Scaling laws and topological exponents in Voronoi tessellations of intermittent point distributions

LETTER TO THE EDITOR

Scaling laws and topological exponents in Voronoi tessellations of intermittent point distributions

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Abstract.   Voronoi tessellations of scale-invariant fractal sets are characterized by topological and metrical properties that are significantly different from those of natural cellular structures. As an example we analyse Voronoi diagrams of intermittent particle distributions generated by a directed percolation process in $2 + 1$ dimensions. We observe that the average area of a cell increases much faster with the number of its neighbours than in natural cellular structures where Lewis' law predicts a linear behaviour. We propose and numerically verify a universal scaling law that relates shape and size of the cells in scale-invariant tessellations. An exponent, related to the topological properties of the tessellation, is introduced and estimated numerically.

Planar random mosaics are often encountered in nature, as, for example, in cuts of biological tissues [1, 2] and two-dimensional soap froths [3, 4]. Thorough investigations of the topological and metrical properties of a huge variety of natural mosaics show a surprising similarity despite the fact that the moulding forces are completely different [5].

The Voronoi construction [6] allows us to generate a mosaic from a set of arbitrarily distributed seeds. It assigns to each seed a cell which is defined as the set of all points of the plane which are at least as close to this seed as to any other seed. Since the resulting Voronoi tessellation is a space-filling cellular structure it allows us to introduce the notion of neighbourhood, i.e. two seeds are neighboured if their cells share at least one side. Thus the Voronoi construction enables us to investigate the neighbour statistics by analysing the topological properties of the corresponding tessellation. This approach has been successfully applied to experimental monosize disk assemblies on an air table [7, 8], confirming the universality of random mosaics. In addition, the Voronoi construction offers an alternative tool for the characterization of the order–disorder transition which occurs when the density is reduced.

So far all experimentally investigated cellular structures are characterized by a typical scale, namely the mean size of a cell. The following question therefore arises: How are the mentioned universal laws affected in the case of *scale-invariant* mosaics? In scale-invariant tessellations one expects that the area distribution of the cells exhibits a power-law behaviour. Moreover, it was shown in [9] that in Sierpinski cellular structures the distribution of edge numbers also behaves algebraically. In this work we are particularly interested in the relation between topological and metrical properties of scale-invariant mosaics. For this purpose we investigate Voronoi tessellations of intermittent particle distributions with algebraic long-range correlations that are generated by planar stochastic point processes.
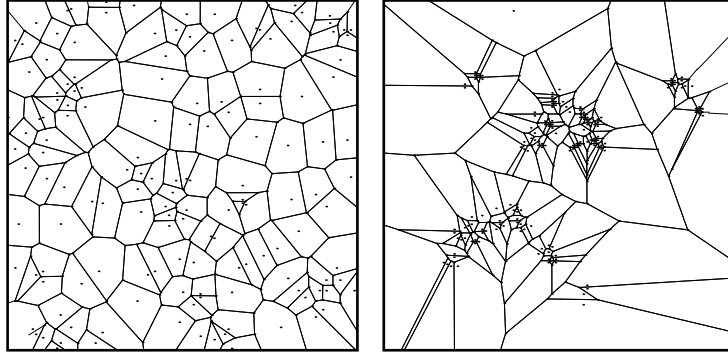
Figure 1. Ordinary and scale-invariant cellular structures. The figure shows parts of Voronoi tessellations for a Poissonian distribution of points (left) and for a point set generated by an almost critical DP process (right).

As an example we consider a directed percolation (DP) process in $2 + 1$ dimensions [10, 11]. In DP—interpreted as a time-dependent stochastic process—particles on a lattice either produce an offspring or self-destruct. Depending on the rates of offspring production and self-destruction this process exhibits a phase transition from a fluctuating active phase into an absorbing state without particles from where the system cannot escape. We use directed bond percolation [10] which is controlled by a single parameter, namely the percolation probability p. Below a critical threshold $p < p_c$ the system approaches the absorbing state in an exponentially short time whereas in the active phase with $p > p_c$ a fluctuating stationary state exists on the infinite lattice. Close to criticality such a stationary DP process evolves through configurations that are characterized by spatially intermittent patterns with long-range correlations [12], i.e. very few particles form highly localized clouds separated by large empty regions.

In this work we use spatial configurations (snapshots) of active particles generated by an almost critical stationary DP process as point sets for a Voronoi construction and study the properties of the resulting tessellations. As shown in figure 1, such a tessellation appears to be very different from natural cellular structures. The reason is that point sets generated by an almost critical DP process approximate a fractal set, apart from lower and upper cut-offs due to system size and lattice spacing. In contrast to natural cellular structures the corresponding Voronoi tessellations are invariant under rescaling. Here we address the question: How do the topological and metrical properties of scale-invariant tessellations differ from those of natural structures? To this end we focus on the correlations between shape and size of the cells.

The simplest quantity describing these correlations is $\langle A \rangle_k / \langle A \rangle$, the normalized average area of an arbitrarily chosen k-sided cell. As pointed out first by Lewis [1], $\langle A \rangle_k$ increases *linearly* with k for a huge variety of tessellations. Deviations from Lewis' law have so far been observed in soap froth experiments [3, 4] and air table tessellations in the dense packing regime [8]. These deviations, however, are restricted to cells with only a few neighbours whereas for large k the linear law appears to be asymptotically valid [4].

In this work the distribution $P(k, A)$ of k-sided cells with area A generated by an almost critical DP process is studied numerically. To this end a directed bond percolation process with parallel updates is simulated on a 500×500 square lattice with periodic boundary conditions. The simulations are performed in the active phase with a small
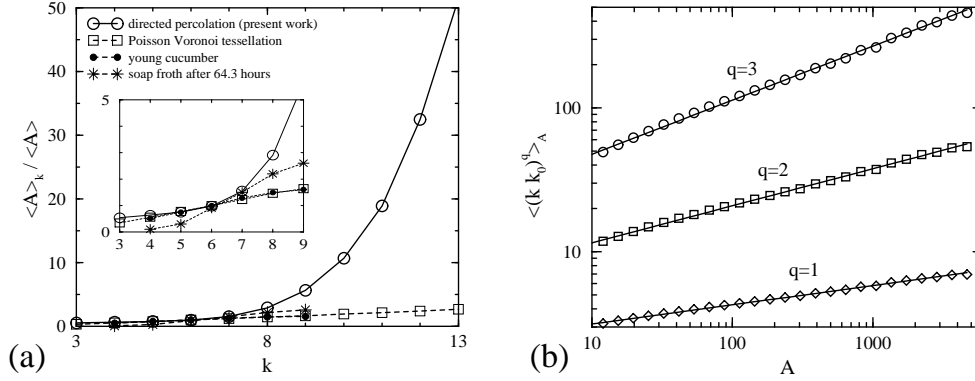
Figure 2. (*a*) Normalized average area $\langle A \rangle_k / \langle A \rangle$ of k-sided cells generated by a DP process in comparison with a Poisson distribution [14], a cucumber tissue [1], and a soap froth [3]. The inset shows a magnification for small values. (*b*) First three moments $\langle (k - k_0)^q \rangle_A$ of the edge number distribution for cells with area A. The shift $k_0 = 3$ is explained in the text.

reduced percolation probability $p - p_c = 10^{-4}$, using the critical percolation threshold $p_c = 0.287\,338$ [13]. After equilibration over 10^5 time steps the system evolves in a practically stationary state with on average about 10^3 particles. The value of $p - p_c$ and the large lattice size ensure that the systems stays in the active phase. At intervals of 100 time steps the actual configuration of active particles is converted into the corresponding Voronoi tessellation†. Averaging over 1000 of such tessellations we determine the relative frequency $P(k, A)$ as well as the mean area $\langle A \rangle_k$ of a k-sided cell.

As shown in figure 2(*a*), the curve for $\langle A \rangle_k / \langle A \rangle$ shows an untypical behaviour as it increases significantly faster than in cellular structures with a typical scale [1, 3, 14]. It neither resembles Lewis' linear law nor a quadratic behaviour that has been discussed in the context of a perimeter law for metallurgical grain structures [4]. The numerical results instead suggest an algebraic increase. In order to check the quality of the scaling, we measured the first three moments of the edge number distribution of cells with area A (see figure 2(*b*)). We observed that the best results are obtained if the edge numbers are measured with respect to their natural minimum $k_0 = 3$. The slopes are estimated by 0.13(2), 0.26(3), and 0.38(5) for $q = 1, 2, 3$, respectively, indicating simple scaling.

Our measurements for $P(k, A)$ are shown in figure 3(*a*). As A increases, the maximum of the curves is shifted along the broken line to higher edge numbers k. The observation that—despite the discreteness of k—all curves roughly have the same shape supports the conjecture that the cell topology in DP may exhibit universal scaling.

Let us recall the scaling properties of DP. As usual, we denote the directed dimension (time) by the index $\parallel$ and the other spatial dimensions by $\perp$. In the active phase close to criticality the stationary particle intensity ρ and the spatial and temporal correlation lengths $\xi_\perp$, $\xi_\parallel$ scale as [10]

$$\rho \sim (p - p_c)^\beta \qquad \xi_\perp \sim (p - p_c)^{-\nu_\perp} \qquad \xi_\parallel \sim (p - p_c)^{-\nu_\parallel}. \qquad (1)$$

The exponents β, $\nu_\perp$, and $\nu_\parallel$ are the three basic critical exponents of DP which usually determine other DP exponents by simple scaling relations. In $2 + 1$ dimensions they have been numerically estimated [11] by $\beta = 0.584$, $\nu_\perp = 0.734$, and $\nu_\parallel = 1.295$.

† The Voronoi construction was performed with the help of the LEDA software package. We extended the algorithm in a way such that periodic boundary conditions are taken into account.
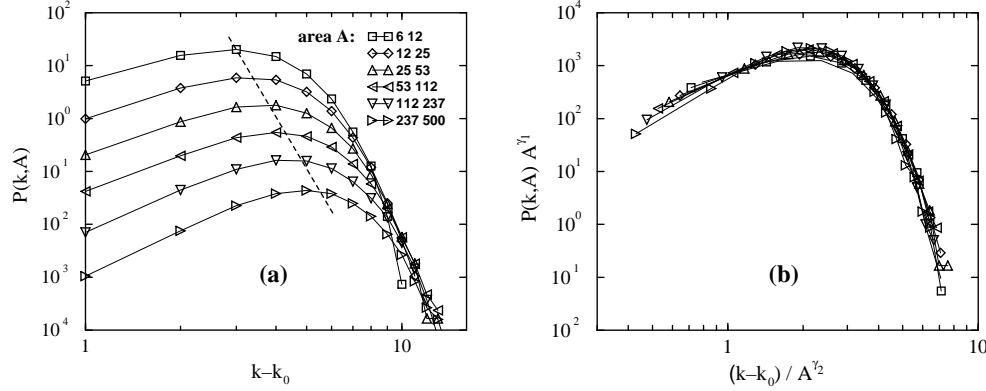
Figure 3. Numerical results from Monte Carlo simulations in the scaling regime. (*a*) Double-logarithmic representation of the relative frequency $P(k, A)$ of cells with k edges and area A in units of squared lattice spacing. The broken line indicates the location of the maxima. (*b*) Collapse of the same data using the scaling form (6), visualizing the scaling function Φ. The best collapse is obtained for the topological exponent $\tau = 0.20(4)$.

The Voronoi construction maps each active site onto a cell with a certain area A and a topology k. The cell topology, i.e. the number of neighbours, can be regarded as an additional quantity for the description of a DP process in more than one spatial dimension. In order to find an appropriate scaling relation, we conjecture—in analogy to equation (1)—that the cell topology exhibits scaling properties similar to those of distances and densities. This means that the corresponding scaling regime is restricted to $k < \kappa$, where κ diverges close to the transition as

$$\kappa \sim (p - p_c)^{-\tau}. \tag{2}$$

Here κ limits the scaling regime of edge numbers similarly as $\xi_\perp$ limits the scaling regime of distances in the active phase. τ is a critical exponent which will be referred to as a *topological exponent* of DP. At present it is not clear whether τ is a novel exponent or related to the bulk exponents β, $\nu_\perp$, $\nu_\parallel$ by a scaling relation (as, for example, the critical initial slip exponent of DP [15]).

In the scaling regime we expect the probability distribution $P(k, A)$ for cells with topology k and area A to obey the scaling form

$$P(k, A) \sim A^{-\gamma_1} \Phi(kA^{-\gamma_2}) \qquad (k < \kappa, A < \xi_\perp^2) \tag{3}$$

where $\Phi(z)$ is a universal scaling function. The exponents γ_1, γ_2 can be derived as follows. Summing over the edge numbers k we obtain the area distribution

$$P(A) = \sum_k P(k, A) \simeq \int_0^\infty \mathrm{d}k\, P(k, A) \sim A^{\gamma_2 - \gamma_1}. \tag{4}$$

Assuming that $0 < \gamma_2 - \gamma_1 + 2 < 1$ we can compute the average area of cells for a given scaling length $\xi_\perp$ by

$$\langle A \rangle \sim \int_{a^d}^{\xi_\perp^d} \mathrm{d}A\, A P(A) \sim \xi_\perp^{d(\gamma_2 - \gamma_1 + 2)} \tag{5}$$

where a is the lattice spacing and $d = 2$ the spatial dimension. Since $\langle A \rangle$ is inversely proportional to the intensity of points $\rho \sim \xi_\perp^{-\beta/\nu_\perp}$ we obtain the relation $\gamma_1 = \gamma_2 + 2 - \beta/d\nu_\perp$

by comparing the exponents. On the other hand, the argument of the scaling function Φ in equation (3) should be invariant under rescaling $A \to b^d A, k \to b^{\tau/\nu_\perp} k$, leading to $\gamma_2 = \tau/d\nu_\perp$. Thus the expected scaling relation reads

$$P(k, A) \sim A^{2+(\tau-\beta)/d\nu_\perp} \Phi(kA^{-\tau/d\nu_\perp}) \qquad (k < \kappa, A < \xi_\perp^2). \tag{6}$$

In order to verify this scaling law, we numerically estimated the topological exponent τ by data collapse (see figure 3(b)). Again, the best collapse is obtained if one replaces k by $k - k_0$ with $k_0 = 3$, which is the natural minimum for the number of sides of a cell. Our best estimate is $\tau = 0.20(4)$, corresponding to $\gamma_1 = 1.73(3)$ and $\gamma_2 = 0.13(3)$. The collapse is fairly convincing, supporting the above scaling hypothesis. In addition, the result is in agreement with the measurements in figure 2(b). We note that this result should not depend on the specific choice of the DP dynamics (e.g. bond or site percolation). Rather we expect τ to be a universal exponent characterizing the DP class in $2+1$ dimensions. The question of whether τ is independent or related to the other scaling exponents is still open.

It should be noted that the proposed scaling hypothesis needs further verification as the scaling regime of k extends over one decade only. The main limitation is the small value of τ, i.e. each decade of scaling range in k requires about five decades of scaling range in A. However, it can be shown that equation (3) holds exactly in the case of a simple Sierpinski gasket which strongly supports our scaling hypothesis [16]. The scaling form assumes that the underlying point distribution is a simple fractal. This is the case for spatial cuts of critical DP states, as stated in [12]. For multifractal cellular structures, however, the proposed scaling relation should be replaced by appropriate multiscaling laws [16].

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