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DAMAGE NUCLEATION IN Si DURING ION IRRADIATION

O.W. HOLLAND, D. FATHY and J. NARAYAN
Microelectronics Center of North Carolina, P.O. Box 12889, Research Triangle Park, NC 27709

ABSTRACT

Damage nucleation in single crystals of silicon during ion irradiation is investigated. Experimental results and mechanisms for damage nucleation during both room and liquid nitrogen temperature irradiation with different mass ions are discussed. It is shown that the accumulation of damage during room temperature irradiation depends on the rate of implantation. These dose rate effects are found to decrease in magnitude as the mass of the ions is increased. The significance of dose rate effects and their mass dependence on nucleation mechanisms is discussed.

INTRODUCTION

During ion implantation, the incident ions penetrate the surface of a solid and lose energy mainly through inelastic collisions with electrons and elastic collisions with the screened nuclear charge of the atoms. If the energy transferred to the lattice atoms during the collision with an ion is greater than the displacement threshold, then the atom can be removed from the lattice site. Due to the importance of ion implantation for semiconductor device fabrication, the mechanisms whereby irradiation produced point defects lead to the nucleation and growth of extended defects such as defect clusters, voids, dislocation loops and amorphous regions need to be clearly understood. The detailed nature of the residual damage in Si not only determines the annealing cycle necessary for its removal but also the efficiency of the electrical activation of the implanted dopant [1,2]. The mechanisms responsible for the formation of extended defects and the nature of defect reactions during heavy ion irradiation in Si are not well-understood. This uncertainty arises partly as a result of a lack of understanding of the role of the collision cascade in the above processes. A cascade generally encompasses a well-defined region within the solid especially if the damage energy density deposited by the incident ion is large. If the damage density is small the cascade region is more diffuse and the boundary less sharp. It has been proposed that the high spatial concentration of point defects in the cascade can lead to damage nucleation around the ion's track [3-5]. This is essentially a heterogeneous model for damage nucleation. Nonlinear cascade effects such as the increased damage produced by molecular ions compared to the atomic species has been offered as evidence to support this model [6,7]. In contrast, a homogeneous mechanism assumes that damage is nucleated by a random clustering of the point defects generated by the irradiation. The role of the energetic ion in this model is solely to produce displaced atoms while spatial correlation effects are ignored.

In this investigation, we have studied the dependence of damage nucleation mechanisms on the mass of the incident ion and substrate temperature. In samples irradiated at liquid nitrogen (LN_2), damage in the form of amorphous regions is nucleated about individual ion tracks. At room temperature (RT), the nature of the damage produced by Si^+ ion irradiation was found to be markedly different from that produced by heavier ions such as Bi^+. Only dislocation loops and no amorphous regions were observed in samples irradiated with Si^+ ions at RT to moderate doses. Also, the amount of damage produced by the ion irradiation was observed to increase with dose rate. This effect was not observed in samples implanted at LN_2 and was found to decrease with increasing ion mass for RT irradiation. These observations will be shown to be consistent with a damage mechanism at RT which varies from homogeneous to heterogeneous nucleation as the mass of the ions is increased.

EXPERIMENTAL PROCEDURE

High resolution, transmission electron microscopy on planar and cross-sectionally thinned specimens and Rutherford backscattering/channeling spectroscopy were used to investigate cascade effects and damage nucleation phenomena in Si during ion irradiation. The combined analysis techniques yield information not only on the amount and distribution of the damage, but the detailed nature as well. Individual collision cascades were investigated by high resolution imaging. This enabled damage and damage nucleation phenomena to be correlated with the individual ion track. Czochralski grown, n-type Si single crystals with (100) and (110) orientations and a resistivity of ~6 ohm-cm were used in this study. Beam rastering (125 Hz in the horizontal and 25 Hz in the vertical direction) during implantation was employed to ensure uniform coverage.

RESULTS AND DISCUSSION

Discrete damaged regions are shown by the arrows in fig. 1a which is a plane-view micrograph of a (110)Si crystal implanted at LN_2 with 5 x 10^{12} Bi^+/cm^2. A high resolution image (fig. 1b) of one of these regions shows them to have essentially an amorphous structure. The lack of black and white strain contrast at the damaged sites in fig. 1a provides further evidence that the damage consists of amorphous regions with no mismatch stress involved, except in isolated cases. It is felt that the amorphous structure is formed about an individual ion track, in the region of the collision cascade where the damage density is sufficient to stimulate a first-order, crystal-to-amorphous transformation. This is essentially the 'critical defect density' model for amorphization which assumes that the phase transformation occurs spontaneously when the free-energy of a defective crystalline region exceeds that of the amorphous state [8-10]. A critical damage energy for amorphization was determined to be 6.0 x 10^{23} ev/cm^3. The association of the discrete damaged regions with individual ion tracks is justified by the similarity between their areal density. The density of the damaged regions in the micrograph in fig. 1a is 4 x 10^{12} cm^{-2}, which is in close agreement with the implanted fluence. Also, the radius of the amorphous damage (~4 nm) agrees with the amorphous radius (<5 nm) determined using the 'critical defect density' model and the lateral damage profile calculated by TRIM [11], a well-known computer simulation program.

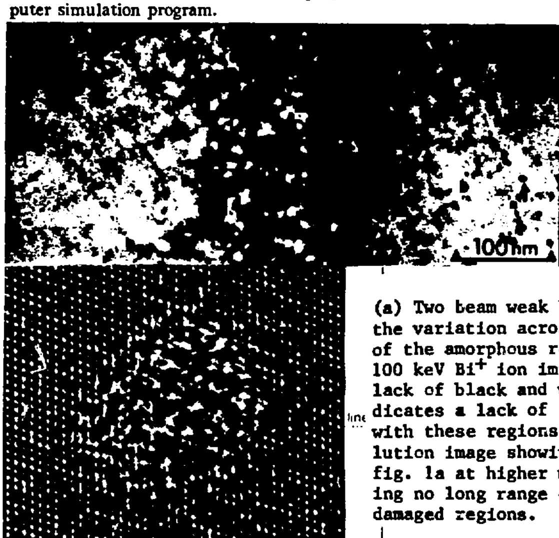

Figure 1

(a) Two beam weak beam image showing the variation across a bent contour of the amorphous regions produced by 100 keV Bi^+ ion implantation. The lack of black and white contrast indicates a lack of strain associated with these regions. (b) High resolution image showing the features in fig. 1a at higher magnification showing no long range order within the damaged regions.

The nature of the damage produced by Si^+ ion irradiation at LN_2 is somewhat similar to that discussed above. Shown in fig. 2 is a high resolution, cross-sectional micrograph from a (100) Si crystal implanted at LN_2 with 100 keV $^{30}Si^+$ ions at a dose of 10^{14} cm^{-2}. Damage regions corresponding to each individual ion track were not observed in this specimen. Rather, small isolated amorphous regions were present below the surface of the sample. Rutherford backscattering/channeling analysis confirmed that the surface was relatively defect-free with the preponderance of the damage centered at 100 nm. Unlike the cascade produced by a Bi^+ ion, the damage density in a Si^+ ion cascade is insufficient to produce amorphization directly [10]. As a result, it is felt that cascade overlap is required before an amorphous transition can occur. An initial cascade is required which pre-damages the lattice sufficiently such that the passage of a subsequent ion will result in damage accumulation in excess of the critical density. The higher implantation dose of light ions needed to produce the amorphous regions reflects the need for cascade overlap in the amorphization process. This interpretation is also consistent with previous observations [12] that, initially, damage in Si increases linearly with dose for light ion irradiation, consistent with linear cascade theory [13,14], but becomes superlinear for higher doses. This superlinear dependency reflects, not only the onset of cascade overlap, but also the collective nature (i.e. phase transition) of the amorphization process.

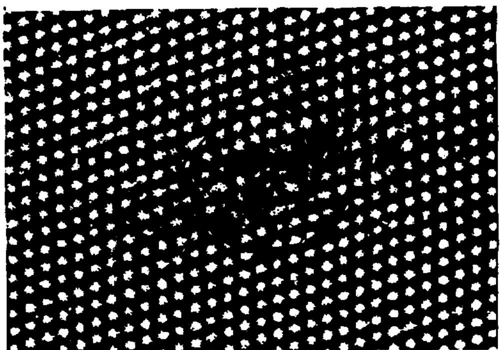

Figure 2

High resolution (110) cross-section micrograph from a (001) specimen implanted with 100 keV, ^{30}Si ions to a dose of 10^{14} cm^{-2}. The amorphous region is encircled.

The damage produced by Si^+ ion irradiation at RT was found to be markedly different from that produced at LN_2. Plane-view micrographs in fig. 3 show two (100) Si samples irradiated at RT with 100 keV Si^+ ions to a dose of 2.5×10^{14} cm^{-2} at different dose rates. In both cases, a high density of dislocation loops is observed in the samples. No evidence was found in either sample of amorphous regions using high resolution imaging and microdiffraction techniques. However, it is clear that at room temperature both the density of loops and the loop radii are influenced by dose rate; the higher the dose rate, the greater the loop density and radii.

The presence of more damage at the increased dose rate was confirmed by channeling spectra from the two samples shown in fig. 4. It is clear that in the damaged region produced by the implantation (~120 nm) the scattering yield is much greater in the sample irradiated at a higher dose rate. Since the scattering yield is related to the density of displaced atoms, it is clear that damage accumulation increases with dose rate. Calculated damage profiles from the channeling data yielded the peak damage density to be 2.1×10^{22} $atoms/cm^3$ in the sample irradiated at a high dose rate compared to 5.0×10^{21} $atoms/cm^3$ in the lower dose rate sample. Dose rates were varied three orders of magnitude (0.1 to 100 $\mu A/cm^2$) in this work. The amount of residual damage was found to increase continuously over the entire investigated range. The following power law dependence was found to fit the data fairly well:

$$\text{damage} \propto (\text{dose rate})^{0.20}.$$

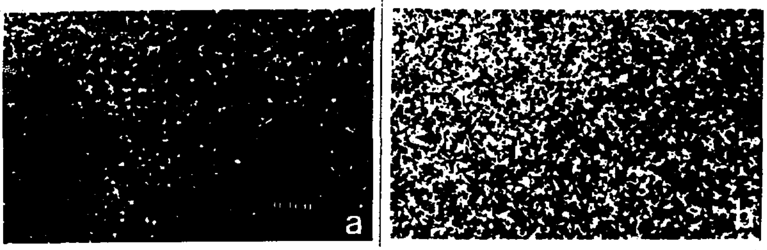

Figure 3

Dark field image taken with s, the deviation parameter, positive for Si irradiated with (a) 0.14 and (b) 70 μA/cm^2.

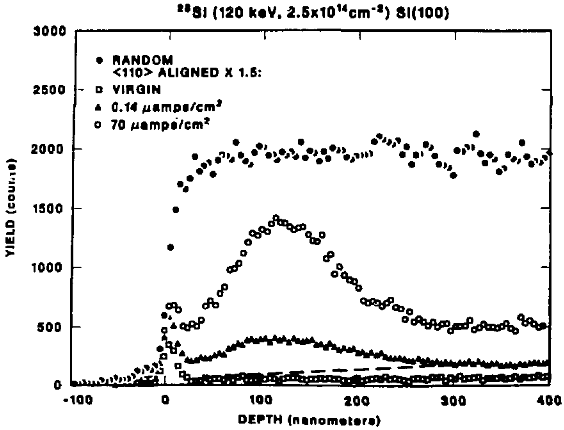

Figure 4

Channeling spectra of samples irradiated at room temperature with 2.5 x 10^{14} Si$^+$/cm^2 at different dose rates.

The magnitude of the dose rate effects was found to decrease with increasing ion mass. For Xe$^+$ ion irradiation the influence of dose rate was minimal. For a dose of 2 x 10^{13} Xe$^+$/cm^2, a change in dose rate by a factor of 50 resulted in only a 15% increase in the observed damage. This corresponds to a power law dependence of 0.04 which is considerably lower than the 0.20 value for Si$^+$ ions in the above expression. Also, for an intermediate mass ion, such as Kr$^+$, an intermediate value of 0.10 was determined. Therefore, as the mass of the ion is increased, the importance of dose rate on damage accumulation decrease.

No such dose rate effects were observed in samples irradiated at LN$_2$, where the basic mechanism for damage nucleation has been shown to be heterogeneous in nature. Channeling spectra in fig. 5 show that the scattering yield from samples irradiated with Si$^+$ ions at LN$_2$ is independent of the dose rate. Even though the dose rate was varied by two orders of magnitude, no difference in the scattering yield (i.e. damage) can be seen.

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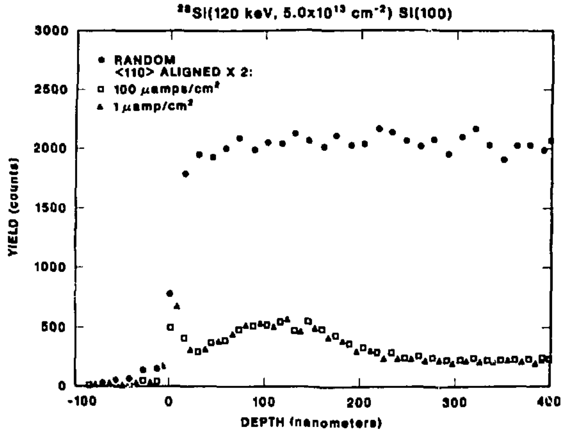

Figure 5. Channeling spectra of samples irradiated at liquid nitrogen temperature with 5 x 10^{13} Si^+/cm^2 at different dose rates.

The presence of a dose rate dependence on damage nucleation in Si at RT indicates a different damage nucleation mechanism is operative than the one previously discussed for LN_2 irradiation. As a result of the increased defect mobility at RT, it is felt that damage is nucleated homogeneously, especially when the defect density within the collision cascade is small as is the case for light ions such as Si^+. This is consistent with the observation of loop-like clusters in the samples irradiated with Si^+ ions, which is very similar to damage observed after electron irradiation at an energy chosen to produce only single atomic displacements (no collision cascade) [15]. Also, we have previously shown [16] that the power law dependence of damage on dose rate, as well as the qualitative differences in the damage (i.e. loop density and radius) in samples, irradiated with widely varying rates, agrees with a homogeneous model of damage nucleation.

CONCLUSION

It has been shown that amorphous damage is produced in Si by ion irradiation at LN_2 with moderate doses of either Bi^+ or Si^+ ions. The damage appears to nucleate about an individual ion track as a result of the passage of a single Bi^+ ion or, in the case of Si^+ ions, cascade overlap. At RT, cascade effects seem to play a decreasing role in damage nucleation, especially for light ions. Clustering of the point defects which survive the cascade quench leads to damage nucleation which can be characterized as homogeneous. The dose rate dependence of damage arises as a result of this homogeneous mechanism. However, as the damage energy density within the cascade increases, a significant amount of damage can be nucleated within the cascade volume. The observed decrease in dose rate effects as the mass of the ion is increased reflects the changing role of the collision cascade in damage nucleation at RT.

ACKNOWLEDGMENT

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