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LETTER TO THE EDITOR

Dynamical phase transitions in cluster growth processes where growth sites have a finite lifetime

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Abstract. We discuss variants of the Eden model for cluster growth, where growth sites have a finite lifetime τ . Time is increased by 1/G when a growth site is transformed into a cluster site; G is the total number of growth sites present at that time. We find by Monte Carlo simulation that the growth process depends drastically on τ . Below a critical lifetime τ_c ($\tau_c \simeq 0.80$ on the square lattice) the clusters are described by the fractal dimension of self-avoiding random walks, $d_f = \frac{4}{3}$, and the number of cluster sites s increases proportional to time t. Above τ_c , we find Eden clusters with $d_f = 2$ and $s \simeq t^2$. Finally, we extend our results to inhomogeneous media (percolation systems) where a fraction (1-p) of sites is not accessible to the growth process, and discuss the phase diagram.

In the past few years, considerable attention has focused on cluster growth models. Cluster growth models have been used to describe a wide variety of spreading phenomena, ranging from the growth of epidemics and forest fires to signal propagation and the formation of diffusion fronts [1-15]; for reviews see [16, 17]. The final structure of the growing cluster can be described by the fractal dimension d_r , which gives the cluster mass s within a distance r from the seed,

 $s \sim r^{d_{\rm f}}.\tag{1}$

The growth sites are defined as that part of the cluster surface where the cluster can grow. The dynamics of the growth process can be characterised by the fractal dimension d_g , which relates G, the mass of the growth sites inside radius r, to r

$$G \sim r^{d_g}.$$
 (2)

In (1) and (2), r is less than the radius of gyration. The dynamic exponent d_g and the static exponent d_f are independent of each other. If, for example, the next growth site to be tested is taken from a probability distribution $P(r) \sim r^{-\beta}$ where r is the distance of the growth site from the most recently added cluster site, then the growth exponent d_g can change continuously with β [10].

§ Present and permanent address: Institut für Theoretische Physik, Universität Hamburg, D-2000 Hamburg 36, Federal Republic of Germany. The growth exponent d_g determines the timescale of the growth process. To describe, e.g., the growth of tumours, epidemics or forest fires it is not realistic to assume that at each timestep one cluster site is added. Rather the timescale is determined by the total number of growth sites G, present at time t. In one timestep all growth sites are occupied in the average. The more growth sites that are present, the faster is the growth process.

In previous works, Leath's cluster growth method or variants of it have been used to describe epidemics and forest fires (see, e.g., [6, 12]). In this method [3, 4] the cluster grows by adding layers of constant path length ('chemical' distance [18]) to the seed, one layer in one timestep. If there are no immune sites in the system where the cluster cannot grow, regular objects are grown which have the structure of the underlying lattice.

Perhaps the simplest growth model for irregular objects is that due to Eden [1] in which empty neighbours of cluster sites, the growth sites, are occupied in random fashion. To describe the appropriate timescale associated with this process we assume that time is enhanced by 1/G,

$$\Delta t = 1/G(t) \tag{3}$$

when a growth site is occupied. G(t) is the total number of growth sites present at time t. Combining (1) and (2) we have $G(t) \sim M(t)^{d_g/d_t}$, where M(t) is the cluster mass (total number of sites) at time t. Hence we obtain from (3)

$$M(t) \sim t^{d_{\rm f}/(d_{\rm f}-d_{\rm g})} \equiv t^{\alpha_{\rm M}} \tag{4}$$

and, accordingly,

$$R(t) \sim t^{1/(d_f - d_g)} \equiv t^{\alpha_R} \tag{5}$$

and

$$G(t) \sim t^{d_{\mathfrak{g}}/(d_{\mathfrak{f}}-d_{\mathfrak{g}})} \equiv t^{\alpha_G} \tag{6}$$

where R(t) denotes the radius of gyration at time t, $M(t) \sim R(t)^{d_t}$.

If this kind of growth process is interpreted as the spreading of an epidemic or a forest fire, then the exponent $\alpha_M = d_f/(d_f - d_g)$ measures the increase of infected individuals or burned trees, while the exponent $\alpha_R = 1/(d_f - d_g)$ measures the increase of affected area.

For the Eden model one has $d_f = 2$ and $d_g = 1$ and thus $M \sim t^2$, $R \sim G \sim t$. If we allow for immune sites with concentration 1 - p, then the exponents change at the critical concentration p_c of the underlying lattice: $d_f = \frac{91}{48}$ and $d_g \simeq \frac{3}{4}$ [10]. Accordingly we have $\alpha_M = \frac{91}{55}$, $\alpha_R = \frac{55}{48}$ and $\alpha_G = \frac{165}{192}$. Compared with the case p = 1 (no immune sites) the size of the infected area increases more strongly in this case, while the number of infected individuals increases slower with time. But note that these exponents are not universal, but can depend on the way in which the growth sites to be occupied are chosen [10].

In the Eden model, an infected site can infect its as yet uninfected neighbours for an arbitrary long time. This assumption is not always satisfied. For example, in a forest fire a tree can burn out before setting its neighbouring trees on fire, or in an epidemic an infected individual can die before infecting the neighbouring individuals. In order to take account of these facts we will consider a finite lifetime τ of growth sites. A growth site becomes inactive τ timesteps after it has been generated. This means a 'sick' site can infect as yet 'healthy' sites only for a certain time τ , i.e. if a neighbour of a site A that was infected at time t_1 has not been infected by A up to time $t_1 + \tau$, it cannot be infected after that time by A anymore. This concept of a finite lifetime of growth sites has been introduced by Bunde *et al* [19] and elaborated further by Miyazima *et al* [20]. However, in these works time was counted differently: time was increased by one unit when the cluster size was increased by one site. It was found that, under this condition, d_f and d_g changed considerably, $d_f = \frac{4}{3}$, $d_g = 0$, for all values of τ and $p \ge p_c$.

In contrast, for the type of spreading phenomena considered here, the exponents depend drastically on τ and p. We find that below a critical lifetime $\tau_c(p)$ the clusters are described by the fractal dimension of self-avoiding random walks, $d_f = \frac{4}{3}$, $d_g = 0$, while above $\tau_c(p)$ we find Eden clusters with $d_f = 2$, $d_g = 1$ $(p > p_c)$ or percolation clusters with $d_f = \frac{91}{48}$, $d_g = \frac{3}{4}$ $(p = p_c)$.

Figure 1 shows typical clusters for p = 1 and $p = p_c$ for two values of lifetime τ in each case. The clusters (a) for p = 1, $\tau = 0.6$ and (c) for $p = p_c$, $\tau = 1.5$ look more like thick chains, while the cluster (b) for p = 1, $\tau = 1.5$ looks like a compact Eden cluster; the cluster (d) for $p = p_c$, $\tau = 5$ is similar to large percolation clusters. To find the fractal dimension d_f we have studied, by Monte Carlo simulations, as a function of the number of cluster sites, the mean square of the end-to-end distance $\langle r^2 \rangle$ between the last added site and the seed at the origin of the square lattice. $\langle r^2 \rangle$ scales as

$$\langle r^2 \rangle \sim s^{2/d_{\rm f}}$$

and therefore by measuring $\langle r^2 \rangle$ as a function of cluster sites s we can determine d_r .

First let us consider the case of p = 1, when there are no immune sites in the lattice. Figure 2(*a*) shows $\langle r^2 \rangle$ as a function of *s* for $\tau = 0.6$, 0.8 and 1. For $\tau = 0.6$ the slope accepts the value 1.5, yielding $d_f = \frac{4}{3}$, while for $\tau = 0.8$ and 1 the slope is 1, yielding



Figure 1. Typical clusters for p = 1 (no immune sites) and $p = p_c$ for two values of lifetime τ of growth sites: (a) p = 1, $\tau = 0.6$; (b) p = 1, $\tau = 1.5$; (c) $p = p_c$, $\tau = 1.5$; (d) $p = p_c$, $\tau = 5$.



Figure 2. Plots of (a) the mean-square end-to-end distance $\langle r^2 \rangle$ and (b) the number of growth sites G against the number of cluster sites s at p = 1 for various values of τ . (c) shows the number of cluster sites s plotted against time t at p = 1 for various τ . (d) shows the probability N(s)/N(1) that s cluster sites can be generated plotted against s at p = 1 for various τ . For most data points, averages over more than 2000 clusters have been made. The asymptotic slopes are denoted by the straight lines.

the fractal dimension of Eden clusters, $d_r = 2$, in two-dimensional systems. Figure 2(b) shows the number of growth sites G as a function of s. Again we observe a drastic change in the asymptotic behaviour between $\tau = 0.6$ and $\tau = 0.8$. For $\tau \ge 0.8$, the asymptotic slopes are equal to 1 (straight lines) as expected for Eden clusters, while for $\tau < 0.8$ the number of growth sites tends to a constant and $d_g = 0$, as expected for clusters belonging to the universality class of self-avoiding random walks [19]. In order to find the critical lifetime τ_c , we have performed extensive computer simulations of clusters up to 20 000 sites. We have found $\tau_c = 0.80 \pm 0.02$ for the square lattice. We expect that τ_c depends on details of the underlying lattice just as the critical concentration does, while the exponents d_f and d_g should not depend on minor structural details.

For $\tau < \tau_c$ clusters belonging to the universality class of self-avoiding random walks are generated, while for $\tau > \tau_c$ Eden clusters are formed. Accordingly, the number of infected sites should scale differently below τ_c and above τ_c . Below τ_c , where $d_g = 0$, we expect $s \sim t$, while above τ_c we expect $s \sim t^2$. Figure 2(c) confirms this expectation. Finally, let us consider the probability that large clusters of size s can be generated. This 'survival' probability is given by the number of successfully generated clusters of s sites divided by the number of trials, N(1). For $\tau \ge 0.8$ practically all clusters survive, while for $\tau < 0.8$ we observe an exponential decay (figure 2(d))

$$N(s) \sim \exp(-s/s_0)$$

where s_0 tends to infinity when τ_c is approached. s_0 is the mean size of a generated cluster. Since $s \sim t$ for $\tau < \tau_c$, $s_0 \sim t_0$ has the meaning of a mean lifetime for the epidemic process. For $t \simeq t_0(\tau)$ there is a high probability that the epidemic stops or the forest fire is exhausted. For $\tau > \tau_c$ the growth process can continue without limitation.

Next we allow for immune sites, considering the case of $p = p_c$ (at criticality). We assume that a fraction (1-p) of sites are immune sites, where the epidemic cannot spread. Below the critical concentration p_c of the lattice only finite clusters of infectable sites exist. At p_c there exists an infinite cluster of infectable sites. For $p \ge p_c$ infinitely extended clusters can be formed. Figures 3(a, b) show, for $p = p_c$, the mean-square end-to-end distance $\langle r^2 \rangle$ and the number of growth sites G as a function of s for τ between 0.75 and 4. For $\tau = 4$, the slope of $\langle r^2 \rangle$ is 1.05, in agreement with the fractal dimension $d_f = \frac{91}{48}$ of percolation clusters for d = 2. Accordingly, the slope of G(s) is about 0.40, in agreement with the prediction $d_g = \frac{3}{4}$ for Eden cluster growth in percolation [10]. For $\tau \le 2$, the asymptotic slopes of $\langle r^2 \rangle$ are 1.5, corresponding to $d_f = \frac{4}{3}$. Accordingly, G(s) approaches a constant for large s values. From this behaviour we expect $s \sim t^{91/55}$ for $\tau = 4$ and $s \sim t$ for $\tau \le 2$, which we confirmed numerically. We have used computer simulations to calculate the critical lifetime for $p = p_c$. We have found $\tau_c(p_c) = 3.75 \pm 0.25$ for the square lattice.



Figure 3. Plots of (a) $\langle r^2 \rangle$ and (b) G against s for $p = p_c$ and several values of τ .

The phase diagram for the whole $\tau - p$ plane is shown in figure 4. Close to p = 1, $\tau_c(p)$ seems to be linear in (1-p), while close to p_c the slope of $\tau_c(p)$ seems to approach infinity. For $p < p_c$ only finite clusters can grow and epidemics or forest fires cannot spread.

In summary, then, we have discussed a cluster growth model with finite lifetime of growth sites which can serve as a model for forest fires and epidemics. We have found dynamical phase transitions at critical lifetimes $\tau_c(p)$ which depend on the fraction (1-p) of immune sites in the system. For $p \ge p_c$ we found clusters with fractal



Figure 4. Phase diagram of our growth model, where growth sites have a finite lifetime τ , on a square lattice. There is a critical line separating a region where one finds Eden clusters and a region where the clusters belong to the universality class of self-avoiding random walks (SAW). For $p < p_c$ only finite clusters can be generated. At the critical concentration p_c , percolation clusters are generated.

dimensions $d_{\rm f} = \frac{4}{3}$ for $\tau < \tau_{\rm c}(p)$, while for $\tau > \tau_{\rm c}(p)$ Eden clusters were formed. To describe the kinetic process we introduced the fractal dimension of the growth sites d_g , which determines the timescale of the growth process. For $\tau < \tau_{\rm c}$ we found $d_g = 0$, while for $\tau > \tau_{\rm c}$ we obtained $d_g = \frac{3}{4}$ ($p = p_{\rm c}$) and $d_g = 1$ ($p > p_{\rm c}$). Our computer simulations were performed on the square lattice. For finite lifetime of growth sites, we expect dynamical phase transitions between clusters having the universality class of self-avoiding walks and Eden clusters also in higher dimensions.

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