The role of the bond defect on silicon amorphization: a molecular dynamics study

Luis A. Marqués *, L. Pelaz, M. Aboy, J. Vicente, J. Barbolla

Departamento de Electrónica, Universidad de Valladolid, ETSI de Telecomunicación, 47011 Valladolid, Spain

Abstract

We have studied the influence of the so called “bond defect” in the silicon amorphization process using molecular dynamics simulation techniques. The bond defect consists in a local distortion of the silicon lattice with no excess or deficit of atoms, and it can be formed during ion-beam irradiation. Even though the bond defect lifetime is too short to justify damage accumulation at usual implantation temperatures, we have observed however that the interaction between close bond defects can generate more stable structures which behave as the amorphous pockets created by ion irradiation. We have seen as well that the recombination of a given amount of damage created by bond defect accumulation depends of its spatial distribution.

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1. Introduction

Modelling the silicon amorphization is important for process simulation because of the use of increasingly high ion implantation doses in the microelectronics industry. The ions generate collision cascades which leave damage in the silicon lattice. The damage configurations range from isolated point defects or point defect clusters to extended amorphous zones, depending on the ion mass and energy, dose, dose rate and temperature [1]. Due to all these possibilities for the damage topology, ion-beam amorphization of silicon is a process difficult to characterize and model.

The interaction between the interstitials and vacancies created by irradiation is usually described by instantaneous annihilation of the two defects. However, some authors have proposed instead that a defect, known as bond defect or IV pair, may be formed [2]. The bond defect can also be directly generated by a collision sequence during the cascade regime of the irradiation, with no need of pre-existing interstitials and vacancies in the lattice for its formation [3]. Its properties have been studied by different simulation techniques, such as tight binding [2] and Hartree–Fock first principles [4]. In this work we analyze the role that the bond defect may have on the silicon amorphization process by the use of molecular dynamics...
simulation techniques. From this study it is possible to deduce an atomistic model to be readily used in kinetic Monte Carlo codes [5].

2. Simulation model

The molecular dynamics simulation technique consists in the resolution of the equations of motion for a set of particles using a computer. By averaging in time, it is possible to extract meaningful physical properties concerning the system under study [6]. In this kind of simulations, the interactions among the particles determine the dynamics of the system. Consequently, it is important to use an interatomic potential that represents as closely as possible the interactions in the real material. We have used in our simulations the Tersoff 3 potential [7], which has been shown to reproduce fairly well the crystal, liquid and amorphous states of silicon [8,9]. The only drawback is that the Tersoff 3 potential gives a melting temperature for silicon 750 K above the value found in the experiments (1685 K). Even though the temperature scale is not well predicted by the Tersoff 3 potential, at least it is possible to infer qualitative information from the simulations.

In our calculations we have used system samples consisting of 576 silicon atoms in a computational cell whose dimensions were \(4a \times 3\sqrt{2}a \times 3\sqrt{2}a\), \(a\) being the basic unit cell length (5.43 Å). The cell, of approximately the shape of a cube, 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. We solved the classical equations of motion using the fourth-order Gear predictor-corrector algorithm [10] with a variable time step.

3. Results and discussion

The bond defect consists of a local distortion of the silicon lattice without any excess or deficit of particles. In Fig. 1 we show the structure given for this defect by Tersoff 3. There is a bond switching between atoms A and B, and another between \(A'\) and \(B'\). The five and seven membered rings typical of the amorphous state can be clearly seen. The gray scale indicates the atom potential energies, where darker tones correspond to higher energies.

![Fig. 1. Structure of the bond defect given by Tersoff 3. Atoms A and A' switch their bonds with atoms B and B', respectively. The gray scale indicates the atom potential energies, where darker tones correspond to higher energies.](image-url)
of 0.43 eV. This implies a lifetime at room temperature in the order of a few microseconds. For the usual implantation parameters, this time is too short for the bond defect to survive between successive cascades, so damage can not accumulate. Consequently, the bond defect by itself can not be responsible for amorphization.

However, when bond defects are not isolated, they interact strongly and do form more stable complexes. In Fig. 2 we show the evolution of the potential energy per atom of samples with bond defect concentrations of 10%, 20%, 25% and 30% annealed at 1200 K. The bond defects are generated randomly through the system to a given concentration. Solid lines represent the mean potential energy that an atom has in the amorphous \((E_{\text{AM}})\) and crystalline \((E_{\text{C}})\) phases. As can be seen, samples with concentrations of 10% and 20% of bond defects recrystallized upon annealing. In the case of 10%, the decay is exponential, with a time constant of the order of the lifetime of the bond defect. This indicates that bond defects recombine as if they were isolated. For a concentration of 20%, the evolution of the potential energy per atom shows a plateau followed by a steep decrease, indicated by the arrow in Fig. 2. In this case, bond defect interaction has produced a more stable complex, responsible for the plateau in the curve. Crystallization then requires the collective movement of several atoms which produces a sudden decrease in the potential energy per atom. This type of behavior has been also observed during the recrystallization of amorphous pockets generated by heavy ion irradiation [12]. On the other hand, the sample with 25% of bond defects became polycrystalline, and for a concentration of 30% it remained amorphous. These results indicate that when the defect concentration is low, they recombine upon annealing and damage can not accumulate. On the contrary, for high damage concentrations homogeneous amorphization takes place.

But the recrystallization or amorphization processes do not depend on the concentration of bond defects alone, but also on its spatial distribution. In Fig. 3 we represent several snapshots taken during the annealing at 1200 K of two samples with a concentration of 20% of bond defects. Unlike previous simulations where bond defects were randomly distributed along the sample, in Fig. 3(a) the bond defects are scattered and in Fig. 3(b) they are concentrated in its center. The former distribution case would be equivalent to electron or light ion irradiation, and the second case to heavy ion irradiation. After 100 ps of annealing at 1200 K the scattered damage has disappeared, but still remains some in the case of the concentrated damage. This means that for the same amount of bond defects, if they are concentrated, it is more likely for them to survive between successive cascades. That explains why it is easier to amorphize when irradiating with heavy ions than in the case of electrons or light ions. These results suggest a method to generate damage in the lattice in a controlled manner, and whose behavior compares fairly well with the experimentally observed for damage generated by irradiation.

4. Conclusions

We have studied the influence of the bond defect on the silicon amorphization process using molecular dynamics simulation techniques. Even though its lifetime at room temperature is too...
short to produce damage accumulation at the usual implantation temperatures, we have shown that bond defects interact strongly to each other and generate more stable complexes. As higher is the concentration of bond defects in the lattice, more likely is the amorphization to occur. Besides, we have observed that amorphization depends on the topology of the damage: when bond defects are isolated they recombine very fast as if they were alone, but if they are concentrated the damage can survive longer times and thus accumulate giving rise to amorphization. The former case can be compared with electron or light ion irradiation and the latter with heavy ion irradiation. These results can be used to define an atomistic model of amorphization based on the bond defect, in which its time for recombination depends on the number of neighbouring bond defects. This model has been readily implemented in a kinetic Monte Carlo code able to simulate the irradiation process in a real time scale [5].

**Fig. 3.** Snapshots taken during the annealing at 1200 K of samples with the same amount of bond defects but distributed differently. In (a) the damage is scattered and in (b) concentrated. Colors indicate atom potential energies. After 100 ps the scattered damage has disappeared, but the concentrated damage still remains.

**References**