SOC in a population model with global control

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Abstract

We study a plant population model introduced recently by J. Wallinga [OIKOS \textbf{74}, 377 (1995)]. It is similar to the contact process (‘simple epidemic’, ‘directed percolation’), but instead of using an infection or recovery rate as control parameter, the population size is controlled directly and globally by removing excess plants. We show that the model is very closely related to directed percolation (DP). Anomalous scaling laws appear in the limit of large populations, small densities, and long times. These laws, associated critical exponents, and even some non-universal parameters, can be related to those of DP. As in invasion percolation and in other models where the rôles of control and order parameters are interchanged, the critical value $p_c$ of the wetting probability $p$ is obtained in the scaling limit as singular point in the distribution of infection rates. We show that a mean field type approximation leads to a model studied by Y.C. Zhang \textit{et al}. [J. Stat. Phys. \textbf{58}, 849 (1990)]. Finally, we verify the claim of Wallinga that family extinction in a marginally surviving population is governed by DP scaling laws, and speculate on applications to human mitochondrial DNA.
1 Introduction

The main concepts in the description of critical phenomena are the control parameter (e.g., the temperature $T$ or a wetting probability $p$) which is typically an intensive quantity, and the order parameter (magnetization, density, ...) which is typically the density of an extensive quantity. As the control parameter passes through the critical point, correlation lengths and times diverge and the order parameter shows a singular (albeit continuous) behavior.

These concepts apply not only to equilibrium physics but also to systems intrinsically off equilibrium. Maybe the best known example of a critical phenomenon which has no static analogue is directed percolation (DP) with the preferred axis taken as time $t$. In this interpretation, DP describes an epidemic process without immunization or death, the so-called ‘contact process’ or ‘simple epidemic’ \cite{1, 2, 3}. The infection rate can be chosen as control parameter in this interpretation, and the density of infected individuals is the order parameter. The critical point corresponds then to a transition from a regime where any finite epidemic dies out finally, to a regime where the epidemic can grow sufficiently fast that it lives forever in spite of statistical fluctuations. Exactly at the critical point the size of the epidemic, both in terms of the number $N$ of infected individuals and in terms of its spatial extent $R$, increases with time, while the density $\rho \sim N/R^d$ decreases to zero.

But, as pointed out by P. Bak \textit{et al.} \cite{4}, there exist also nonequilibrium models which show anomalous scaling related to diverging length scales without possessing any obvious control parameter. This phenomenon, called self-organized critical (SOC), has been vigorously studied in the last ten years. It appears in models for sand piles \cite{4}, earthquakes \cite{5}, biological evolution \cite{6} and forest fires\cite{7, 8}, among others. A common signature of those models is that they are slowly driven into an unstable regime. In the limit of zero driving speed and infinite system size, the system is forced into a critical state regardless of the initial conditions. Another model which shows the same behavior (and which can therefore be regarded as a forerunner of SOC) is invasion percolation \cite{9}.

Actually, there are substantial conceptual differences between different models with SOC. While in some of them (like the sand pile and forest fire models) the slow driving appears to be ‘genuine’ and hard to replace by anything else without destroying criticality, this is not so in other models. Prototypes of the latter are invasion percolation and other models with ‘extremal dynamics’ \cite{6, 10}. In these models, each site on a lattice carries some ‘fitness’ $f$, and evolution takes place only at the site with minimal $f$. Obviously, one could in these models replace the evolution by one where all sites evolve whose fitness is below some critical value $f^*$. This $f^*$ would then play the role of control parameter, and critical behavior is found when $f^* = f_c$. The close connection between this non-self organized critical models and their SOC cousins is demonstrated by the fact that fitness distributions in SOC models with extremal dynamics show singularities precisely at $f = f_c$.

Therefore, some SOC models are just characterized by an interchange between order and control parameters \cite{1, 2}. Indeed, this is true \textit{cum grano salis} also in other SOC models like the sand pile model. Also there the essential feature is that control is not exerted via an intensive parameter, but directly on a global flux. In equilibrium systems, a similar situation would prevail if we would switch from a (grand-) canonical ensemble...
to a microcanonical ensemble. In general this has no substantial effect on equilibrium phase transitions and critical behavior, although there exist exceptions [13, 14]. On the other hand, it is e.g. well known that replacing voltage control by current control can have dramatic effects in nonlinear (and non-equilibrium!) electric circuits.

In the present paper we shall discuss a model which can be understood as ‘control-switched’ directed percolation. Instead of controlling the infection rate as in ordinary DP, we control the size of the epidemic. Essentially (with some minor differences outlined below) our model was introduced first by Wallinga [15] as a model for a population of weeds. In this model a farmer’s field is treated as a regular \(d\)-dimensional lattice of size \(L^d\), with at most one weed per lattice site. Seeds from these plants fall onto all neighboring sites and in spring a new weed is growing on all those sites. This alone would lead to supercritical growth of the population. But the farmer has decided that he will not tolerate more than \(N\) weeds in the entire field. Thus he eradicates all plants in excess of this number. He does this in a completely random way, by picking plants from random sites until exactly \(N\) plants are left over.

The critical regime in this model is reached for \(N \to \infty\) and \(L \to \infty\), with \(\rho = N/L^d \to 0\). As we shall see in the next section, the case of finite \(N\) and \(\rho = 0\) corresponds to subcritical DP, while that of \(N = \infty\) and finite \(\rho\) is equivalent to supercritical DP.

The model will be defined more formally in the next section. There, we shall also formulate scaling laws, and test them in one spatial dimension (simulations were also performed for \(d = 2\), but will not be shown here). We will find excellent agreement, with one notable exception. This exception shows that we have not yet fully understood the model. On the other hand, some of the observed scaling laws have also non-trivial consequences for ordinary DP. A discussion of our results and a comparison with previous results is presented in sec.3. Among others, we will treat there a mean field type approximation where we relax the constraint that each site is occupied by one plant at most. We will see that this leads to a model studied by Zhang et al. [16] in which all critical exponents assume their mean field values.

### 2 The model and its scaling laws

#### 2.1 The model

Consider a population of \(N\) plants distributed on a \(d\)-dimensional hypercubic lattice of linear size \(L\), such that any site is either empty or occupied by one plant. Boundary conditions are assumed to be periodic. The reproduction and distinction of the plants happens in discrete time steps and is controlled by the following rules:

1. At the beginning the \(N\) plants are randomly distributed onto the lattice.
2. At each time step \(t\) every plant places \(2d\) seeds at its neighboring sites and dies.
3. On every site which has received at least one seed a new plant is growing. Notice that this implies that all seeds die which fall onto sites which contain already another seed. Nevertheless, it is easy to see that the number \(M(t)\) of new plants is strictly larger than \(N\).
4. The size of the population is kept constant by randomly removing \(M(t) - N\) plants, and leaving \(N\) in place. The fraction of surviving plants is denoted by \(\tilde{p}(t) = N/M(t)\).
This model differs from that in [15] in the assumption that seeds fall only onto neighbors of the parent plant, not onto the site of the parent plant itself. This is done in order to make closer contact to existing treatments of DP.

A variant of this model is obtained by replacing steps (i)-(iii) by the following steps:
(i') Plants are chosen at random, and for each chosen plant a random nearest neighbor is chosen as well. If this neighbor had already been chosen in the same time step, another plant is chosen. This is repeated until a free neighbor is found, and a seed is put there.
(ii') This is repeated until $N$ seeds have been placed.
(iii') After that, each of the seeds grows into a new plant. The number of bonds (pairs of different neighbors) tested during this time step is denoted as $M(t)$, and $	ilde{p}(t) = N/M(t)$.

Just as the first variant will be seen to correspond to directed site percolation, this variant corresponds to bond percolation.

In the following we shall present numerical results from simulations for $d = 1$. We have also made simulations in two dimensions. They show essentially the same results. But they are less significant since deviations from mean field behavior are less pronounced, they are harder to analyze, and they are more affected by finite size corrections. Therefore we shall not present them in this paper. We shall present results only for the first variant, although again we have also performed extensive simulations for the second variant.

Actually, instead of starting with a random distribution, we started in some runs with all plants located at even sites of the lattice. As we shall show in subsec. 2.4, for $\rho < 1/2$ and $t \to \infty$ only such configurations survive where the number of empty sites between two plants is odd. Starting already with such a configuration can then shorten transient times.

### 2.2 Supercritical case

Let us first study the case where $N$ is large, and $\rho = N/L^d$ is non-zero in the limit $N \to \infty$. In this case it is obviously irrelevant whether we control $N$ rigidly or just in average, with fluctuations of order $N^{1/2}$. Such a soft control can be implemented by killing new plants completely at random, with probability $0 < 1 - p < 1$ for each new plant. As long as $p$ is larger than the critical threshold for directed site percolation (which is $0.705485$ in $d = 1$ [L] – notice that $d = 1$ in our notation corresponds to 2 dimensions of space-time), this will be exactly the case of supercritical DP. In this case correlations are indeed short range and fluctuations of $N$ are of order $N^{1/2}$, as we required. Thus we conclude that our model and supercritical DP coincide in the limit $N \to \infty$, $\rho = \text{const} > 0$.

In particular, in this limit the long time average of the fraction of surviving plants should coincide with the ‘wetting’ probability. Let us denote the ‘equation of state’ for DP as
\[
\rho = \Phi(p)
\] (1)
where $\rho$ is the density of infected sites in an infinitely extended epidemic. For $p \to p_c$ from above, one has
\[
\Phi(p) \sim |p - p_c|^\beta.
\] (2)
We predict then that the time average of $\bar{p}(t)$ satisfies exactly the same relations,

$$\langle \bar{p} \rangle \equiv \langle N/M(t) \rangle = \Phi^{-1}(\rho) \tag{3}$$

and

$$\langle \bar{p} \rangle - p_c \sim \rho^{1/\beta} \tag{4}$$

for $N \to \infty$, $\rho = N/L^d > 0$. Deviations from eq.(3) for finite $N$ should be analytic, since the model is not critical. Indeed we found that these deviations decreased as $1/N$. The function $\Phi(\rho)$ obtained from eq.(3) agreed within statistical errors with that obtained from DP. A log-log plot showing $\rho$ against $\langle \bar{p} \rangle - p_c$ for very large $N$ and $t$ is shown in fig.1. Also plotted in this figure is a line with slope $\beta = 0.2765$. We see perfect agreement.

\begin{figure}[h]
\centering
\includegraphics[width=0.5\textwidth]{fig1.png}
\caption{Double logarithmic plot of $\langle \bar{p} \rangle - p_c$ versus $\rho$. The dashed line has the slope $1/\beta$ with $\beta = 0.2765$. Here $N$ ranged from 1024 ($\rho = 1/8$) to 2048 ($\rho = 1/3$). Simulation times $t$ ranged from $10^8$ ($\rho = 1/8$) to $5 \cdot 10^7$ ($\rho = 1/3$), with the first $10^6$ iterations discarded. Statistical errors are less than the sizes of the symbols.}
\end{figure}

In [15], the author found $\langle \bar{p} \rangle - p_c \sim \rho^{1.66}$ for a model which should be in the universality class of DP in 2+1 dimensions. This is in reasonable agreement with the best estimate [12] $\beta^{-1} = 1.71 \pm 0.01$. We should, however, point out that we found for his model $p_c = 0.3178 \pm 0.002$, while he had found $p_c = 0.36$. In view of this, the good agreement for the exponent is a bit surprising.

### 2.3 Critical case

The above argument tells us that the average survival rate tends to $p_c$ when $\rho \to 0$. In this limit fluctuations of $N$ become large in DP, and the very close relationship between DP and the present model breaks down. Nevertheless, we shall argue that there is a non-trivial scaling law with an exponent determined by DP.

Let us start at $t = 0$ with a random, infinite, and infinitely dilute population of plants. Since in the first time step all plants have $2d$ free neighbors, the survival rate for the first
Figure 2: Double logarithmic plot of $p_c - \tilde{p}(t)$ versus $t$. Data are averaged over 100 runs with $N = 2^{14}$ and $L = 100 \cdot 2^{14}$ each. The dashed line has slope $-1/\nu_{||}$ with $\nu_{||} = 1.7338$ [17].

step is $\tilde{p}(1) = 1/2d$. This is smaller than $p_c$ for any dimension. For the next time step it is no longer true that each plant has $2d$ free neighbors. Therefore $M(2)$ will be less than $2dN$, and $\tilde{p}(2) > 1/2d$. During successive time steps the clustering due to the locality of off-spring production will increase, and $\tilde{p}(t)$ will increase with it, until $\tilde{p}$ reaches $p_c$ for $t \to \infty$. We expect thus a scaling law

$$p_c - \tilde{p}(t) \sim t^{-\alpha}$$

with a yet unknown exponent $\alpha$. But $\alpha$ can be related to the exponent $\nu_{||}$ of DP if we assume that there is only one divergent time scale in critical DP, and that this time scale is uniquely related to $p - p_c$ as

$$t \sim |p_c - p|^{-\nu_{||}}.$$  

If we identify here $p$ with $\tilde{p}$ in eq. (6) then obviously

$$\alpha = 1/\nu_{||}. \quad (7)$$

Alternatively, assume we start with the same infinitely diluted initial population, but let it evolve according to DP. Since plants are infinitely far apart, their offsprings will evolve independently. If we would use $p = p_c$, the total population size would grow as $N(t) \sim t^{\eta}$ [1, 2]. In order to prevent this and to keep at least $\langle N(t) \rangle$ fixed, one has to use $t$-dependent values of $p$ which are slightly below $p_c$, $p = p_c - \epsilon(t)$ with $\epsilon(t) \to 0$ for $t \to \infty$. In order to obtain eq. (6), we again have to postulate that the relation between $\epsilon$ and $t$ is the same as between a fixed distance from the critical point and the corresponding correlation time.

Neither of these two arguments is of course rigorous, but simulations shown in fig. 2 give again perfect agreement.
2.4 Subcritical case

The case \( N < \infty, \ L \to \infty \) corresponds to the subcritical case of DP. In this case fluctuations of DP are strongest (indeed, if no precautions are taken, all epidemics die with probability 1), and the connection between DP and the present model is the most delicate. It is indeed in this regime that we found problems that we have not yet fully understood.

For finite \( N \) and \( t \to \infty \), it is easy to see that all plants must have a common ancestor \[^{[16]}\]. Assume that there were \( k \) different lines of ancestry, with \( k > 1 \). Although the rules of the game prevent the total population from going extinct, they do not prevent any of the \( k \) subpopulations from dying. Indeed, since their sizes will fluctuate, any of them has a finite chance to die, and thus \( k \) decreases with a finite rate until \( k = 1 \) is reached.

Notice that the same argument leads also to the fact that plants survive only on one of the even/odd sublattices stated in sec.2.1.

![Figure 3: Typical run with \( N = 10 \). Notice that all clusters die sooner or later except for one which is indicated by heavy dots. The surviving cluster has an internal width \( R \), a characteristic time \( \tau \), and its center of gravity follows essentially the random walk performed by the common ancestor.](image)

Let us consider a finite population at some late time \( t \). If we take any two plants \( i \) and \( j \), they will have a common ancestor \( t_{ij} \) time steps back. The maximum over \( i \) and \( j \) is the time it took the common ancestor to wipe out all its competitors. This is a fluctuating number, its average value \( \tau \) is assumed to scale with \( N \),

\[
\tau = \left\langle \max_{i,j} t_{ij} \right\rangle \sim N^a
\]  \hspace{1cm} (8)

(see fig.3). Thus \( \tau \) is a characteristic scale for the time it took for all other lines of ancestry to die out.

During each time step, a daughter plant cannot move more than one lattice spacing from its parent. Therefore, all plants in the population must form a cluster of finite extent \( R \leq \tau \) \[^{[5], [16]}\]. Again we assume a scaling law

\[
R \sim N^b, \quad b \leq a.
\]  \hspace{1cm} (9)
Unless $b = 1/d$, the cluster is fractal with dimension $d_f = 1/b$.

Notice that $\tau$ and $R$ must become stationary for large $t$. This is not so for the center of mass $X$. Due to translation invariance, the difference $\Delta X_T(t) = X(t + T) - X(t)$ is a stationary random variable for large $t$. Also, we should expect its autocorrelation time to be finite. Therefore, $X(t)$ makes essentially a random walk \[16, 18\].

$$\langle (X(t + T) - X(t))^2 \rangle \sim DT \quad \text{for} \quad T \to \infty .$$ (10)

For $N = 1$ one easily sees that $D = 1$. For large $N$ we again make a scaling ansatz

$$D \sim N^\nu .$$ (11)

Finally, we can look at the average value of $\bar{p}(t)$,

$$\langle \bar{p} \rangle = \lim_{T \to \infty} \frac{1}{T} \sum_{t=1}^{T} \bar{p}(t) .$$ (12)

For $N = 1$ one obviously has $\langle \bar{p} \rangle = 1/2d < p_c$, while $\bar{p} \to p_c$ for $N \to \infty$. It seems thus natural to assume again a scaling law

$$p_c - \langle \bar{p} \rangle \sim N^{-x} .$$ (13)

Let us now try to predict these exponents in terms of DP critical exponents. The closest analogy is with DP in the subcritical regime, but conditioned on those rare clusters which have not yet died out. Let us denote $\epsilon = p_c - p$. The general ansatzes for the average population size, survival probability, and spatial extent of DP clusters (or, rather contact model clusters; notice that in terms of DP proper we are actually dealing with cross sections of clusters at fixed $t$) are \[18\]

$$n(t, \epsilon) \sim \epsilon^{2\beta - d\nu_\perp} \psi(\epsilon^{\nu_\parallel} t) ,$$ (14)

$$P(t, \epsilon) \sim \epsilon^{\beta} \phi(\epsilon^{\nu_\parallel} t) ,$$ (15)

and

$$X(t, \epsilon) \sim \epsilon^{-\nu_\perp} \chi(\epsilon^{\nu_\parallel} t) .$$ (16)

Notice that we denoted the spatial extent by $X$, anticipating that a subcritical DP cluster has finite internal width, so that the total width is dominated by the spread of the center of gravity of the cluster.

For the long-time behavior at $\epsilon > 0$ one needs the behavior of the scaling functions when their argument $\epsilon^{\nu_\parallel} t \to \infty$. Assuming that surviving clusters perform random walks \[16, 18\], we have $X \sim \sqrt{t}$ and therefore

$$\chi(z) \sim \sqrt{z} , \quad z \to \infty .$$ (17)

This gives us immediately

$$D \sim \epsilon^{\nu_\parallel - 2\nu_\perp}$$ (18)

or

$$\frac{c}{x} = 2\nu_\perp - \nu_\parallel .$$ (19)
Since we are interested only in the subcritical DP process conditioned to surviving clusters, we are not interested in $n(t, \epsilon)$ and $P(t, \epsilon)$ themselves, but only the ratio $\bar{N}(t, \epsilon) = n(t, \epsilon)/P(t, \epsilon)$. Since this has to converge to a finite constant for $t \rightarrow \infty$, we see that $\psi(z)/\phi(z) \rightarrow \text{const}$ for $z \rightarrow \infty$, and

$$\bar{N} \sim e^{\beta-d\nu_\perp}, \quad (20)$$

leading to the predictions

$$x = \frac{1}{d\nu_\perp - \beta} \quad \text{and} \quad (21)$$

$$c = \frac{2\nu_\perp - \nu_\parallel}{d\nu_\perp - \beta}; \quad (22)$$

Equations (18) and (20) could indeed have been obtained by simple dimensional arguments. In addition to assuming that there is only one characteristic time scale $\sim \epsilon^{-\nu_\parallel}$, we assume similarly that there is only one length scale $\sim \epsilon^{-\nu_\perp}$ and one characteristic density $\sim \epsilon^\beta$. Then, eq.(18) follows simply by noting that $D$ has dimension $[\text{length}]^2/[\text{time}]$, while eq.(20) follows from $[\bar{N}] = [\text{density}] \times [\text{length}]^d$. Accepting this argument, we can immediately predict also the exponents in eqs.(8) and (9): \begin{equation}
 a = \frac{\nu_\parallel}{d\nu_\perp - \beta}, \quad b = \frac{\nu_\perp}{d\nu_\perp - \beta}. \quad (23)
\end{equation}

Figure 4: Double logarithmic plot of $R = \langle x_{\text{max}} - x_{\text{min}} \rangle$ versus $N$ (crosses). In these simulations, long transients (typically $t = 10^6$) were discarded, and averages were taken over $10^8$ time steps. Statistical errors are smaller than the size of the symbols. The dashed line has slope 1.337 as predicted by eq.(23). We also show data for subcritical DP (rectangles). They not only increase with the same power of $N$, but within error bars they have even the same amplitude.
Figure 5: Double logarithmic plot of the diffusion coefficient \( D \) versus \( N \). The dashed line has slope 0.561 as predicted by eq.(22).

Again we show only numerical tests for \( d = 1 \), although we made also some simulations for \( d = 2 \). In more than one dimension of space, the most natural definition of \( R \) is in terms of the gyration radius (rms. spread) of the population cluster. In \( d = 1 \) an alternative is the end-to-end spread of the cluster, \( R = \langle x_{\text{max}} - x_{\text{min}} \rangle \). We checked that both definitions give compatible results. Results obtained with the latter definition are shown in fig.4. In this figure we also show data for subcritical DP, obtained with an enrichment trick [19]: in order to compensate the exponential decrease of the number of surviving clusters, we made copies of these clusters in regular time intervals. By judiciously adjusting the copying rate, one can keep the number of surviving clusters roughly constant, allowing them to be studied up to very long times. Surprisingly, we found that the DP data not only showed the same scaling as the data of the present model (this was expected), but they have also the same amplitude within the measured error bars. We don’t have any good explanation for this.

Results for the diffusion coefficient \( D \) obtained from the same runs are shown in fig.5. Notice that \( D \) increases with the population size. Naively, one might have guessed that it would decrease. The latter would be true if the wandering of the cluster were a cooperative effect. But as we have seen, it is due to the diffusive motion of a single plant line, namely the common ancestor of the entire cluster. This line of ancestry is singled out by having had better chances of survival than its competitors. Without local saturation (i.e. the fact that seeds falling onto sites which contain already a seed are lost), each line would have had the same chance of survival, and \( D \) would be independent of \( N \) [16]. With local saturation it is clear that a plant in a less dense region has a larger chance to have long lasting offspring. Thus survivors will generally be seeds which fall further from the center of the cluster, and which lead thus to faster diffusion.

The fact that \( D \) increases with \( N \) holds also for \( d > 1 \). It seems at odd with the observation that “clusters of weeds tend to remain on the same spot for a long time” of
but this shows just that diffusion is a slow process and can be easily missed if the
diffusing object itself is fuzzy and changing its shape.

Figure 6: Double logarithmic plot of $\langle \tau \rangle$ versus $N$. The dashed line has slope 2.114 as predicted
by eq.(23). Statistical errors are again smaller than the size of the symbols.

Instead of showing data for $\tau$ defined exactly as above, we show in fig.6 data for
a slightly different average. Instead of averaging over all times $t$, the data shown in
fig.6 were obtained as follows. To each plant we attach a label which is inherited to
all successors. We start with different labels for all plants and iterate until the entire
population carries the same label. At this moment we measure $\tau$, relabel the plants so
that each has again a different label, and continue with the evolution. Thus the results
shown in fig.6 present averages taken only at those times when all plants have for the
first time a common ancestor. While this should affect the prefactor in eq.(8), it should
not affect the exponent. We see perfect agreement with the prediction.

On the other hand, it seems that eq.(21) is wrong, at least if we identify $\epsilon$ with the
difference $p_c - \langle \tilde{p} \rangle$. Simulations show a clear power law dependence of $p_c - \langle \tilde{p} \rangle$ on $N$
(see fig.7), but the exponent seriously disagrees with the prediction. Numerically we
found $x = 0.94 \pm 0.02$, while eq.(21) would give $x = 1.2191$. This suggests that simple
linear averaging is not the right procedure to obtain the characteristic deviation of $\tilde{p}(t)$
from $p_c$ [21]. We checked that taking geometric or harmonic averages of $\tilde{p}(t)$ did not
improve the situation, since they gave essentially the same results. Rather, it seems that
one should take some non-linear average of the difference $p_c - \tilde{p}(t)$. A priori, the most
natural candidate might seem the geometric average of $p_c - \tilde{p}(t)$, but this is ruled out by
the fact that $p_c - \tilde{p}(t)$ is not positive definite. Distributions of $p_c - \tilde{p}(t)$ are plotted in
fig.8 for several values of $N$. We see that the average value is positive, but as $N$ increases
the width of the distribution becomes much larger than this difference. In this limit, the
distribution approaches a Gaussian whose width scales roughly as $1/\sqrt{N}$.
2.5 Offspring Statistics, Family Survival, and Mitochondrial Eve

Let us consider again the critical case, and let us label at some given time \( t_0 \) all plants with different labels. We can then study the evolution of the labeling pattern. As some branches of the population die, this pattern will coarsen increasingly. We are in particular interested in the limit where the coarsening is followed during a time \( T \) which is \( \gg 1 \) but much smaller than \( t_0 \). In this limit the system can be considered as stationary during \( t_0 < t < t_0 + T \), although it of course never becomes strictly stationary at the critical point.

This is essentially the famous Galton-Watson problem of survival of family names \(^{[21]}\), but in a marginally surviving total population with local competition and local offspring production. This problem was studied in \(^{[15]}\), and it was mainly because of the results obtained thereby that this author claimed that his model is in the DP universality class.

Other phenomena and models related to this problem are the voter model \(^{[22]}\), and the mitochondrial Eve problem \(^{[23, 24]}\). The voter model is essentially the extremely supercritical version where each lattice site is occupied by a plant, and sites receive in the next generation a seed from a randomly chosen neighbor. Its name derives from a caricature of political opinion formation where a citizen adopts at each time step the former opinion of one of his \( 2d \) neighbors. Its upper critical dimension is \( d = 2 \). For \( d > 2 \), the number of different opinions in a large population with no two individuals sharing originally the same opinion decrease as \( N_{\text{opinion}} \sim 1/t \). This is the same as in the Galton-Watson problem which is essentially the mean field (or random neighbor) version of this model. For \( d = 2 \) one has logarithmic corrections, \( N_{\text{opinion}} \sim t^{-1} \ln t \), and for \( d = 1 \) one has \( N_{\text{opinion}} \sim 1/\sqrt{t} \).

The Eve problem arises from the fact that mitochondrial DNA (mtDNA) is inherited

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Figure 7: Double logarithmic plot of \( p_c - \langle \tilde{p} \rangle \) versus \( N \). The dashed line indicates the exponent predicted by eq.(21). Obviously it does not fit the data.
Figure 8: Plots of the distribution of $\tilde{p}(t)$ versus $(p_c - \tilde{p}(t))\sqrt{N}$, for $N = 30$ (⋆), $N = 60$ (+), and $N = 120$ (×). The lines are spline fits to the data points. Normalization is arbitrary.

only from mother to child, without any contribution from the father. Thus different strands of mtDNA act like different labels in our model. Variations present humans suggest that all humans have a surprisingly recent common female ancestor, called mitochondrial Eve, who lived ca. 200,000 years (i.e. 100,000 generations) ago in southern Africa. The problem is whether this is simply explained as a statistical effect (for any single trait, one expects that only one line of ancestry survives finally), or whether this points to a common origin of the human race. This depends on whether the characteristic time $\tau$ discussed in the last subsection - which can be defined also for the voter and Galton - Watson processes - is smaller than 10,000 generations or not. We should mention that similar arguments apply also to the Y chromosome which is inherited only from the father [25].

If the human populations would have been well mixed and of stationary size during most of the last 100,000 generations, we could use the Galton - Watson process to argue that a common root at about this time was to be expected if the population size was $N < 100,000$. In this case it would be a mere coincidence that this root is in Africa, and ancestry lines involving males would most likely point to other origins. If, however, the population size was larger, then this finding is non-trivial and could be only explained by important migrations. It would then point to a relatively recent African origin of all humans. If we base our estimate on the voter model, on the other hand, we would estimate $N \sim 1100$ as critical population size, and the findings of [23, 24] would seem even more significant.

Let us now come back to the present plant model. The mitochondrial Eve problem deals indeed with a finite population, treated in the last subsection. But one can also study subpopulations with identical traits (e.g. identical mtDNA) in an infinite pop-
ulation, and that is what we shall do in the following. Since we are interested in this problem exactly at the critical point, the following discussion applies in exactly the same way to DP.

We denote by $P(t)$ the survival probability of a family, and $N(t)$ the average size of surviving families. Dealing with a critical phenomenon, we expect of course that both should follow power laws. Due to stationarity, the average number of plants carrying the same label (i.e., the number of offspring in surviving families) must increase such that this decrease of the survival probability is exactly compensated, $N(t)P(t) = \text{const}$. Finally, we expect also that the size $R(t)$ occupied by a family should scale.

On the other hand, our previous argument that there are unique length, time, and density scales suggests that

$$R(t) \sim t^{\nu_\perp/\nu_\parallel}$$  \hspace{1cm} (24)$$

and

$$N(t) \sim t^{(d\nu_\perp - \beta)/\nu_\parallel}$$  \hspace{1cm} (25)$$

Thus the spatial growth of a family cluster in a critical population should scale in the same way as an isolated population, and $N(t)$ should grow as for an isolated population conditioned on survival. This is not trivial; the analogous is not true, e.g., for the survival probability $P$. While an isolated 1-d population dies with an exponent $\delta = -\beta/\nu_\parallel = 0.16$, a family has to compete with other families and dies with the same exponent as $1/N(t)$.

![Figure 9: Log-log plot of $R(t)$ versus $t$, where $R(t)$ is the average end-to-end size of families in a stationary critical population. The dashed line has the slope 0.6326 predicted by eq.(24). Error bars are again smaller than symbol sizes.](image)

To test these predictions, we show numerical results (again for $d = 1$) in figs. 9 and 10. These data were obtained with $N = 8192$ and $L = 2^{23}$. After discarding a transient with $t_0 = 5 \times 10^4$, plants were labeled and followed over 8192 time steps. After that...
each plant was given again a different label, and the population was followed for another 8192 time steps, etc., for a total of $3 \times 10^5$ time steps. The data shown in figs. 9 and 10 are in very good agreement with the prediction. Small discrepancies can be explained as finite size effects. Notice that these data seem at first sight to be inconsistent. For $d = 1$ one can easily see that families must occupy non-interleaved territory. If two plants at sites $x_1$ and $x_2$ carry the same label, all plants with $x \in [x_1, x_2]$ must also carry the same label. Thus, $R(t)$ must be smaller than the average distance between families. The latter grows as $1/P(t) \sim t^{-0.4731}$, while $R(t)$ grows faster, $R(t) \sim t^{0.633}$. The answer to this seeming contradiction is that DP clusters are fractals. They contain holes of all sizes, and thus $R(t)P(t) \ll 1$ for small $t$.

Finally we want to mention that $(d\nu_\perp - \beta)/\nu_\parallel = 0.68$ for $d = 2^{[12]}$. Thus, if we assume the present model for the human population during the last 200,000 years, the critical population size for the observed mitochondrial Eve phenomenon would be $N \approx 500$. This estimate is of course highly uncertain, but it indicates again that an estimate based on the Galton-Watson process is most likely too high.

3 Discussion

We studied a population model which can be understood as a variant of the simple epidemic model without immunization. The latter is in the same universality class as DP in $d + 1$ dimension. Numerically, we studied in most detail that version in one dimension of space which corresponds to directed site percolation on the square lattice. Although slightly different in details, this model was first introduced as a model for weeds.

![Log-log plot of $P(t)$ versus $t$, where $P(t)$ is the survival probability of a family. The dashed line has the slope $-0.4731$ predicted by eq. (23). The average family size increases as $1/P(t)$. Error bars are again smaller than symbol sizes.](image)
The crucial point is that it is not the fertility of the weeds which is controlled, but control is exercised directly on the population size.

In contrast to DP one does not, therefore, control the ‘wetting’ probability. Instead, the global population size (i.e., the integral over the order parameter) is kept fixed, and the usual control parameter is allowed to fluctuate. This is analogous to controlling a thermal phase transition not by means of the temperature but by means of the energy. While such an exchange of control makes usually no big difference in equilibrium critical phenomena, it leads to SOC in the present case. Even without fine tuning any parameter, critical behavior is found in the limit of large populations and low densities.

Although our model is not governed by extremal dynamics, it shares with such models (as, e.g., invasion percolation) the fact that the threshold of the standard (non-self-organized) version is recovered in the critical limit.

The same features are also shared with a recent model studied by Tadic and Dhar [26]. In both models the system is driven into the critical state of DP without any fine tuning. In both, the dynamics is not extremal. Finally, in both models some scaling laws and critical exponents are identical to those of DP, while others are only related to DP. But the detailed mechanisms which drive the system into the critical state are completely different in both models.

A more direct relation exists to a model studied by Zhang et al. [16]. The main difference between that model and ours is that these authors have assumed no local saturation. Any seed has the same chance to grow, whether it is far from other seeds or whether it fell into a densely crowded region. Thus population growth is only controlled globally in the model of Zhang et al., while there is a local aspect in our model by the requirement that each lattice site is occupied by at most one plant. Thus the model of [16] can be considered as a mean field version of ours where the average offspring number is the same for all plants, independent of their location with respect to the bulk of the population. It is easily seen that all scaling laws discussed in the present paper are satisfied in the model of Zhang et al., but with ‘mean field’ exponents $\nu_\parallel = 1$, $\nu_\perp = 1/2$, and $d\nu_\perp - \beta = 1$ [27]. Thus, the diffusion of the cluster is independent of its size, and $R \sim \sqrt{N}$, $\tau \sim N$. On the other hand, $p$ is independent of $N$ in the model of [16]. This shows again that the relation between $p_c - \langle \tilde{p} \rangle$ and $N$ is not as robust as the other scaling relations.

We verified most of the results of [15], but formulated and tested the scaling laws in much more details. The main true discrepancy is the fact that the diffusion of the population cluster in the case of finite populations was missed completely in [15]. On the other hand we verified the non-trivial claim of [15] that the growth of surviving labeled clusters inside a large critical population obeys exactly the same scaling laws as the growth of surviving isolated clusters. In 2 dimensions of space this means that specific traits die out in a marginally surviving population with probability $\sim 1/t^{0.68}$. This is to be compared to $\sim 1/t$ for the Galton-Watson process, and to $\log(t)/t$ for the voter model [22], i.e. for the case of a supercritical DP.

Studies of human mitochondrial DNA indicate a common female African ancestor some 200,000 years ago [23, 24]. The last result suggests that this might be more significant in favor of a recent African origin than one might have argued on the basis of the Galton-Watson process.
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