

Astonishing Cellular Automata

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Cellular automata arise naturally in the study of physical systems, and exhibit a seemingly limitless range of intriguing behaviour. Such models lend themselves naturally to simulation, yet rigorous analysis can be notoriously difficult, and can yield highly unexpected results.

Bootstrap percolation is a very simple model, originally introduced in [13], which turns out to hold many surprises. Cells arranged in an L by L square grid can be either *infected* or *healthy*. Initially, we flip a biased coin for each cell, declaring it infected with probability p . At each subsequent time step, any healthy cell with 2 or more infected neighbours becomes infected, while infected cells remain infected forever. (A cell's neighbours are the cells immediately to its North, South, East and West - interior cells have 4 neighbours while boundary cells have fewer). See Figures 1 and 2.

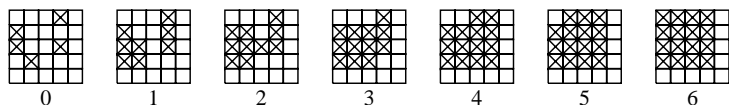


Figure 1: *Bootstrap percolation on a square of size 5 at time steps 0, ..., 6. Infected cells are shown by Xs.*

How does the probability

$$I(L, p) := \mathbb{P}(\text{the entire square is eventually infected})$$

behave for large L ? It is easy to guess that for p sufficiently large we have $I \rightarrow 1$ as $L \rightarrow \infty$, since the infection can always invade finite healthy islands. More surprisingly, it turns out that for *any* fixed $p > 0$ we have $I \rightarrow 1$ as $L \rightarrow \infty$. This was the first rigorous result in the subject, proved in [31]. The intuition is that certain rare local configurations act as “nucleation centres”, which can spread to infect any region (given a background of randomly infected cells), so any square large enough to contain a nucleation centre will become entirely infected.

Clearly for fixed L we have $I \rightarrow 0$ as $p \rightarrow 0$, so it is natural to ask what happens when L and p are varied simultaneously – how large must a square be, as a function of p , to have high probability of becoming entirely infected? An early breakthrough was the following.

Theorem 1. [3] *There exist positive constants C_{\pm} such that, as $(L, p) \rightarrow (\infty, 0)$,*

- $I(L, p) \rightarrow 1$ if $p \log L > C_+$;
- $I(L, p) \rightarrow 0$ if $p \log L < C_-$.

Other aspects of the model were subsequently studied in detail (e.g. [4, 9, 28, 30]), but the question of whether C_{\pm} could be replaced by a single sharp constant remained open for 15 years until the following result in 2003, which furthermore establishes the constant's value.

Theorem 2. [21] *Theorem 1 holds for all $C_- < \frac{\pi^2}{18} < C_+$.*

The above is an example of a *phase transition* – a sudden change in behaviour as a parameter (here $p \log L$) crosses a *critical value*

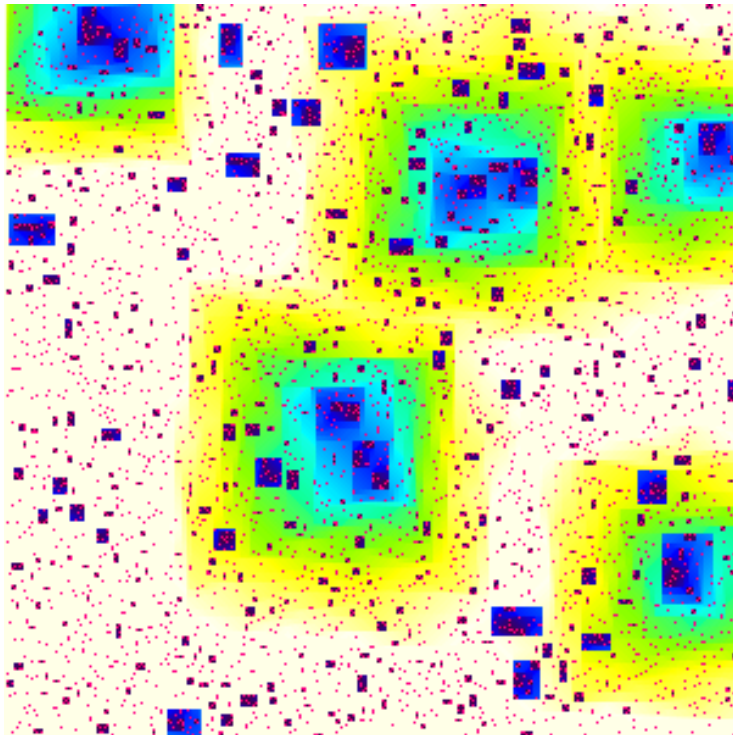


Figure 2: *Bootstrap percolation on a square of size $L = 300$. Cells were initially infected with probability $p = 0.05$. In this example all cells eventually became infected, and the colours indicate time of infection: red (initial infections), black (earliest), followed by dark blue, light blue, green, yellow (latest).*

($\pi^2/18$). The proof involves finding accurate bounds on the density of nucleation centres.

Bootstrap percolation seems ideally suited to computer simulation. Indeed this can be a valuable tool – Figure 2 immediately suggests the idea of nucleation (a vital ingredient of the proofs). Amazingly however, the resulting numerical predictions differ greatly from the above rigorous result. On the basis of simulation of squares up to size $L = 28800$, the critical value was estimated earlier in [2] to be 0.245 ± 0.015 . (This conclusion was natural given the data, and subsequent larger simulations appear at first sight to confirm it.) However the rigorous value $\pi^2/18 = 0.548311 \dots$ is larger by more than a factor of two! The discrepancy seemingly arises because convergence to the limit $\pi^2/18$ is extremely slow. In fact, combining simulation data with Theorem 2 suggests that a square of size at least $L = 10^{20}$ would be needed in order to get close. This points to further intriguing questions which we shall discuss later.

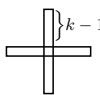
Cellular automaton models may be loosely defined as uniform local rules for updating the states of an array of cells. Aside from their intrinsic mathematical interest, such models have been applied to wide variety of physical problems. In the case of bootstrap percolation these include magnetic alloys, hydrogen mixtures, computer storage arrays, and crack formation (see e.g. the references in [1]).

For the last example, imagine a lattice of atoms, with a small fraction p “missing” or “defective”, and where any atom with two or more defective neighbours becomes defective. Rather than accurately approximating reality, the aim is to capture phenomena of interest (such as nucleation), and study them in the simplest setting possible. Interestingly, a relevant scale for some applications might be $L \approx 10^{10}$, where the most accurate predictions currently arise from interpolation between simulation and theoretical results. A further motivation for studying simple models is for use as tools for the rigorous analysis of more complicated systems. See [10, 15] for recent examples in the case of bootstrap percolation. The model also arose independently in the following (see [32] for background).

Puzzle. *Prove that the entire square cannot become infected with fewer than L cells initially infected. (There is a one-word solution!)*

Theorem 1 has been extended to several variant models, sometimes with other functional forms replacing the parameter $p \log L$. These include: $p \log \dots \log L$ for the d -dimensional model [11, 12]; $p^\alpha \log L$ for 2-dimensional models with various symmetric neighbourhoods [17, 18]; $p(-\log p)^\beta \log L$ for certain asymmetric versions [18, 29]; $pn^2 4^{\sqrt{n}}$ for the hypercube [8]; $p\sqrt{q}$ for a “ q -polluted” lattice [20]. The existence of a sharp constant as in Theorem 2 is conjectured in all the cases above, but proved only for two other models below. In the *modified model*, a healthy cell becomes infected if it has at least one infected neighbour in each dimension.

Theorem 3. [22] *For the modified model in $d \geq 2$ dimensions, Theorem 2 holds with parameter $\underbrace{p \log \dots \log L}_{d-1}$ and critical value $\frac{\pi^2}{6}$.*

Theorem 4. [23] *For the cross-shaped neighbourhood  in dimension 2 with an infection threshold of $k(\geq 2)$ neighbours, Theorem 2 holds with parameter $p \log L$ and critical value $\frac{\pi^2}{3k(k+1)}$.*

It is an open problem to extend Theorem 3 to the standard model in dimensions ≥ 3 , and more generally to find a unified method for proving the existence of sharp critical values. (The latter is reminiscent of the famous random k -SAT problem.) The combinatorial structure behind the constants $\frac{\pi^2}{3k(k+1)}$ is somewhat subtle and mysterious. A by-product of Theorem 4 was the following, which stimulated further investigations in [5].

Theorem 5. [23] *The number $a_k(n)$ of integer partitions of n not containing any k distinct consecutive parts satisfies*

$$\log a_k(n) \sim \pi \sqrt{\frac{2}{3} \left(1 - \frac{2}{k(k+1)}\right) n} \quad \text{as } n \rightarrow \infty.$$

Adding randomness to the *evolution* of cellular automata often makes them more tractable. Interacting particle systems (spatial models involving Markovian evolution in continuous time) have been studied with great success (see [27]). In contrast, models involving deterministic evolution from random or deterministic initial conditions, while physically natural, can be very challenging, and their rigorous study is in its infancy. Besides bootstrap percolation, Figure 3 gives tastes of a few recent advances. Traffic in the BML model (i) is proved to jam at high densities, but nothing is known about low densities. The n th stage of the rotor aggregation (ii) is known to be within distance $n^{1/4+\epsilon}$ of a disk, but believed to be within distance 2. Certain Packard snowflake models (iii) fill only a proper fraction of the plane, yet would completely fill any simulation up to size 10^9 . In a different direction, limiting trajectories in the random sorting network (iv) are believed to be random sine curves, and known to be Hölder(1/2).

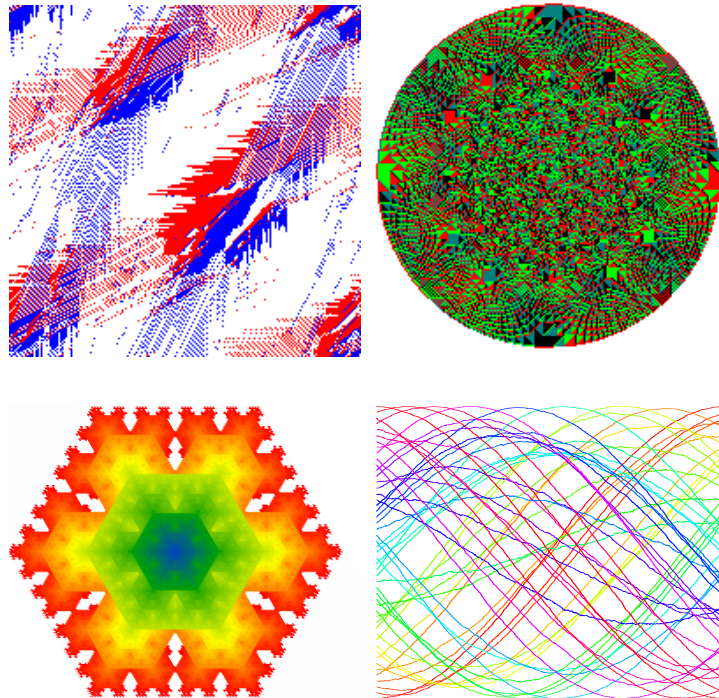


Figure 3: (i) Biham-Middleton-Levine traffic model [6]; (ii) Propp’s rotor-router aggregation [26, 25] (also see [24]); (iii) Packard’s snowflake model [16]; (iv) random sorting network [7].

Returning to bootstrap percolation, it is of interest to explain the apparent discrepancy between simulations and limiting behaviour. These issues have been investigated partly non-rigorously in [14], and some rigorous answers are proved in [19].

Theorem 6. [19] *For bootstrap percolation in 2 dimensions, there exists a positive constant c such that, as $(L, p) \rightarrow (\infty, 0)$,*

$$I(L, p) \rightarrow 1 \quad \text{if } p \log L > \frac{\pi^2}{18} - c(\log L)^{-\frac{1}{2}}.$$

Suppose one tries to estimate the critical value $\pi^2/18$ using $p_{1/2} \log L$, where $p_\alpha(L)$ is defined via $I(L, p_\alpha) = \alpha$. In order to halve the error term $c(\log L)^{-1/2}$, one must raise L to the power 4. In the case of the modified model in 2 dimensions, explicit estimates are available, and imply for example that $p_{1/2} \log L$ is not within 1% of $\pi^2/6$ even when $L = 10^{3000}$ (see [19]).

While Theorem 6 shows that the distance from the critical parameter $p_{1/2} \log L$ to its asymptotic value is $\Omega((\log L)^{-1/2})$, the width of the critical window $p_{1-\epsilon} \log L - p_\epsilon \log L$ is much smaller, $O(\log \log L / \log L)$ (or $\theta(1/\log L)$ if we fix p rather than L) – see [3, 8, 19]. This contrasts with models such as the Erdős-Renyi random graph, where the window shrinks more slowly than its centre converges.

Bootstrap percolation also seems to exhibit *crossover* – the approximate critical value 0.245 estimated in [2] appears accurate over a wide (but bounded) interval of L . It is a fascinating challenge to understand this phenomenon rigorously. One possible approach is to prove a limiting result for some sequence of models in which this interval becomes longer and longer.

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