DESCRIPTION OF MICRODOMAIN GROWTH IN COMPETITIVE 3D-AGGLOMERATES*

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A model of microdomain growth in three-dimensional systems like metals or ceramics is adapted to describe the growth kinetics and structure formation in competitive mass exchanging systems like biomembranes, liquid crystalline materials or polymers. The theory proposed assumes that the material in question can be partitioned into pieces (microdomains, clusters, grains) and concerns with modelling of the growth process in a time-dependent regime (*i.e.*, when the so-called long tail kinetics is introduced). As a result, power and logarithmic laws of the average radius of the growing domain against time are obtained and some other probabilistic characteristics of the process are analyzed. An extension to disruption or defect processes in biosystems is presented. The approach developed can serve to elucidate some experimental results got *e.g.* for multilamellar lipid bilayers which till now are exclusively interpreted in terms of the Kolmogorov–Avrami model.

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1. Introduction

Many different growth and structure formation processes have carefully been studied in recent years by physicists, materials scientists as well as by technologists (*cf.* , [1, 2] and references therein). The normal grain growth, till now related preferably to metallic and ceramic materials [3–5] may stand for a proper example of the microdomain growth in which both competition of the grain population within the system as well as complexity of it are permanently observed. Till now, this phenomenon has mostly been modelled by means of computer simulations using the Monte Carlo technique (*cf.* [5]) and by providing an analytical description based on a diffusion-type equation for evolution of the grain (domain) population when fluctuations of the grain boundary are imposed on the system. One may find some other descriptions of this process, *e.g.* the mechanism of reduction of the grain boundary energy [6] or very recent modelling based on the Voronoi tessalation concept [7, 8]. Since the systems described are interacting systems of grains it seems to be quite natural that such modelling could also be applied to more complicated physical situations. Namely, we think of the growth and/or some disruption (in particular defect) processes occurring in agglomerates understood here as objects made of smaller subunites which are somehow glued together strongly enough to keep the structure as a whole. Examples of such agglomerates are model biomembranes or bilayers (treated as three-dimensional objects) being the systems of lipid domains (grains, clusters) which may interact with each other [9]. Because, however, these complex diffusion-relaxation or diffusion-reaction [10] systems are recently understood in terms of the so-called long tail or fractal kinetics [11, 12], therefore a certain modification within the classical description is needed just for better reflection of the cooperative kinetic behaviour of those systems (*cf.* [13] and references therein). In this work we wish to describe two kinds of growing processes: a purely growth process which does not lead to creation of a final structure (unlimited growth) and a growth process which leads to the structure formation (limited growth). In Section 2 we briefly sketch a model of the normal grain growth of materials introduced by Mulheran and Harding [4, 5]. Next, we modify this model in order to adapt it for a description of anomalous kinetic behaviour of the "soft matter" system [12, 14] and microstructure (pattern) formation [8]. In Section 3, analysis of the model is carried out. The last Section 4 contains final remarks.

2. Description of the domain growth kinetics in agglomerates like biomembranes or liquid crystalline materials

There are two main theories of the normal grain growth with differing mechanisms driving the growth, *i.e.* caused by the surface tension of the curved boundaries [6] and caused by random fluctuations of the grain boundaries (it is named the random walk model) [4, 5]. In the latter case, the mechanism for the growth is the migration of particles across the boundaries. Individual atoms (or rather clusters of atoms) move from one grain to its neighbour changing grains volume and only surface atoms take part in the process. So, grains grow by gaining or losing atoms and if any grain shrinks to zero size then it cannot re-nucleate and is lost for ever. The second mechanism is correct when the grain boundary energy is negligible in comparison to the thermal energy of the boundary atoms and this is the case when the thermal fluctuations are important for the growth kinetics. In this theory, the spatio-temporal evolution of the system consisting of grains of volume v is represented by the following equation of diffusion type [4, 5],

$$\frac{\partial}{\partial t} f(v,t) = \frac{\partial^2}{\partial v^2} \left[D(v,t) f(v,t) \right], \tag{1}$$

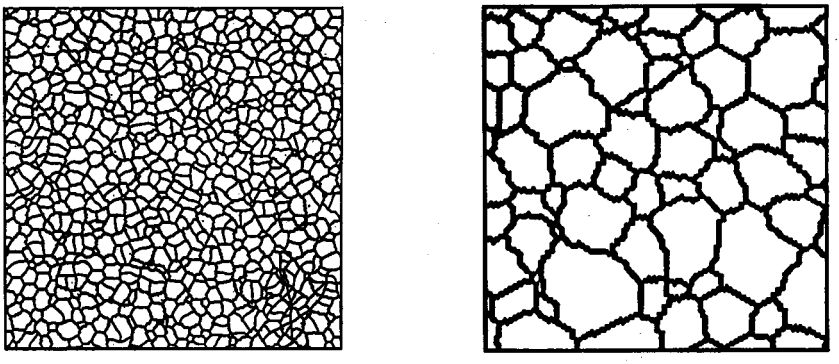
where $f(v,t)$ is the distribution function of grains of volume v at time t (a number of grains of volume v). The diffusion coefficient $D(v,t)$ has to reflect the fact that the net flux of the migrating particles is proportional to the surface of an individual grain [5],

$$D(v,t) \sim v^{2/3}. \tag{2}$$

Notice that this term represents the *scaling* of the number of available surface sites (atoms) with grain volume (the basic ideas of the scaling concept can be found *e.g.* in [15]). One can prescribe the boundary conditions as follows [5]

$$f(0,t) = f(\infty,t) = 0\,, \tag{3}$$

which means that: no new grain may nucleate when a certain one is shrunk to size zero and no single grain has to dominate the structure as a whole. A more careful and detailed presentation of the aforementioned approach can be found elsewhere [4, 5, 16]. Phenomena described by the formalism are kinetic, which mostly relies on exchanging individual atoms or molecules among grains (microdomains, clusters) , so a certain grain can grow or shrink in its size simply by gaining or losing atoms or molecules. The afore presented formalism can be adapted to the description of such phenomena as growth, structure formation or even disruption in biomembranes, *cf.* Fig. 1.



(A) (B)

Fig. 1. Sketch of a possible realization of the growth process: (A) at the early stage of evolution; (B) for "long" times. In the case of unlimited growth, the growing process is still continued. In the case of limited growth, the situation in (B) corresponds to the final structure (a limiting state of the system).

In biosystems or in "soft matter" systems like biomembranes, the co-operative structural changes in lipid bilayer membranes can be associated with either the growth of lipid domains or with some kind of disruption (defection, lysis, phase separation) of the membrane material (or a part of it) caused by certain species like proteins, anaesthetics, impurities, *etc.* [9]. Both are closely connected to the strength of interactions in the system which are in general the lipid-protein interactions [9]. We know [12] that if the biological process involves diffusion of proteins within the lipid matrix and the protein thermodynamically prefers certain lipid domains then the size of these domains and their lifetime are of great importance. It can lead to the fractal reaction kinetics of the process and cannot be understood in classical reaction-kinetic terms [12]. To be more concrete let us recall the gel-to-liquid crystalline phase transformation of some multilamellar lipid bilayers [17] or the defect process of lipid model membranes, *i.e.*, the fluid mosaic model of the structure of biomembrane due to Singer and Nicolson or formation of liposomes [9]. The latter can be done on the basis of certain experimental [18, 19] and theoretical [20] studies of the defect process in model phospholipid membranes (for review see also [13] and references therein) where interactions between melittin and some lipid molecules cause the formation of defects in the bilayer. These defects result from the fact that each group of lipids within the range of action of a protein molecule, which also likely tends to diffuse in the lipid environment, constitutes a domain with different thermodynamic and structural properties compared to the nonaffected lipids. Defects would emerge at the interfaces between the affected domains and the nonaffected surroundings resulting in a desta-

bilization of the gel phase of the lipid bilayer [19]. In such systems also another kind of disruption may take place like lysis, phase separation or even gel-to-liquid crystalline phase transformation [21, 14]. Under these physical circumstances, however, the system in question tends to fall into pieces, separated subunits or subdomains and for long times a limiting stationary state is eventually reached [18]. An example of how the small helical polypeptides poison a lipid bilayer structure is carefully studied in [18] for states close to equilibrium. In this case, a possible scenario of the structure formation is roughly sketched. The main conclusion which comes from the investigation is that the whole defect process would lead to the formation of a "frozen" or "static" essentially time-independent structure of the poisoned piece of material. Just for encountering those observations that, in general, seem to be rather quite time-sensitive, we assume that the diffusion function $D(v,t)$ is of the form

$$D(v,t) = C_0 \frac{v^{2/3}}{(1+t)^h}, \quad h \geq 0, \tag{4}$$

where C_0 is a positive constant (it ensures correct units). The v-dependence of $D(v,t)$ follows from the same arguments as leading to the relation (2). The explicit dependence of the diffusion function (4) on time might have its origin in the random nature of growth or disruption (fragmentation) processes: mass of microdomains changes stochastically in time due to random attachment or detachment of molecules or atoms (or adsorption-desorption processes [22] at the grain boundary). In particular, the exponent h might be related to probabilistic characteristics of a model put on the migration process across domain boundaries just as it has been justificated in two Refs [23, 24] for other kinds of processes. In general, it could be related to the spectral dimension of the process studied (*e.g.*, when chemical reaction is taken into account; *cf.* batch reactions described by [25, 26]).

3. Analysis of the model

Our model is based on the random walk model (1) with the diffusion function (4) and with the boundary conditions (3). This model can be solved exactly with the result

$$f(v,t) = A v^{1/3} a^{-7/4}(t) \exp\left(\frac{-9v^{4/3}}{16 C_0 a(t)}\right), \tag{5}$$

where A is a constant which takes into account normalization of the distribution function $f(v,t)$ and

$$a(t) = \begin{cases} \frac{(1+t)^{1-h}-1}{1-h} & \text{for } 0 \leq h < 1, \\ \ln(1+t) & \text{for } h = 1, \\ \frac{1-1/(1+t)^{h-1}}{h-1} & \text{for } h > 1. \end{cases} \tag{6}$$

Now, let us present the first three moments $\langle v^n(t)\rangle$ of the process for $n = 0, 1, 2$. The zero-moment, $\langle v^0(t)\rangle$, is equivalent to the number of microdomains in the system and for long times it has the asymptotics

$$\langle v^0(t)\rangle \propto a^{-3/4}(t). \tag{7}$$

The first moment, $\langle v^1(t)\rangle$, is a total volume of all microdomains and is constant,

$$\langle v^1(t)\rangle = V = \text{const}. \tag{8}$$

From this equation one can calculate the constant A in (5) which is proportional to V,

$$A = \left(\frac{9}{16C_0}\right)^{7/4} \frac{V}{\Gamma(\frac{3}{4})}. \tag{9}$$

The second moment, $\langle v^2(t)\rangle$, behaves asymptotically as

$$\langle v^2(t)\rangle \propto a^{3/2}(t). \tag{10}$$

The most important physical characteristics of the process is the mean radius $r_{\mathrm{av}}(t)$ of the microdomains defined by the relation

$$V \propto \langle v^0(t)\rangle r_{\mathrm{av}}^3(t) \tag{11}$$

and asymptotically $r_{\mathrm{av}}(t)$ behaves as

$$r_{\mathrm{av}}(t) \propto a^{1/4}(t). \tag{12}$$

From the relations (6) it follows that one should distinguish two cases. Namely, the case which represents a pure growth of the agglomerate, *i.e.* a dynamic case, for $h \in [0,1]$, and the case of limited growth for $h > 1$. As to the first case, we can distinguish three interesting types of behaviour of the system. Namely, for $h = 0$ one gets the classical behaviour of the Mulheran and Harding model which was successfully applied to the normal

grain growth in metals and ceramics. This may also be a sub-case reflecting the growth of unaffected domains. The next sub-case,

$$r_{\mathrm{av}}(t) \propto t^{(1-h)/4}, \tag{13}$$

where $0 \leq h < 1$, would still be responsible for the growth of unaffected domains or grains, but then the growth is slower because of some more subtle and "time-consuming" effects as *e.g.* interactions, pH-changes of the environment, water penetration, diffusion of proteins or convection of clusters, chemical reactions at boundaries, *etc.* [9, 27, 28]. Note that these two sub-cases differ substantially from the "conventional" case $r_{\mathrm{av}}(t) \propto t^{1/2}$ and which is also valid for one-dimensional systems because then the normal grain growth process is represented by the standard diffusion equation with a constant diffusion function (*cf.* , [29, 30] for details). The last sub-case ($h = 1$) is given separately by

$$r_{\mathrm{av}}(t) \propto \ln^{1/4}(t). \tag{14}$$

As to the second case ($h > 1$), one can observe that

$$r_{\mathrm{av}}(t) \to \text{const.} \quad \text{as} \quad t \to \infty \tag{15}$$

which means that microdomains stop to grow! It may be interpreted as a formation of structures (frozen or "static" ones). It is worth to stress that in the case of unlimited growth, the volume fluctuations $\langle v^2(t)\rangle - \langle v^1(t)\rangle^2$ grow with increasing time as it is expected (see Eqs (10) and (8)). In the case of limited growth, in turn, the volume fluctuations saturate for a limiting stationary state. It is another supporting argument for the formation of a stable final structure like that obtained *e.g.* in the experimental work [18]. Let us also repeat once more that our model is purely kinetic and has no microscopic details of the process included. Some speculations concerning more detailed mechanisms of the growth or fragmentation process could be done by *e.g.* more careful analysis of values of the exponent h [25, 26, 31], but it seems to be beyond the scope of this paper.

4. Final remarks

Let us first summarize the results obtained in the paper. Namely, in Section 2 we have presented the description of the grain growth kinetics in agglomerates (biomembranes or liquid crystalline materials) that is based on the model of normal grain growth (*cf.* , [4, 5]). The basic extension relies on the modification of the diffusion function $D(v,t)$ given by Eq. (4). It has serious physically interesting consequences which lead to broader

as well as more accurate description of many types of growth and structure formation phenomena not necessary being limited to the growth of ceramics or metallic materials. Experimental examples concerning the growth and pattern formation processes are reported elsewhere (*cf.* [9, 21, 17] and references therein). Looking at the results revealed in Section 3, it is clear that the formalism proposed can be applied for the description of not only the pure (unlimited) growth process in materials in a more "subtle" way (at least, the logarithmic sub-case of $h = 1$ can be mentioned here), but also the limited growth process (some kind of structure formation) represented by the sub-case of $h > 1$ (for details, see Eq. (6)) which, in general, may likely occur in biomaterials or "soft matter" systems [28, 32, 13].

Let us also make here a general comment to the situation that we studied. Namely, we are of the opinion that Eq. (1) with an adequately chosen function $D(v,t)$ could even be accepted as a generalization of the phenomenological Avrami (or Kolmogorov–Avrami) equation [33, 17]

$$\frac{d}{dt}F(t) = N[1 - F(t)]\frac{dV_n}{dt}, \tag{16}$$

where N is a number of randomly distributed nuclei per unit volume each of which will grow to a volume V_n at time t, and $F(t)$ is a time-dependent fractional completion of a sample transformed to a new phase [17, 33] (*cf.* , [16] for a critical discussion). It may probably provide an interesting explanation for understanding the phase transformations in lipid bilayers (model lipid membranes) which follow classical kinetics but with small fractional dimensionalities [21]. Such results, but based on more realistic physical foundations (*e.g.*, that the number of lipid domains is never constant during the transformation process), can easily be recovered by means of the description presented above. Moreover, it can be believed that some subtle effects like *e.g.* lipid-lipid interactions or presence of other molecules (*e.g.*, mobile or immobile proteins or even anaesthetics) and their influence on the behaviour of the whole system, *etc.*, are possible to be accommodated in Eq. (1), especially when performing a reasonable modification of the formalism due to Mulheran and Harding (which is sketched above). This can at least elucidate some discrepancies between the values measured and those which may be obtained on the basis of the simple Avrami model [21]. Note also that both descriptions, *i.e.* done by Eq. (1) and by Avrami equation, are here of interest in the regime of the interface-controlled growth (*cf.* , [34], especially Chap. 5) represented in this model by the function $D(v,t)$ in Eq. (1).

An important problem, which is associated to the modelling of the process under investigation, is related to the microscopic justification (from the "first principles") of the form of the diffusion function $D(v,t)$, in particular,

determination of the value of h-exponent. We hope that the problem will attract future attention.

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REFERENCES

[1] K. Birdi, *Fractals in Chemistry, Geochemistry and Biophysics*, Plenum Publishing Corporation, New York 1993.
[2] B.H. Kaye, *A Random Walk Through Fractal Dimensions*, VCH Verlagsgesellschaft mbH, Weinheim, 1989.
[3] M.P. Anderson, D.J. Srolovitz, G.S. Grest, P.S. Sahni, *Acta Metall.* **32**, 787 (1984).
[4] P.A. Mulheran, J.H.Harding, in *Defects in Materials*, eds P.D. Bristowe, J.E. Epperson, J.E. Griffith and Z. Liliental-Weber, Materials Research Society, Pittsburgh, 1991, Vol.209, pp.251-256; P.A. Mulheran, J.H. Harding, *Acta Metall. Mater.* **39**, 2251 (1991).
[5] P.A. Mulheran, J.H.Harding, *Materials Science Forum*, Vols.94-96, Trans Tech Publications, Switzerland 1992.
[6] M. Hillert, *Acta Metall.* **13**, 227 (1965).
[7] P.A. Mulheran, *Phil. Mag. Lett.* **65**, 141 (1992); **66**, 219 (1992).
[8] P.A. Mulheran, *Phil. Mag. Lett.* **68**, 63 (1993).
[9] O.G. Mouritsen, R.L. Biltonen, in *Protein-Lipid Interactions*, ed. A. Watts, Elsevier Science Publishers B.V., Amsterdam 1993, Ch.1, pp.1-39.
[10] A. Bunde, Sh. Havlin, *Fractals in Science*, Springer-Verlag, Berlin 1994.
[11] J.F. Nagle, *Biophys. J.* **63**, 366 (1992).
[12] R.L. Biltonen, *J. Chem. Thermodynamics* **22**, 1 (1990).
[13] P. Laggner, M. Kriechbaum, *Chem. Phys. Lipids* **57**, 121 (1991).
[14] S. Frey, L.K. Tamm, *Biophys. J.* **60**, 922 (1991); M.J. Kanehisa, T.Y. Tsong, *J. Amer. Chem. Soc.* **100**, 424 (1978).
[15] P.J. De Gennes, *Scaling Concepts in Polymer Physics*, Cornell University Press, Ithaca, New York 1979.
[16] A. Gadomski, *Phil. Mag. Lett.* **70**, 335 (1994).
[17] Q. Ye, W.W. van Osdol, R.L. Biltonen, *Biophys. J.* **60**, 1002 (1991).
[18] M. Posch, U. Rakusch, C. Mollay, P. Laggner, *J. Biol. Chem.* **258**, 1761 (1983).
[19] A. Colotto, D.P. Kharakoz, K. Lohner, P. Laggner, *Biophys. J.* **65**, 236 (1993); A. Colotto, K. Lohner, P. Laggner, *Progr. Colloid Polym. Sci.* **89**, 334 (1992).
[20] A. Gadomski, M. Kriechbaum, P. Laggner, *Nuovo Cimento* **D**, (1995).
[21] C.P. Yang, J.F. Nagle, *Phys. Rev.* **A37**, 3993 (1988).
[22] J.F. Douglas, H.E. Johnson, S. Granick, *Science* **262**, 2010 (1993).

[23] A. Gadomski, J. Łuczka, *Fractals* **1**, 875 (1993).
[24] J. Łuczka, P. Hänggi, A. Gadomski, *Phys. Rev.* **E51**, April (1995).
[25] R. Kopelman, *Science* **241**, 1620 (1988).
[26] R. Kopelman, in *Fractal Approach to Heterogeneous Chemistry*, ed. D. Avnir, John Wiley and Sons, Chichester 1989, Ch.4.1.3, pp.295-309.
[27] J.N. Israelachvili, *Intermolecular and Surface Forces*, Academic Press, London 1985.
[28] N.A. Peppas, R. Langer, *Science* **263**, 1715 (1994).
[29] N.P. Louat, *Acta Metall.* **22**, 721 (1974).
[30] A. Gadomski, Z.J. Grzywna, J. Łuczka, *Chem. Engng. Sci.* **48**, 3713 (1993).
[31] L.S. Liebovitch, J.M. Sullivan, *Biophys. J.* **52**, 979 (1987).
[32] G. Cevc, D. Marsh, *Phospholipid Bilayers*, John Wiley and Sons, New York 1987.
[33] M. Avrami, *J. Chem. Phys.* **7**, 1103 (1939); A.N. Kolmogorov, *Bull. Acad. Sci. USSR, Phys. Ser.* **3**, 355 (1937).
[34] D.A. Porter, K.E. Easterling, *Phase Transformations in Metals and Alloys*, Van Nostrand Reinhold Company, New York 1981, Ch.5.4, pp.287-289.