Atomistic modeling of ion beam induced amorphization in silicon

Lourdes Pelaz *, Luis A. Marqués, Maria Aboy, Juan Barbolla

Department of Electronics, Campus Miguel Delibes s/n, University of Valladolid, ETSI Telecommunication, 47011 Valladolid, Spain

Abstract

We have developed an atomistic model to describe ion beam induced amorphization in silicon. The building block for the amorphous phase is the bond defect or IV pair, whose stability increases with the number of surrounding IV pairs. This feature explains the regrowth behaviour of different damage topologies and the kinetics of the crystalline to amorphous transition. Amorphous regions start their regrowth by the atoms in the interface, as they have less surrounding IV pairs. The model provides excellent quantitative agreement with experimental results. It captures the critical transition temperature as a function of the ion mass and dose rate. The superlinear increase of the accumulated damage with dose is also reproduced by the model.

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1. Introduction

Ion beam induced damage exhibits different configurations, going from isolated point defects or point defect clusters surrounded by crystalline silicon, to continuous amorphous layers. Amorphization is thought to occur spontaneously when the free energy of the defective lattice is higher than that of the amorphous material. Under traditional phenomenological models, amorphization is envisaged to occur as a result of the overlap of discrete amorphous regions created directly from individual collision cascades [1], or via the local accumulation of simple defects followed by the sudden collapse of the region into the amorphous phase [2]. The critical behaviour of the amorphous transition in situations where significant dynamic anneal occurs require nucleation limited models, involving the production of suitable amorphization sites and the subsequent interaction of simple defects with these sites [3]. Ion beam induced damage is generally described in terms of Frenkel pairs, i.e. Si self-interstitials and vacancies. However, molecular dynamic (MD) calculations show that a single collision cascade contains a large variety of configurations, size and shape of the damage [4]. This variety makes their characterization very arduous, and it is difficult to identify the defect or defects responsible for amorphization.
The divacancy has been considered by several authors as the defect most likely to be controlling the transition to the amorphous phase [5,6]. Their idea was based on the coincidence of the dissociation energy of divacancies, 1.2 eV, with the activation energy measured for the onset of amorphization. A second-order defect production process (for example, the divacancy) was inferred from the quadratic dependence of the generation rate of defects on the density of displaced atoms in the collision cascade. Jackson determined that the defects that control recrystallization annihilate each other in pairs [7]. He suggested two possible candidates, a combination of vacancies and interstitials in the crystalline phase, and a dangling bond in the amorphous phase. Theoretical calculations indicate that a defect known as bond defect or IV pair was formed by incomplete interstitial-vacancy recombination [8] and also may be formed during implantation as a result of a ballistic process [9]. This defect contains no excess or deficit of atoms but introduces some disorder in the lattice, with the five and seven member rings characteristic of the amorphous phase.

Goldberg et al. determined a series of apparent activation energies for amorphization ranging from 0.7 to 1.7 eV for irradiation with ions going from 12 amu (C) to 132 amu (Xe) [3]. The existence of several different defects capable of controlling the phase change for different implanted ions seems unlikely because of the similarity in the defect structure of bombarded crystal for all ion species, just before the onset of amorphization. However, the energy deposited by the collision cascade changes with ion type, so will the relative populations of these residual defects and the combination of different reactions between defects.

The detailed description of the amorphization process in terms of defect reactions is important both from fundamental and practical points of view, since Si pre-amorphization is frequently used to achieve improved dopant activation with low thermal budget [10]. In this work we present an atomistic model that governs the defect evolution in time and space and provides a quantitative description of ion beam induced amorphization in Si. This model allows us to extend atomistic process simulators to amorphizing doses.

2. Atomistic modeling of amorphization

MD calculations have shown that when IV pairs are present in the Si lattice to a concentration above 25% homogeneous amorphization takes place, which indicates that amorphization could be achieved without the intervention of any additional defect [11]. Amorphous pockets generated by ion implantation have similar features to the ones artificially generated as agglomerated of IV pairs. The activation energy for IV pair recombination was determined to be of 0.43 eV, which implies that the IV pair alone is not stable enough to justify damage accumulation at the typical dose rates. MD simulations show that interactions among this type of defects lead to more stable structures or amorphous pockets. The stability of a given IV pair within an agglomerate increases with the number of surrounding IV pairs [12].

In order to extend the study of the amorphization kinetics to scales comparable to experiments we have implemented these ideas in a non-lattice kinetic Monte Carlo simulator using the IV pair as the building block for the amorphous phase. Coordinates of Si self-interstitials and vacancies are determined using the binary collision approximation for the cascade generation. Then they are transferred to the non-lattice kinetic Monte Carlo Diffusion Code DADOS [13]. The new feature of this model is the formation of the IV pair, instead of assuming instantaneous interstitial-vacancy recombination. This may happen within the ion collision cascade and so we reproduce the amorphous pockets formed in the ballistic process [9] or as a result of interstitial-vacancy encounter after some diffusion hops [8].

Amorphous pockets are modelled as agglomerates of IV pairs. The activation energy for recombination, $E$, of a given IV pair increases with the number of surrounding IV pairs, $n$, according to the following expression: $E(n) = 0.6 + 0.2n + 0.0012n^3$ (in eV) for $n > 0$, $E(0) = 0.43$ eV. This expression is in accordance to MD calculations for isolated IV pair (0 neighbors) [11]. The recombination rate of the IV pairs with about half of the total coordination number corresponds to the experimental recrystallization velocity of a planar a/c interface (2.4 eV) [14]. IV pairs embedded into
an amorphous matrix (completely surround by neighboring IV pairs) have an activation energy of 5 eV [15].

For subamorphizing implants, the presence of these metastable defects before their annihilation does not change the basic understanding for defect evolution since the IV pair is very unlikely to break up into separate Si self-interstitials and vacancies [8]. When amorphous pockets recrystallize, they leave the local concentration of excess defects unchanged, similar to what happened when we considered instantaneous recombination of interstitials and vacancies. However, the inclusion of IV pairs allows the damage to accumulate during implant and make possible the formation of a continuous amorphous layer. Thus, amorphization is the result of the simulation itself and it is not imposed as a parameter or initial condition.

The local characterization of the IV pairs allows the model to capture any damage topology that may arise from irradiation cascades and the different annealing behaviour of damage structures. Highly damaged but sub-amorphizing implants produce regions that are seen as amorphous in structure and show similar annealing behaviour to those of continuous amorphous layers but the regrowth occurs at lower temperatures [16]. In the model, the continuous amorphous layer is just a particular case of amorphous pocket. Experimentally determined damage generated at cryogenic implant temperatures is very similar to that predicted by theoretical calculations [17]. However, for implants done at room temperature the experimental damage is significantly lower than that predicted by theoretical calculations. This implies that significant dynamic anneal already occurs during the implant and that many defects recombine at temperatures as low as 25 °C (IV pairs with $E_{\text{act}} < 1$ eV). Typical regrowth temperatures of the tails of the damage profiles are in the order of 200–400 °C ($E_{\text{act}} \sim 1.2–1.8$ eV) while continuous amorphous layers regrow at around 550 °C ($E_{\text{act}} \sim 2.4–2.7$ eV). Amorphous pockets shrink in size starting from the outer regions in contact with crystalline material that can act as a seed. In the model IV pairs in the outer part of the aggregate are more likely to recombine first, as they have less surrounding IV pairs. For the same reason, finite-size amorphous pockets (convex in several sides) naturally regrow at lower temperatures than they would have in a planar interface.

3. Simulation results

3.1. Temperature and dose rate dependence: the critical transition

The rate of defect accumulation during irradiation is the result of the balance between its generation and its recombination. Temperature and time determine the thermal budget between cascades. Amorphization can be reached at higher implant temperatures when the annealing time between cascades is reduced (higher dose rates). The effect of the dose rate appears only in a temperature window at which the damage generation is comparable to its annihilation, as shown in Fig. 1. At low temperatures there is little dynamic annealing and most damage survives, independently of the dose rate. 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 [18].

![Fig. 1. Amorphous fraction versus substrate temperature for 1 MeV Si implants to a dose of 10^15 cm^-2 for several dose rates. Solid symbols correspond to the experimental data of [18]. Open symbols represent our simulation results.](image)
3.2. Ion mass dependence: different effective activation energies

The critical temperature for amorphization increases with ion mass [3]. The IV pairs have different activation energies for recombination as a function of its local coordination, which explains the diversity of energies extracted from the experiments. Heavier ions produce denser damage (IV pairs surrounded by more IV pairs) and, therefore, more stable. The balance between damage generation and its recombination is governed by more stable amorphous pockets and, therefore, occurs at higher implant temperatures, as shown in Fig. 2.

3.3. Dose dependence: superlinear behaviour

The accumulated damage versus dose presents a superlinear behaviour, as can be seen in Fig. 3. For low doses the amorphous fraction grows slowly, until a sharp increase occurs near a given (transition) dose [6]. 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 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.

4. Conclusions

We have developed a model that explains the characteristics of the ion beam induced amorphization transition at an atomistic scale which is also able to reproduce times and sizes directly comparable to experiments. The building block for the amorphous phase is the bond defect or IV pair, whose stability increases with the number of surrounding IV pairs. The model captures the regrowth behaviour of different damage topologies and the kinetics of the ion beam induced crystal to amorphous transition. It provides excellent quantitative agreement with experimental results related to the critical transition temperature as a function of dose rate and ion mass and the superlinear behaviour of the accumulated damage versus dose.

References