Defect-induced nucleation and growth of amorphous silicon

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We propose a microscopic model of the amorphization of silicon such as that resulting from ion implantation. We demonstrate that amorphization can be induced by the presence of defects provided they form clusters embedded in a defective crystalline matrix. Our results are in striking agreement with transmission-electron microscopy measurements and confirm the superlinear dependence of damage on deposited energy, supporting the view that the crystal-to-amorphous transition proceeds via nucleation and growth. [S0163-1829(96)02324-7]

Ion implantation is routinely used for doping semiconductors. During irradiation, however, the samples suffer damage to an extent that depends on the energy of the incoming particles as well as other experimental parameters. In extreme situations, damage may be so large that amorphization takes place. Evidently, there is considerable interest in understanding implantation damage in materials, including amorphization.

Amorphization, i.e., the transition of a crystal (c) into amorphous (a) material, is also a problem of fundamental interest which, in the case of silicon, has been the object of intense discussions over the last two decades (see, e.g., Ref. 1). Two competing views of the phenomenon have been proposed: (i) heteregeneous nucleation, whereby amorphization proceeds by the coalescence of the quenched-in amorphous ''collision cascades'' left by the implanted ions, and (ii) homogeneous nucleation, where the amorphous phase *grows* following the accumulation, above a certain threshold, of damage resulting from the passage of ions; this is similar to the well-characterized growth of a-Si at a/c interfaces.[1]

In a recent Letter,[2] Diaz de la Rubia and Gilmer (DG) have shown, using molecular-dynamics (MD) simulations, that energetic (5 keV) Si ions could give rise, via a melt-and-quench process, to the formation of localized amorphous regions, or ''spikes.'' The role played by these spikes in the c-to-a transition was not established by DG, but it can be conjectured that overall amorphization will take place when the local displacement cascades begin to overlap, consistent with the heteregeneous-nucleation model. This, evidently, does not involve the nucleation and subsequent growth of the amorphous phase. In fact, the recrystallization of the cascade was observed upon annealing the sample at high temperature. Nevertheless, DG have shown, through an analysis of stress fields, that the spikes can enhance the damage rate.

A model of amorphization by nucleation and growth was found to explain very well the high-energy Ge implantation data of Campisano and co-workers.[1] In these experiments, it was observed that the rate of production of amorphous material increases in a superlinear way with deposited energy, indicating that amorphization is more effective when the host sample is already damaged. This cannot be explained by heterogeneous nucleation. The nucleation-and-growth character of the c-to-a transition was established clearly by Ruault, Chaumont, and Bernas using *in situ* transmission electron microscopy (TEM).[3] These authors have found that *overall* amorphization does not originate in the core of the cascades but, rather, results from the overlap of disordered regions outside the cores, which they refer to as ''grey zones.'' These grey zones are proposed to be the building blocks of amorphous matter.[3]

In the MD simulations of DG (see also Ref. 4), the sample is brought into a highly nonequilibrium state by concentrating the ''incident'' energy into a small number of particles [''primary knock-on atoms'' (PKA)]. As demonstrated by DG, amorphization in this case results from melting and subsequent fast quenching of the material, and is thus purely kinetic in origin. Perhaps more relevant to the nucleation-and-growth process, a purely static model of point-defect amorphization was proposed by Colombo and Maric:[5] In a perfect crystal, interstitials were inserted at random positions until amorphization took place. It is, however, not clear how this model relates to real ion-beam amorphization. In particular, the density of defects is so large ($\sim$30%) that one can argue, on the basis of percolation arguments alone, that the system has no other choice but to disorder. Also, interstitials do not leave vacancies behind, precluding the possibility of defect recombination. A similar model, where amorphization was induced by the insertion of divacancy–di-interstitial pairs, was proposed by Motooka.[6]

In this paper, we report the results of MD simulations that provide a unifying view of defect-induced amorphization of silicon. More precisely, we demonstrate the following: (i) Amorphization *cannot* be induced by the accumulation of point defects if these are distributed uniformly. (ii) Localized regions of defects, i.e., clusters, which can be assimilated to the implantation cascades, lead to the (homogeneous) nucleation and growth of the amorphous phase, at thermal energies, provided that the embedding crystal contains a minimal amount of defects, corresponding to the grey zones observed in TEM experiments. (iii) Conversely, nucleation of the amorphous phase will *not* proceed if the cluster is embedded in a crystalline matrix that is free of defects; rather, recrystallization is observed at high enough temperature. This situation corresponds to a subcritical concentration of amorphous spikes, below the threshold for overlap and overall amorphization. (iv) Amorphization does not take place for clusters with energies below a certain threshold, determined roughly by the difference in energy between the crystal and the amorphous phase; our study, therefore, reconciles the

view that amorphization can be induced by defects with the existence of a threshold energy for amorphization.[4,7]

In the model described below, the density of defects is not so large that the system loses memory of its crystalline nature under static conditions. However, and this is central to our approach, the number of defects is such that the system (before relaxation) is in a state of energy higher than that of the amorphous phase, i.e., clearly out of equilibrium. Given thermal energy, it will therefore go to the nearest available phase, whether it is metastable or not, and in spite of the fact that it would be more favorable for it to recrystallize (''phase selection rule'').

Because long runs and large systems are necessary for our purposes, we use the empirical Stillinger-Weber potential[8] to describe the interactions between Si atoms. This model has been used with success in the simulation of various states of silicon and reproduces reasonably well the energetics of many point defects.[9] In view of this, we expect the model to deliver the essential physics of amorphization, while the quantitative features can only be examined using more precise, quantum-mechanical, models. The results described below were obtained using supercells containing, in all cases, 4096 atoms. Runs as long as 164 ps were carried out, depending on ''experimental'' conditions. In the following discussion, different types of structures are considered. In all cases, before proceeding with the dynamical finite-temperature runs, the structures were optimized (i.e., their energy minimized) by subjecting them to a steepest-descent search at zero temperature.

As mentioned earlier, it has been conjectured that amorphization of Si could be induced by the accumulation of (uniformly distributed) point defects in the crystalline lattice.[5] This idea was in fact originally proposed, and amorphization observed, by Hsieh and Yip[10] in the context of metallic glasses. The bonding in these materials is very different from that in covalent systems, however, and point defects are not as clearly defined. In fact, it has been demonstrated that vacancies anneal out rapidly in Lennard-Jones glasses,[11] while they are mechanically stable in a-Si at low enough temperature.[12]

In an attempt to verify this conjecture, we have first constructed a number of different models with uniform distributions of defects. The aim is to fabricate a model whose energy lies above that of a-Si. The minimum concentration of defects required to achieve this is simply $n_\Omega=(\epsilon_a-\mu)/\Omega$, where ϵ_a is the energy per atom of the amorphous phase, μ that of the crystal, and Ω is the formation energy of the defects. The appropriate values of μ and ϵ_a are given in Table I. Taking the defects to be vacancy-interstitial pairs, for which $\Omega=9.15$ eV, we obtain $n_\Omega=2.7\%$, quite close in fact to the ''critical'' concentration of atoms displaced for ion-beam amorphization.[13]

The above value of Ω, however, is for the case where the vacancy and the interstitial are at infinite separation. In practice, they interact with one another, and Ω decreases somewhat, which means that the n_Ω required for the energy of the defective crystal to exceed that of a-Si increases. In turn, the probability that recombination will take place also increases, meaning that even more defects are necessary. This makes it necessary to define an exclusion sphere (of radius R_X) around each defect so as to avoid overlap and minimize recombination, which limits the number of defects that can be created. (We remain here below percolation, which, as mentioned above, can cause amorphization).

TABLE I. Energies (in eV per atom) at 0 and 300 K of the fully relaxed samples with uniform distribution of defects, as well as for the reference c-Si and a-Si samples. R_X is the radius of the exclusion sphere (in units of $a=5.43$ Å) and n_Ω is the nominal concentration of defects.

Sample	n_Ω (%)	R_X	E_p (0 K)	E_p (300 K)
c-Si			$-4.334=\mu$	-4.295
a-Si			$-4.088=\epsilon_a$	-4.063
120 VI	2.9	1.8	-4.163	
200 VI	4.9	1.5	-4.094	
300 VI	7.3	1.1	-4.094	-4.129
100 diVI	2.4	1.5	-4.119	
140 diVI	3.4	1.45	-4.119	
195 diVI	4.8	1.10	-4.090	
220 diVI	5.4	0.9	-4.078	-4.073
250 diVI	6.1	0.9	-4.062	-4.067
300 diVI	7.3	—	-4.068	

In Table I we illustrate these ideas by listing the energies of several model structures with different values of n_Ω and R_X. We consider both the case of vacancy-interstitial pairs (VI) and divacancy–di-interstitial pairs (diVI; $\Omega=12.1$ eV). It is clear from this table that it is extremely difficult to construct a model with energy-per-atom values larger than ϵ_a. The energy of the defective crystal rapidly saturates to a value, at zero temperature, of about -4.06 eV, i.e., only slightly above $\epsilon_a=-4.088$ eV. Increasing the density of defects and/or decreasing R_X does not help, as extensive recombination then takes place. Upon increasing the temperature moderately, e.g., to room temperature, some defects anneal out, and the energy in all cases drops below the amorphous level. Close examination of the structures reveals the unequivocal presence of the underlying crystalline lattice: amorphization will not take place. Similar conclusions were reached by Stock *et al.* using the PKA approach,[4] distinct from our total-energy arguments.

It is clear from the above discussion that uniform distributions of defects are too permissive to recombination and do not constitute a proper route for amorphization. Uniform distributions can only result from ''gentle'' treatment of the material, and this may be the reason why electron irradiation fails to amorphize Si.[7] In the case of ion implantation, the initial distribution of defects (immediately following irradiation) is known to be strongly inhomogeneous, or clusterlike. In order to simulate such a situation, we have constructed a number of models containing extended defects; by ''extended defect'' we mean a region in space where point defects have clustered. We have examined both spherical and cylindrical clusters. We discuss only the latter here, which can be regarded as a simple model of a collision cascade.

Samples were generated as follows: We consider a perfect silicon crystal and define a cylindrical region of radius R_C. Atoms (chosen at random) from ideal lattice sites are then moved to tetrahedral interstitial positions (also at random) *within* the cylinder. Atoms displaced in this manner can originate either from the region outside (case I) or inside (case II) the cylinder. We consider examples of both situations here; their (zero-temperature) relaxed configurations are displayed in Figs. 1(a) and 2(a), respectively. (To im-

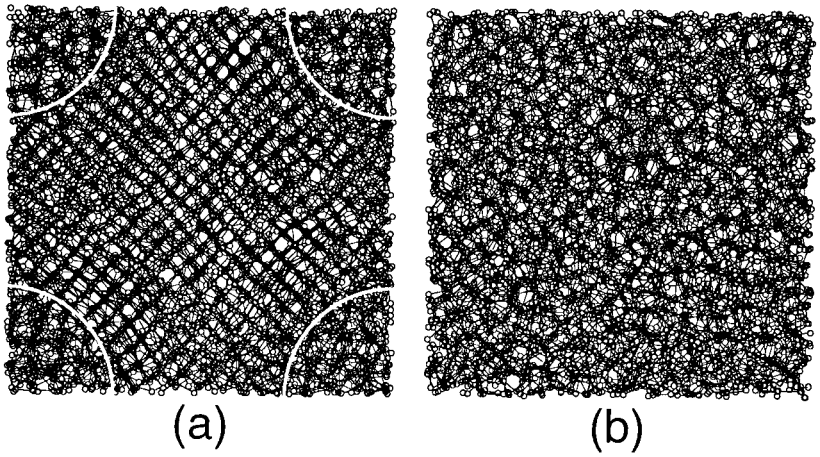

FIG. 1. Initial (a) and final (b) structures of the Cyl-I sample; in (a), the cylinder is marked by white lines.

prove presentation, the cylinders are centered at a corner of the cell, and show up as four quadrants). The region within the cylinders, by virtue of the percolation arguments mentioned above, are amorphous. Clearly, however, the regions outside the cylinders remain crystalline, though evidently, in case I, defects are present and the lattice is distorted. Case II corresponds, loosely speaking, to an amorphous cluster embedded in a perfect crystal. The cluster can be assimilated to the implantation cascades. In case I, the amorphous cluster is embedded in a defective crystalline matrix, corresponding to the grey zones observed with TEM by Ruault, Chaumont, and Bernas;[3] clearly, these are not amorphous.

The parameters and energies of the two models—Cyl-I and Cyl-II—are listed in Table II. The parameters n_Ω and R_C were chosen such that, again here, the energy exceeds that of a-Si. In case I, recombination is difficult (because the interstitials are clustered), and a relatively small cylinder is needed. In case II, in contrast, extensive recombination takes place (within the cylinder), and a much larger concentration of defects is required, leaving only a small crystalline region (but see below). Because their energy is higher than that of the amorphous phase, one would expect both samples to relax into a-Si. We demonstrate next that this is not the case.

We show in Fig. 1(b) the structure of sample Cyl-I after

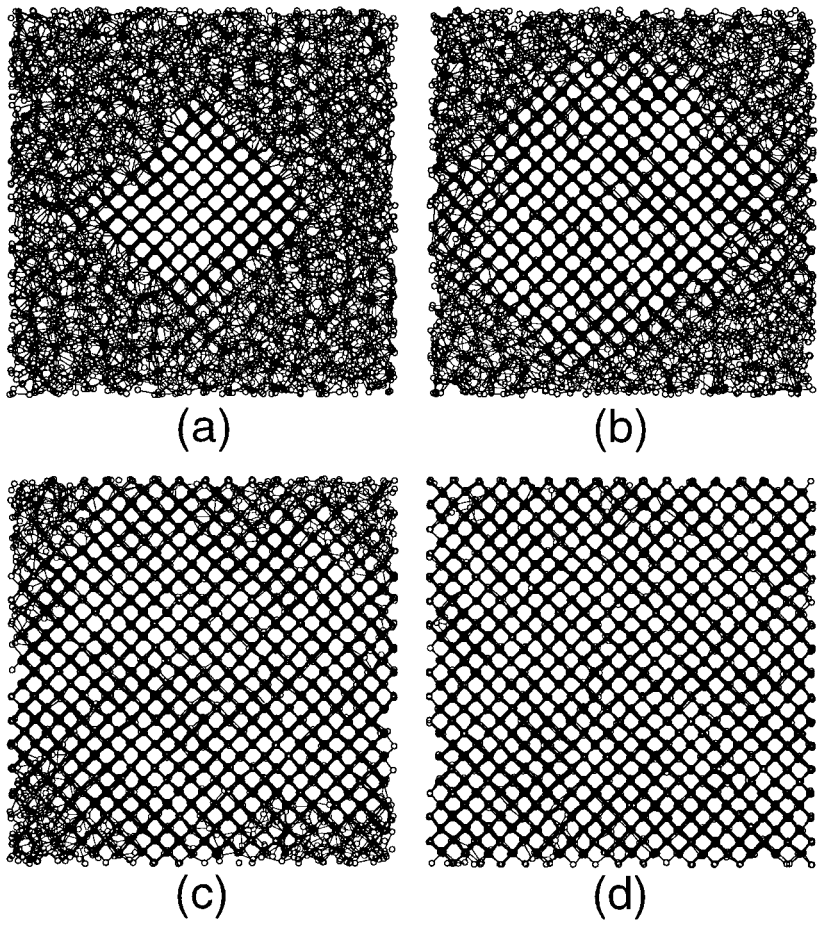

FIG. 2. Initial (a) structure and evolution in time of the Cyl-II sample; (b)–(d) correspond to 84, 124, and 164 ps, respectively.

TABLE II. Energies at 0 and 1200 K, in eV per atom, for the two samples with cylindrical clusters of defects, as well as the reference *c*-Si and *a*-Si samples, after relaxation. The radius of each cylinder, R_C, is given relative to the cubic supercell side; n_Ω is the nominal concentration of defects.

Sample	n_Ω (%)	R_C	E_p (0 K)	E_p (1200 K)
c-Si			−4.334	−4.169
a-Si			−4.088	−3.913
Cyl-I	12.2	0.28	−4.031	−3.905
Cyl-II	36.6	0.50	−4.059	−4.163

relaxing at 1200 K for 36 ps. The system, evidently, has lost complete memory of its original crystalline state. This is in fact confirmed by the data of Table II, as well as by the radial distribution function, displayed in Fig. 3, which overlays exactly that of a reference *a*-Si sample. We have carried out a corresponding simulation at 900 K, and obtained similar results, though complete amorphization was not observed, probably because of time limitation — the process is much slower at this temperature. We therefore conclude that nucleation of *a*-Si can indeed be induced by the accumulation of defects, provided they form clusters, corresponding to the displacement cascades of energetic ions.

We turn now to the badly damaged sample II, which has no point defects in the region exterior to the cylinder. The evolution in time of this sample (over a period of 164 ps), again at a temperature of 1200 K, is shown in Fig. 2. In spite of the extent of the damage in the cylinder (much worse in fact than in sample I), which exhibits initially no trace of crystallinity, Fig. 2 very clearly shows that the system is able to anneal out the damage and recover its crystalline ground state.

The above results are in striking agreement with the observation by Ruault, Chaumont, and Bernas that amorphization nucleates in defective areas of the crystalline matrix (grey zones):[3] when embedded in a perfect crystal, the amorphous cluster chooses to recrystallize rather than to grow. They are also fully consistent with the observation of a superlinear damage rate with dose,[1] and the concomitant conclusion that amorphization is a nucleation-and-growth process: if the crystal surrounding the cluster is free of defects, amorphization can only take place (at sufficiently low temperatures) heterogeneously by the overlap of the displacement cascades. We note that recrystallization of sample II proceeds from the edges inwards (see Fig. 2), confirming that

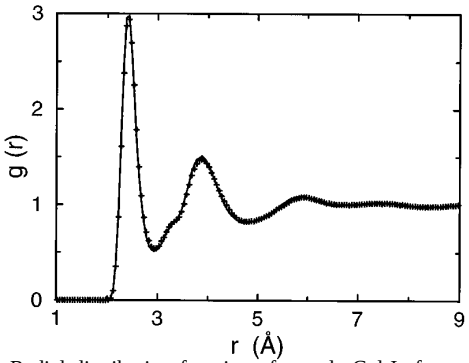

FIG. 3. Radial distribution function of sample Cyl-I after annealing at 1200 K (crosses) and of a reference *a*-Si sample, also at 1200 K (full line).

the processes are growthlike. We have also examined several samples of type I clusters (i.e., embedded in a damaged crystal) with defect concentrations *below* the threshold for amorphization, and found these *not* to induce growth of the amorphous phase (as could be expected), and *not* to recrystallize on time scales comparable to that needed to recrystallize Cyl-II: the nearest available phase, the crystal, is more difficult to reach in this case, that is without the help of a ''seed'' crystal, as in case II.

Our model, we conclude, constitutes a true representation of ''microscopic amorphization'' by nucleation and growth, appropriate to ion-implanted material, distinct from the (kinetic) melt-and-quench process. It establishes unambiguously that amorphization can be induced by defects, provided they belong to clusters embedded in a defective crystalline matrix. This is in remarkable agreement with the observation by *in situ* TEM of the role of grey zones in nucleating the transition,[3] as well as the superlinear dependence of damage with deposited energy.[1] Our results are also consistent with the existence of a threshold energy for amorphization,[4,7] necessary to the creation of extended defects, i.e., collision cascades.

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