

# Kinetically Driven Point-Defect Clustering in Irradiated MgO by Molecular-Dynamics Simulation

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An approach for performing molecular-dynamics simulations of radiation damage by inserting Frenkel pairs directly into the crystal is presented. By by-passing the ballistic phase of the radiation cascade of traditional radiation-damage molecular-dynamics simulation, this approach focuses entirely on defect evolution and not on defect creation. In addition to replicating the nature of interstitial clustering observed from the conventional collision-cascade radiation-damage simulations, the simulations reveal an intricate cluster-formation mechanism that involves not only the originally introduced Frenkel defects but also formation of new Frenkel pairs that stabilize a defect lattice.

Atomic-level simulation has long provided important insights into the fundamental processes associated with radiation damage. In particular, simulation has revealed that radiation damage usually involves two distinct phases. First, the “ballistic phase” involves the formation of the collision cascade arising from the high-energy “primary knock-on atom” (PKA). This phase is typically only a few picoseconds long, its net effect being the generation of Frenkel pairs (FPs) and small point-defect clusters.<sup>1</sup> Second, the “kinetic phase” captures the diffusion-controlled dynamical evolution of these vacancies and interstitials, their annihilation, and the formation of defect clusters. Thus it is this kinetic phase that determines the long-time, experimentally accessible behavior of the material: while the recombination events promote radiation tolerance, the cluster-formation process produces the irreversible damage that degrades materials performance and ultimately limits lifetime. Previous simulations of collision cascades followed by rather long temperature-accelerated dynamics (TAD) simulations<sup>2,3</sup> have shown that the defect evolution during the kinetic phase is largely independent of the detailed nature of the initial damage created during the ballistic phase, suggesting that the role of the cascade simulation is mostly to introduce nonequilibrium point-defects into the system.

The standard PKA simulations introduce only a rather small number of point defects into the system. Thus a key question is whether they are able produce the wide

range of defect environments that are present in experiment. Here we describe an approach that, by circumventing the ballistic-phase simulation entirely, allows a much wider range of different defect environments to be explored. In this approach, the initial damage is introduced by randomly distributing a specific (variable, PKA-energy dependent) concentration of stable Frenkel pairs into the lattice and subsequently following their diffusion-controlled kinetic evolution at elevated temperature. When applied to MgO as a model material, this approach replicates the defect clusters seen in the full cascade simulation followed by the TAD simulation.<sup>2,3</sup> More importantly, it also exposes an intricate cluster-formation mechanism that involves not only the original, radiation-induced Frenkel pairs but also formation of new “structural” FPs as a lattice response that stabilizes the larger clusters. The new approach also has the advantage of being a simpler, computationally less cumbersome and physically more transparent way of introducing the initial ‘collisional’ damage into the material.

We choose MgO as the model material in which to demonstrate this approach, as the point-defect evolution due to the collision-cascade PKA simulation has already been reported by Uberuaga *et al.*<sup>2,3</sup> Their work showed that in relatively low PKA-energy (0.4-5.0 keV) cascade simulations, most of the FPs formed in the ballistic phase recombine during the kinetic phase; those that do not recombine kinetically evolve to form charged, highly stable clusters containing a maximum of seven ions. Particularly noteworthy is the fact that these clusters are not formed during the ballistic phase but as a consequence of interstitial diffusion during the kinetic phase during which the defects diffused long distances and were not confined to the spatial region affected by the initial ballistic cascade.

We have performed MD simulations in a single crystal consisting of 20x20x20 cubic rocksalt crystal structure unit cells containing 64,000 ions. A classical Lewis and Catlow<sup>4</sup> Buckingham-type potential is chosen to describe interionic short-range interactions. The long-range Columbic interactions are calculated by using Wolf’s 1/r-summation method<sup>5</sup> with a spherical truncation at the cut-off radius of 8.139 Å. The time step of 0.5 fs ensures good energy conservation in the tests in the NPE ensemble. All the simulations are performed at 1000K. The FPs are created randomly and every vacancy and interstitial is initially separated by at least 5 lattice parameters to minimize the uninteresting rapid recombinations. However, no constraint is put on interstitial-interstitial or vacancy-vacancy distance.

To elucidate the defect evolution and clustering mechanisms, we have performed two different types of simulations, differentiated by the FP defects present on the either or both sublattices. The first type involves FPs on the Mg and O sublattices separately; in the second type, FPs are created simultaneously on both sublattices.

Fig. 1 shows the time evolution of a system into which initially 100 FPs were inserted into only the Mg sublattice at  $T = 1000$  K. At  $t = 0$  ps and  $t = 2450$  ps, the number of Mg FPs are 100 and 18 respectively. As we can see in Fig. 1b, no clustering of the defects takes place. Similarly, all the FPs when created only on the O sublattice recombine without forming clusters. In a collision-cascade simulation it is, of course, not possible to selectively create defects on only one of the sublattices; this demonstrates a unique aspect of our approach in understanding the defect evolution. These simulations provide a baseline against which to compare the results of simulations of FPs on both sublattices.

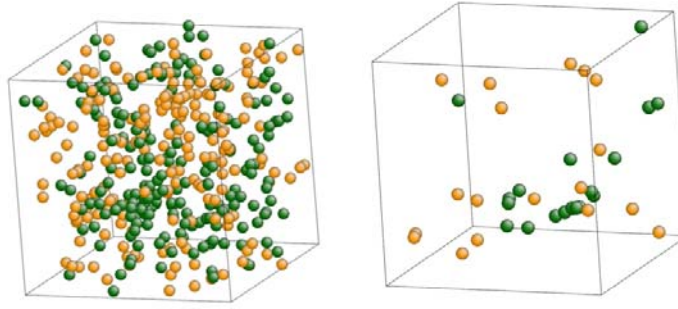


Fig. 1. Evolution of the 100 initially-introduced Frenkel pairs in only the Mg sublattice at  $T = 1000$  K. Color scheme used in all figures - dark green spheres represent Mg interstitials and orange spheres represent Mg vacancies. Shown are two snapshots at (a)  $t = 0$  ps (containing 100 FPs), and (b)  $t = 2450$  ps (18 FPs). Due to the absence of O point defects (see also Fig. 2) no Mg interstitial clustering occurs during the annihilation by interstitial-vacancy recombination on the Mg sublattice. (*Lattice atoms are not shown.*)

Next, we create FPs on both sublattices simultaneously. Figure 2 shows snapshots taken at (a)  $t = 0$  ps and (b)  $t = 2450$  ps for a system containing initially 100 FPs on each of the two sublattices. Compared to the case of Mg or O FPs only, this annealing simulation reveals much less interstitial-vacancy recombination but, instead, significant interstitial aggregation into clusters. This aggregation starts quickly by the formation of highly mobile di- and tri-interstitials. Identical di- and tri-interstitial clusters were observed in the lower-temperature, combined collision-cascade and TAD simulations by Uberuaga *et. al.*<sup>2,3</sup>

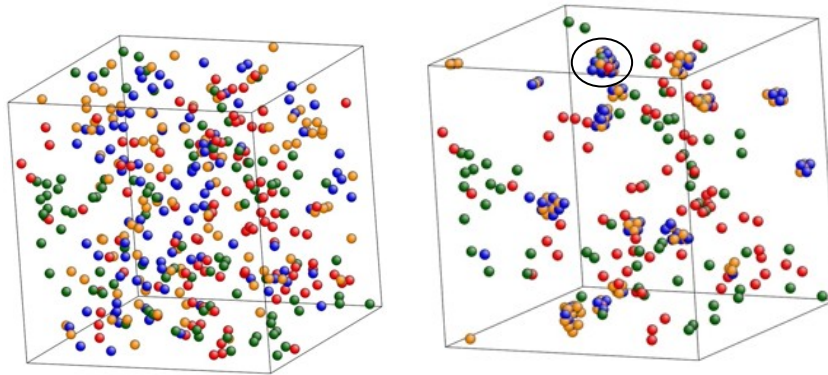


Fig. 2. Snapshots taken at (a)  $t=0$  and (b)  $t=2450$  ps showing the evolution at  $T = 1000$  K of 100 FPs initially introduced into each of the two sublattices. Of the initially 100 Mg and 100 O FPs in (a), 72 Mg and 76 O FPs are still present in (b). Dark green and orange spheres represent Mg interstitials and vacancies; red and blue spheres represent O interstitials and vacancies. The circled cluster in (b) is analyzed in Fig. 3.

The comparison between the two simulations (Figs. 1 and 2) demonstrates that, when the FPs are present on only one sublattice, all the vacancies and interstitials eventually recombine due to the absence of clustering; hence, after 2450 ps only ~20% of the initial FPs remain (see Fig. 1). By contrast, in the system containing FPs on both sublattices, about 80% of the FPs become immobilized by precipitation into highly stable vacancy-interstitial clusters (see Fig. 2).

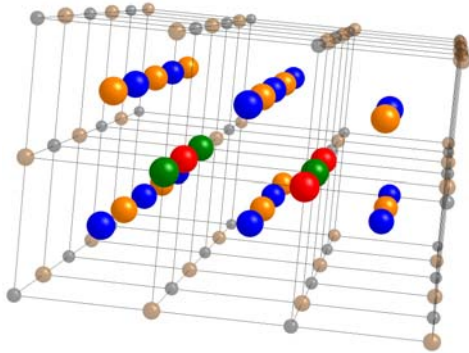


Fig. 3. Closeup of the all-interstitial cluster circled in Fig. 2(b). The cluster forms an interstitial rocksalt lattice that is displaced with respect to the parent lattice (not shown).

A closeup snapshot of one of the larger interstitial clusters, circled in Fig. 2(b), is displayed in Fig. 3. Investigation of the dynamic growth mechanism of this representative cluster reveals *two* distinct, coordinated mechanisms that control its formation and growth.

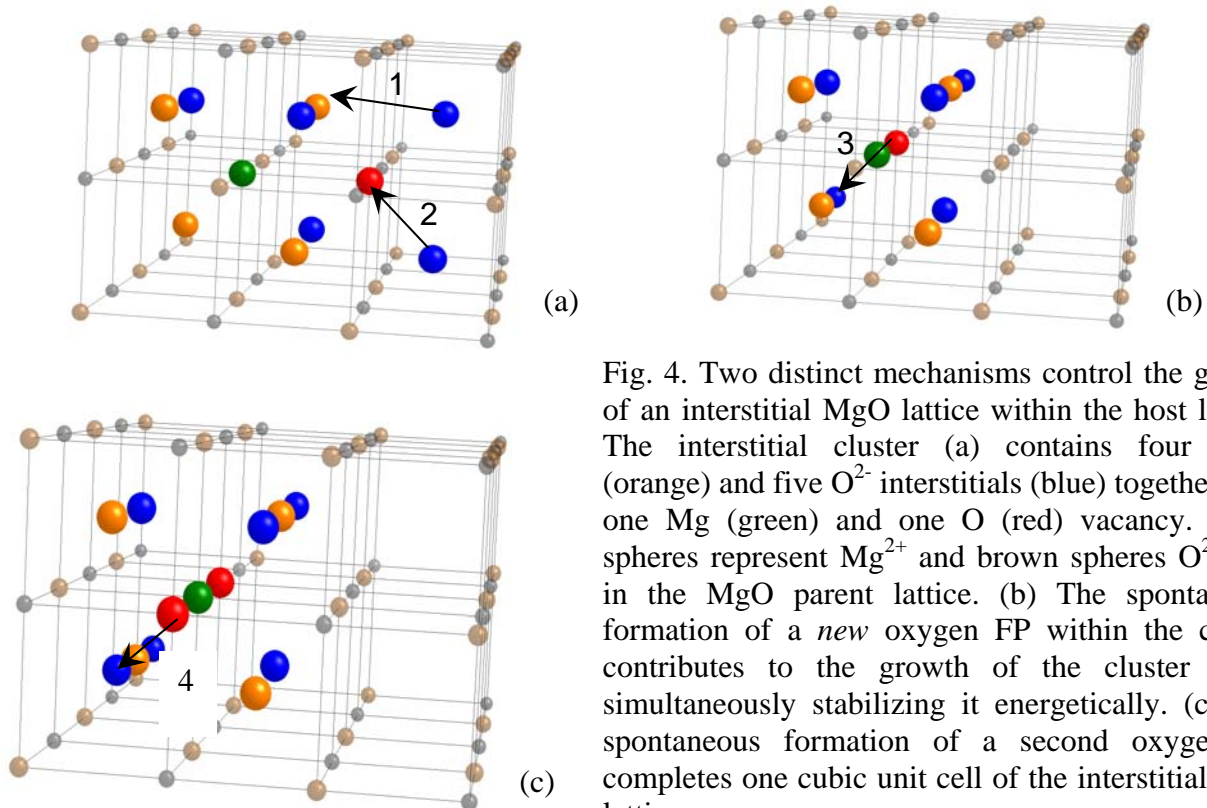


Fig. 4. Two distinct mechanisms control the growth of an interstitial MgO lattice within the host lattice. The interstitial cluster (a) contains four  $\text{Mg}^{2+}$  (orange) and five  $\text{O}^{2-}$  interstitials (blue) together with one Mg (green) and one O (red) vacancy. Black spheres represent  $\text{Mg}^{2+}$  and brown spheres  $\text{O}^{2-}$  ions in the MgO parent lattice. (b) The spontaneous formation of a *new* oxygen FP within the cluster contributes to the growth of the cluster while simultaneously stabilizing it energetically. (c) The spontaneous formation of a second oxygen FP completes one cubic unit cell of the interstitial MgO lattice.

These are: (i) the conventional mechanism involving diffusion-controlled aggregation and annihilation of the originally introduced interstitials, and (ii) the spontaneous formation of *new*, “structural” FPs by the coordinated displacements of lattice atoms within the cluster core.

The coordinated operation of the two mechanisms is illustrated in Fig. 4 which captures four sequential hopping events (see arrows) during the growth and stabilization of a cluster containing initially four  $\text{Mg}^{2+}$  and five  $\text{O}^{2-}$  interstitials together with one Mg and one O vacancy (see Fig. 4(a)). Arrow 1 indicates oxygen diffusion to a neighboring interstitial site while arrow 2 captures the annihilation of an oxygen FP by interstitial-vacancy recombination. Both are conventional diffusion events of interstitials already present at the beginning of the kinetic phase. By contrast, Fig. 4(b) reveals a novel mechanism involving the spontaneous creation of a new (not initially present) FP as a regular-lattice O ion hops to an interstitial site (arrow 3), leaving behind a new vacancy within the cluster core; this event is then repeated (Fig. 4(c), arrow 4). Two additional ions already present at the back of the interstitial cube in this figure act as nucleation sites for the completion of the next, adjacent cube. The net outcome of these events is a complete cube of an interstitial MgO crystal that has been formed and that continues to grow, on the displaced, interstitial lattice.

From the viewpoint of simulation methodology, temperature-accelerated dynamics (TAD) simulations<sup>2,3</sup> have largely overcome the issue of MD simulations only reaching out to nanosecond time scales. However, their application to single cascades and hence low defect concentrations limits the complexity of the defect structures that can thus be observed. By injecting a very high density of defects, the method proposed here allows large clusters to develop. Moreover, this high density essentially allows nanosecond simulations to produce defect structures only previously seen in much longer simulations. The initial defect concentrations introduced in this approach are extremely high. It might thus be argued that the dynamically generated complex defect structures seen here may in reality be rather uncommon. However, given that single defects and small clusters are annihilated as the system evolves, it is these large, structurally complex clusters that will survive and constitute the long-term radiation damage in the system.

To conclude, this robust and straightforward approach enables elucidation of the kinetics of radiation-damage development unencumbered by the initial damage due to one specific cascade. By following only the kinetic evolution at elevated temperature, it enables observation of defect aggregation up to significant cluster sizes, and with mechanistic detail not previously possible. In addition, the ability to selectively create defects on only one of the sublattices provides an additional perspective on the nature of radiation-damage development. Since it does not require particularly large system sizes, the approach thus provides a relatively simple and direct route for elucidating the competing roles of point-defect annihilation and aggregation in the development of radiation damage, including host-lattice responses in the process.

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