# Universality Classes for Spreading Phenomena: A New Model with Fixed Static but Continuously Tunable Kinetic Exponents

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A new and quite tractable model for spreading phenomena is proposed, which contains as a special case the Eden model and a model for epidemics. Two exponents are defined, one static and one kinetic ("growth"). The surprising feature is that the kinetic exponent can be continuously tuned while the static one does not change. *Thus the dynamic universality classes are quite independent of the static one.* This is the first one-cluster growth model showing dynamic universality classes unrelated to static ones and thereby yields insight into a generic feature for growth models.

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How does a disease (fluid, etc.) "spread" through a randomly heterogeneous material? This simple question is of relevance to a wide range of disciplines, ranging from epidemiology, oncology, and cardiovascular physiology on the one hand to signal propagation and network mechanics on the other. In this work, we propose a model for such "spreading phenomena" that is simple but has a rich range of behavior. Using Monte Carlo simulations, we find nontrivial values of both static *and kinetic* exponents. Moreover, we find that the dynamics universality classes are independent from the static universality classes. This property seems to be generic for a wide class of growth models.

For specificity, we consider a square lattice. Each site can be empty or can be occupied by three types of particles that we shall call sick ($S$), immune ($I$), and growth ($G$). At time $t=1$, place an $S$ particle at the origin and occupy the four nearest-neighbor (nn) sites with $G$ particles.[1] At time $t=2$ any of the $G$ particles is chosen randomly and converted into an $S$ particle with probability $p$ or an $I$ particle with probability $1-p$. If an $S$ particle was created again all of its not-yet-determined nearest neighbors become $G$ particles and the time is increased by one. The infection spreads (i.e., the cluster grows) by the successive conversion of a fraction $p$ of the $G$ particles into $S$ particles, and after $t$ time steps a large ramified tumor (cluster) has been formed. The static properties of this cluster are in the same universality class as percolation, since the process of *randomly* making a site into an $S$ or $I$ particle with weights $p$ or $1-p$ is the same as for percolation. The kinetic or "growth" properties need not be the same, since the statics is a function of only the final product (a large ramified cluster), while the kinetics is a function of *how* the final product is reached (the spatial sequence in which the cluster sites are added).

The Leath[2] method is the most widely used and accepted method of cluster growth; in this classic approach, one grows a cluster by first considering the first chemical shell[3]—the set of all sites that may be connected to the origin by a single ("chemical") bond. One randomly converts a fraction $p$ of these to cluster sites ($S$ particles) and blocks the rest ($I$ particles). Then one proceeds to the next chemical shell and repeats the same process. At any given instant of time, all sites under consideration are on the same chemical shell.

The purpose of this Letter is to propose an altogether different growth kinetics—not because it is more efficient than the Leath method in arriving at the final product, a percolation cluster, but because the kinetics depends continuously upon a parameter so that the actual cluster growth process can be tuned.[4] In contrast to the classic "shell-by-shell" growth kinetics of Leath, we choose the next growth site to be tested from a probability distribution $P(r)$, where $r$ is the distance from the most recently added sick particle and[5]

$$P(r) \sim 1/r^{\alpha}. \tag{1}$$

Pictorially speaking, we can imagine the infection spreading by an infected butterfly who flies from one $G$ site to the next, randomly choosing the next from the distribution (1).[6] Representative clusters for $\alpha=0$, $\alpha=-4$ (long range), and $\alpha=+8$ (short range) are shown in Fig. 1. For the short-range $P(r)$, the next sick particle tends to be close by while in the long-range case it tends to be far away. Accordingly, for $\alpha$ negative the $G$ sites are largely localized on the external surface, while for $\alpha$ positive they occur on the internal surface as well.

How can one *quantitatively* describe this large tumor? The static (geometric) properties are reflected in the fractal dimension, $d_f$, which governs how the tumor mass increases with its radius of gyration,

$$M = s \sim R^{d_f}. \tag{2a}$$

For $p=1$ (no immune particles), we expect that $d_f=d$ since our model reduces to the much-studied Eden model. For $p \lesssim 1$, we also expect that $d_f=d$ since a nonzero fraction of our realizations will continue to

 

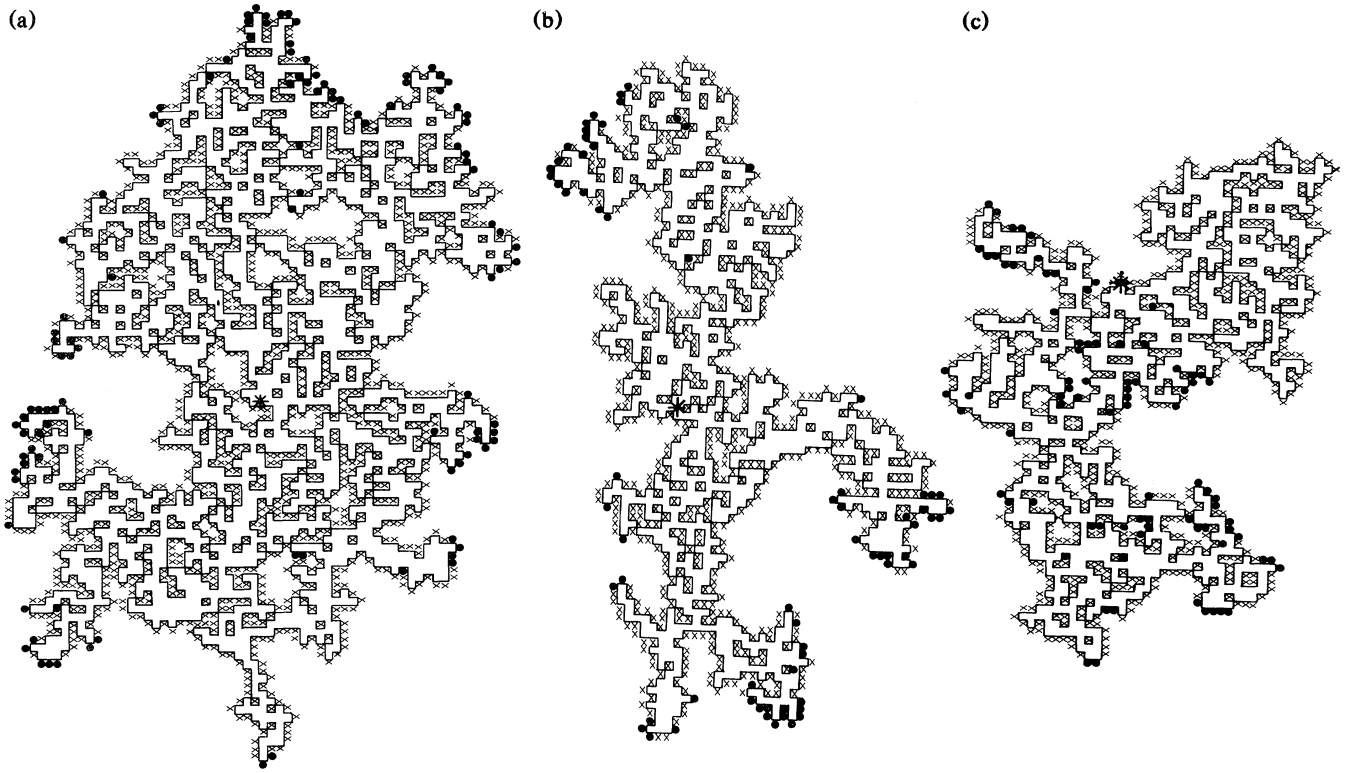


FIG. 1. Typical tumors, each of roughly 1500 sites, for (a) $\alpha = 0$, (b) $\alpha = -4$ (long range), and (c) $\alpha = +8$ (short range) at $p = p_c$. The cross denotes an immune particle (blocked site), while the dot denotes a growth particle (neighbor of the growing tumor that can be infected). The seed of the clusters is denoted by an asterisk.

grow forever. Indeed, the ultimate connectivity of the $S$ particles is *identical to the connectivity of occupied sites in percolation,* since both are randomly present with probability $p$. For the same reason, at $p_c$ we expect $d_f$ to assume the value $\frac{91}{48}$ corresponding to percolation clusters.[7] This reason, to be precise, is that after a very long time the cluster perimeter has a vanishingly small fraction of growth sites—since, as we shall shortly see, the fractal dimension of the growth sites is distinctly smaller than the fractal dimension of the perimeter. In the regions of the cluster where there are no growth sites left [such regions are already apparent in the relatively small 1500-site clusters of Figs. 1(a)–1(c)] the clusters must have precisely the statistics—including fractal dimension—of percolation clusters since each of the sites has been occupied or been blocked once and only once, *independently* of the other sites (since the process is random by definition). Henceforth $p = p_c$ unless otherwise stated.

In addition to the *static* properties of this growth process, we wish to describe quantitatively the *kinetic* properties. To this end, we note that the $G$ sites themselves form a "volatile fractal,"[8] whose identity changes with time. Its fractal dimension is given by $d_G$, where

$$G \sim R^{d_G} \sim s^{d_G/d_f}. \tag{2b}$$

For $\alpha \leq 0$ we find that $d_G/d_f = 0.40 \pm 0.015$ for $p = p_c$, 20% smaller than the value $d_G/d_f = 0.493$ recently found for percolation clusters grown by the ant mechanism.[9] Accordingly, the growth mechanics of what are ultimately percolation clusters depends sensitively on the rules by which the clusters are grown.

The variation of $d_G/d_f$ with $\alpha$ is shown in Fig. 2. The result for the long-range limit might be interpreted by the following simple argument.[10] For $\alpha$ negative the $G$ particles form a subset of the hull of the fractal; by definition, the hull is the set of fractal sites that form the entire external perimeter [see Figs. 1(a) and 1(b)]. If the $G$ sites were randomly chosen from the hull, then $d_G = d_H$, where $d_H$ is the fractal dimension of the hull. In fact, for long-range $P(r)$, the $G$ particles are biased to be at a maximum distance, so that they generally lie at the extremities of the hull. Hence we expect that the $G$ particles more nearly form a fractal cut of the hull, and $d_G = d_H - 1$. Using $d_f = \frac{91}{48}$ and the recent result[11] $d_H = \frac{7}{4}$, we then have

$$d_G/d_f = (d_H - 1)/d_f = \tfrac{36}{91} \cong 0.396. \tag{3}$$

This argument obviously breaks down for $\alpha$ above some limiting value $\alpha_c$, i.e., for sufficiently short-range $P(r)$. To estimate $\alpha_c$, consider the mean length of one jump, $\bar{r} = \int dr\, rP(r)$. Only if $\bar{r} \to \infty$ can we

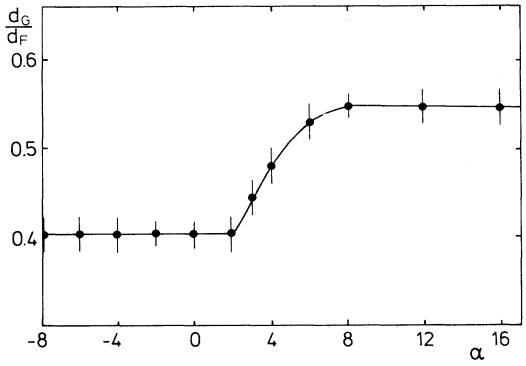


FIG. 2. Dependence on the parameter $\alpha$ of $d_G/d_f$. The data for $\alpha=0$, $\alpha=-4$, and $\alpha=+8$ are based on 20 000, 8000, and 8000 Monte Carlo runs, respectively, while for the other $\alpha$ values 2000 runs have been made. In all cases, clusters have been grown up to $4\times10^3$ $S$ particles.

jump to the extremities of a huge cluster, which implies that $\alpha_c \simeq 2$. From Fig. 2 it appears that $\alpha_c = 2$ is indeed the limiting value of $\alpha$ above which the long-range result $d_G/d_f \simeq 0.4$ fails. This argument may be more general than for the $\alpha=0$ case of our model, since its prediction also holds for a fractal set of frontier sites of the Eden model generated on a percolation-cluster substrate.[12]

At the present time we have no argument to support our finding that $d_G/d_f$ approaches a constant value $0.55\pm0.02$ for the limit of short-range interactions. It is an intriguing result, however, since it implies that $d_G$ approaches unity in the limit of large $\alpha$. Note that our error bars exclude the possibility that our short-range value is the same as for another "short-range" model, the de Gennes "ant in a labyrinth" problem (for which $d_G/d_f=0.49$). *Thus also in the short-range limit the growth process is in a different kinetic universality class than the diffusive growth process.*

We found the probability distribution $N(r,s)$ giving

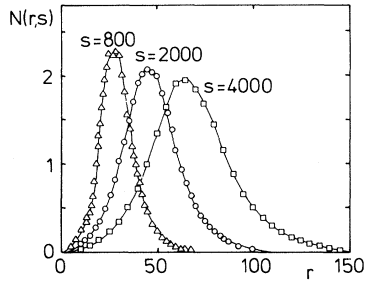


FIG. 3. The number $N(r,s)$ of $G$ particles at a distance $r$ from the seed of a growing $s$-site tumor, for $s=800$, 2000, and 4000; $\alpha=0$.

the number of $G$ particles at a distance $r$ from the seed of the tumor. A representative example ($\alpha=0$) is shown in Fig. 3. We found that the mean $\langle r\rangle$ and also the width $\Delta$ (variance) scale with tumor mass with the same power. This finding is in agreement with the case of the diffusive growth process, in contrast to the case of diffusion-limited aggregation (DLA) where the width of the distribution of "growing sites" increases less quickly than the mean.[13] We also confirmed that $N(r,s)$ obeys scaling in the two active parameters $r$ and $s$: $N(r,s)=Gs^{-1/d_f}N_\alpha(rs^{-1/d_f})$ (Fig. 4). For different values of $\alpha$, $N(r,s)$ cannot be made to collapse. The scaling function $N_\alpha(x)$ depends strongly on $\alpha$ and cannot generally be described by a simple Gaussian, as can be seen by comparison of the location of the maxima of $N_\alpha(x)$ with $\langle r\rangle s^{-1/d_f}$, denoted by an arrow in Fig. 4. Moreover, we find for $\alpha>4$ that growth particles are deposited near the starting point of the walk, which is nonzero even at large times. This is in accord with our understanding that, in the short-range limit, the butterfly prefers to explore new territories, leaving behind untouched growth particles buried deep inside the cluster.

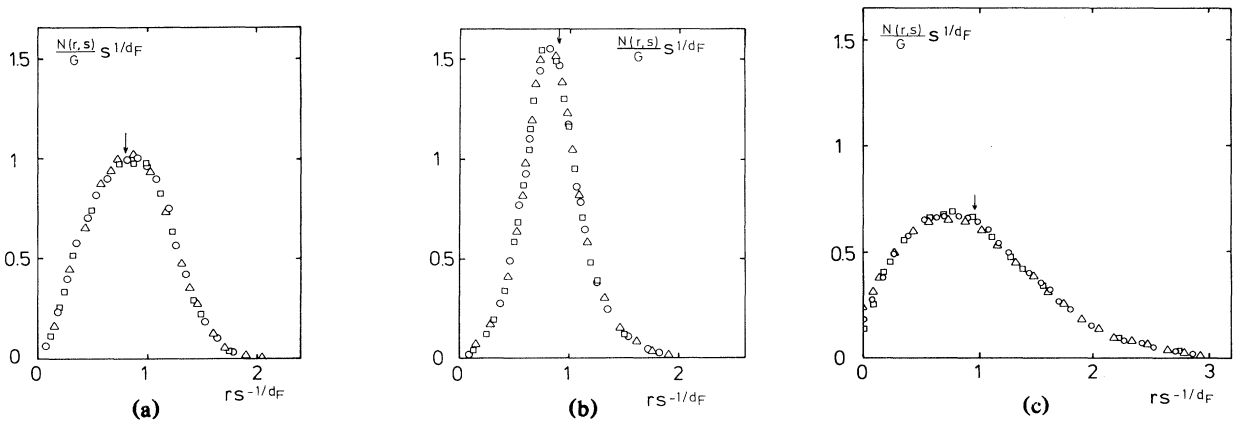


FIG. 4. The probability distribution for (a) $\alpha=-4$, (b) $\alpha=0$, and (c) $\alpha=8$, scaled so that all curves collapse upon a single curve ($s=800$, triangles; $s=2000$, circles; $s=4000$, squares).

In summary, then, we have introduced a new "epidemic" growth model, characterized by a parameter $\alpha$ that determines the mean distance between successively infected tumor sites. We found that the static exponents are independent of $\alpha$, but that dynamic exponents depend strongly on $\alpha$; our model is like a cluster growth analog of the Fisher-Ma-Nickel model of long-range spin-spin interactions. We studied the spatial distribution of growth sites $N(r,s)$ and found that a single length is sufficient to describe this function, unlike the situation in DLA.[13]

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[3]A site is said to belong to chemical shell $L$ if the shortest connected path to the origin has $L$ bonds.

[4]An analogy might be useful at this point. The Fisher-Ma-Nickel model [M. E. Fisher, S. K. Ma, and B. G. Nickel, Phys. Rev. Lett. **29**, 917 (1972)] is another model in which the critical exponents can be continuously tuned by variation of a model parameter; in this case the exchange interaction $J(r)$ has the same power-law decay with distance as $P(r)$ has in Eq. (1).

[5]Note that the butterfly flight is not a Lévy flight, which is a random walk on a Euclidean lattice whose step length is chosen from the distribution (1). Rather, the butterfly moves on a volatile (time-dependent) fractal, composed of the $G$ particles. Shortly we shall calculate the fractal dimension $d_G$ of this fractal, and shall find that it is quite different from $d$, the dimension of the space on which the Lévy flight moves. Moreover, the fractial dimension of the Lévy flight itself is equal to the parameter $\alpha$ and hence is tunable, while the fractal dimension of our walk is $d_f = \frac{91}{48}$ and is not tunable. This last statement follows from Eq. (2a) by the setting of $s = t$. We found that the fractal dimension $d_f$ of the cluster could most easily be determined by the measurement of $d_w$, the fractal dimension of the walk.

[6]In contrast, in the classical ant problem, a "drunk" ant can sample $S$ sites as well as $G$ sites; the ant chooses his next step with equal probability from among the nearest neighbors ($G$ and $S$ particles) that are not immune.

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[10]This argument appears to be known in other branches of physics.

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