Complex dynamics with threshold: SOC, generalized epidemic and percolation processes

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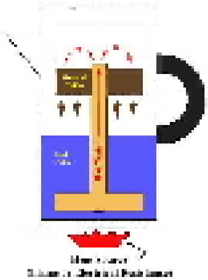
Overview

1. classical percolation
2. bootstrap percolation
3. SOC versus bootstrap
4. bootstrap on Erdös&Renyi
5. BJR graphs
6. bootstrap on BJR graphs
7. some applications

What is percolation?



real life percolation

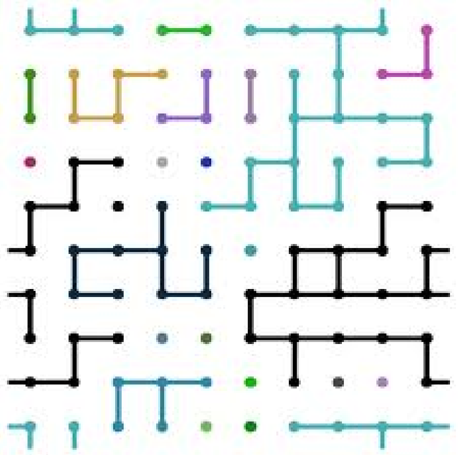
Percolation

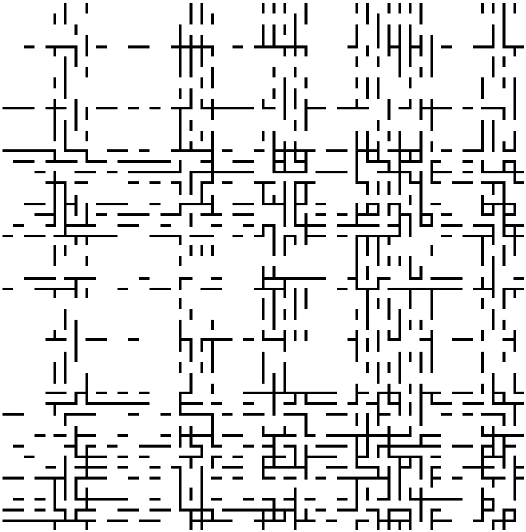
- studied since 1957 (Broadbent&Hammersley)
- on the $\mathbb{Z}^2-$ lattice graph:
  - with prob. p keep edges
  - with prob $1-p$ remove edges
- the resulting object is a random subgraph $\Lambda_p \subset \mathbb{Z}^2$
- percolation question: is there an infinite connected subgraph of Λ_p
- Theorem:

$$\begin{aligned} \exists\ p_c &\in (0,1) \text{ such that} && (1)\\ \text{for } p &< p_c \Rightarrow \text{with prob 1 no infinite cluster} && (2)\\ \text{for } p &> p_c \Rightarrow \text{with prob 1 is an infinite cluster} && (3) \end{aligned}$$

- Kesten 1980: $p_c = 1/2$
- the above model is called bond percolation
- removing/keeping sites instead of edges defines site percolation

some pictures





variants of the classical percolation problem

- different lattices $\mathbb{Z}^n$ with different neighborhood structure
- random graphs
  - giant component
  - on complete graph $K_N : p_c = \frac{1}{N}$
- on trees it is closely related to critical branching processes
- continuum percolation
- directed percolation
- first passage percolation
- for all these generalizations is a critical threshold p_c

- very active field of research
- many open questions
    - what happens at p_c
    - how to determine p_c
    - theory is well developed for lattices in high dimensions
- applications:
    - material science (erosion of materials)
    - spreading phenomena: forest fire, epidemics,....

The origin of the name

- the American version of "the adventures of Baron Münchhausen"
- the Baron pulled himself out of a swamp by his bootstraps (on his hairs in the German version)
- 

Bootstrap percolation

- two states 0 and 1 (infected)
- infect initially at random vertices with probability p_0
- infected vertices remain infected forever
- deterministic discrete dynamics: vertex gets infected if it has $\geq \Delta$ infected neighbors
- how large is the finally infected set?
- studied in physics literature since 70s (mainly dual process)
- mainly studied on lattices, hypercube (Balogh,Bollobas,Morris 2009) and regular trees (Biskup, Schoneman 2009)

some bootstrap percolation pictures

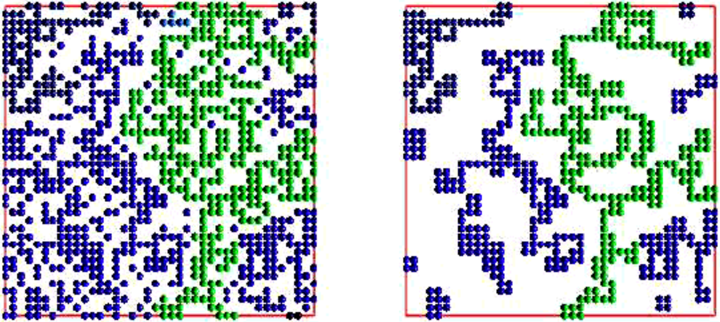
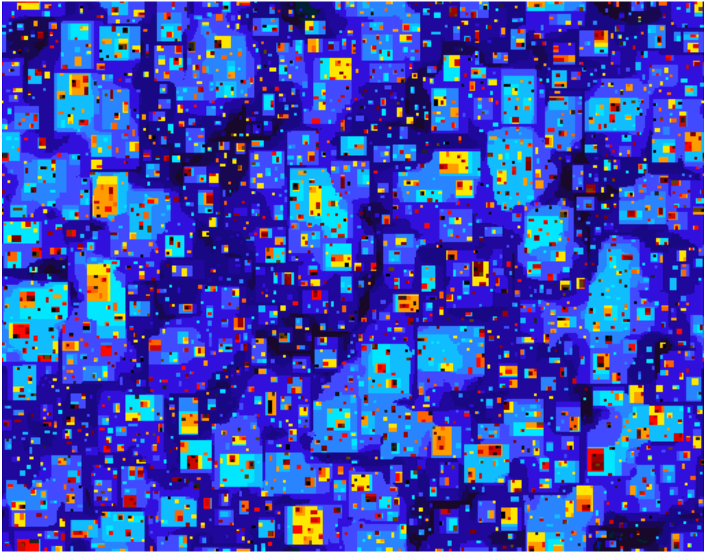


Figure 1. The original freshly occupied lattice shown on the left as well as the more compact clusters obtained after culling, on the right for the case of $m = 2$ on the square lattice, with initial concentration of $p = 0.55$. Although this concentration is below p_c for the infinite lattice this particular realization on the finite lattice does have a cluster (light grey) thats spans top to bottom.



SOC versus Bootstrap

- Formal similarities:
  - local dynamics highly nonlinear e.g. threshold function
  - SOC: if "infection" of site is larger $\Delta \Rightarrow$ infect neighbors
  - Boot: if "infection" of neighbors is larger $\Delta \Rightarrow$ infect site
  - this is a kind of local duality
- Differences:
  - SOC is not restricted to local threshold dynamics (example: Bak&Sneppen model of punctured equilibrium)
  - in general SOC is : a dynamical balance between
    - triggering the system slowly
    - dissipation at small set/boundary
  - this can cause the system to stabilize at a critical state (phase transition)
  - to make Bootstrap-SOC one would have to include such effects (loss of infection + feed in of infection)
  - possible candidate: competing bootstrap infections

Bootstrap percolation on regular trees

- find p_c s.t. for $p > p_c$ the whole $d-$tree becomes infected eventually with probability one
- $\Delta = 2, d > 3$: (Blanchard, Krueger 2005) and (Balogh,Peres,Pete 2006):

$$p_c = 1 - \frac{(d-2)^{2d-5}}{(d-1)^{d-2}(d-3)^{d-3}} \tag{4}$$

- for random trees with GF $g(z)$ and min-outdegree $\geq \Delta = 2$: $p_c = 1 - q$ where $q \in (0,1)$ is the smallest value s.t. the following equation has a real positive solution

$$\frac{z}{q} = (1-z)\,g'(z) + g(z) \tag{5}$$

(Blanchard,Krueger 2005)
- similar results for regular random graphs (Balogh,Pittel 2007)

Bootstrap on fat trees

- local clustering accelerates the infection spread (in classical epidemics clustering slows down the spread)
- $p_c^{fat} = \frac{3}{2} - \sqrt{2} < p_c^{tree} = \frac{1}{9}$
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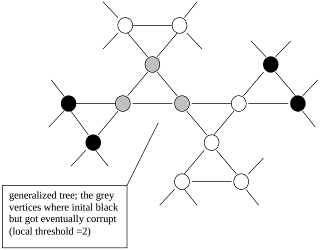
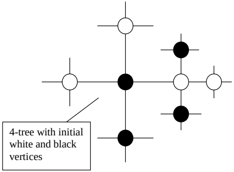




Bootstrap on Erdös&Renyi graphs

- for $G\left(n, p = \frac{c}{n}\right)$ one has

$$\begin{aligned} s_{t+1} &= 1 - (1 - p_0)\, e^{-cs_t} \sum_{l=0}^{l=\Delta-1} \frac{(cs_t)^l}{l!} && (6) \\ &= 1 - (1 - p_0) \Pr\left\{Pois\left(cs_t\right) < \Delta\right\} && (7) \end{aligned}$$

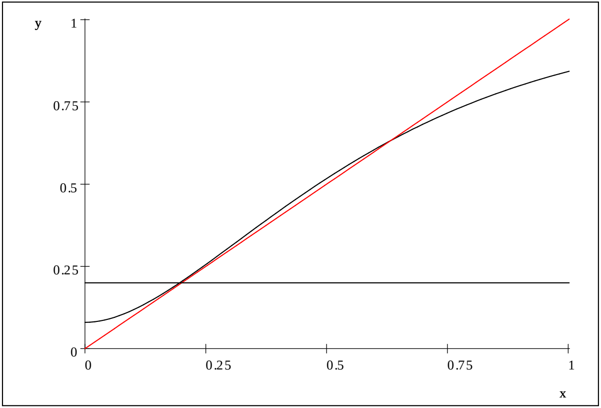
- there is at most one critical p_c
- for $\Delta = 2$

$$p_c = 1 - \frac{2e^{\left(-\frac{1}{2}+\frac{1}{2}c-\frac{1}{2}\sqrt{c^2-3-2c}\right)}}{c\left(-1+c-\sqrt{c^2-3-2c}\right)} \qquad (8)$$

- the critical window around p_c is of order $n^{-\frac{1}{2}}$

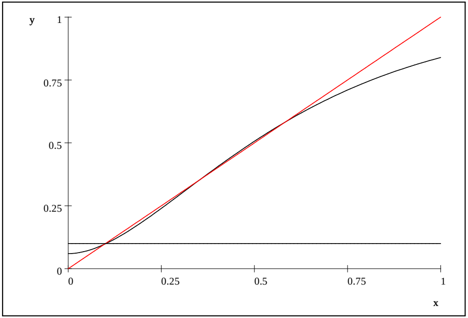
Dynamics at the critical density

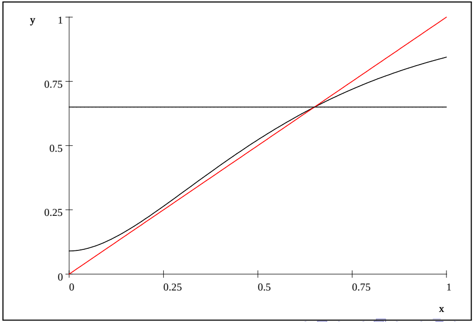
- Example: $p_0 = 0.08$, $c = 3.2$, $\Delta = 2$



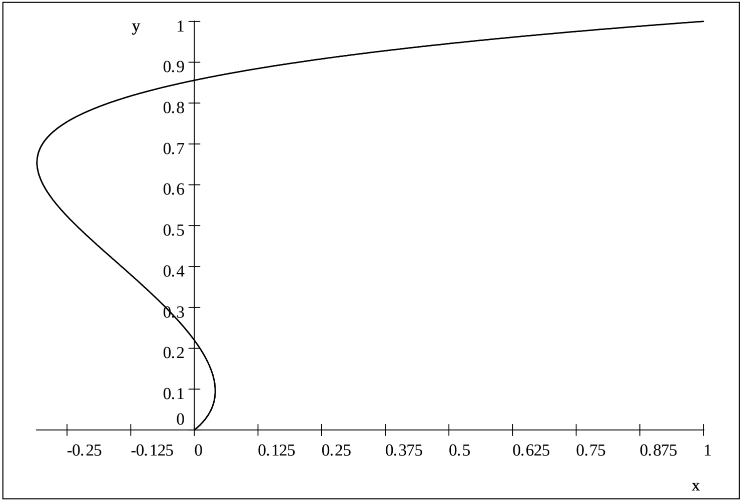
- around p_c long transient phases
- for $p_0 > p_0^c$: s is close to the $\Delta-$ core of the graph

above and below the critical density





The fixed point set as a function of the initial density (c=4)



Bollobas-Janson-Riordan graphs (2005)

- generalizations of Erdös-Renyi graphs
- good balance between simplification and complexity
- provide gauge models for more real life graph models
- inhomogeneous in vertex properties but independent edges
- exact mathematical estimations for many graph properties possible
- some parts of theory easy to extend to weighted graphs
- includes also evolving graphs
- appropriate for SIR processes with individual dependent infection rates

general setting for BJR graphs

- the n vertices have properties $\{\omega_i\}$ with values in ground space S
- the values $\{\omega_i\}$ are asymptotically μ distributed
- the edge probabilities for vertices of given types are independent and defined via a kernel κ

$$\Pr(i \sim j) = \frac{\kappa(\omega_i, \omega_j)}{n} \tag{9}$$

the giant component

- let $\rho(\omega) =$ $\Pr\{\text{Poisson process starting with type } \omega \text{ survives}\}$
  - $\rho(\omega)$ is solution of

$$f = 1 - e^{-Tf} \tag{10}$$

$$\text{with } Tf(\omega) = \int \kappa(\omega, \eta) f(\eta) \, d\mu(\eta) \tag{11}$$

- the giant component is given by $n \int \rho(\omega) \, d\mu(\omega)$ (vertices where the associated Poisson process does not die out join the giant component
- threshold: $\|T\| = 1 \Longrightarrow$ for $\|T\| = \infty$ no epidemic threshold!

Bootstrap percolation on BJR graphs

- Let $\kappa(\omega, \eta)$ be of finite type
- **Theorem** (Bl,D,Kr,S-S 2010):
  - Let p_0 be disjoint from a finite critical set $\left\{p_c^1, \ldots, p_c^l\right\}$, and let S be the asymptotic size of the bootstrap process on $\mathcal{G}(n, \kappa, \mu)$: **whp** $S = ns + o(n)$ where $s = \int s(\omega)\, d\mu(\omega)$ and $s(\omega)$ is the smallest solution $> p_0$ of

$$
\begin{aligned}
s(\omega) &= 1 - (1 - p_0)\, e^{-\int \kappa(\omega,\eta) s(\eta) d\mu(\eta)} \cdot \\
&\quad \cdot \left(\sum_{l=0}^{l=\Delta-1} \frac{1}{l!} \int \left[\kappa(\omega, \eta)\, s(\eta)\right]^l d\mu(\eta) \right)
\end{aligned}
$$

- Remark: For $\Delta = 1$ and $p_0 \searrow 0$ the formula reduces to the survival probability for the associated Poisson process

generalized epidemic processes

- the states of infection are encoded by a finite alphabet
  - e.g. $\left\{0 \triangleq \text{ not infected}, 1 \triangleq \text{ infected}, 2 \triangleq \text{ immune}\right\}$
- the infection process takes place on a graph $G\left(V, E\right)$ along edges $e \in E$
  - the dynamics is defined via local transition probabilities e.g.:

$$\Pr\left\{\chi_i\left(t+1\right) = k \mid \chi_i\left(t\right) = k'\right\} \quad (12)$$

$$= \quad f_{loc}\left(\chi_{B_1(i)}\left(t\right)\right) \oplus f_{mean}\left(\chi_G\left(t\right)\right) \quad (13)$$

    - $B_1\left(i\right)$: set of neighbors of i + vertex i
    - $\chi\left(t\right)$: state function at time t
  - classical assumption on f_{loc} : the infection events along edges are independent

local threshold processes as bootstrap percolation

- infection events along edges no longer independent
  - in many applications there are local thresholds $\Delta(i)$

$$\begin{aligned} \#\text{ infected neighbors} &< \Delta(i): \\ &\Longrightarrow \text{small infection rate } \varepsilon \end{aligned}$$

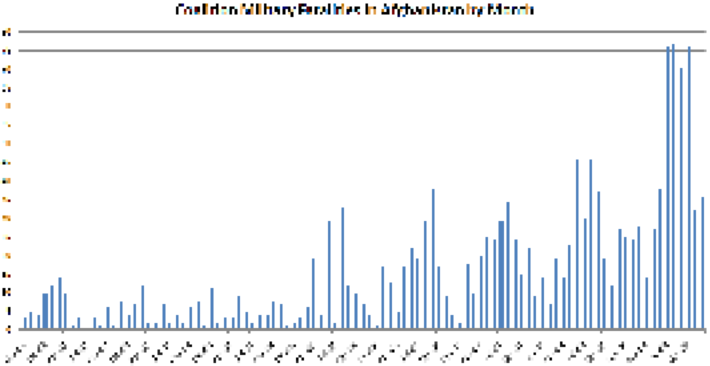
$$\begin{aligned} \#\text{ infected neighbors} &\geq \Delta(i): \\ &\Longrightarrow \text{high infection rate } \alpha \end{aligned}$$

- $\varepsilon = 0, \alpha = 1$ corresponds to bootstrap percolation
- examples: neuron dynamics, corruption, prejudices, knowledge spread, opinion spread

radical opinion formation and terrorism

- first studied by S.Galam in 2002 as a classical percolation problem
- his model: terrorist wants to move (spatially, on a lattice) from A to B
- he needs to follow a "continuos" path of passive supporters
- the lattice is determined by social dimension
- below p_c : terrorism is regionally restricted
- above p_c : terrorism becomes a global phenomena

the war in Afghanistan



passive supporters and terrorism

- Q: what is the effect of "collateral damages" on the prevalence of passive supporters?
  - example: Afghanistan
- Warning:
  - it is not a real life approximation
  - the model shows a possible qualitative dynamic process
- assume that on average:
  - to capture/eliminate an active terrorist causes m civilian victims
  - relatives/friends of victims are likely to become passive supporters (state 1)
- hence a counter terrorist action induces about rm new passive supporters
  - typically $rm \sim 10 - 1000$