Acta of Bioengineering and Biomechanics
Vol. 1, No. 1, 1999

Critical growth of fractal patterns in biological systems

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The paper presents the fractal model of biological tissue growth comparing surface evolution with bulk processes. Thin surface layer has been modelled by fractal and characterised by external fractal dimension. The evolution equation compares cell multiplication in surface tissue from bulk development. When tissue grows inside some limiting volume other critical effect may appear entailing singularity of characteristic measures.

Key words: fractals, critical effects

1. Introduction

From the very beginning fractals were applied to model various biological systems [1]. The basic reason was the complexity of biological patterns and the recurrent construction of fractals reducing the amount of information necessary to create a given form. Even at present time the discussion about fractality or nonfractality of some tissues has been continued.

On the other hand, the reasons to make use of fractal language can be very different. Suppose that one models some pattern in terms of smooth manifold. According to differential geometry any suitably small fragment of such a manifold is equivalent to the fragment of the space $\Re^n$ for some n (equal to the manifold dimension). In turn, the fragments of $\Re^n$ look in the same way from the both sides (or more sides in spaces with larger dimension). For definiteness let's think about the surface surrounding some volume. The plane surface does not favour any side and the same holds for fragments. The global geometry of smooth manifold (like sphere, for example, with clearly different internal and outer directions) becomes invisible in fragments at suitably short scales of length. At the same time, the differentiation of sides may origin at short scales and subsequently it disappears when one approaches longer scales. In all such situations, fractals appear to be the only tool able to describe differentiation of sides coming from zero scales of length. This difference is visible in the one-sided fractal dimensions. To some extend, the one-sided fractal dimensions are the characteristics complementary to the tools of differential geometry (the global one).

Suppose that we have some (three-dimensional) geometrical object A with (possibly) fractal boundary ∂A. Let B_δ denote the δ-parallel body of the given set B (i.e., the union of ball with centres belonging to B and radii equal to δ: $B_\delta = \bigcup_{b\in B} K(b,\delta)$, where $K(b,\delta)$ denotes the ball with the centre at b and the radius δ). Independently of the initial geometrical set B, its δ-parallel body B_δ will have a finite volume (or finite Lebesgue'e measure to be more rigorous). However, this volume can be spread in the sides of B in very different ways. The one-sided fractal dimensions describe the distribution of the above volume with respect to the sides of boundary:

$$\dim_{\text{int}} \partial A = 3 - \lim_{\delta\to 0} \frac{\ln Vol(A\cap(\partial A)_\delta)}{\ln \delta},$$
$$\dim_{\text{ext}} \partial A = 3 - \lim_{\delta\to 0} \frac{\ln Vol(A^c\cap(\partial A)_\delta)}{\ln \delta}, \tag{1}$$

where subscripts int and ext denote the respective internal and external fractal dimensions and A^c is the complement of the set A. Clearly, internal and external dimensions depict power dependence of volume upon δ. The greater value among internal and external dimensions coincides with the box-counting or the Minkowski dimension for the entire fractal set.

The correspondence between bulk growth process (in A) and surface evolution of ∂A allows comparing cell multiplication via labile internal fractal dimension of ∂A. On the other hand, in the usual physical approximation one assumes that the number of cells (or other biological units) is proportional to the size of geometrical pattern filled with cells. In turn, the sizes of fractal patterns are depicted by their suitable fractal measures. Therefore:

$$N = a(D)\mu_D, \tag{2}$$

where N is the number of cells, D denotes the fractal dimension and μ_D depicts the corresponding fractal measure. Note that the generalised cell density $\alpha(D)$ cannot be constant with respect to D. The small (multiplicative) shifts of all quantities entering (2): $N' = (1 + \varepsilon_N)N$, $D' = (1 + \varepsilon_D)D$, $\mu_D' = (1 + D\varepsilon_\mu)\mu_D$ entail the following (evolution) equation:

$$\varepsilon_N = D\varepsilon_\mu + \varepsilon_D \ln\frac{\mu_D}{A(D)} \qquad A(D) = \left(\frac{\pi a(D)}{\pi D}\right)^{-1} \qquad \frac{\pi f(x)}{\pi x} = \lim_{\varepsilon\to 0}\left(\frac{f((1+\varepsilon)x)}{f(x)}\right)^{1/\varepsilon} \tag{3}$$

where $\pi f/\pi x$ denotes the multiplicative derivative of the function $f(x)$. In contrast to the generalised densities $\alpha(D)$, the characteristic measures $A(D)$ define the characteristic size of the examined system, which change their (fractal) structure in the uniform

way (uniform with respect to scaling, i.e. such that it does not favour any length scale). Essentially the above equations (2) and (3) exactly coincide with equations describing defects evolution during fatigue process. The only difference consists in replacing energy by cell number. However, when examin fractal models some surface layer of biological tissue, the fractal dimension D should be replaced by internal fractal dimension D_{int}. The parameter δ of the δ-parallel body plays now the role of surface layer thickness and also the fractal measure should be derived from the power dependence of (internal) volume upon δ (passing to the limit $\delta \to 0$). Even for elementary fractal patterns, the internal fractal dimension may drastically differ from D.

Suppose now that the examined tissue A, bounded by some fractal surface ∂A grows changing its number of cells. The bulk growth constant is λ (i.e., during unit time, the number of cells changes from N to $(1 + \lambda)N$ and in similar way, the surface growth constant equals γ. When cell densities per unit volume (in both: bulk and surface layer) are constant:

$$\gamma - \frac{D}{3}\lambda = \varepsilon_D \ln \frac{\mu_D}{A(D)}. \tag{4}$$

This may be used to compare growth processes by means of the measurement of fractal dimension D. Of course all said above about one-sided dimensions remains also valid for (4). However, (4) describes only free growth process, without any limitations (coming from other bounding tissues of living biological system). In turn, such additional limitations are often present, for example, the growth (and complexity) of brain becomes limited by skull. On the other hand, fractals do not take into account these limitations. In finite volume one may generate large (without any mathematical limitations) fractal with lower fractal dimension. Some other physical postulate becomes necessary. The basic problem can be briefly explained in the following way. As a rule various fractal measures are singular one with respect to another. This means that relative densities do not exist and any physical quantity possessing density with respect to some fractal measure does not have densities with respect to other measures. Therefore some other method to characterise spatial distribution should be applied (without referring to densities).

2. Critical effects in the growth of biological systems

In equation (2) the quantity characterising the structure of surface layer was just the generalised cell density $a(D)$. When other limitations of growth process are present it appears natural to incorporate them via modification of $a(D)$. Suppose that the volume V limits the available space for tissue evolution. Then

$$V \approx (\partial A)_\delta \tag{5}$$

for some value of the parameter δ. In the mathematical sense, the above δ depicts the (Hausdorff) distance between the examined surface layer ∂A and the limiting volume

V. In turn, if the growth process of tissue becomes sensitive to limitation, the generalised cell density should also depend upon δ from equation (5). When δ is large, the correction to equation (2) will be negligible. There is enough space for evolution.

Suppose now that at some moment δ approaches small values (the tissue "fills" limitation imposed upon growth). Then the two things happen. At first, corrections coming from cell density on δ may become large $a(D) \rightarrow a(D,\delta)$. Secondly, the Minkowski estimation of the equation (5) may begin to work according to the dependence

$$V \approx (\partial A)_\delta \approx \mu_D \delta^{3-D}, \tag{6}$$

where μ_D denotes the corresponding fractal measure of surface layer. Once more, expanding equation (2) with respect to multiplicative shifts of all quantities and taking into account the limiting equation (6) we derive the evolution equation. Now both D and δ should be treated as independent characteristics of (fractal) tissue structure. The direct calculations give:

$$\varepsilon_N = (1-\xi)D\varepsilon_\mu + (1-\xi)\varepsilon_D \ln\frac{\mu_D}{B(D)} \qquad B(D) = A(D)^{\frac{1}{1-\xi}}\left(\delta^D\right)^{\frac{-\xi}{1-\xi}} \qquad \xi = \frac{\ln\left(\frac{\pi a}{\pi\delta}\right)}{3-D}, \tag{7}$$

where the new, effective characteristic measure $B(D)$ has appeared. The first equation (7) has the structure similar to (2), apart from a factor $(1 - \xi)$. However, there is crucial difference between (2) and (7). The characteristic measures $A(D)$ were finite for all dimensions D (at least if generalised cell densities are differentiable). In contrast, the effective characteristic measures $B(D)$ may approach infinite or zero values. Such critical behaviour corresponds to points at which the value of ξ becomes close to or equal to 1. In turn the variable ξ carries all corrections coming from limitations given by the bounding volume V. Note that the transition of ξ through 1 does not entail critical evolution of the cell density N since the right-hand side of equation (7) is proportional to the factor $(1 - \xi)$ vanishing for ξ. The entire (critical) effect of ξ corresponds to the singularity of effective characteristic measures $B(D)$.

As has been explained before, the characteristic measures define the size of (fractal) surface tissue undergoing uniform transformation of fractal structure. When $B(D) \approx 0$, the surface tissue evolves as the collection of infinitely small, uncorrelated patterns. At the opposite limit, when $B(D) \rightarrow \infty$, the entire structure grows as the one correlated pattern. To some extent this singularity of characteristic measures reminds a phase transition, however this is a point singularity.

It should be underlined that the described critical growth of biological pattern is quite similar to the evolution of fatigue defects in materials [2]. In the case of defects, the energy accumulated at defects replaces the generalised cell density (and of course energy density occupies the place of cell density). This coincidence reflects the simi-

lar nature of energy and cell number since both quantities are scalars. However there is also a difference. In the case of a surface layer the fractal dimension D is the internal dimension, whereas the box-counting dimension describes defects. Moreover, the fractal dimension D in the equation (6) may differ from the one-sided internal dimension in equation (1). This entails another critical behaviour not presented in this paper.

3. Conclusions

In contrast to the popular opinion that fractals describe well biological patterns we must say that only very specific geometrical fractal sets have been met in biological systems. Fractals describing such patterns have another important property: the one-sided fractal dimensions are different. This reflects the fixed direction of evolution of biological tissues: inside or outside.

The cell multiplication may entail shift of fractal dimension of the surface layer. In this case, the growth constant of bulk and surface tissues can be related via equation including fractal dimensions as well as characteristic fractal measure.

The characteristic fractal measures define the size of fractal pattern undergoing uniform, with respect to scaling, transition of fractal structure. When external geometrical bounds are imposed upon growth of biological tissues, effective ones replace the characteristic measures. In turn, the effective measures may become singular, approaching zero or infinite values.

References

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