# Cross-sectional transmission electron microscopy analysis of {311} defects from Si implantation into silicon

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(Received 31 October 1997; accepted for publication 13 March 1998)

Transient enhanced diffusion (TED) below the amorphization dose threshold is thought to be caused by the release of interstitials from {311} defects. The interstitials are annihilated by diffusion to and then recombination with the surface of the wafer. This would suggest that the layer of {311} defects formed from an implantation and anneal would dissolve from the surface down. Cross-section transmission electron microscopy (TEM) was used to investigate this hypothesis. It is shown that the {311} defects dissolve uniformly across the layer, and the width of the layer does not change until the {311} defects nearly completely dissolve. The total population was also measured using plan-view TEM, so that the dissolution and distribution functions could be plotted from the same annealing conditions. These data suggest that surface is not the limiting factor in the interstitial removal from {311} defects. © *1998 American Institute of Physics.* [S0003-6951(98)00320-9]

Transient enhanced diffusion (TED) in silicon below amorphization is thought to be controlled by dissolution of {311} defects.[1,2] The Frenkel pair damage from the implant is annealed quickly, leaving behind a concentration of interstitials that mimics the concentration profile of the implanted species. These leftover interstitials are due to dopants (which are primarily found on substitutional sites) displacing silicon lattice atoms. This +1 dose condenses into observable rodlike defects with a {311} habit plane. As the {311} defects dissolve, interstitials are released which enhance the diffusion of dopants. These interstitials are annihilated by diffusion to and recombination with the surface of the wafer. Previous experimental work has indicated that the TED of dopants depends on energy[3–5] and distance to the surface.[6] It has therefore been assumed that the {311} defects would dissolve from the top down, since the surface is a strong sink for interstitials.[6]

To test this hypothesis, a $10^{14}/cm^2$ Si implant at 40 keV was performed into a ⟨100⟩ Czochralski wafer. This implant was then annealed at 750 °C for 15, 45, 90, and 135 min. Both plan-view and TEM cross-sectional samples were prepared. To measure the width of the layers, multiple points were sampled in the cross-sectional pictures. To measure the defect sizes, distribution, and atomic concentration plan-view samples were highlighted, scanned, and then image processing was used to extract the defect and atomic densities.

Figure 1 shows the dissolution curves at 750 °C for both the number of atoms and the number of defects. The {311} defects dissolve with a characteristic time of ~55 min, which is similar to previously reported dissolution times.[1,2] TEM of the defects indicates that they dissolve normally compared to other studies.

Cross-sectional TEM was used to analyze the width of the layers. Repeated measurements at different positions were made to obtain error bars for the approximate width of the layers. Figure 2 plots the distance from the surface of the top and bottom of the defect layer as a function of anneal time at 750 °C. The top and bottom of the layer were obtained by making ten measurements of the position of the layer at different points in the cross-sectional TEM images. These measurements were averaged and are plotted in Fig. 2. The error bars were computed using pooled variance, and represent plus and minus one standard deviation. The layer thickness does not change appreciably until the final time point, 135 min. The {311} defect layer also does not show appreciable decay from either side. The top and bottom positions remain fixed within error bars until the final time point.

If we assume that the surface is an infinitely strong sink

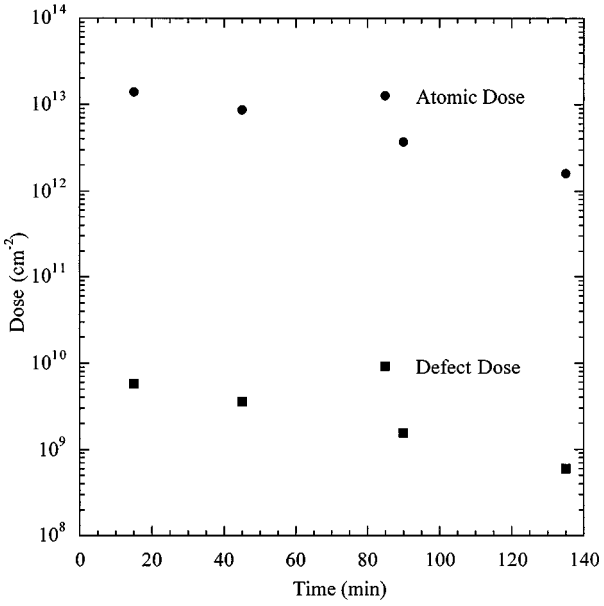


FIG. 1. The number of atoms and defects measured in plan-view TEM as a function of anneal time at 750 °C. Defect dose is the number of defects measured per $cm^2$, and the atomic dose is the number of atoms bound to the defects per $cm^2$.

 

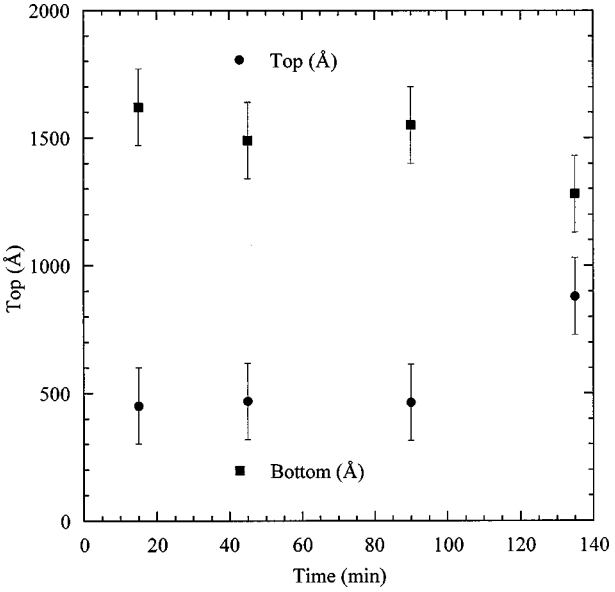


FIG. 2. The position of the top and bottom of the {311} defect layer as measured from the surface of the wafer (depth 0).

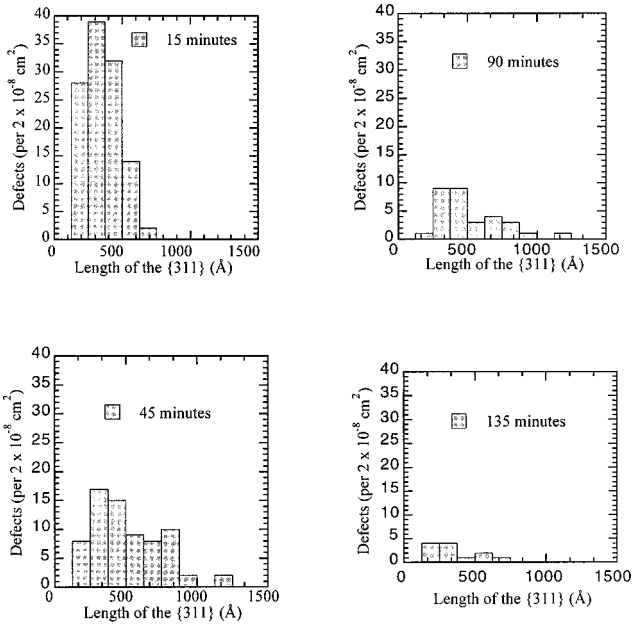


FIG. 3. Histograms of the number of {311} defects found sorted by length.

for interstitials, the interstitial removal from the dissolving layer would be limited by diffusion to the surface. This assumption is supported by previous work on TED.[6,7] In this case, the surface concentration would be pinned at the equilibrium concentration, and interstitial removal would be limited by the rate of diffusion to the surface. The diffusion flux to the surface from any point in the bulk would be inversely proportional to the distance from the surface. The top {311} defects are four times closer to the surface than the bottom {311} defects. The rate of interstitials leaving the front of the defect-rich region should be four times higher than the rate leaving the back of the defect-rich region. Since the source of the interstitials is {311} defects, the defects at the top of the layer must lose atoms at a rate of four times faster than the defects at the back of the layer. Under these conditions, we would expect the defects at the top of the layer to dissolve first. Since 90% of the atoms are removed from the {311}s before the top interface moves, it appears that atoms are equally likely to be lost from the back as from the front. Therefore, the surface does not appear to be the controlling sink for interstitials released from the {311}s.

Figure 3 shows histograms of the length of {311}s as they evolve during time. With the exception of the 15 min time, the defect distributions are very non-Gaussian and exhibit significant skewness to higher defect sizes. The distributions at 45 and 90 min nearly mirror each other except that the 90 min distribution has fewer defects. At 135 min, the defect distribution has been significantly reduced. Figure 4 plots the mean, standard deviation, and maximum size as a function of time. The mean increases slightly from 15 to 45 min, but only on the order of one standard deviation of distribution. The standard deviation remains relatively constant as a function of time, within error bars for the measurement statistics. The maximum size shows marked increase from 15 to 45 min. For the 45 and 90 min case, the maximum defect size is on the order of 75% of the cross-sectional layer thickness.

The standard model of TED assumes that the {311} defects dissolve and release interstitials. These released interstitial are responsible for TED, and recombine at the surface. This model is supported by two separate experimental results. First, TED depends strongly on energy[3,4] and distance to the surface.[6] Therefore, the surface seems to be a likely candidate for the controlling mechanism of interstitial removal. Second, the {311} defect dissolves with a time constant activation energy similar to that for TED.[1,2] This work shows that at 750 °C, the {311} defect dissolution does not appear to be controlled by the surface. This evidence weakens the connection between {311} defects in TED, since the {311} dissolution does not appear to be controlled by the surface and TED is controlled by the surface.[6]

In summary, the {311} dissolution does not appear to be controlled by the surface recombination rate, since the defect layer does not narrow from the surface down. Almost 90% of

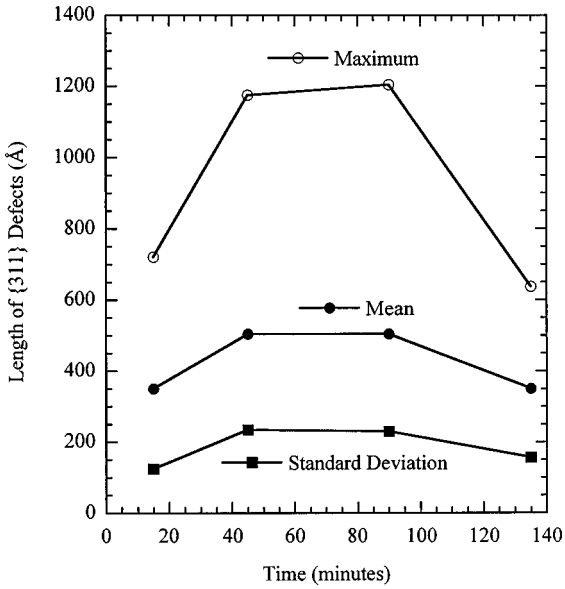


FIG. 4. Plot of the mean, standard deviation, and maximum size of the defect length as a function of time.

the atoms are lost from the {311} defect before the layer dissolves. Histograms show that the distribution of defects is relatively flat at long times. The mean size increases only slightly with time before decaying at long times.

The authors would like to thank the support of the Semiconductor Research Corporation and National Science Foundation.

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