

The effect of dose rate on interstitial release from the end-of-range implant damage region in silicon

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(Received 8 May 1997; accepted for publication 23 September 1997)

Low temperature molecular beam epitaxy was used to grow boron doping superlattices DSLs in Si, with peak boron concentrations of $1 \times 10^{18}/\text{cm}^3$, and spike widths of 10 nm. Amorphization of these DSLs was achieved using a series of Si^+ implants of 30 and 112 keV, each at a dose of $1 \times 10^{15}/\text{cm}^2$, which placed the amorphous to crystalline interface between the first and second doping spikes. The dose rate of the Si^+ implants was varied from 0.13 to 1.13 mA/cm². Post-implantation anneals were performed in a rapid thermal annealing furnace at 800 °C, for times varying from 5 s to 3 min. Secondary ion mass spectrometry was used to monitor the dopant diffusion after annealing. Increasing the implant dose rate appears to increase the amount interstitial flux toward the surface but has no observable effect on the flux into the crystal. Transmission electron microscopy was used to study the end of range defect evolution. Increasing dose rate was observed to decrease the end of range defect density. These observations are consistent with previous findings that indicate the amount of backflow toward the surface decreases as the end of range loop density increases. © 1997 American Institute of Physics. [S0003-6951(97)00347-1]

Amorphization by ion implantation is a widely used technique to prevent channeling of lighter elements, such as B, during implantation in silicon integrated circuit manufacturing. Following amorphization, a damage layer, which is termed the end of range (EOR) damage region, exists below the amorphous to crystalline interface (*a/c*). This layer contains a supersaturation of interstitial point defects¹ created during the implantation by recoiled atoms and transmitted ions. Solid phase epitaxial (SPE) regrowth of the amorphous region occurs upon annealing at low temperature (600 °C). At higher temperatures the excess interstitials in the EOR both diffuse away to regions of lower interstitial concentration and precipitate into EOR (type II) extended defects.² These extended defects can lead to the two main difficulties in creating shallow junctions in amorphized layers; leakage current from defects in the space charge region of the device and junction depth increase by transient enhanced diffusion of the dopant.

It has been reported that EOR defects maintain a supersaturation of point defects in the EOR region.³ Some studies report that during annealing, interstitials from the EOR may induce transient enhanced diffusion (TED) of the dopant contained in the regrown Si layer.³⁻⁷ The relation between implantation beam current or dose rate and residual damage (which leads to amorphization and extended defects) has been explored for both Si and GaAs. Many groups have found that as the dose rate increases, the amount of residual damage also increases.⁸⁻¹¹ In this letter, we seek to elucidate the relation between dose rate and both dopant diffusion and defect evolution.

Low temperature molecular beam epitaxy (MBE) grown boron doping superlattices (DSLs) in $\langle 100 \rangle$ silicon were implanted using an Eaton NV-GSD 200. In the DSL the boron peaks occurred at depths of 105, 313, and 521 nm, each with a concentration of $1 \times 10^{18}/\text{cm}^3$. Each specimen of the DSL was mounted on a 150 mm Czochralski (CZ) grown *n*-type silicon wafer ensuring a planar fit and thermal conductivity. The entire wafer was then implanted with 112 keV Si^+ at a dose of $1 \times 10^{15}/\text{cm}^2$, followed by a 30 keV, $1 \times 10^{15}/\text{cm}^2$ implant. The two overlapping implants were performed to produce a continuous surface amorphous layer. The dose rate of the 112 keV implant was varied from 0.13 to 1.13 mA/cm² while the endstation temperature was maintained at $20 \text{ °C} \pm 1 \text{ °C}$. The tilt/twist angles for each implant were $5^\circ/0^\circ$. Post-implantation annealing was performed in an A.G. Associates Thermopulse 410T rapid thermal anneal (RTA) in a N_2 ambient. Each wafer was annealed at 800 °C for either 5, 30, or 180 s. Secondary ion mass spectrometry (SIMS) profiles were performed on a Cameca IMS-4f. The counts of $^{11}\text{B}^+$ were obtained using an 8 keV O_2^+ beam rastered over a $225 \mu\text{m}$ by $225 \mu\text{m}$ area, with an $80\text{-}\mu\text{m}$ -diam detected area. Plan-view transmission electron microscopy (TEM) of the samples was performed on a JEOL 200CX with images taken in g_{220} weak beam, dark field. Cross-sectional TEM was used to measure the amorphous layer depth. The majority of the TEM was performed on CZ wafers that were used as backing wafers to mount small, 4 cm^2 , pieces of the DSL material in the implanter. Selected SIMS samples were also prepared into TEM samples to correlate the EOR defect evolution in the MBE samples with the

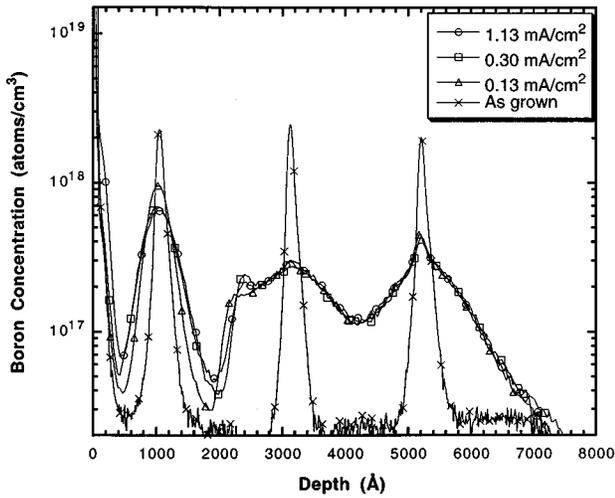


FIG. 1. SIMS profiles of DSL after Si^+ implantation at different dose rates and annealing at 800°C for 3 min.

CZ wafers. The quantification methodology of the trapped interstitials in $\{311\}$ and loop defects have been discussed elsewhere.¹²

It has previously been shown that TED of boron can occur in preamorphized silicon.⁷ The profiles of the samples implanted with Si^+ at dose rates of 0.13, 0.30, and 1.13 mA/cm^2 and annealed at 800°C for 3 min are shown in Fig. 1, along with the as implanted profile. As the dose rate is increased the amount of interstitial flux into the regrown layer also increases. Plots of the diffusivity enhancements in the regrown layer of the three dose rates versus annealing time are shown in Fig. 2. Peak 1 is the doping spike closest to the surface and peak 2 is the middle spike. In the regrown silicon, the diffusion enhancement increases with increasing dose rate. There is no measurable change in the diffusion enhancement into the material below the a/c as a function of dose rate. The diffusivity enhancements indicate that the flux into the crystal is an order of magnitude greater than the flux toward the surface for these implant conditions.

Microstructural changes in the samples as a function of dose rate were also characterized by TEM. The amorphization threshold has previously been observed to decrease with increasing dose rate.^{8,9} Under the implant conditions of this study, variation in dose rate has a strong effect on amorphous layer depth. The a/c depth was found to increase linearly with dose rate from 2000 \AA at 0.13 mA/cm^2 , to 2200 \AA at 1.13 mA/cm^2 . Beyond the a/c interface is a transition region of dark strain contrast (in bright field) between the amorphous layer and perfect crystal. It was found that although the amorphous layer depth increased with increasing dose rate, the transition region thickness remained constant at 300 \AA . Figure 3 shows TEM micrographs of the EOR defects of the three different dose rates after rapid thermal anneal (RTA) at 800°C for 3 min. It is apparent from the micrographs that the EOR defect density decreases with increasing dose rate. In Fig. 4, the number of both types of EOR defects versus annealing time at 800°C for the different dose rates is presented. The number of $\{311\}$ defects remains constant while the number of dislocation loops decreases with increasing dose rate. Figure 5 shows a plot of

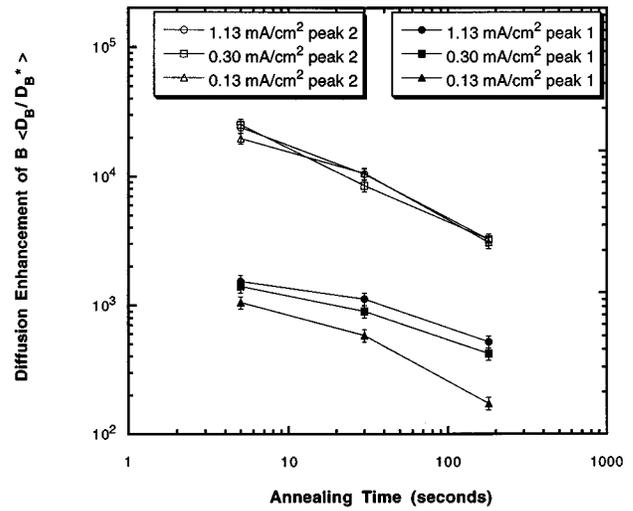


FIG. 2. Diffusion enhancement of boron vs annealing time at 800°C .

the number of interstitials bound by the EOR defects versus annealing time for different dose rates. Although the defect density did vary with dose rate the total number of interstitials in the EOR damaged region remained constant as a function of dose rate. It is also clear that the interstitial content of the loops increases significantly for these annealing times.

Explanation of the variation in diffusion enhancement as a function of dose rate is centered around the a/c interface. The observation that the transition region thickness is independent of dose rate is consistent with the observation that the number of interstitial point defects in the EOR extended defects after annealing is relatively independent of dose rate. The excess interstitials in the wafer after implantation are widely believed to be the source of TED of boron in ion implanted silicon.^{3-7,12} Since the number of interstitials appears to be constant as a function of dose rate, the diffusion differences are presumably not the result of a shift in the concentration of EOR interstitials but, instead, may be a function of the EOR defect nucleation/evolution process. It is interesting to note that, as stated, the EOR loop density varies significantly ($3\times$) with dose rate, whereas the interstitial content in the defects does not vary substantially with dose rate. This implies the loops nucleation process may be heterogeneous. However, the $\{311\}$ density appears to be independent of dose rate which is consistent with homogeneous

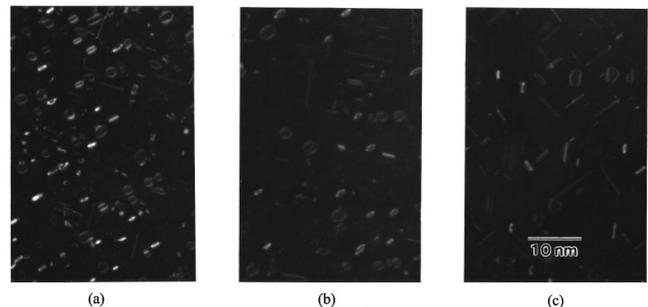


FIG. 3. Plan-view TEM micrographs of samples implanted at three different dose rates after annealing at 800°C for 3 min: (a) 0.13 mA/cm^2 , (b) 0.30 mA/cm^2 , (c) 1.13 mA/cm^2 .

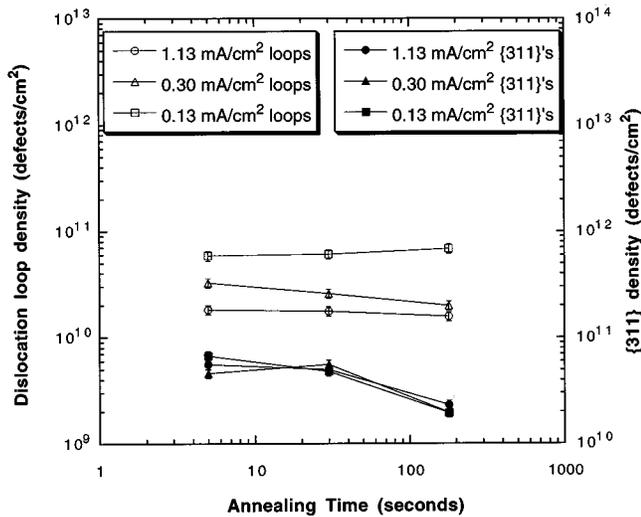


FIG. 4. End of range defect density as a function of annealing time at 800 °C.

nucleation of these defects. It has been proposed that {311} unfauling may be the source of the loops, however we found no correlation to suggest that this process accounts for any significant fraction of loops. The EOR loop density and the diffusivity enhancement follow inverse trends. This is consistent with a model that the EOR loops are acting as traps and screening the flux of interstitials toward the surface. This argument which correlates EOR loop density with the amount of interstitial flux to the surface was recently reported by Jones *et al.*¹²

The shift in amorphization depth may be attributed to dynamic annealing differences with dose rate variation. As set forth by Morehead,¹³ the lifetime of a collision cascade is on the order of 10^{-13} s. Assuming a collision cascade diameter of approximately 100 Å, the minimum time between collisions for this dose rate regime varies between 1.8×10^{-4} and 1.6×10^{-3} s, making the probability of cascade overlap negligible. If it is also assumed that the jump frequency of free interstitials and vacancies is 10^{-6} s and that on average 100 successful random jumps are required for

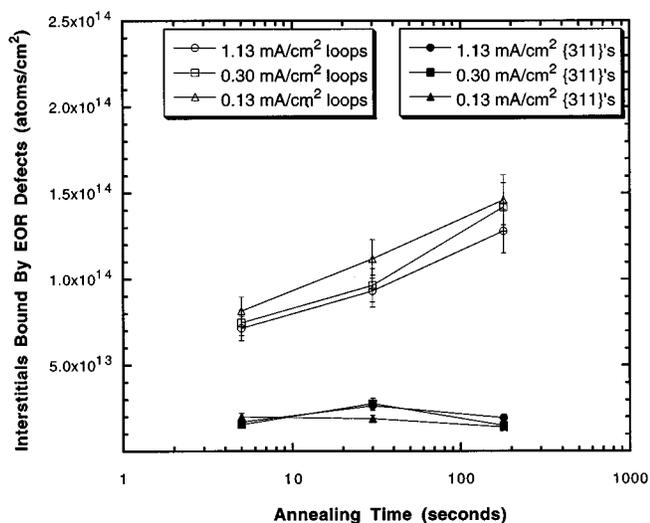


FIG. 5. Interstitials bound by the end of range defects as a function of annealing time at 800 °C.

interstitial-vacancy recombination, the lifetimes of displaced Frenkel pairs in the collision cascade are approximately 10^{-4} s.¹¹ Since the time constants for interstitial-vacancy recombination and the time between collisions within the damage region of the collision cascade are the same, variation of the time between collisions by an order of magnitude could logically effect the recombination process. Therefore, by decreasing the amount of dynamic annealing through increasing the dose rate by an order of magnitude, the threshold for amorphization is achieved at deeper regions of the crystal. This change in the dynamic annealing process also appears to change the morphology of the amorphous/crystalline interface such that if the loops are nucleating heterogeneously, there are fewer nucleation sites at higher dose rates. It will require further work to discern the source of the loop nucleation sites.

In conclusion, upon annealing, increasing the dose has the effect of increasing interstitial flux from the EOR damage region toward the surface and decreasing the EOR defect density. For these room temperature implants the interstitial flux into the crystal is approximately an order of magnitude greater than toward the surface. It was found that increasing the dose rate of the amorphizing implant increases the amorphous layer thickness while maintaining a constant transition region thickness. The observation that the EOR dislocation loop density varies with dose rate despite a similar trapped interstitial content implies the loop nucleation process may be heterogeneous. The dependence of the interstitial flux toward the surface on the EOR loop density is consistent with the loop layer acting as a barrier to backflow. This effect must be accounted for in process simulators in order to correctly model device processing.

The authors would like to thank Kathryn Moller and Michael Wright for assistance with TEM sample preparation, and Jewon Lee for assistance in annealing. This work was supported by the SRC under Contract No. BP 707.

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