LETTER TO THE EDITOR

Generalised theory of diffusion-controlled defect annealing

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Abstract. Recently derived kinetic equations for joint distribution functions of defects are discussed. Defects are assumed to possess a diffusion motion and to annihilate instantly when two dissimilar defects approach a clear-cut recombination radius. Along with the spatial correlation of dissimilar defects (produced by both an initial correlated distribution within 'genetic' Frenkel pairs and a further annihilation), the correlation of similar defects has been taken into account for the first time. The latter arises indirectly from an annihilation of dissimilar defects. Its contribution to the annealing kinetics has been reported. The present theory is compared with earlier developed theories by Waite and Antonov-Romanovskii.

Many reactions between mobile defects in both solids and liquids may be considered in terms of a diffusion-controlled process. At present there are two main theories for the diffusion-controlled defect annealing $A + B \rightarrow C$, developed by Waite (1957) and Antonov-Romanovskii (AR) (1942, 1966, 1970). In both of them the spatial correlation of dissimilar defects (A–B) is taken into account only, whereas similar defects (A–A, B–B) are assumed to be randomly distributed in a medium. The main difference between these theories is that in the latter one a *single* joint (two-particle) distribution function (DF) is employed, whereas in the former, *two* DFs of dissimilar defects are used. These two DFs are used to distinguish between the initially correlated ('genetic' or proper) pairs created in the same radiation event (e.g. Frenkel defects, i.e. interstitials–vacancies in crystals) and uncorrelated defects from different pairs. On the other hand, the DF used in the AR theory is a sum of these DFs.

A relation between these two theories was established by Kalnin and Pirogov (1977). They have shown these theories are equivalent if an initial, genetic correlation is negligible.

Quite recently we have developed a more complete (but restricted by the joint DFs) system of kinetic equations describing both defect accumulation under irradiation and further annealing taking also into account defect diffusion (Kuzovkov and Kotomin 1980a, b).

Without going into their derivation, let us discuss here the corresponding annealing kinetics. Two kinds of DF are employed: (i) the one-particle DF is the probability of finding that an elementary cell of a crystal (equivalent to a volume per lattice site) contains v kinds of defects ($v = 1$ for vacancy and 2 for interstitial†), (ii) the three independent joint DFs: Y, X_1, X_2. Here $y(|r - r'|)$ is the probability density of finding two dissimilar

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† For correctness we restrict ourselves to the case of Frenkel defects in ordered crystals. A lattice constant is used hereafter as a length unit.

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defects in unit volumes at r, r', whereas the X_v describe a relative distribution of *similar* defects.

So the kinetic equations read

$$\partial c / \partial t = -4\pi R_0^2 D y'(R_0) \equiv -\phi \tag{1}$$

$$\frac{\partial X_v}{\partial t} = 2D_v \hat{L} X_v - \frac{\phi X_v}{r R_0 c^3} \int_{|r - R_0|}^{r + R_0} y(\rho)\rho \, d\rho \tag{2}$$

$$\frac{\partial y}{\partial t} = D\hat{L}y - \frac{\phi y}{r R_0 c^3} \int_{r - R_0}^{r + R_0} X(\rho)\rho \, d\rho \qquad r \geqslant R_0 \tag{3}$$

where $\hat{L} = r^{-2}(\partial/\partial r)(r^2 \partial/\partial r)$ is the diffusion operator, $X = \frac{1}{2}(X_1 + X_2)$, and the D_v are defect diffusion coefficients ($D = D_1 + D_2$). When deriving equations (1) to (3), the usual Kirkwood superposition approximation for the three-particle DFS is used, along with the limiting case of an instant annihilation of defects separated by a distance less that the clear-cut radius R_0. This limit leads to the well known Smoluchowski' boundary condition in equation (3) $y(r \leqslant R_0) \equiv 0$. The equation for a *finite* annihilation time is more complicated (Kuzovkov and Kotomin 1980a, b).

It should be noted that omitting equation (2) and assuming $X_v \equiv c^2$ in equation (3) (i.e. a random distribution of similar defects at *any* moment), one obtains for the y the AR equations. Liebfried (1965), as well as Kalnin and Pirogov (1977), have shown that such equations fail to describe correctly an annealing of initially strongly correlated pairs of defects in the case of low defect density (see also Shroeder 1973), which in fact has a monomolecular character. We shall discuss shortcomings of this theory below, but let us first consider a test problem simulating the monomolecular annealing of pairs just referred to. Let us separate a whole volume by unit cubic cells with a length L and put defects of the first kind (e.g. vacancies) in their centres. The defects of the other kind are placed at a distance r from a centre with a probability density $f(r)$ of finding a correlated pair. Thus we have produced correlated pairs in unit cells having volumes L^3. An initial defect concentration is $c_0 = L^{-3}$, and in the limiting case $L \to \infty$ (i.e. $c_0 \to 0$) the mixing between different pairs is negligible, which leads to monomolecular annealing kinetics within genetic pairs. It is easy to find that here $y(r) = c_0 f(r)$ for $r < L$, whereas one can expect oscillations with an asymptotic c_0^2 at large r due to uncorrelated pairs. A strong correlation of dissimilar defects obviously leads also to a strong correlation for *similar* defects: $X_v = 0$ (if $r < L$) and X_v tend towards an asymptotic c_0^2 at large r. For the limiting case $L \to \infty$ we introduce the new variables $s = c/c_0$(survival probability for a pair) and $\bar{y} = \lim_{c_0 \to 0} y/c_0$, with the initial condition $\bar{y}(t = 0) = f(r)$. In this limiting case $X_v/c_0^2 = 0$. Thus equations (1) and (3) yield the well known relations for a monomolecular annealling

$$\partial s / \partial t = -4\pi R_0^2 D \bar{y}'(R_0) \tag{4}$$

$$\partial \bar{y}/\partial t = D\hat{L}\bar{y} \qquad r \geqslant R_0. \tag{5}$$

It may be concluded from what has been said that a strong initial correlation within pairs of dissimilar defects leads to a correlation of similar defects, which contradicts the approximation $X_v = c^2$ employed in the theories by both Waite (1957) and Antonov-Romanovskii (1942)†.

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† The same conclusion was recently drawn for the kinetics of localised defect accumulation (Kuzovkov and Kotomin 1980a, b).

Let us now change the variables:

$$r' = r/R_0, \qquad t' = Dt/R_0^2, \qquad s = c/c_0, \qquad y_0 = y/c^2, \qquad X_{0v} = X_v/c^2.$$

In the new variables (the primes are omitted below) $\lim_{r \to \infty} y_0, X_{0v} = 1$. Equations (1) to (3) now take the form

$$\partial s^{-1}/\partial t = (1/\tau)y'(1) \equiv \phi_0 \qquad \tau^{-1} = 4\pi R_0^3 c_0 \tag{6}$$

$$\frac{\partial X_{0v}}{\partial t} = \frac{2Dv}{D} \hat{L} X_{0v} - s\phi_0 \frac{X_{0v}}{r} \int_{r-1}^{r+1} (y_0(\rho) - 1)\rho \, d\rho \tag{7}$$

$$\frac{\partial y}{\partial t} = \hat{L}y - s\phi_0 y_0 r^{-1} \int_{r-1}^{r+1} (X_0(\rho) - 1)\rho \, d\rho \qquad (r \geqslant 1). \tag{8}$$

Consider now the initial state with a random distribution of similar defects but a strongly correlated distribution within genetic pairs, i.e.

$$X_{0v} = 1, \qquad y_0 = 1 + \tau f(r).$$

An analysis of the set of non-linear integro–differential equations (6) to (8) shows *three* distinctive time intervals (Kuzovkov and Kotomin 1980(b): (i) $t_1 \simeq \tau^{-1}$ for an appearance of a spatial correlation between similar defects; (ii) t_2 (independent of τ) for a mono-molecular annealing; and (iii) $t_3 \simeq \tau$ for a bimolecular annealing of defects from different pairs. The value $\tau \gg 1$ (for the validity of a joint DFS description level, which is usually the case) and hence for a small initial concentration

$$\tau^{-1} \sim c_0 \to 0.$$

Therefore a correct description of annealing kinetics necessitates a *complete* set of equations (1) to (3) or (6) to (8). As has been shown above, these equations—contrary to those in AR's theory—are correctly reduced to monomolecular kinetics in the limiting case of a small concentration and strong initial correlation within pairs. Therefore the shortcoming of the AR theory is the neglect of the correlation or *similar* defects. It should be stressed that, even in the case of no initial correlation within genetic pairs, the mentioned correlation of similar defects *arises* with time in the course of defect annealing (Kuzovkov and Kotomin 1980b).

In addition to the shortcoming similar to that of AR, the theory by Waite has a more fundamental shortcoming. It claims to describe annealing kinetics of defects affected by their initial spatial correlation. In order to see that this theory fails to de-scribe the kinetics sought for, let us consider the following simple test problem.

Complete information about defect distribution may be given by the coordinates of all the defects. In doing so let us choose a model of a regular defect distribution similar to an NaCl lattice. Then the question arises: can Waite's theory for such cases formulate uniquely the initial conditions for kinetic equations describing an annealing? The answer is no. In our theory and in AR's theory the knowledge of initial defect distribution is quite enough to calculate the DFS c, y and X (through statistical development of given information about defects). Moreover, such information is *not* necessary because the one- and two-particle DFS employed in these theories contain only a part of the complete information on the defect distribution and hence the shortcomings. In other words, a correct probabilistic way of describing the kinetics assumes a one-to-one correspondence between a given defect distribution and the DFS. On the other hand, even knowledge of all the information on defect distribution (coordinates) is not enough to obtain the

initial conditions in Waite's theory. Waite separates the DF for dissimilar defects into two parts: $y = y_c + y_u$, i.e. describing the initially correlated and randomly distributed defects. Each of y_c, y_u separately cannot be regarded as y because they contain additional (excess and subjective) information about the system's evolution with time ('prehistory').

Well known elementary events in a system (defect annihilation, diffusion) are stochastic Markovian processes. For a Markovian process (in the present case, for an annealing) the probability of a transfer between initial and final states (i.e. between different defect distributions) is fully determined by these states only, and is independent of how the final state has been produced (i.e. independent of the system's prehistory). Waite's approach disregards this condition because it requires additional information on correlated defects. Such information can be obtained only by detailed supervision of how the system develops in time. The separation of the y into two parts by itself would not lead to mistakes if the solution of Waite's equations were invariant with respect to the y separation, required for determining its initial conditions. However, such an invariance does *not* take place. Both AR's and Waite's theories have a similar two-particle level of describing the defect distribution. By summing equations for the DFs y_c and y_u in Waite's theory and comparing the results with those of the equation for y in AR's theory, one can find the terms which break down the invariance referred to. The appearance of these terms is due to the pseudo-probabilistic way of how Waite derives the equations, in which the DFs are not determined *uniquely* by a given defect distribution just because the number of equations (three) exceeds that of the independent DFs (c and y).

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