Microscopic Description of the Irradiation-Induced Amorphization in Silicon

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We have investigated the atomistic mechanism behind the irradiation-induced amorphization in Si using molecular dynamics simulation techniques. The microscopic description of the process is based on the defect known as *bond defect* or *IV pair*. IV pairs recombine very fast when isolated, but if they interact to each other they survive longer times and thus accumulate giving rise to amorphization. This fact accounts for the superlinear behavior of the accumulated damage with dose and the different activation energies for recrystallization observed in the experiments. The molecular dynamics results have been used to define an atomistic model for amorphization and recrystallization which has been implemented in a kinetic Monte Carlo code. The model is able to reproduce quantitatively the dependence of the critical crystal-amorphous transition on the irradiation parameters.

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Ion implantation is the most common technique to introduce dopants in Si for the fabrication of integrated circuits. The ion irradiation creates a large amount of defects in the lattice, and the balance between its generation and recombination determines the rate of defect accumulation. Amorphization is thought to occur spontaneously when the free energy of the defective lattice is higher than that of the amorphous material. A number of interesting experimental observations have been reported on the crystal-amorphous (c/a) transition, such as the existence of a critical temperature (depending on the ion mass) above which it is not possible to amorphize [1], a superlinear trend in the damage accumulation with dose [2], and the influence of the dose rate at room temperature but not at cryogenic temperatures [3]. Various phenomenological amorphization models have been suggested in an attempt to explain the experimental observations. Under traditional models, amorphization is envisaged to occur through the overlap of isolated damaged regions created by individual ions [4], or via the buildup of simple defects, leading to a sudden collapse of a large region of material into the amorphous phase [5].

In order to model and provide a microscopic understanding of the irradiation-induced c/a transition in Si, it is necessary to identify the lattice defects involved in the process. However, the nature of these defects is still not clear despite the many experimental and theoretical works devoted to this study. Molecular dynamics (MD) calculations show that a single ion can produce a collision cascade with a large variety in the configurations, size, and shape of the damage [6]. This variety makes its characterization very arduous, besides making it difficult to identify the defect or defects responsible for amorphization [7]. From the experimental perspective, the annealing behavior of the damage has been extensively analyzed and tried to be correlated to the annealing behavior of known defects. Si interstitials have been considered to have an influence on the amorphization process, through their interaction with the lattice defects created by the irradiation or by forming interstitial clusters that would act as amorphous embryos [8,9]. Linnros *et al.* observed that the onset for amorphization by irradiation with high energy ions and the movement of a buried c/a interface were both processes with an activation energy of 1.2 eV, which coincides with the dissociation energy of the divacancy [10]. Holland *et al.* also give the divacancy an essential role in controlling the damage accumulation that lead to amorphization [2]. Jackson in turn attributes this energy of 1.2 eV to a dangling bond in the amorphous phase [11]. Goldberg *et al.* determined a series of apparent activation energies ranging from 0.7 to 1.7 eV for different ions [1]. However, it is not likely that different defects control the phase change in each case. It seems more likely that the dominant interactions between the same type of defects may vary with the irradiation parameters.

Another defect that has also been proposed to be responsible for amorphization in Si is the so-called *IV pair*. It consists of a local rearrangement of bonds with no excess or deficit of atoms (see inset of Fig. 1); hence, it is also known as *bond defect*. It introduces in the Si lattice the five- and seven-membered rings typical of the amorphous phase. This defect is present in Si under irradiation, since it can result from a pure ballistic process [12] or by incomplete Si interstitial-vacancy (I-V) recombination [13]. In a previous paper, we demonstrated that the activation energy for IV pair recombination is 0.43 eV, which implies an average lifetime in the order of microseconds at room temperature [7]. This means that the IV pair alone is not stable enough to justify damage accumulation, and thus amorphization, at the typical dose rates. Nevertheless, we proved that, when IV pairs are present in the Si lattice to a given concentration ($\simeq 25\%$), homogeneous amorphization takes place. Recrystallization of such amorphous zones created by IV pair accumulation and those by direct irradiation showed the same features, as far as energy content, internal structure, and recrystallization dynamics are

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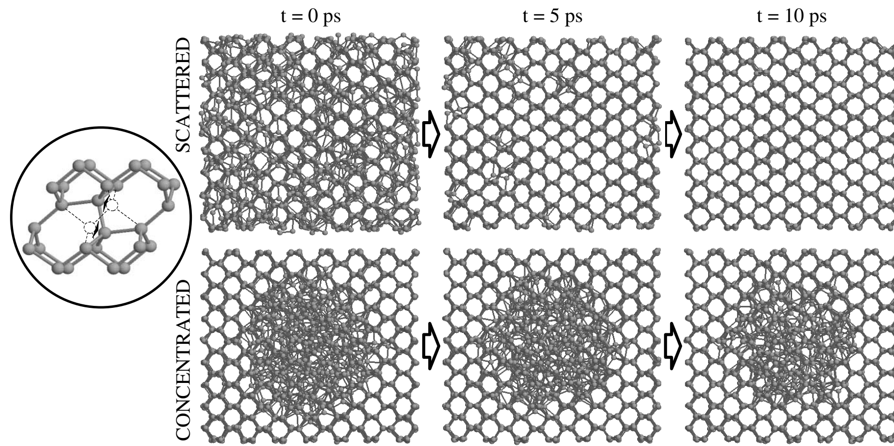

FIG. 1. Snapshots taken during the annealing at 1200 K of samples with the same amount of IV pairs (8%), scattered in one case and concentrated in the other. Each IV pair is introduced in the lattice by randomly choosing two neighboring atoms and displacing them as shown in the inset on the left. The scattered damage has disappeared after 10 ps of annealing, while the concentrated damage has barely shrunk.

concerned. These results indicate that amorphization could be achieved without the intervention of any additional defect, and also that amorphous pocket characterization can be studied by IV pair accumulation. However, how these IV pairs accumulate during irradiation to promote amorphization is a question yet to be answered.

In this Letter, we propose a possible atomistic mechanism for the irradiation-induced c/a transition in Si which uses the IV pair as the building block for the amorphous phase. We study the interactions among this type of defects that lead to more stable structures or amorphous pockets. Those can survive at room or higher temperatures and thus accumulate to induce amorphization. We will also show how this microscopic description can quantitatively explain the features of the c/a transition observed in the experiments.

We analyze the interaction among IV pairs by MD simulation techniques, which have been extensively used to study irradiation effects in Si [6,14]. We have chosen the Tersoff 3 potential to describe the Si atom interactions [15], since it gives a description of the IV pair properties in excellent agreement with tight binding and first principles results [7,13,16]. In our MD simulations, we have used a system consisting of 2268 Si atoms in a computational cell whose dimensions were $7a \times 4.5\sqrt{2}a \times 4.5\sqrt{2}a$, a being the basic unit cell length (5.43 Å). The cell was bounded by two (100) planes in the X direction and by four (110) planes in Y and Z directions. To minimize finite-size effects, we used periodic boundary conditions along the three axes. All simulations were performed at constant temperature by rescaling atom velocities every 1000 steps. We have carried out MD simulations at 1200, 1300, 1400, 1500, and 1600 K, always below the melting temperature predicted by the T3 potential [7]. A concentration of 8% of IV pairs (16% of the atoms displaced from their equilibrium lattice positions) were introduced following the scheme indicated in the inset of Fig. 1. In one set of simulations, IV pairs were distributed randomly in the MD cell, separated from each other by a distance of at least 4 Å. In another set of simulations, IV pairs were arranged in a sphere located in the center of the cell, with a radius of 12 Å. The former case can be assimilated to damage induced by electron or light ion irradiation, and the latter with amorphous pockets produced by heavy ion irradiation [6,7].

Figure 1 shows several snapshots taken during the annealing of the two types of samples at 1200 K. After a simulated time of 10 ps, the scattered damage has disappeared completely, while the concentrated damage still remains. Even though the amount of IV pairs was the same, the dynamics of the recrystallization process is different. This result indicates that, when IV pairs are close to each other, they interact strongly forming more extended defect clusters whose lifetime is higher. The rate of IV pair recombination then depends on the local concentration of surrounding IV pairs.

Atoms in the defective (amorphous) zones have a higher energy content than atoms in the crystal [7]. When crystallization takes place, the excess energy is freed in the form of latent heat. Our MD simulations are carried out at constant temperature, therefore total energy decreases in time as crystallization proceeds. Knowing the energy content difference of the perfect crystal and the defective zones at each temperature [7], it is possible to extract the crystallization velocities from the slope of the potential energy curves. The recrystallization velocities are represented in the Arrhenius plot of Fig. 2 for both sets of simulations, along with the corresponding to a planar Si(100) c/a interface. The calculated activation energy for the scattered damage is 0.44 eV, which is very close to that corresponding to isolated IV

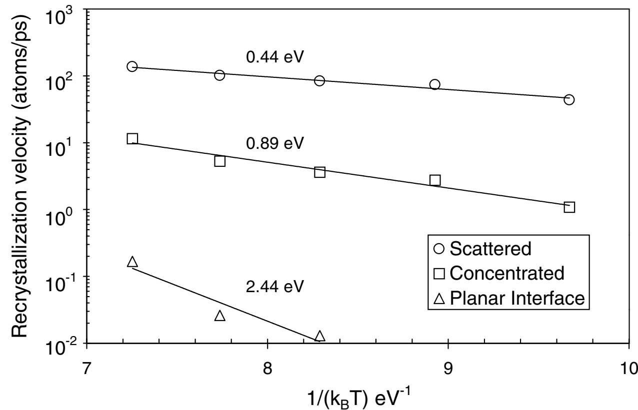

FIG. 2. Arrhenius plot of the recrystallization velocity in samples with scattered and concentrated damage. The recrystallization velocity of a c/a interface is also shown. Lines are best fits to each data set. Activation energies are represented besides the corresponding line.

pair recombination, 0.43 eV [7]. This indicates that the separation of 4 Å among IV pairs was long enough to prevent their interaction. In the case of the concentrated damage the activation energy is higher, 0.89 eV, and the recrystallization dynamics slower. Recrystallization starts from the c/a interface, as it is also observed in experiments [17,18]. This is because IV pairs in contact with crystalline atoms are less stable than IV pairs near the center of the sphere. However, their strong interaction with IV pairs in the amorphous side of the interface make them more stable than if they were isolated. This effect is increased in the case of the planar interface, which shows an even higher activation energy for recrystallization, 2.44 eV, in very good agreement with experiments [17]. IV pairs that lie on the planar interface are surrounded by more IV pairs than those on the amorphous sphere. In the limit, a pure amorphous matrix is represented in this model by an IV pair completely surrounded by other IV pairs [7]. The recombination energy of such an IV pair would imply simulation times out of the reach of the MD technique. However, this energy can be experimentally extracted by monitoring the crystal nucleation in a pure amorphous Si matrix, which has been determined to be 5 eV [19]. Consequently, the higher the number of surrounding IV pairs, the higher the activation energy for IV pair recombination. These energies range from 0.43 eV for an isolated IV pair to 5 eV for a fully coordinated IV pair. Because the network rearrangement responsible for recrystallization occurs through the simultaneous breaking of several bonds (collective atomic motion [7]), it is reasonable that the recrystallization kinetics depends on the number of disordered neighbors.

In order to extend the simulations to times and sizes comparable to experiments, we have implemented these ideas in a kinetic Monte Carlo code. The simulation scheme and parameters are as in Ref. [20], but instead of assuming instantaneous I-V recombination when they are within second neighbor distance, we create an IV pair. This may happen to interstitials and vacancies generated within the ion collision cascade [12] or as a result of their encounter after some diffusion hops [13]. The IV pair is the elementary unit to describe the amorphous material in our model. The activation energy for IV pair recombination, E, increases with the number of surrounding IV pairs, n, following the expression: $E(n) = 0.6 + 0.2n + 0.0012n^3$ (in eV) for $n > 0$, and $E(0) = 0.43$ eV. The cubic term is added to strengthen the stability of IV pairs in the core of amorphous pockets. This phenomenological expression has been fitted to give the aforementioned experimental values for crystal nucleation in the amorphous phase at $n = 12$ (maximum coordination value that results from the interaction distance of the model, the Si atomic density, and that each IV pair involves two disordered atoms) and the planar c/a interface for $n = 7$ (since an IV pair lying on a planar c/a interface would have a coordination number a little higher than half the previous maximum value). It is worth noting that, for nonamorphizing conditions, IV pairs recombine very fast and the simulation results coincide with the ones obtained using the previous model with direct I-V annihilation [20].

This simple model based on the IV pair quantitatively captures the remarkable features related to the ion-induced amorphization in Si: (i) It has been experimentally observed that the amorphous pockets have similar structure and annealing behavior to continuous amorphous layers [18]. In the model, the continuous amorphous layer is just a particular case of amorphous pocket. Finite-size amorphous pockets (convex in several sides) naturally

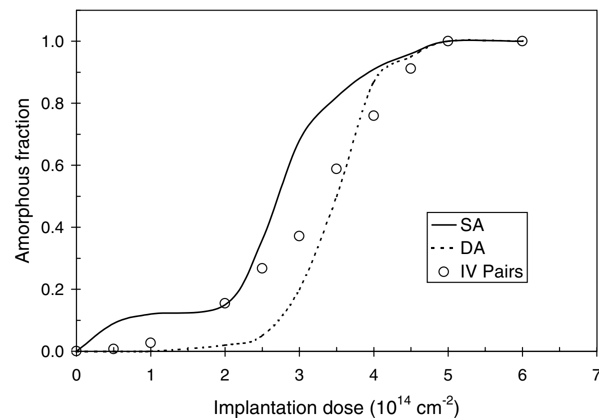

FIG. 3. Dose dependence of the damage produced by 100 keV Si^+ ions at room temperature. Solid and dashed lines correspond to the single- (SA) and double-alignment (DA) Rutherford backscattering spectra from experiments of Ref. [2], respectively. Symbols represent our simulation results. The amorphous fraction is obtained as the ratio of the maximum IV pair concentration to that corresponding to a pure amorphous Si matrix (25% of IV pairs, Ref. [7]).

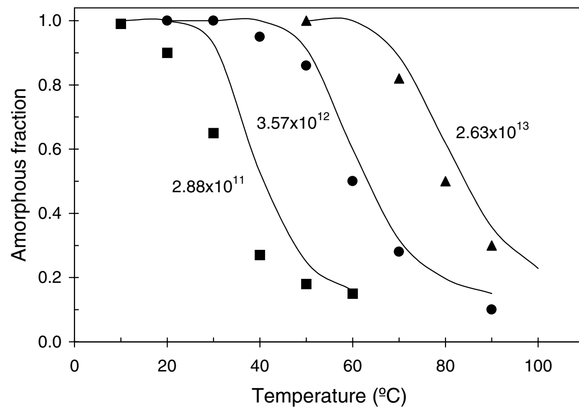

FIG. 4. Amorphous fraction vs substrate temperature for 1 MeV Si^+ implants to a dose of 10^{15} ions/cm^2 with several dose rates (in ions/cm^2 s). Solid symbols correspond to the experimental data of Ref. [21], obtained from Rutherford backscattering spectra. Solid lines represent our simulation results calculated as in Fig. 3.

regrow at lower temperatures because IV pairs at the interface have fewer neighbors than they would have in a planar interface. (ii) The superlinear behavior of the accumulated damage vs dose (experiment [2] and simulation) is shown in Fig. 3 for a 100 keV Si^+ implant at room temperature. For low doses, the amorphous fraction grows slowly, until a sharp increase occurs near a given (transition) dose. This happens because the damage is dilute for low doses, so only a small percentage of the generated damage survives. As the dose increases and the damage reaches a certain level, IV pairs start interacting and become more stable. Therefore, a larger percentage of the generated damage survives, resulting in its superlinear increase with dose. This cooperative mechanism is the result of the increased stability of IV pairs with the number of IV neighbors. (iii) The effect of the dose rate appears only in a temperature window that depends on the ion mass [1]. At low temperatures, there is little dynamic annealing and most damage survives, independently of the dose rate [3]. At high implant temperatures, the lattice recovers quickly and it becomes difficult to start nucleating the amorphous sites (amorphous pockets with enough number of IV neighbors to be stable), even for high dose rates. In the case of Si implants, the critical temperature for amorphization lies around room temperature [21]. The effect of the dose rate for 1 MeV Si^+ implants is represented in Fig. 4. As can be seen, dose rate has an effect in this temperature window as the time between successive cascades determines the amount of surviving IV pairs. Amorphization can be reached at higher implant temperatures when the annealing time between cascades is reduced (higher dose rates). The critical temperature for amorphization also increases with ion mass [1]. Heavier ions produce denser damage (IV pairs surrounded by more IV pairs), and therefore, more stable.

In summary, we have developed an atomistic model for the irradiation-induced amorphization of Si based on MD simulations. The building block for the amorphous phase is the IV pair, whose stability increases with the number of surrounding IV pairs. This single type of defect shows different activation energies for recombination as a function of its local coordination, which explains the diversity of energies extracted from the experiments. The model has been implemented in a kinetic Monte Carlo simulator able to monitor the defect evolution in time and space. The model provides excellent quantitative agreement with experimental results related to the critical c/a transition in terms of ion mass, dose, dose rate, and temperature dependencies.

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