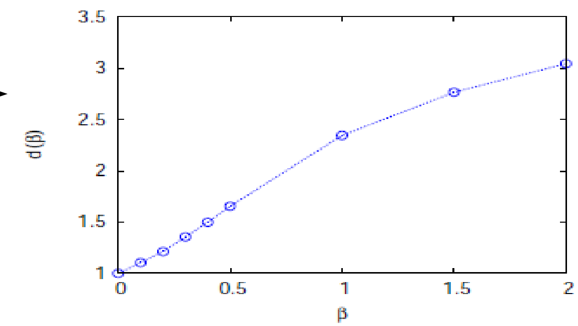
- In Erdős-Rényi networks below the percolation threshold
- In generalized small-world networks for finite topological dimension

# CP + Topological disorder results

**Generalized Small World networks**: $P(l) \sim \beta\, l^{-2}$
(link length probability)




- *Top. dim:* $N(r) \sim r^{d}$ $d(\beta)$ **finite:**




$\lambda_c(\beta)$ *decreases* monotonically from
$\lambda_c(0) = 3.29785$ *(1d* **CP***) to:*
$\lim_{\beta\to\infty} \lambda_c(\beta) = 1$ towards mean-field **CP** value

$\lambda < \lambda_c(\beta)$ inactive, there can be
locally ordered, **rare regions** due to more than average, active, incoming links

- **Griffiths phase:** *$\lambda$ – dep. continuously changing dynamical power laws:*
  for example : $\rho(t) \sim t^{-\alpha(\lambda)}$

  Logarithmic corrections !

- **Ultra-slow** ("activated") scaling: $\rho \propto \ln(t)^{-\alpha}$ at $\lambda_c$
- As $\beta \to 1$ Griffiths phase shrinks/disappears
- **Same results for** **cubic, regular random networks higher dimensions**

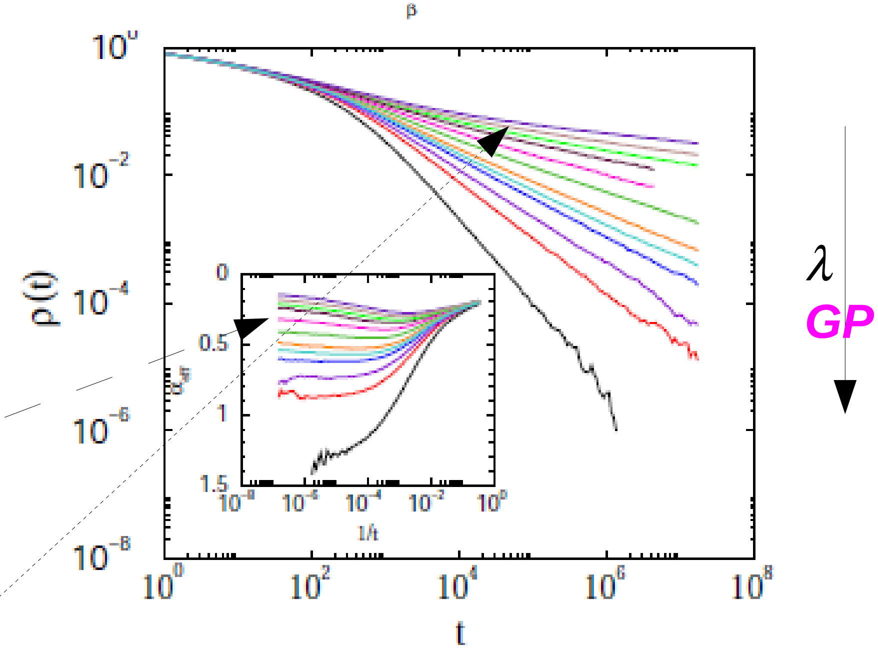



FIG. 3: Density decay in Benjamini-Berger networks with $s = 2$ and $\beta = 0.2$ for different values of $\lambda$ (from top to bottom: $2.81, 2.795, 2.782, 2.77, 2.75, 2.73, 2.71, 2.70, 2.69, 2.67, 2.65, 2.6$). Straight lines lie in the Griffiths phase. Inset: Corresponding effective exponents, illustrating the presence of corrections to scaling.

# Contact process on Barabási-Albert (BA) network

- Heterogeneous mean-field theory: conventional critical point, with linear density decay:

$$\rho(t) \sim [t \ln(t)]^{-1},$$

with logarithmic correction

- Extensive simulations confirm this
- No Griffiths phase observed
- Steady state density vanishes at $\lambda_c$ =~*1*

linearly, HMF: $\beta = 1$

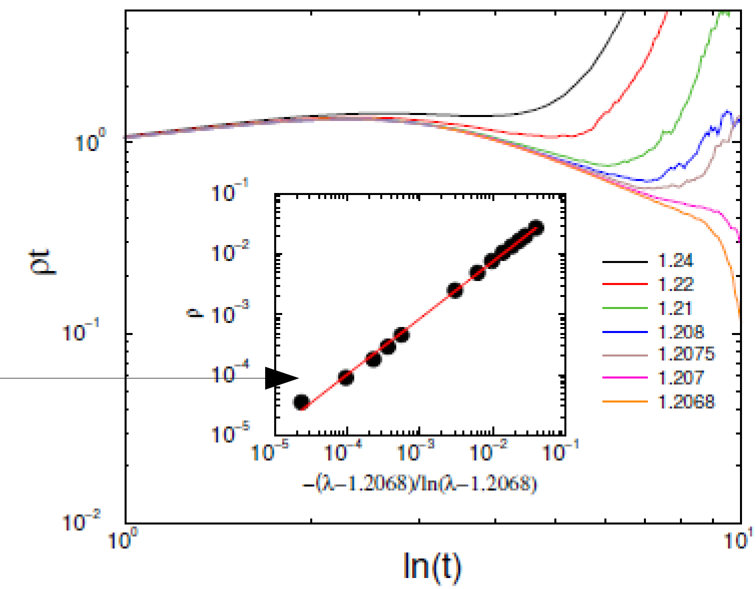



FIG. 1. Density decay ($t\rho(t)$) as a function of $\ln(t)$ for the CP on unweighted looped BA networks with $m = 3$ of size $N = 8 \times 10^7$. The different curves correspond to $\lambda = 1.2068, \ldots, 1.24$ (bottom to top). Inset: Steady state density, showing agreement with HMF theory scaling. The full line shows a power-law fitting to the data points in the form $-0.36(5)x^{0.98(2)}$.

# CP on Barabási-Albert trees

## hunt for GP-s, by slowing the propagation

- Lack of loops slows propagation
- For $\langle k \rangle = 3$ : $\lambda_c > 0$

Weighted networks:

$$\omega_{ij} = \omega_0 (k_i k_j)^{-\nu} \qquad \omega_{ij} = \frac{|i-j|^x}{N}$$

Strong size corrections

$$k_i \propto (N/i)^{1/2}$$

Non mean-field transition :

Power-laws for: *x = 2,3*

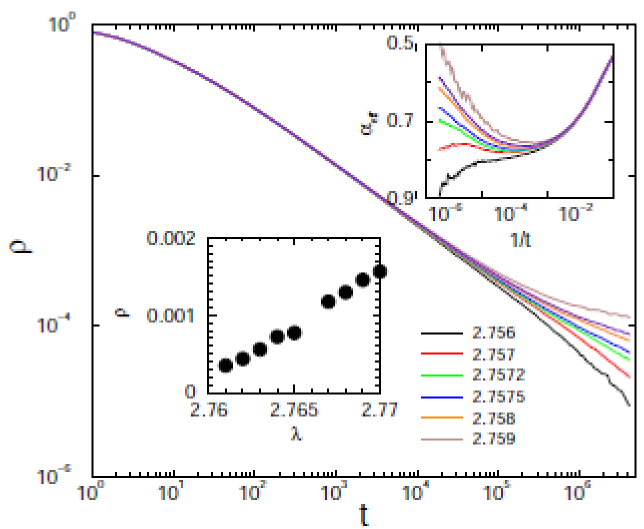



FIG. 1: Density decay in a pure BA CP model, $m = 1$, $m_0 = 10$, $N = 4\times10^7$ for $\lambda = 2.756, ..., 2.759$ (bottom to top). Right insert: the corresponding effective exponents. Left insert: steady state density in the active phase.

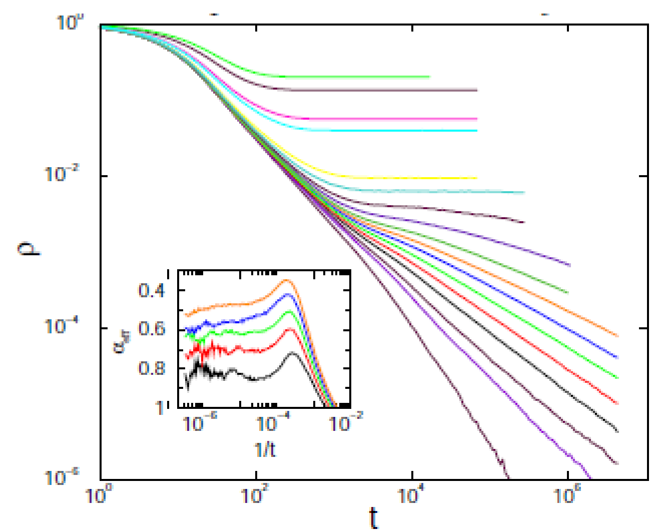



FIG. 5: Density decay in model B $m = 1$, $m0{=}20$, $N = 10^5$ for $\lambda = 6.8, ..., 15$ (top to bottom). Inset: corresponding local slopes in the GP region.

Heterogeneous mean-field theory: critical point, with linear density decay: *ρ µ 1/t*
can't describe frozen disorder !

# Do power-laws survive the thermodynamic limit ?

- Finite size analysis shows the disappearance of a power-law scaling:

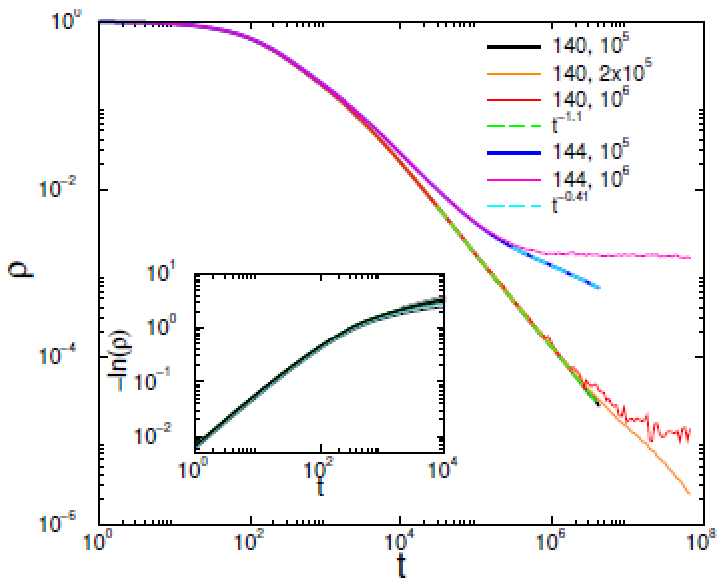



FIG. 5. Density decay as a function of time $\rho(t)$ for the CP on weighted BA trees with a multiplicative weighting scheme (WBAT-I) with exponent $\nu = 1.5$. Plots correspond to two sets of $\lambda$ (upper branch: $\lambda = 144$, lower branch $\lambda = 140$) at different network sizes $N$. Dashed lines represent PL fittings. Inset: Initial time region of the same data, showing an stretched exponential behavior.

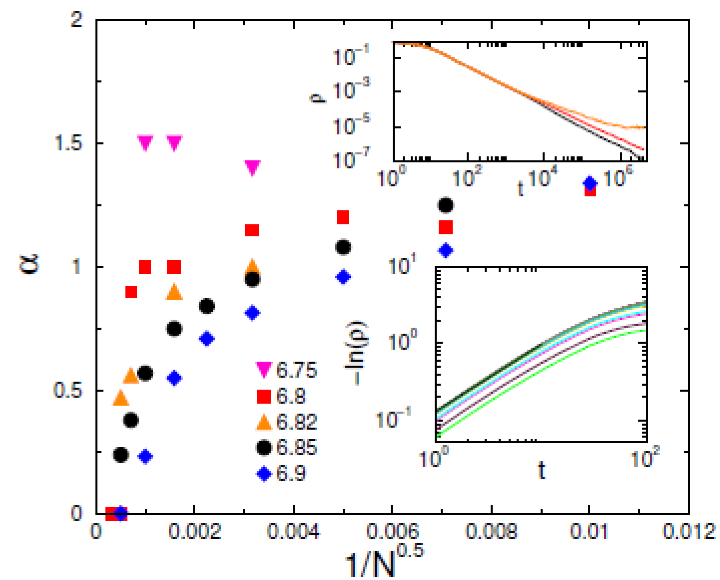



FIG. 8. Finite-size scaling analysis of the density decay exponent for $\lambda = 6.75$ (triangles), $\lambda = 6.8$ (boxes), $\lambda = 6.82$ (triangles), $\lambda = 6.85$ (bullets), $\lambda = 6,9$ (rhombes) in the CP on weighted BA trees with a age-dependent weighting scheme (WBAT-II) with exponent $x = 2$. Top inset: $\rho(t)$ for $\lambda = 6.82$ ($N = 10^6$, $N = 4x10^5$, $N = 10^5$ top to bottom). Bottom inset: Initial time density.

- Smeared phase transition: power-law → saturation:
- Rare sub-spaces, but infinite dimensional ?

# Percolation analysis of the weighted BA tree

We consider a network of a given size N, and delete all the edges with a weight smaller than a threshold $\omega_{th}$.

For small values of $\omega_{th}$, many edges remain in the system, and they form a connected network with a single cluster encompassing almost all the vertices in the network.

When increasing the value of $\omega_{th}$, the network breaks down into smaller subnetworks of connected edges, joined by weights larger than $\omega_{th}$.

The size of the largest ones grows linearly with the network size N

↔ standard percolation transition.

These clusters, which can become arbitrarily large in the thermodynamic limit, play the role of correlated **RR**s, sustaining independently activity and smearing down the phase transition.

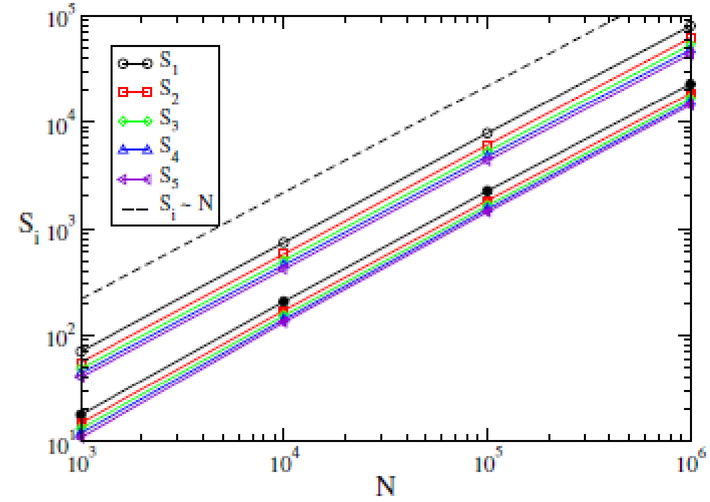



FIG. 6. Size $S_i$ of the 5 largest clusters in a percolation analysis of the WBAT-I model with $\nu = 1.5$ for $\omega_{th} = 100\omega_{min}$ (hollow symbols) and $\omega_{th} = 1000\omega_{min}$ (full symbols), where $\omega_{min}$ is the minimum weight in the network. The size of all components grows linearly with network size $N$, and is therefore infinite in the thermodynamic limit.

# Summary

- *Quenched disorder in complex networks can cause slow dynamics :*
  *Rare-regions → (Griffiths) phasess → no tuning or self-organization needed !*

- *In **finite dim**. (for CP) GP can occur due to topological disorder*

- *In **infinite dim**, scale-free, BA network mean-field transition of CP with logarithmic corrections (HMF+simulations)*

- *In BA **trees** non mean-field transition observed*

- *In **weighted** BA **trees** non-universal, slow, power-law dynamics can occur for finite N, but in the N $\to\infty$ limit saturation is observed*

- *Smeared transition can describe this,*
  *percolation analysis confirms the existence of arbitrarily large dimensional sub-spaces with (correlated) large weights*

- *Acknowledgements to : HPC-Europa2, OTKA, Osiris FP7*

*[1] M. A. Munoz, R. Juhasz, C. Castellano, and G, Ódor, Phys. Rev. Lett. 105, 128701 (2010)*
*[2] G. Ódor, R. Juhasz, C. Castellano, M. A. Munoz, AIP Conf. Proc. 1332, Melville, New York (2011) p. 172-178.*
*[3] R. Juhasz, G. Ódor, C. Castellano, M. A. Munoz, Phys. Rev. E 85, 066125 (2012)*
*[4] G. Ódor and Romualdo Pastor-Satorras, Phys. Rev. E 86, 026117 (2012)*