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Theoretical investigation of microstructure evolution and deformation of zirconium under neutron irradiation

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ABSTRACT

The radiation growth of zirconium is studied using a reaction–diffusion model which takes into account intra-cascade clustering of self-interstitial atoms and one-dimensional diffusion of interstitial clusters. The observed dose dependence of strain rates is accounted for by accumulation of sessile dislocation loops during irradiation. The computational model developed and fitted to available experimental data is applied to study deformation of Zr single crystals under irradiation up to hundred dpa. The effect of cold work and the reasons for negative prismatic strains and co-existence of vacancy and interstitial loops are elucidated.

1. Introduction

The radiation growth (RG) of Zr-based materials with the hexagonal close-packed (hcp) crystal lattices is one of the damaging mechanisms which affect safe and economical operation of commercial nuclear reactors.

Experiments have demonstrated that deformation of these materials at temperatures below ~300°C is driven by nucleation and growth of dislocation loops on both the prismatic (a) and basal (c) planes. In annealed Zr crystals, the RG is characterized by expansion along a axes and contraction along c axis. Radiation growth typically consists of three distinct stages [1]. Stage I exhibits a high strain rate and lasts for ~0.1–1.0 displacements per atom (dpa). Stage II demonstrates a very low strain rate, often interpreted as strain saturation, and proceeds up to ~3 dpa. At higher doses, during stage III, usually referred to as the *breakaway growth* stage, the strain rates increase with increasing dose and reach values as high as $\sim 10^{-3}$ dpa^{-1}. The dose dependence of these rates is debated. Transmission electron microscopy exanimation of irradiated samples revealed formation of interstitial-type prismatic loops with $(1/3)\langle 11\bar{2}0\rangle$ Burgers vectors during stages I and II, and vacancy-type c loops during stage III. No basal interstitial-type loops are ever observed.

In cold-worked materials the strain rates are relatively high from the very beginning and no strain saturation occurs [2]. In some cases, both a and c strains have been found to be negative. Moreover, vacancy- and interstitial-type prismatic loops of similar densities and sizes may co-exist, which is another intriguing feature of the RG phenomenon. This co-existence violates the well-known loop property: vacancy- and interstitial-type loops of large enough size have almost the same efficiencies for absorption of point defects, hence, cannot grow at the same time. This is the reason why their co-existence is never observed in cubic crystals. To our knowledge, these two observations: the negative a strains and co-existence of the vacancy- and interstitial-type prismatic loops have never been explained in a self-consistent way by any model.

Several theoretical models of RG have been published since the first model by Buckley [3]; these models have been reviewed by Holt [4]. All of them, with one exception discussed below, are based on a simplified implementation of mean-field reaction rate theory in which the primary damage is in the form of Frenkel pairs, i.e. single vacancies and self-interstitial atoms (SIAs), both migrating three-dimensionally (3-D). Such an approach is not consistent with current knowledge of the primary damage generated in atomic displacement cascades, where significant defect clustering occurs. In our view, this is the reason why several important observations remained unexplained. For example, the conventional concept of dislocation bias employed by these models suggests that the strains must have opposite signs to those generally observed, i.e. positive/expansion in basal c and negative/contraction in prismatic a directions. This is because the Burgers vector of c dislocations is larger than that of a dislocations, which creates stronger interaction of c dislocations rather than a dislocations with SIAs.

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A significant step in understanding RG was made by Woo and Gösele [5,6] by introducing anisotropic diffusion of single SIAs. In the diffusion anisotropy difference (DAD) model [6] it is assumed that the vacancy diffusion is isotropic, whereas the SIAs migrate preferentially along the basal planes. This provided an explanation of the contraction of *c* axes and the crucial role of *c* loops in the breakaway growth of annealed crystals.

Nevertheless, we argue that the DAD model does not correctly describe RG in neutron-irradiated materials because of the assumption that the primary damage consists of only Frenkel pairs. Both experiments [7] and molecular dynamics (MD) simulations [8,9] show that a large fraction, ~20–50%, of the point defects produced in displacement cascades are found in small clusters. The SIA clusters are highly mobile and migrate one-dimensionally (1-D) along close-packed directions in all crystals including Zr [8,10]. This leads to a mixture of the second-order (for the point defects) and the third-order (for the SIA clusters) reaction kinetics [11], rather than just second order, as in the DAD model. The third-order reaction kinetics of SIA clusters with dislocations arises because the reaction rate is proportional to the product of the cluster concentration and the dislocation density squared. This contrasts with the second-order reaction kinetics between point defects and dislocations, which is proportional to the point defect concentration and dislocation density. In the calculations presented in [6], the DAD bias factor for SIAs was required to be equal to 200% in order to fit the experimental data. This implies a very high anisotropy of single SIA diffusion, with the ratio of diffusion coefficients parallel and perpendicular to the basal planes $D_a/D_c \approx 10^2$. Such a high ratio is not supported by a study using a combination of Monte Carlo and *ab initio* calculations in [12], where it was estimated to be between three and four at temperatures in the range from 300 K to 1000 K. Moreover, the anisotropy was found to be higher for vacancies than for SIAs at temperatures below 900 K, so that the basic assumptions of the DAD model seem to be invalid.

Holt *et al.* [13] made an attempt to generalize the DAD model by accounting for the cascade-produced SIA clusters, but assumed the clusters to be immobile, which contradicts the MD simulation results mentioned above. One common failure of all these models is that they do not explain the co-existence of vacancy and SIA loops which is observed in irradiated samples.

Recently we have proposed a reaction–diffusion model of RG [14,15] which is based on the Production Bias Model (PBM) [16–19]. The PBM represents a significant step in the development of theory of void swelling in materials with cubic lattices. Its predictions are consistent with a broad range of experimental results, and account for such observations as enhanced swelling near grain boundaries, and void-lattice formation, which could not be explained by earlier models. This success is due to inclusion of the cascade production and 1-D migration of SIA clusters into the theory.

The displacement cascades in hcp Zr are similar to those in cubic crystals; hence the PBM should provide a realistic framework for the behavior of hcp metals. For example, the observations of basal-plane alignment of vacancy loops at low temperature [20,21] and voids at high temperature [22] in zirconium alloys are analogous to void ordering in cubic metals. Such a similarity gives additional support to the idea that, with certain modifications accounting for the hcp lattice structure, the PBM will be capable of describing RG.

The PBM model [14,15] reproduces all the RG stages observed, including the break-away growth in pre-annealed samples at high irradiation doses. In addition, it accounts for such striking observations as negative strains in prismatic directions and the co-existence of vacancy- and interstitial-type prismatic loops, both of which are unexplainable by any model based on the assumption that Frenkel pairs are the only form of initial damage created by incident particles [15]. It follows from the model that accumulation of vacancy- and interstitial-type dislocation loops with increasing irradiation dose changes the relative fractions of dislocations with different Burgers vectors, which leads to the dose dependence of the radiation growth rates. There is no analytical description of this process, and the development of a computational model is the main aim of the present work.

This paper is organized as follows. In Section 2, the model is described. The model was implemented in a computer code named RIMD-ZR.V1 (Radiation Induced Microstructure and Deformation of Zr, Version 1). In Section 3, selected results obtained using the RIMD-ZR code are presented. A summary is given in Section 4.

2. The model

2.1. Model formulation and assumptions

The model was implemented using the following assumptions:

1. Initial microstructure consists of *a*- and *c*-type edge dislocations; densities of dislocations with the Burgers vectors along different $\mathbf{a}_1$, $\mathbf{a}_2$ and $\mathbf{a}_3$ prismatic directions and lying in the basal ($\mathbf{c}$) plane, ρ_j ($j = \mathbf{a}_1, \mathbf{a}_2, \mathbf{a}_3, \mathbf{c}$), may be unequal.
2. The primary damage produced in displacement cascades consists of mobile point defects and SIA clusters with the Burgers vectors along $\langle 11\bar{2}0\rangle$ prismatic directions.
3. The point defects execute 3-D random walk on the lattice.
4. The SIA clusters migrate 1-D along their Burgers vectors, i.e. one of the $\langle 11\bar{2}0\rangle$ directions, parallel to the basal planes.
5. The basal interstitial-type loops are not formed, which is in agreement with experiments.
6. The SIA clusters interact with dislocations of the same Burgers vector only, while the much weaker interaction with other dislocations is ignored. An analysis supporting this assumption is discussed in detail elsewhere [14,15].
7. The dislocation bias factor for point defects, mutual recombination of point defects, and thermal vacancies are ignored.

The population of edge dislocations and dislocation loops, which evolve during irradiation, is characterized by the total dislocation length per unit volume for each Burgers vector ρ_j ($j = \mathbf{a}_1, \mathbf{a}_2, \mathbf{a}_3, \mathbf{c}$), thus neglecting the difference in the absorption properties of loops and dislocations with both point defects and SIA clusters:

$$\rho_j = \rho_d^j + 2\pi r_v^j N_v^j + 2\pi r_i^j N_i^j, \tag{1}$$

where ρ_d^j is the edge dislocation density, and $r_{v,i}^j$ and $N_{v,i}^j$ are the mean radius and number density of vacancy (subscript v) and interstitial (subscript i) type loops of j orientation of the Burgers vector.

2.2. Concentrations of mobile defects

The system evolves due to reactions involving mobile defects, which cause dislocation climb, and sessile loops grow or shrink. The concentrations, C, of mobile defects: single vacancies (subscript v), single SIAs (i) and SIA clusters (cl) are obtained from the steady-state balance equations ($m = \mathbf{a}_1, \mathbf{a}_2, \mathbf{a}_3$):

$$\dot{C}_v = G - \rho D_v C_v = 0, \tag{2}$$

$$\dot{C}_i = G(1 - \varepsilon_i^g) - \rho D_i C_i = 0, \tag{3}$$

$$\dot{C}_{cl}^m = G\frac{\varepsilon_i^g}{3n_i^g} - k_m^2 D_{cl} C_{cl}^m = 0, \tag{4}$$