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LETTER TO THE EDITOR

Mean-field self-organized criticality

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Abstract. We consider a variant of directed percolation in which the set of nodes is itself dependent on the progresss of the percolation process. For the mean-field model studied, the system is driven to its critical point from a broad range of initial conditions. An advantage of the model is its explicit and easy solvability.

The notion of self-organized criticality has the potential to explain a variety of natural phenomena. The first striking metaphor used was the sandpile [1, 2] and other more analytically tractable models have also been proposed [3]. In this letter we give a model that has the virtue of being *extremely* simple. It is a mean-field model and everything can be done analytically (see [4] but note that this is a mean-field theory of the sandpile and is quite different from ours). We do cite computer studies, but mainly to show that fluctuations do not overwhelm the mean-field predictions. Since there is no spatial dependence, the criticality is expressed through power law decay of the order parameter.

The model is a variant of directed percolation on a finite set in which bonds connect all elements of the set. It is convenient and also suitable, for the variant we introduce, to phrase the model in epidemiological terms, and the underlying disease has been termed percolitis [5]. The variant to be introduced here will be called population modulating percolitis (PMP) and can show self-organized criticality as well as other epidemic properties. The appearance of self-organized criticality in systems equivalent to epidemiological models has been noted before, [6, 7] and in these works will be found discussions providing a broader perspective for the present letter.

Let $\mathcal{N}$ be a collection of N objects and consider an infinite set of labelled copies, $\mathcal{N}_t$, $t = 0, 1, \ldots$, with directed bonds from every element of $\mathcal{N}_t$ to every element of $\mathcal{N}_{t+1}$. These bonds are occupied with probability p. It is convenient to define $\{0, 1\}$-valued bond occupation variables $A_{\beta\alpha t}$ with $\beta \in \mathcal{N}_t$ and $\alpha \in \mathcal{N}_{t+1}$. The usual directed percolation question in terms of this model is the finding of a path of occupied bonds from a point in $\mathcal{N}_0$ to points in $\mathcal{N}_t$ for arbitrarily large t. Because N is finite this cannot happen and in this model the phase transition is an asymptotic property. Let $p = x/N$. Then for $x < 1$ paths are O(1) for $N \to \infty$, while for $x > 1$ path length can grow unboundedly (like $e^{N\cdot\text{constant}}$) for $N \to \infty$. In the epidemic picture the starting point in $\mathcal{N}_0$ is a sick individual, and an occupied bond ($A_{\beta\alpha t} = 1$) corresponds to disease transmission from β to α from time step t to time step $t+1$. (For $A_{\beta\alpha t} = 1$, if β was sick at t, then α will be sick at $t+1$.) It is useful to define another set of $\{0, 1\}$-valued variables $\sigma_\alpha(t)$ which take the value 1 if $\alpha \in \mathcal{N}_t$ is sick, 0 otherwise. Progress of the disease is then given by

$$\sigma_\alpha(t+1) = 1 - \prod_{\beta \in \mathcal{N}_t} (1 - A_{\beta\alpha t}\sigma_\beta(t)). \tag{1}$$

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We further define $\rho(t) \equiv \langle \Sigma \sigma_\alpha(t)/N \rangle$. Because the As are all independent it is clear from (1) that

$$\rho(t+1) = 1 - e^{-x\rho(t)}. \tag{2}$$

(For finite N, $1-\rho(t+1)$ is given by $(1-x/N)^{Np(t)}$.) For given x, the equilibrium value of ρ, $\bar{\rho}(x)$, satisfies

$$\bar{\rho}(x) = 1 - e^{-x\bar{\rho}(x)} \tag{3}$$

which has a strictly positive solution only for $x > 1$, indicating that the critical point is at $x_c = 1$.

The characteristic critical property for our purposes will be the relaxation of $\rho(t)$ to $\bar{\rho}$. Linearization of (2) ($\eta(t) \equiv \rho(t) - \bar{\rho}$) yields

$$\eta(t+1) = x\, e^{-x\bar{\rho}} \eta(t) \tag{4}$$

so that we find $\xi_\parallel = -1/\log(x\, e^{-x\bar{\rho}})$ to be the relaxation time or 'correlation length' for this problem. For all (positive) $x \neq 1$, $x\, e^{-x\bar{\rho}} < 1$, and for $x \to x_c$, $\xi_\parallel \to \infty$. Away from criticality, relaxation is thus exponentially fast. At $x = 1$ we retain quadratic terms in $\rho(t)$ ($= \eta(t)$), since $\bar{\rho}(1) = 0$, to get

$$\rho(t+1) = \rho(t) - \tfrac{1}{2}\rho(t)^2 \tag{5}$$

which has the (approximate) solution

$$\rho(t) = \frac{2}{t + t_0} \tag{6}$$

with t_0 arbitrary. This time dependence is a characteristic manifestation of criticality in this system.

In previous publications [5, 8] the time steps were a day or a week and each sick individual was to have come into contact with every individual for potential disease transmission. In the present model, each time step is a single generation and we have in mind that successive copies of $\mathcal{N}$ are *different* people, $\mathcal{N}_{t+1}$ comprising the descendants of $\mathcal{N}_t$. The new feature is that the size of $\mathcal{N}$ will be allowed to vary and that the size of $\mathcal{N}_{t+1}$ will depend on the number of sick individuals in $\mathcal{N}_t$. The picture is that percolitis is a childhood disease and affects the fertility of stricken individuals. In particular we will assume that couples that have *both* had percolitis have slightly fewer offspring. Other models are certainly possible and at the end of this letter we will mention rules that simulate aspects of sickle cell anaemia.

The full PMP model is defined as follows: an initial population $N(0)$ and an initial number of sick individuals $S(0)$ are given. The population at time 1 ($N(1)$) is obtained by a series of random processes. N will always be even and we first do a random pairing of the elements of $\mathcal{N}_0$ to produce $N/2$ couples. By a second random process we ascribe to each couple 0, 2 or 4 descendants. For couples in which zero or one partner was sick the mean number of descendants is 2. For couples in which both partners were sick the probabilities are adjusted to put the mean slightly below 2. This produces $\mathcal{N}_1$. Next, each of the $N(1)$ individuals (in $\mathcal{N}_1$) has the opportunity to contract percolitis from the $S(0)$ sick individuals of $\mathcal{N}_0$. This random process is described by (1) with $\beta \in \mathcal{N}_0$, $\alpha \in \mathcal{N}_1$ and $t = 0$. The entire procedure continues with alternate determination of $N(t)$ and ($S(t)$).

Mean-field equations for this process are easy to derive. Let $p = 1/M$; scaling by 'N' (i.e. our previously defined $x = pN$) is inappropriate since N varies in time. The expectation of (1) yields

$$\rho(t+1) \equiv \frac{S(t+1)}{N(t+1)} = 1 - \left(1 - \frac{1}{M}\right)^{S(t)} = 1 - e^{-\rho(t)\nu(t)} \tag{7}$$

where $\nu(t) \equiv N(t)/M$ and we have ignored order $1/M$ corrections. We will also freely replace random variables by their expectations. The check that fluctuations do not alter our conclusions was made numerically and will be reported below. For the population size, $N(t)$, if percolitis were absent its expected value would be the same on successive time steps, $\langle N(t+1)\rangle = \langle N(t)\rangle$. For double-percolitis couples there will be reduced fertility so that there will be an effective reduction proportional to $\rho(t)^2$. For (the expectation of) $\nu(t+1)$ we therefore have

$$\nu(t+1) = \nu(t)[1 - \alpha\rho(t)^2] \tag{8}$$

where α is a proportionality constant reflecting the percolitis induced fertility defect.

Equations (7) and (8) can be iterated numerically and it is found that for small enough α the system converges to $\rho = 0$, $\nu = 1$. Moreover, that convergence is *not* exponential and has the inverse power law characteristic of critical percolitis.

Before analysing the equations we provide a simple explanation of what is happening. For large initial population ($N_0 \gg M$) percolitis is rampant. $\nu(0)$ ($\gg 1$) acts like the 'x' of simple percolitis and $\bar{\rho}(\nu(0))$ is near unity. Therefore most couples are percolitis doubles and $M(t)$ gradually diminishes. However, the reduced population has fewer double-percolitis couples so that the *rate* of diminution is itself reduced. In this process the population gradually tends toward M, the value at which the disease itself could no longer be sustained. As one approaches M, however, the number of double-percolitis couples is severely reduced ($\sim\rho^2$) so that, as we shall see, the result is the same inverse power law that characterizes critical ($x = 1$) simple percolitis.

For (7) and (8), all points (ρ, ν) with $\rho = 0$ are fixed points and there are no others. For $\nu > 1$ these are not stable (as for $\bar{\rho} = 0$ for $x > 1$), while $(0, \nu)$ is definitely an attractor for $\nu < 1$. The question of interest is whether $(0, 1)$ has a non-trivial basin of attraction and if so how that point is approached. Writing $\nu(t) = 1 + \delta(t)$ and expanding up to terms quadratic in ρ and δ, (7) and (8) yield

$$\rho' = \rho + \rho\delta - \tfrac{1}{2}\rho^2 \qquad \delta' = \delta - \alpha\rho^2 \tag{9}$$

where unprimed quantities are for t and primed for $t+1$. Bearing in mind the irrelevance of the time scale and using the smallness of ρ and δ, (9) becomes

$$\dot{\rho} = \rho\delta - \tfrac{1}{2}\rho^2 \qquad \dot{\rho} = -\alpha\rho^2. \tag{10}$$

The forms $\rho = R/(t + t_0)$ and $\delta = D/(t + t_0)$ satisfy (10) provided R and D satisfy

$$-R = RD - \tfrac{1}{2}R^2 \qquad -D = -\alpha R^2 \tag{11}$$

which implies for non-trivial (i.e. >0) R

$$R = \frac{1}{4\alpha}(1 \pm \sqrt{1 - 16\alpha}). \tag{12}$$

For $\alpha > \frac{1}{16}$ the attempted form does not provide a real solution, but for $\alpha < \frac{1}{16}$ we get the critical behaviour that we seek. The population approaches M, the disease nearly

disappears, the rate of approach to M is slowed yet further and the disease is further reduced; and so it goes. The path actually followed by the asymptotic solution is given by the positive root in (12). The negative root provides the separatrix: Even for $\alpha < \frac{1}{16}$, if you begin with ν too small, the solution tends to $\nu < 1$. By numerical means we determined the actual position of the separatrix and (for $\alpha = 0.5/16$) it lies close to its linear approximant. Because the time is discrete one could in principle have paths crossing the separatrix (in effect, not have a separatrix) but this did not seem to happen (and the occurrence of this phenomenon at large ρ or $\nu - 1$ would not affect our conclusions).

Besides the exact solution given above to (10), the changing character, as a function of α, of the fixed point $(\rho, \delta) = (0, 0)$ can be seen by examining the equation $d\delta/d\rho = -\alpha\rho/(\delta - \rho/2)$. The phase portrait (but not the time dependence!) is given by the equations $d\rho/ds = \delta - \rho/2$, $d\delta/ds = -\alpha\rho$, where s is a new parameter. Standard stability analysis shows that for $\alpha < \frac{1}{16}$ one trajectory goes *into* $(0, 0)$, one *out* (corresponding to the roles of asymptote and separatrix, seen above). For $\alpha > \frac{1}{16}$ the trajectories are spirals tending to 0. However, in the original time variable these spirals never get past their first loop since it takes infinite time (t) to reach $\rho = 0$.

This power law time dependence demonstrates self-organized criticality in our system. To see the phenomenon graphically, and also to be sure that fluctuations do not vitiate the equations we derived for the average quantities ρ and ν, we performed computer simulations of PMP. The results are shown in figure 1. We plot both $N(t)$ and $S(t)$. Even for the relatively small M used (4000) the system settles into power law decline. We also did a straight line fit to (the noisy) $1/S(t)$ and show this in figure 2. The slope gives reasonable agreement with the theory of (9). For the α we used (0.1/16), $(R)_{+\text{root}}$ is 77.95 while the effective value obtained from figure 2 is 79.5. (Another run gave 90, so 79.5 is coincidentally close.)

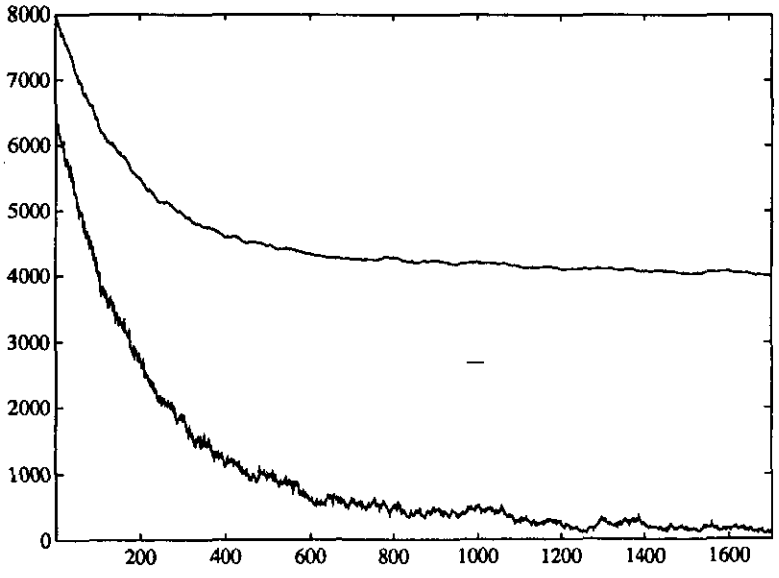

Figure 1. $N(t)$ and $S(t)$. For this simulation M $(=1/p)$ was taken to be 4000 and α was 0.1/16. The level of noisiness in the population variation was kept fairly low (although its randomness is evident in the figure) so as to get reliable results even for fairly small overall population.

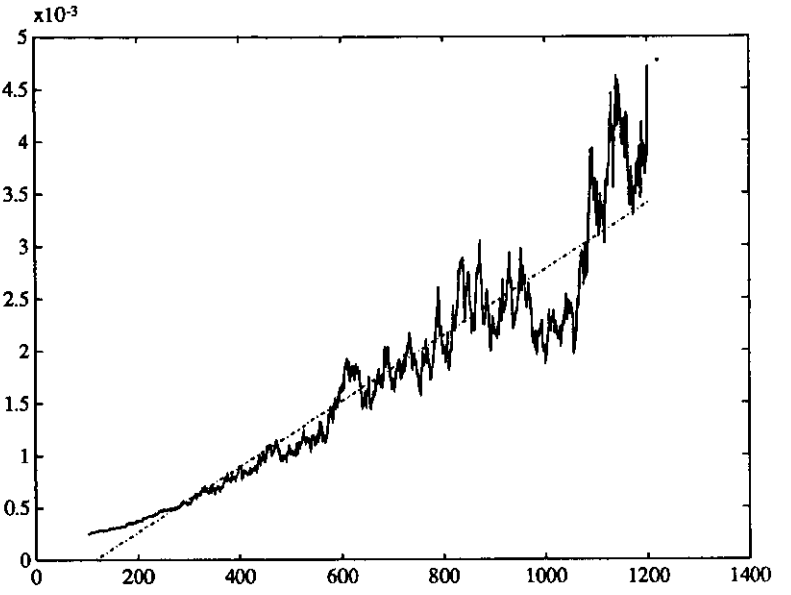

Figure 2. $1/S(t)$ (from the data of figure 1) and its fit (dotted line).

In the pure percolitis model, criticality (in the sense of power law decay) is surely not generic. However, when the 'disease' influences its own propagation the feedback is able to drive the system precisely to the critical point. For a different kind of feedback, one in which the fertility defect was linear in ρ, one would not get criticality. For the model that we do define, a small (linear) deviation from the quadratic ρ dependence cannot occur; this restriction is analogous to the constraints imposed by symmetries in other contexts.

By virtue of its simplicity, this model allows examination of general ideas on self-organized criticality. Of course there are aspects that are beyond its scope, for example the relation of spatial features to time features and to $1/f$ noise. Our model has no space. One of the important general ideas that can be studied in our model is the way linear response theory makes or does not make its appearance. In its simplest form, linear response theory would predict exponential decay. How does our model depart from that simplest form? One way to look at this is to say that the coefficients in the linear response theory themselves become functions of the system variables. This viewpoint is consistent with the intuitive discussion above in which the population changes are pictures as gradually modifying the percolitis process, which effectively means a time dependent 'x.' A second way is to recall the power law (in time) decay of correlations in hydrodynamics; these arise because of an infinite number of modes with vanishing energy differences. It is likely that the same situation exists here as well. In studies of ordinary percolitis it is found [9] that the transfer matrix (time evolution operator) has an infinity (as $N \to \infty$) of eigenvalues approaching 1. The PMP transfer matrix is a more elaborate object, but if one imagines it used for time evolution, then our demonstration of a power law dropoff shows that it necessarily has a zero mass gap [6, 7, 10].

There is a known physical example where modified directed percolation drives the effective percolation parameter toward its critical point. This is the stochastic stimulated star formation model described in [11] and [12]. One defines there an effective

percolation parameter for the probability that a supernova explosion in one region of a galaxy induces star formation a few hundred light years away. That parameter, in turn, is influenced by the recent and, to some extent, local, star formation history of the galaxy. This *feedback* process drives the system to an effective parameter *just a bit* above the percolation threshold. (This proximity to the critical point, by the way, accounts for the beautiful spiral arms seen in many disc galaxies.) This system does not quite exhibit self-organized criticality although the feedback modified phase transition does show some peculiar features.

In terms of the percolitis model, this feedback can be achieved by allowing a degree of immunity subsequent to a disease episode. (In the neuronal model [13] this corresponds to post-activation refractoriness.) For the mean-field percolitis described here, immunity does not lead to self-organized criticality (even with protracted immune periods) because of the long-range interactions. However, with finite-dimensional directed percolation (e.g. $(1+1)$- or $(2+1)$-dimensional) and long term, perhaps power law, immunity, we conjecture that the system would be driven to criticality.

The following example is analogous to the situation for sickle cell anaemia, where an individual homozygous in the sickle cell allele does not survive but those heterozygous for this allele have an unusually high resistance to malaria. The analogy is not complete because for the percolitis model the probability of falling 'victim' to the disease is independent of the individual's parentage.

Let the reduced fertility of a double again be described by α. For a couple with exactly one percolitis partner let the enhanced fertility (or survival of offspring) be described by a parameter β. The mean-field equations become

$$\rho(t+1) = 1 - e^{-\rho(t)\nu(t)}$$

$$\nu(t+1) = \nu(t)[1 - \alpha\rho(t)^2 + \beta\rho(t)(1-\rho(t))].$$

For equilibrium, the second equation implies $\rho = 1 - \alpha/\beta$. Inserting this in the first equation we have in turn $\nu = -\log(\alpha/\beta)/(1-\alpha/\beta)$. The equilibrium population is thus νM. This is not a critical phenomenon, but illustrates another aspect of the model.

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