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Amorphous layer depth dependence on implant parameters during Si self-implantation

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Abstract

Preamorphization followed by low temperature solid phase epitaxial regrowth has been proved to provide a high activation of the dopants with minimal diffusion. However, the end of range damage present after regrowth beyond the initial amorphous/crystalline interface causes diffusion and deactivation of dopants during subsequent annealing. In this paper, we study the influence of implant conditions on the depth of the amorphous layer during Si self-implantation. We compare experimental data with our simulation results obtained using an atomistic amorphization–recrystallization model recently developed. We show that the amorphous/crystalline interface depth initially increases with dose but saturates at high doses. Beam current and wafer temperature also alter the depth of the amorphous layer and the amount of residual damage by affecting the dynamic annealing of the damage. These parameters are not always well controlled or specified in experiments and can explain differences observed in dopant profiles.
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1. Introduction

Ultra-shallow junction formation required for the fabrication of modern electronic devices demands an accurate control of dopant profiles. Ion implantation is nowadays the main technique to introduce dopants in the lattice, due to its capability of impurity profile control and shallow penetration. However, this technique produces considerable damage in the lattice, ranging from point defects to continuous amorphous layers (*a*-layers), as a consequence of the collisions between incident ions and lattice atoms. In this process, dopants are usually left into non-substitutional positions. Thermal treatments required to cure the damage and to activate dopants cause a significant redistribution of the impurity profile due to the accompanying diffusion [1,2].

Preamorphization of the wafer prior to dopant implantation allows the suppression of channeling and a high activation of the dopants with low temperature anneals and thus with minimal diffusion [3]. During solid phase epitaxial regrowth (SPER) dopants within the amorphous region are activated up to a concentration of few times 10^{20} [4]. After regrowth, the end of range (EOR) damage, mainly {3 1 1} defects and dislocation loops, is present beyond the initial amorphous/crystalline (a/c) interface. These defects are responsible for transient enhanced diffusion that takes place during subsequent thermal treatments and may increase the junction leakage if they are not completely removed [5,2]. An adequate prediction of the a/c interface depth, and thus of the position and quantity of EOR defects, is necessary as a starting point to evaluate the redistribution of dopants previously activated during SPER.

In this work, we perform atomistic simulations to study the influence of implant parameters on the a/c interface. An amorphization/recrystallization model recently developed allows us to analyze the role that dynamic anneal plays on the accumulation of generated damage and the formation of extended amorphous regions [6].

2. Simulation model

In our simulation scheme, implantation cascades are simulated with the binary collision computer code MARLOWE

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[7], and the defect evolution with the kinetic non-lattice Monte Carlo diffusion code DADOS [8]. Annealing at the implant temperature is carried out after each implantation cascade during a time defined by the dose rate of the implant. This scheme is followed until the specified dose is reached. In the model self-interstitials, vacancies and their clusters are considered as well as the defect known as *bond defect* or *IV pair* [9,10]. The IV pair is formed by the interaction of Si interstitials and vacancies, instead of undergoing instantaneous annihilation. It is considered as the building block to describe the amorphous phase in our model. The recombination rate of the IV pair is defined to decrease as the number of neighboring IV pairs increases. This feature allows the model to capture any damage topology that may arise from irradiation cascades, as well as the characteristic regrowth behavior observed in experiments [6]. The simulation of the dynamic annealing that takes place during the implant together with the amorphization/recrystallization model makes our atomistic simulation code a very useful tool to study the formation of amorphous regions.

3. Results and discussion

Dose, mass and energy of the implant ion and substrate temperature control the quantity and the position of generated damage during ion implantation. Since the damage increases with the number of cascades, high implant doses may cause amorphization of the Si lattice. Additional cascades widen the amorphous region. The energy of the incident ions defines the position of the mean projected range, affecting the depth of the amorphous region. As the ion implantation proceeds a fraction of the generated damage may anneal out. In fact, the intensity of the dynamic annealing during the implant may allow the accumulation of the generated damage or, on the contrary, the recombination of most of it, avoiding the creation of an amorphous region. Very low temperature implants allow the analysis of the evolution of the *a*-layer depth when the dynamic annealing is negligible.

First of all we study the influence of dose on the width of amorphous regions. Maszara and Rozgonyi measured the depth of the a/c interface from cross-section transmission electron microscopy micrographs for a Si 150 keV $1.56 \times 10^{12}\,cm^{-2}\,s^{-1}$ implant, with doses ranging from 3×10^{14} to $3 \times 10^{15}\,cm^{-2}$ [11]. Implant temperature was 82 K, discarding the influence of the dynamic annealing. The measurements from Maszara and Rozgonyi, reproduced in Fig. 1, show that there is an initial fast increment of the a/c interface depth with dose until a given amorphous depth is reached, and then, the increase of the *a*-layer width with dose is very slow. Our simulation results, performed with the same implant conditions as Maszara and Rozgonyi's experiments and also plotted in Fig. 1, reproduce the experimental data showing that the a/c interface depth saturates with dose at high doses. Since all damage is stable at this temperature, the observed saturation is caused by the spatial distribution of the generated damage. It is known that during the implant some ions may find a path where they undergo few interactions with lattice atoms. This mechanism, called channeling, allows the implanted ions to reach deep regions of the wafer. The existence of a wide

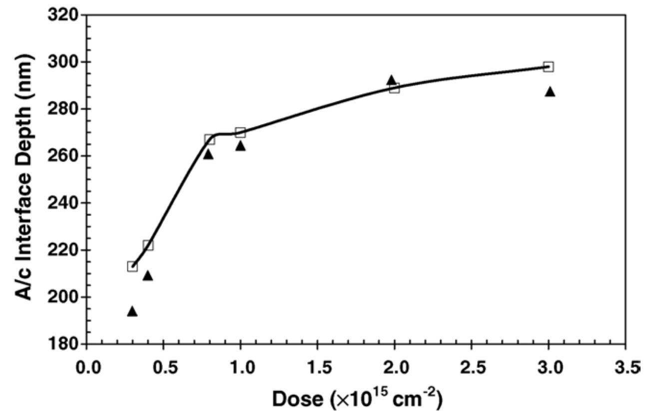

Fig. 1. A/c interface depth as a function of dose for a Si 150 keV $1.56 \times 10^{12}\,cm^{-2}\,s^{-1}$ implant performed at 82 K. Open symbols correspond to our simulations results. Solid symbols represent experimental data extracted from Maszara and Rozgonyi [11]. Two regions are observed: at low doses the interface depth increases with dose, whereas at high doses the *a*-layer depth saturates. Since dynamic anneal is negligible at this temperature, the saturation of the a/c interface depth with dose is caused by the distribution of the generated damage.

amorphous layer reduces the channeling and makes the damage profile more abrupt. Most of cascade damage falls within the *a*-layer and only a small fraction of it contributes to the widening of the amorphous region. The slow increase of the a/c interface at high doses shows the low efficiency of increasing the dose to form wide *a*-layers. Multiple energy implants are preferred to achieve a deep amorphous layer that extends to the surface.

A more detailed study of this process indicates a correlation between the mean projected range (R_p) and the longitudinal straggling (ΔR_P) of the implant with the saturation of the *a*-layer depth. We have simulated the variation of the a/c interface depth with dose for the same implant conditions as in Fig. 1, but with three different implant energies. The data, shown in Fig. 2, follow the same trend as experimental data reported by Maszara and Rozgonyi. For all the simulated implant energies two different regions can be observed: at low doses the a/c interface depth increases with dose, whereas at high doses the width

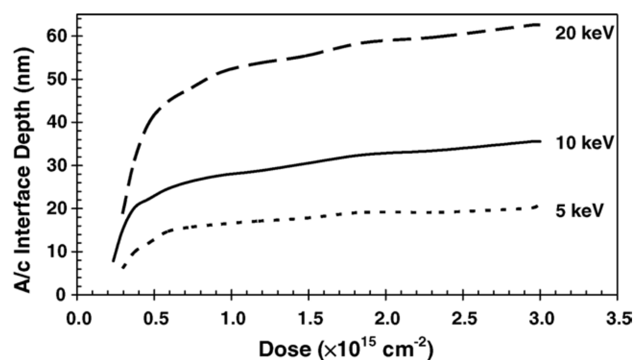

Fig. 2. Evolution of the a/c interface depth with dose as a function of implant energy for a Si $1.56 \times 10^{12}\,cm^{-2}\,s^{-1}$ implant performed at 82 K. Implant parameters are the same as in Fig. 1. The width of the amorphous region is larger as the implant energy increases and saturates at high doses. The depth at which a change in the slope of the evolution of the a/c interface depth with dose is observed, fits approximately the sum of the mean projected range and the longitudinal straggling.

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