Localization, Griffiths phases and burstyness in neural network models

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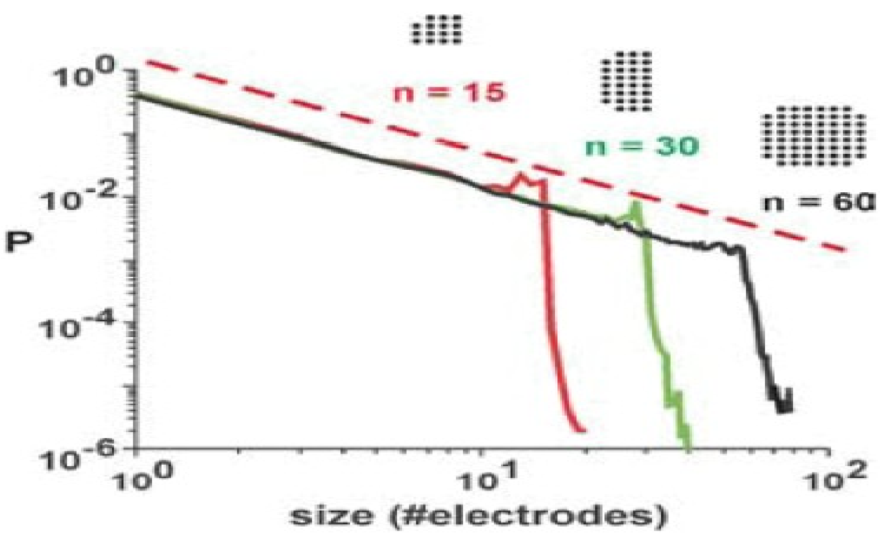
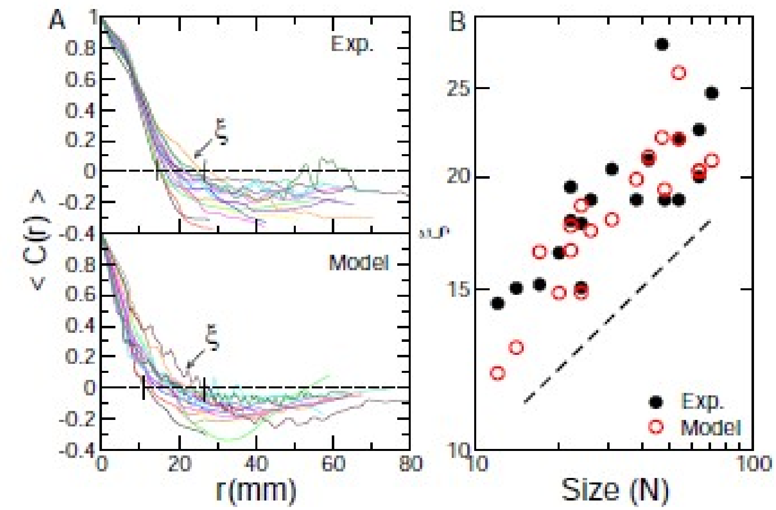
Power-laws & critical slow dynamics in networks

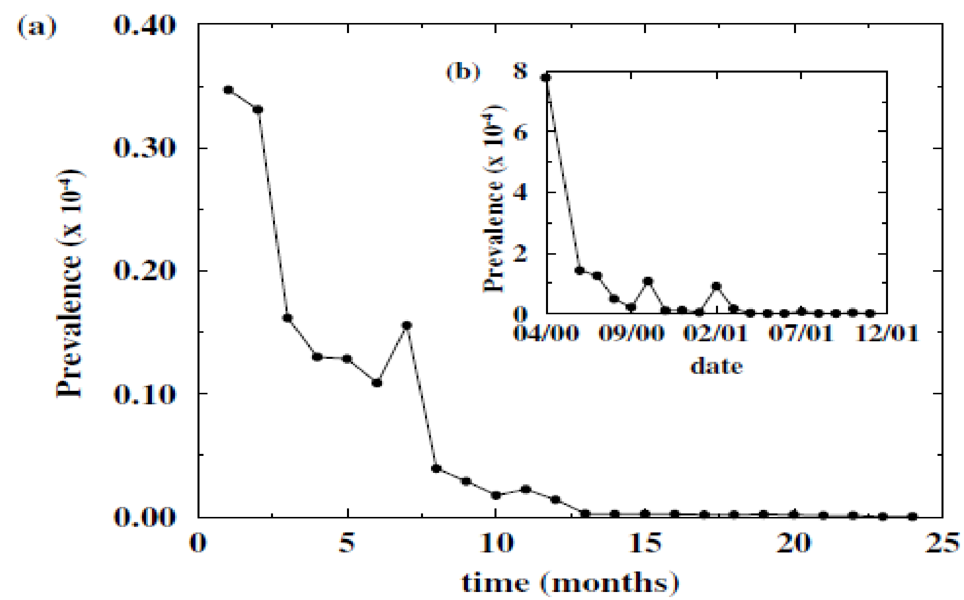
- Brain : PL size distribution of neural avalanches
  G. Werner : Biosystems, 90 (2007) 496,



- *Correlation length (ξ) diverges*
  Haimovici et al PRL (2013) :
  *Brain complexity born out of **criticality**.*



- Internet: worm recovery time is **slow**:

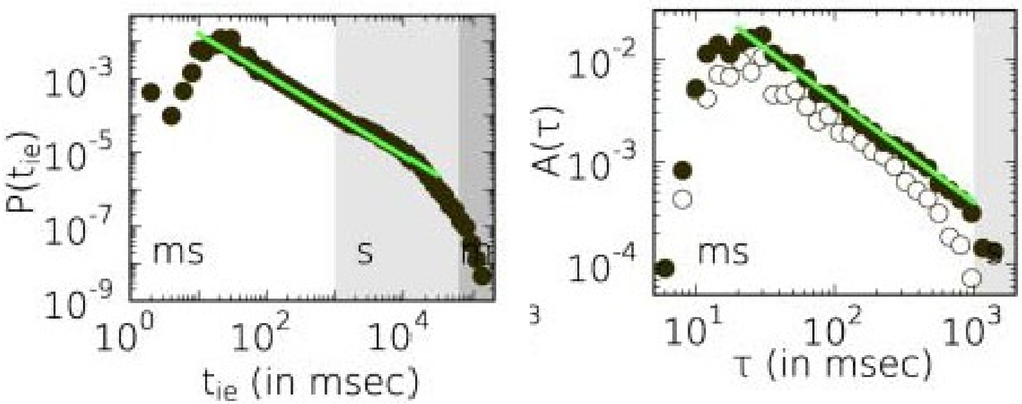


How can we explain power-law dynamics in network models ?

Burstyness observed in nature

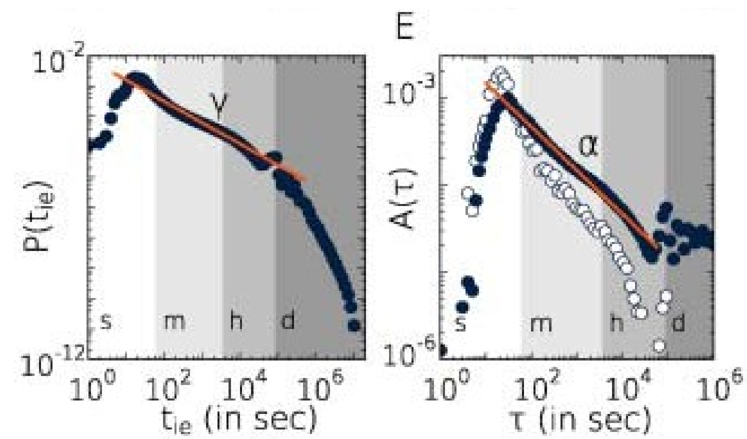
- **Brain** : PL inter-event time distribution of neuron firing sequences & Autocorrelations

  *Y. Ikegaya et al.: Science, **304** (2004) 559,*
  *N. Takahashi et al.: Neurosci. Res. **58** (2007) 219*

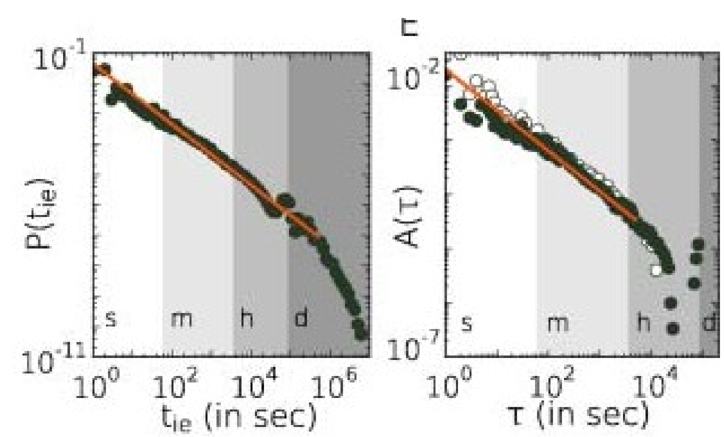


- *Mobile call: Inter-event times and Autocorrelations*

  *Karsai et al. PRE **83** (2013) 025102 : Small but slow world ...*



- **Internet**: Email sequences:
- And many more ….
- **Models** exist to explain internal non-Markovian behavior of agents *(Karsai et al.: Sci. Rep. **2** (2012) 397)*



*J. Eckmann et al.: PNAS **101** (2004) 14333*

Can this occur by **the collective behavior** of Markovian agents **?**

Scaling in nonequilibrium system

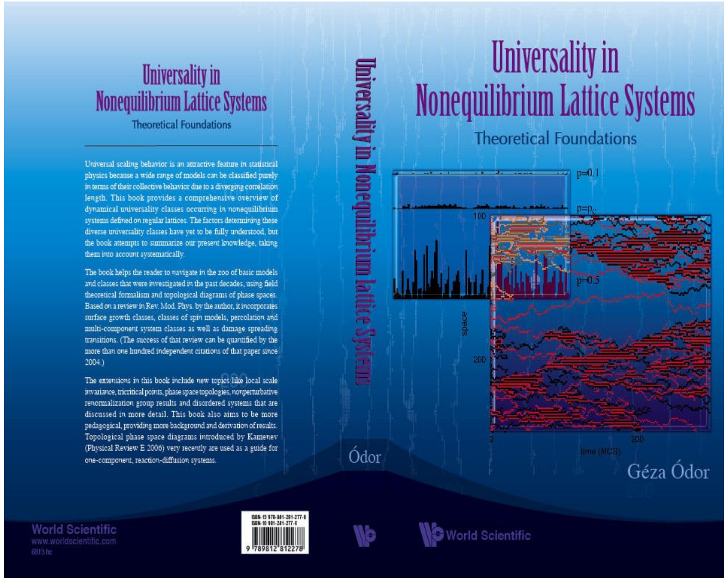
Scaling and universality classes appear in complex system due to : $\xi \to \infty$
i.e: near critical points, due to currents, ...

Basic models are classified by **universal scaling behavior** in Euclidean, regular system

Why don't we find these critical universality classes in dynamical network models ?

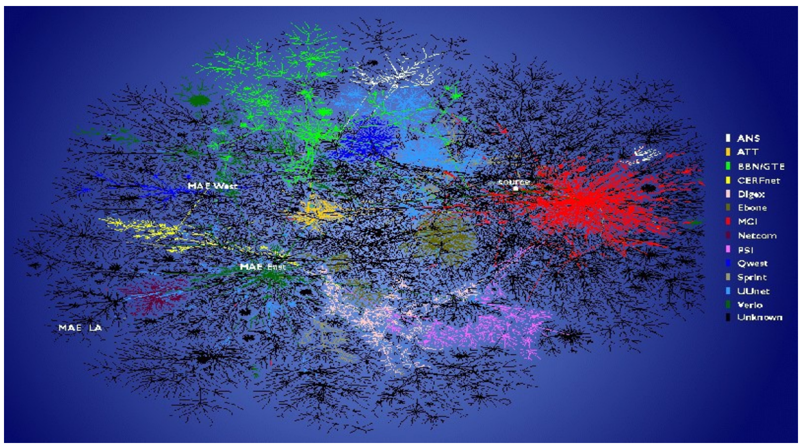
Power laws are frequent in nature $\leftrightarrow$ Tuning to critical point (SOC) ?

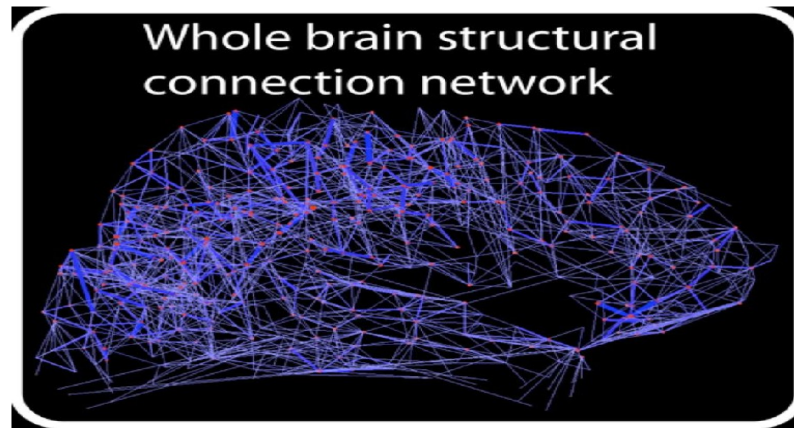
I'll show a possible way to understand these on quasi-static networks



Networks models with fast dynamics

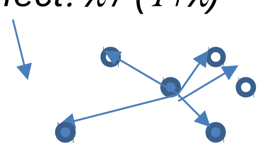
Small world networks: Expectation: mean-field type behavior with fast dynamics



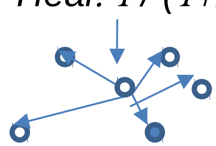


*Prototype: Contact Process or **S**usceptible-**I**nfected-**S**usceptible (**SIS**) two-state model:*

Infect: $\lambda / (1+\lambda)$

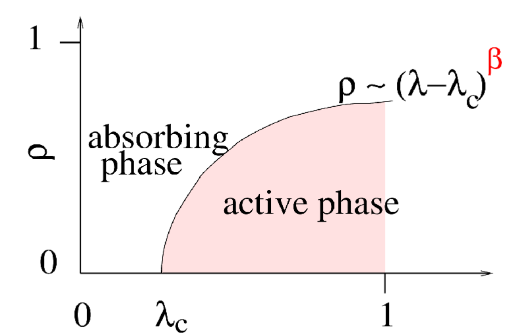


Heal: $1 / (1+\lambda)$

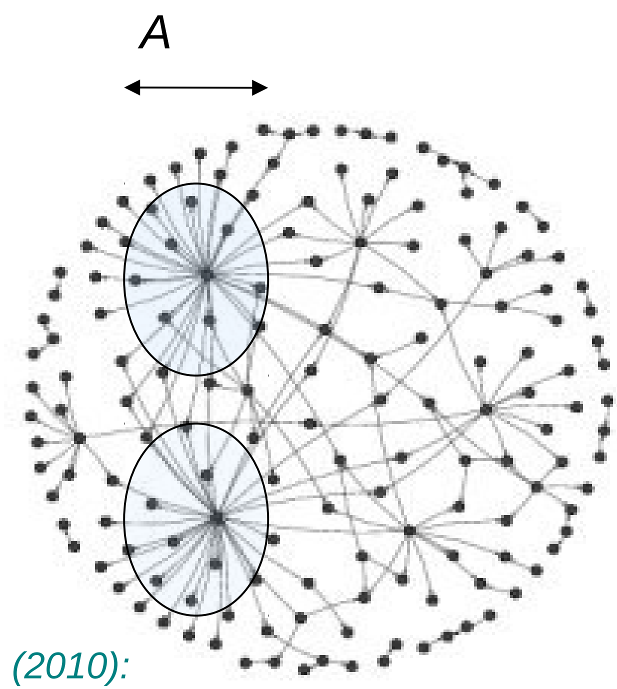


For SIS : Infections attempted for all nn

Order parameter : density of active (●) sites
*Regular, Euclidean lattice: **DP** critical point :*
λ_c *> 0 between inactive and active phases*



Slow dynamics induced by heterogeneities



Rare, active regions below λ_c with: $\boldsymbol{\tau(A) \sim e^{A}}$
→ slow dynamics (Griffiths Phases) ?

$$\rho(t) \sim \int dA_R \;\; A_R \;\; p(A_R) \;\; \exp[-t/\tau]$$

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 fluctuation theory + simulations: YES !

Further studies imply :

- In networks with finite dimension the topological heterogeneity can induce **GP**
- In networks with disordered couplings (Erdős-Rényi … etc.) heavy tails observed: **GP** or at least stretched exponentially slow dynamics
- In finite networks non-universal power-law tails appear, but their exponents depend on the control parameter

G. Ódor, R. Pastor-Satorras, Phys. Rev. E 86 (2012) 026117.
G. Ódor, Phys. Rev. E (2013) 032109.
G. Ódor, Phys. Rev. E 90 (2014) 032110.

Hierarchical **M**odular **N**etworks motivated by connectomes

HMN-s exhibit different clustering properties than structureless networks. Rare region effects ?

Can we see GP-s in small-world brain network models ?

Do we need to tune the networks close to the percolation threshold to keep finite dimension as well as connectivity ?
Moretti & Munoz, Nature Comm. 4 (2013) 2521

In finite dimensions what kind of long-range connections induce RR-s and GP-s ?

Limited sustained activity requires localization of activity. When can localization arise ? Kaiser-Hilgetag, Front. In Neuroinf. 4 (2010) 8

Do we see burstyness in these GP-s ?

Hierarchical **M**odular **N**etwork topology motivated by connectomes

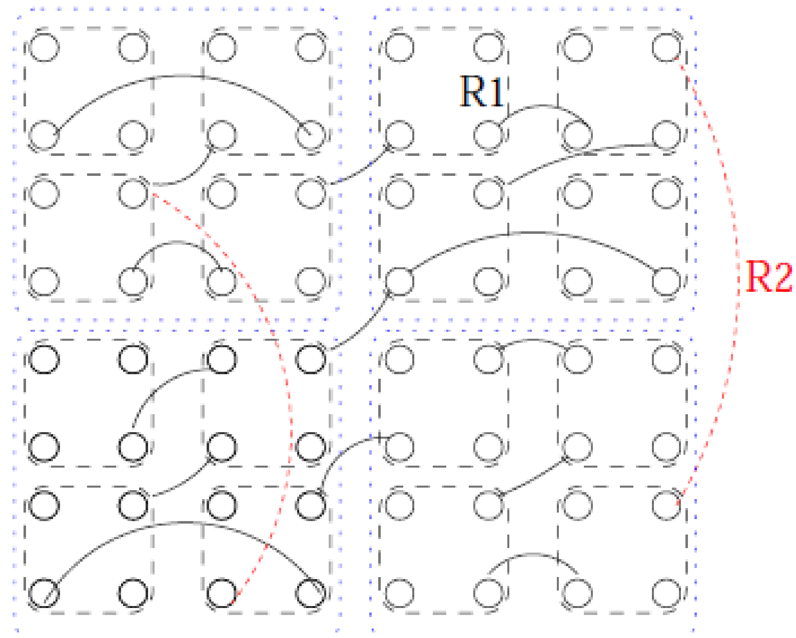
Our HMN constructions

A: HMN2d:

Exponentially decaying connection probabilities with the levels *l* :

$$p_l = \langle k \rangle / 2 \, (½)^{sl}$$

$R \simeq 2^l$,



related to networks with long edge probabilities:

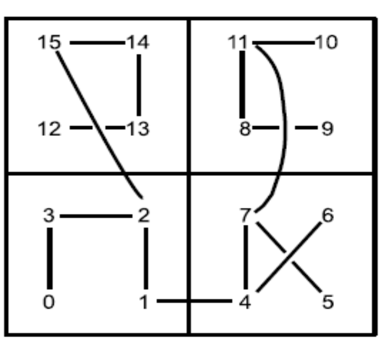
$$p(R) \sim \langle k \rangle / 2 \, R^{-s}$$

where **GP**-s are present (*Juhasz et al PRE 2012*)

FIG. 1: Two lowest levels of the hierarchical network construction with 4 nodes/module. Dashed lines: $l = l_1$, dotted lines: $l = l_2$. The solid lines denoted R1 are randomly chosen connections within the bottom level (fully connected) modules, while those denoted R2 provide random connections on the next level. Links can be directed.

B: **Hierarchical trees :**

Connectedness + finite dimension



Network metrics

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

Effective dimension:

$$d_{\text{eff}} = \frac{\ln[N(r))/N(r')]}{\ln(r/r')}$$

Breadth-first search results, in agreement with the ***2d*** networks with power-law ranged, long edges:

For *s = 4 :* <*k*> dependent continuously changing dimension.
Similar to the MM HMN1 construction

For *s < 4* small-world networks, similar to **KH**

For *s > 4* finite dim., fast decaying long links inducing quenched disorder

A: We study this *(s = 6)* in more detail
+ ***2d*** lattice connectedness (*l = 1)*

B: Hierarchical, random trees: *d ≈ 0.72*

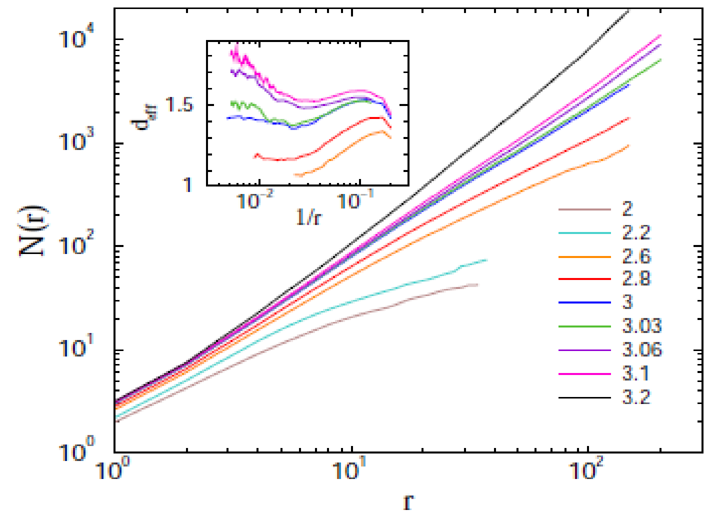
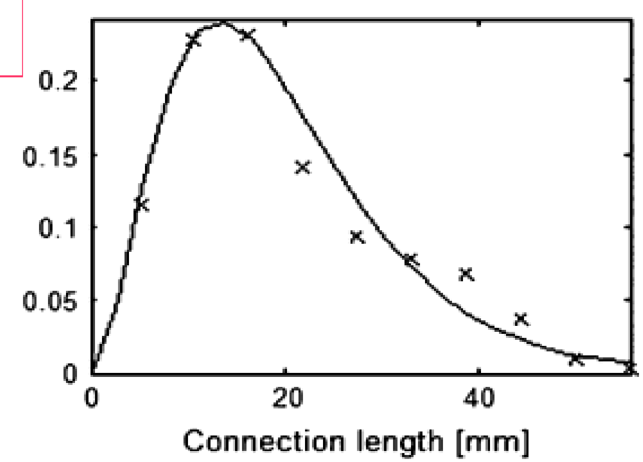


FIG. 3: Number of nodes within chemical distance r in HMN2d networks with $s = 4$ and $l = 9$ levels. Different curves correspond to different $\langle k \rangle$-s. Inset: local slopes d_{eff} of the $N(r)$ curves, defined in Eq. 4.



*Kaiser et al
Cerb. Cort. 2009:*

*Axon length distribution:
Initial large peak +
exponential tail*

Dynamic simulations of the CP on asymmetric HMN2d

Density decay from fully active initial state:

Network size (*l=8, 9, 10*) independent power-laws for *2.45 < λ < 2.53*

Local slopes of the curves:

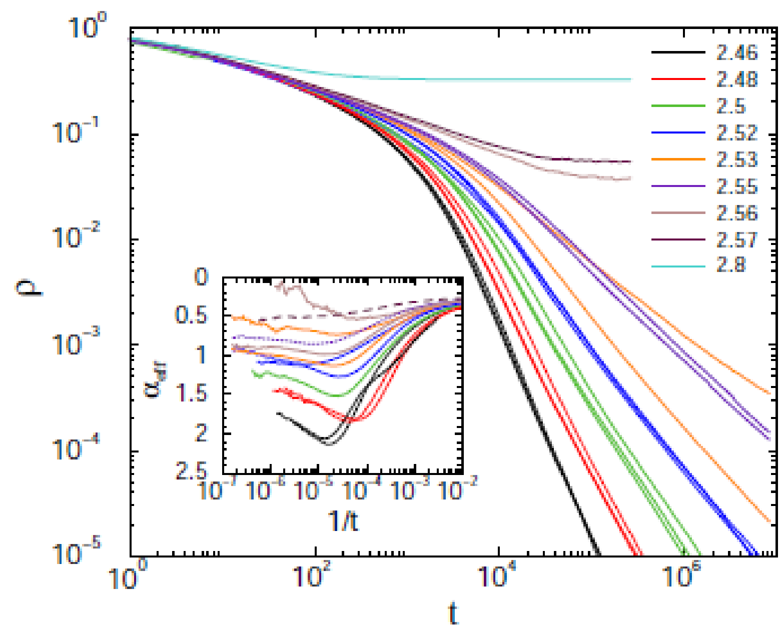
$$\alpha_{\mathrm{eff}}(t) = -\frac{\ln[\rho(t)/\rho(t')]}{\ln(t/t')}$$

Logarithmic corrections (GP)

At λ_c = *2.53(1)* : $\alpha_{eff} \rightarrow 1$

Above this λ the α_{eff} curves of larger systems veer up as $t \rightarrow \infty$

Mean-field critical point instead of activated scaling



Similar results for s = 4 and s = 3 at the percolation threshold

For symmetric HMN2d-s power-laws with size dependency

Dynamic simulations of the CP on random hierarchical trees (RHT)

Density decay from fully active initial state:

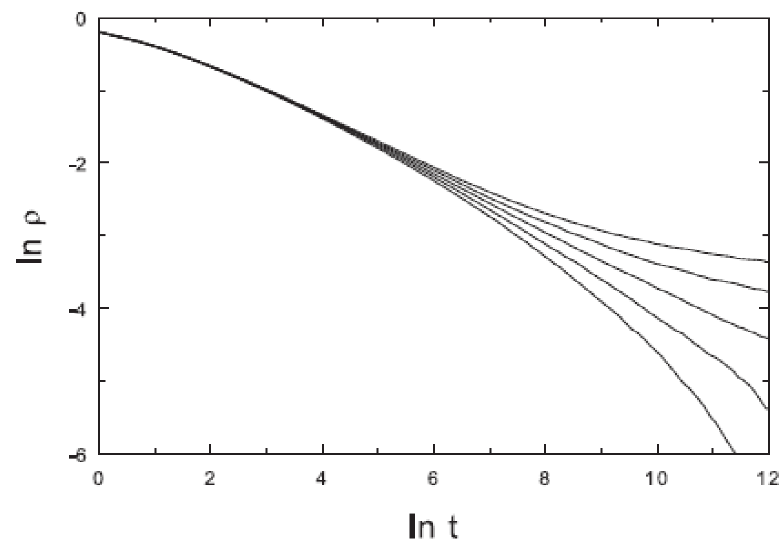
For single tree: no GP !

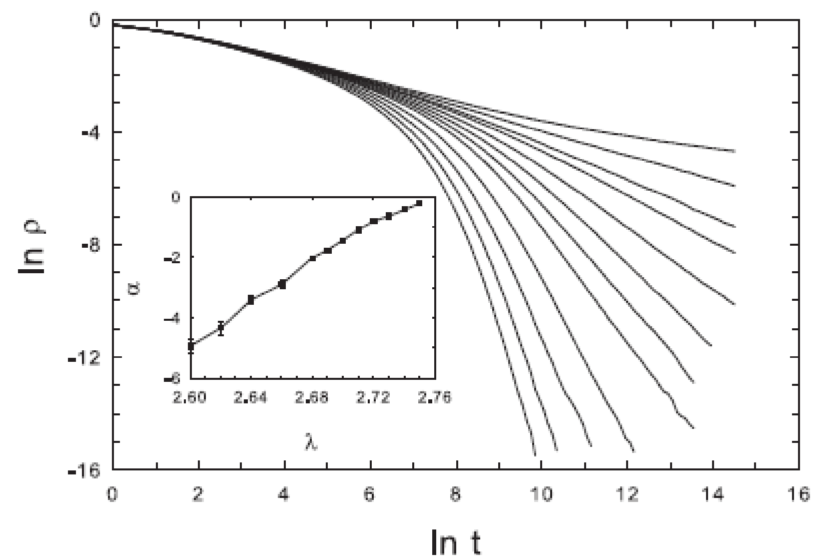
If we average over many independent network realizations:

Power-laws for: *2.6 < λ < 2.75*

GP in case of a modular networks composed of loosely coupled RHT-s ?

Or time dependent networks, where we average over runs of many RHT-s ?





Dynamic simulations of threshold models on HMN2d-s

More realistic neuron model:

k A → *(k+1)A* with: *k > 1*

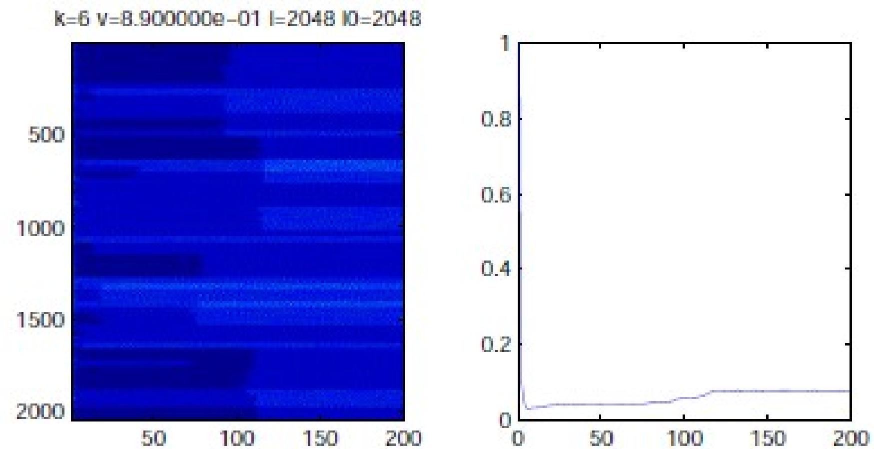
Proposed by **KH** and LSA found

First order transition (no GP) is expected, however due to disorder rounding of transition (see *P.V.Martin et al JSTAT 2015 P01003*)

Stochastic CA simulations

Size independent (*l=8,9*) power-law decays for: *k = 2,3*

Simple connectedness is not enough to provide infinite activity propagation



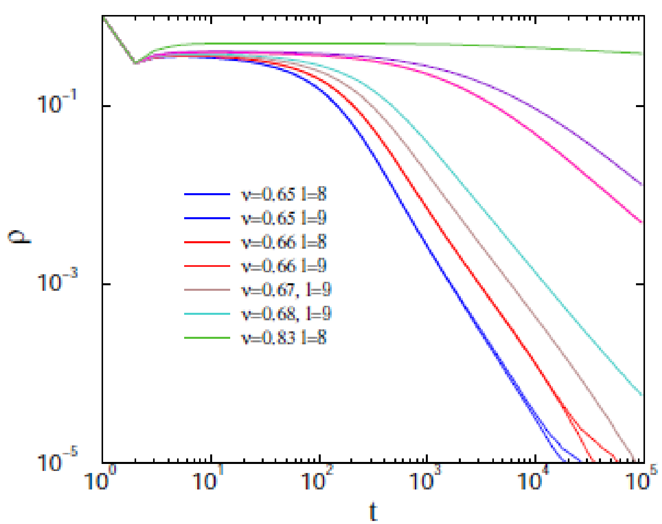


FIG. 13: Decay of activity at different branching rates and $v = 0.7$ fixed in the threshold model with $m = 2$, $s = 6$, and $\langle k \rangle = 24$. Levels: $l_{max} = 8, 9$ (thin, thick lines). Size-independent power-laws, reflecting a GP are observed.

Burstyness of CP in the GP

- Density decay and seed simulations of CP on HMN2d with *s = 6*
- Power-law inter-event time distribution among subsequent interaction attempts
- λ dependent slopes + log. periodic oscillations due to the modularity
- The tail distribution decays as the auto-correlation function for $t \rightarrow \infty$ near *d =1*

Bustyness around the critical point
in an ***extended Griffiths Phase***

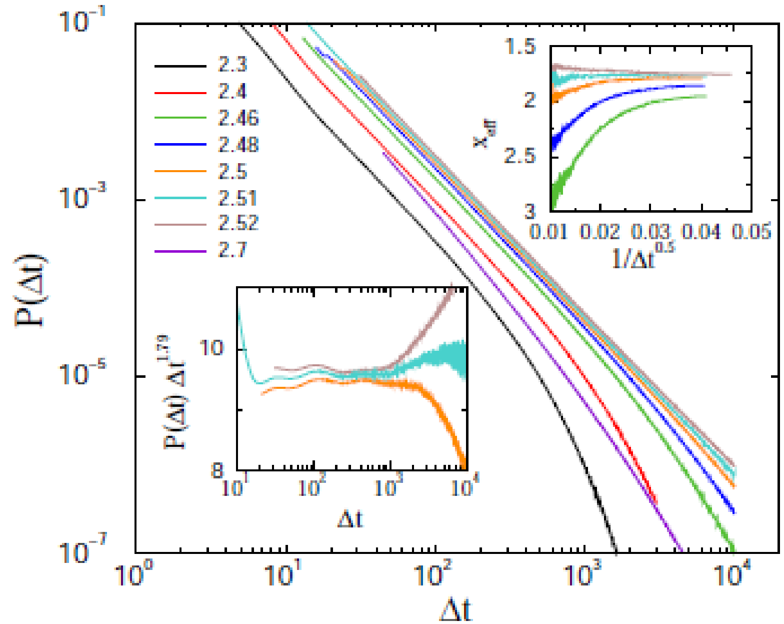


FIG. 9: CP on asymmetric HMN2d networks with $s = 6$: probability distribution, $P(\Delta t)$, of inter-event times for λ values as indicated; system size $l_{max} = 10$. Power-law tails are evident for $2.46 < \lambda < 2.6$, with continuously changing exponents. The dashed lines represent fits : $\simeq \Delta t^{-1.80(4)}$. Inset: $P*(\Delta t) \equiv (\Delta t)^{1.79} P(\Delta t)$ for $\lambda = 2.5, 2.52$.

Quenched Mean-Field (QMF) theory for SIS

Rate equation of SIS for occupancy prob. at site i:

$$\frac{d\rho_i(t)}{dt} = -\rho_i(t) + \lambda(1-\rho_i(t))\sum_{j=1}^{N} A_{ij} w_{ij} \rho_j(t)$$

Weighted (real symmetric) Adjacency matrix: $B_{ij} = A_{ij} w_{ij},$

For $t \to \infty$ the system evolves into a steady state, with the probabilities expressed as

$$\rho_i = \frac{\lambda \sum_j B_{ij}\rho_j}{1+\lambda\sum_j B_{ij}\rho_j} \,. \qquad (5)$$

Express ρ_i on orthonormal eigenvector ($\mathbf{f}_i(\Lambda)$) basis:

$$\rho_i = \sum_{\Lambda} c(\Lambda) f_i(\Lambda). \qquad (6)$$

Mean-field critical point estimate $\lambda_c = 1/\Lambda_1$

Total infection density vanishes near λ_c as :

$$\rho(\lambda) \approx \alpha_1\tau + \alpha_2\tau^2 + \ldots \,, \qquad (8)$$

where $\tau = \lambda\Lambda_1 - 1 \ll 1$ with the coefficients

$$\alpha_j = \sum_{i=1}^{N} f_i(\Lambda_j)/[N\sum_{i=1}^{N} f_i^3(\Lambda_j)]. \qquad (9)$$

To describe the localization of the components of $\boldsymbol{f}(\Lambda_1)$ [19] used the inverse participation ratio

$$IPR(\Lambda) \equiv \sum_{i=1}^{N} f_i^4(\Lambda), \qquad (10)$$

Localization of SIS in HMN2d-s

Weakly coupled case <*k*> = *4*

In small world case (*s=3/2*) KH no localization

For *s=3, s=4* signs of localization

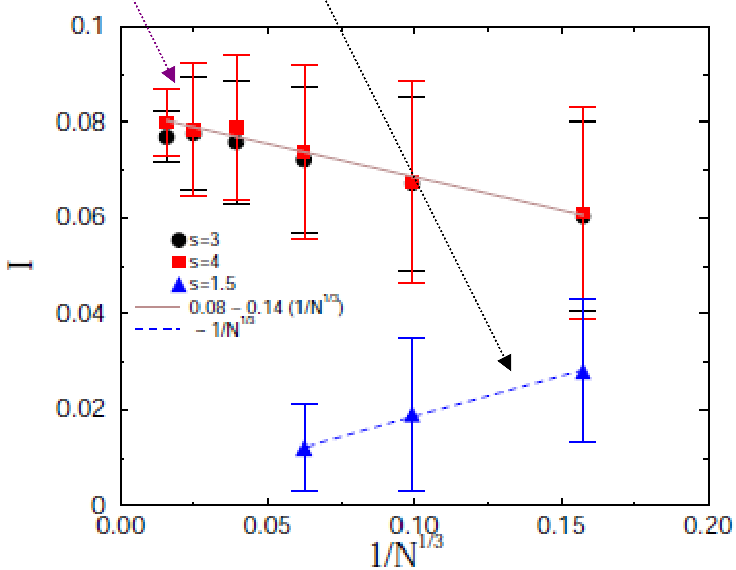


FIG. 14: Finite size scaling of the inverse participation ratio in weakly coupled HMN2d models, with maximum levels $l_{max} = 4, 5, ..9$. The $s = 3$ (bullets) and $s = 4$ (boxes) results suggest localization (finite I) in the infinite-size limit. Lines are power-law fits to the data. For $s = 1.5$, corresponding to the symmetrized, small-world network (model-6 of [41]) no evidence of localization is seen.

For <k> = 50 *and s=3,4 localization goes away*

For weighted links (uniform distributed quenched diso.) localization can be see again

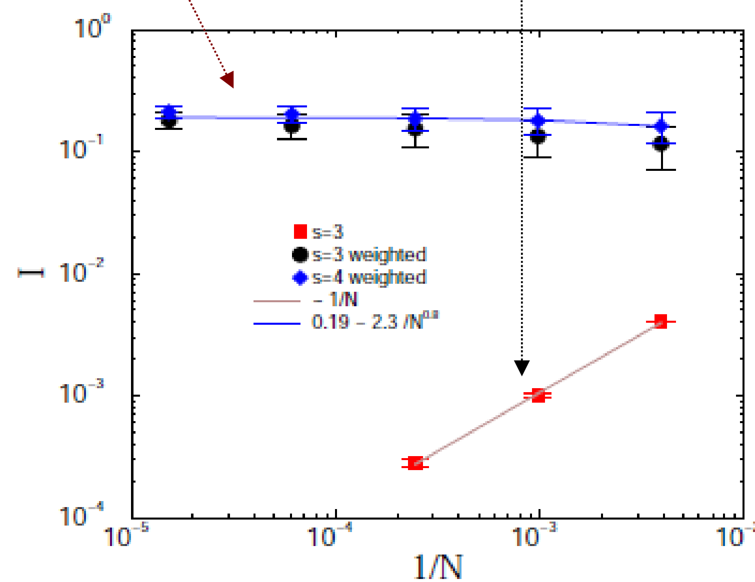


FIG. 15: Finite size scaling of the inverse participation ratio in HMN2d models with higher average degree ($\langle k \rangle \simeq 50$) for maximum levels: $l_{max} = 4, 5, 6, 7, 8$. Bullets: $s = 3$ with uniform randomly distributed weights; boxes: without weights. Diamonds: $s = 4$ with randomly distributed weights. Lines show power-law fits to the data. In the unweighted case, no localization effect can be seen, and I decays linearly with N.

Conclusions

- *Heterogeneities in **HMN** networks can cause **slow (PL)** dynamics :*
  *a **working memory mechanism in brain***
  Rare-regions → Griffiths Phases → even without tuning or self-organization mechanism !
- ***GP-s** arise in case of purely topological disorder, in finite topological dimensions,*
  if we average over many independent network realizations
  → time dependent nets, modules of weakly coupled modules, ...
- *Heterogeneities in the interaction strengths improves **GP** effects and cause **localization***
- *Relation to real brain networks: embedded in 2d space of a short-ranged connected substrate + fast decaying long link connections, disorder in the weights and directions*
- *Bursty behavior in extended **GP**-s, where* $\tau \to \infty$
  Complexity induces non-Markovian behavior of the individual neurons

Géza Ódor, Ron Dickman, Gergely Ódor, arXiv:1503.06307

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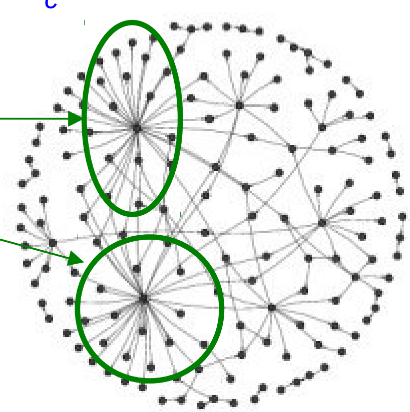
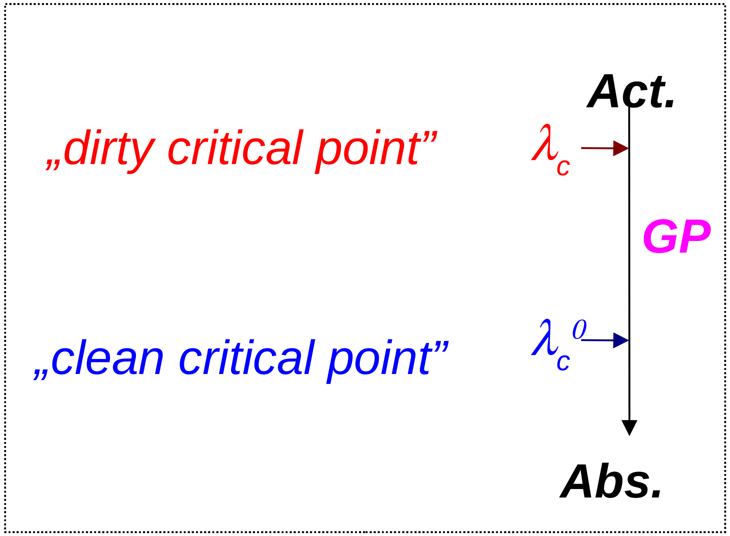
Rare Region theory for quench disordered CP

- *Fixed (quenched) disorder/impurity* **changes the local birth rate** $\Rightarrow \lambda_c > \lambda_c^0$
- ***Locally active***, *but arbitrarily large Rare Regions in the inactive phase due to the inhomogeneities*





- *Probability of RR of size* L_R:

  $w(L_R) \sim \exp(-c\, L_R)$

  contribute to the density: $\rho(t) \sim \int dL_R\; L_R\; w(L_R)\; \exp[-t/\tau(L_R)]$

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

  $\rho(t) \sim t^{-c/b}$ *continuously changing exponents*

- ***At*** λ_c : *b may diverge* $\rightarrow$ $\rho(t) \sim \ln(t)^{-\alpha}$ *Infinite randomness fixed point scaling*
- ***In case of correlated RR-s with dimension*** $> d^-$ ***: smeared transition***