Study of end of range loop interactions with B⁺ implant damage using a boron doped diffusion layer

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A boron doped epilayer was used to investigate the interaction between end of range dislocation loops (formed from Ge⁺ implantation) and excess point defects generated from a low dose 1 \times 10^{15}/cm² B⁺ implant into silicon. The boron doping spikes was grown in by chemical vapor deposition at a depth of 8000 Å below the surface. The intrinsic diffusivity of the boron in the doped epilayer was determined by simply annealing the as-grown layer. The end of range (type II) dislocation loops were created using two overlapping room-temperature Ge⁺ implants of 75 and 190 keV each at a dose of 1 \times 10^{15}/cm². Upon annealing the amorphous layer regrew and a layer of type II dislocation loops formed \approx 2300 Å deep at a density of \approx 8 \times 10^{13}/cm². The enhancement in the buried boron layer diffusivity due to the type II loop formation was observed to increase approximately between 2.5 and 3 min from 1500× to a value 2500× above the intrinsic diffusivity before dropping back to intrinsic levels after 30 min at 800 °C. A low-energy (8 keV) 1 \times 10^{14}/cm² B⁺ (R_p = 320 Å) implant into material without loops resulted in an average enhancement of 1540× in boron epilayer diffusivity after 2.5 min at 800 °C. The enhancement dropped down to intrinsic diffusion levels after 5 min at 800 °C. When a layer of loops was introduced and annealed prior to and deeper than a subsequent low-energy B⁺ implant, annealing of the B⁺ implant produced no measurable enhancement in the buried B layer diffusivity. Taken together this implies that the interaction kinetics between the dislocation loop layer and the damage induced interstitials are primarily diffusion limited and the loops are absorbing a significant fraction of the interstitials produced by the low-energy B⁺ implant. © 1995 American Institute of Physics.

I. INTRODUCTION

In previous reports,¹² dopant transients three orders of magnitude higher than intrinsic diffusion values have been observed following the implantation and anneal treatment of silicon. Studies by Law et al.³ and Packan⁴ suggest that pairing between dopant atoms and the highly mobile point defects generated during the implantation process is responsible for this anomalous movement of the impurity. Because the diffusion transient can induce variations in dopant distribution and concentration that can disrupt normal device operation, enhanced diffusion has become a significant obstacle in attempts to scale down device dimensions.

For nearly two decades, process modeling programs such as the SUPREM³ family of simulators and more recently FLOOPS have proven highly effective in modeling the dopant redistribution associated with implantation and thermal oxidation treatments of silicon. It is recognized that accounting for excess point defects introduced by processing is essential for accurate profile modeling. As device geometries continue to shrink, a knowledge of both the excess point defect concentrations and their interactions with dislocation

loops introduced by ion implantation is essential for accurate simulations using these process modeling codes.

In past studies, methods for measuring point defect perturbations have relied on using either oxidation stacking faults (OSF) or doped marker layers. Antoniadis⁶ used the growth of OSF to monitor the injection rate of point defects (interstitials) during the oxidation of silicon. His calculations suggested that OSF operate in a reaction-rate limited regime and only relative point defect concentrations could be obtained. Though doped marker layers have been successful for deriving point defect (Ci/Ci⁺) values under varying processing conditions, they too are unable to provide actual point-defect flux values.⁷⁸

Previously, we described a technique using type II⁹ (end of range) dislocation loops for quantifying the net flux of interstitials generated from low dose (7 \times 10^{13–2} \times 10^{15}/cm²) B⁺ implants followed by low-temperature (800 °C) annealing treatments.¹⁰ Our results indicated that the number of interstitials trapped by the loops was nearly identical to the respective B⁺ dose, supporting a "+1" model for implant related point defects, but only if the interaction between the interstitials and the loops is diffusion limited.
The interaction kinetics between dislocation loops and point defects has recently been studied during oxidation. Park et al.,11 Roth et al.,12 and Griffin et al.13 used type II dislocation loops in combination with a boron doped marker layer to characterize the point defects generated during wet thermal oxidation of (100) silicon. In these studies, the oxidation-enhanced diffusion (OED) of the marker layer is significantly reduced in the presence of the loop layer. Calculations by Park et al.11 and Roth et al.12 indicate that a larger fraction of the interstitial flux is gettered by the loop layer, in agreement with Meng et al.14 This suggests that point defect-loop interactions are predominantly diffusion limited.14

Oxidation is known to produce a relatively small (10–20X) enhancement in diffusivity that continues for a relatively long time. Ion implantation on the other hand produces a very high super saturation (>1000X) for very short periods of time. In order to study the interaction kinetics between implantation induced point defects and dislocation loops an experimental geometry similar to those used for oxidation studies11,14 was used. This involved the growth of a buried marker layer well below the surface which is used to monitor the concentrations of implanted point defects that get through a layer of dislocation loops. If the interactions between the loops and the interstitials is predominantly diffusion limited, nearly all of the point defects generated during B⁺ implantation/anneal treatment will be captured by the loops and any enhancement of the doped marker due to a lower energy B⁺ implant will be suppressed. In addition, this dual interstitial “detection” system provides a method for comparing the diffusion enhancement (D/Dₜ) value with the net interstitial flux measured by transmission electron microscopy (TEM) under a specific implant/anneal condition.

II. EXPERIMENTS

The substrate used in this study was (100), 11–16 Ω cm, p-type silicon; 5 in. wafers were individually loaded into an atmospheric pressure chemical vapor deposition (APCVD) system outfitted with an Epsilon One Epitaxial Reactor and preannealed at 1150–1190 °C (in a H₂ ambient). An undoped silicon buffer layer 3500–4000 Å in thickness was grown at 850 °C followed by a boron doped marker region, ~1000 Å thick at full width at half maximum (FWHM) with a peak concentration of ~ 7 X 10¹⁷ atoms/cm³. A final 8000 Å undoped epitaxial Si layer was grown over the boron doped epilayer.

A portion of this as-grown boron doped “epi” material was prepared for secondary ion mass spectroscopy (SIMS) analysis while the remainder was subsequently furnace annealed at 800 °C for 10 min (in a dry N₂ ambient) to eliminate any diffusion transient within the doped marker layer prior to postprocessing of the experimental and control samples. In order to characterize the effect of ion implantation damage and/or anneal treatments on marker layer diffusivity, material containing the boron doped epilayer was processed in one of three ways. Samples were either (1) postannealed directly at 800 °C for times of 2.5, 5, 15, 30, 60, and 120 min in a dry N₂ ambient, or (2) implanted with 8 keV B⁺ (1 X 10¹⁴/cm²) then annealed at 800 °C, or (3) amorphpized via dual 1 X 10¹²/cm² Ge⁺ doses at 75 and 190 keV then annealed at 800 °C (Fig. 1). The first set of samples were controls to establish the DB for our material. The second set was used to determine the effect of the low-energy B⁺ implant on the buried marker layer. The third set was used to determine the effect of end of range loop formation on the buried marker layer. All implants were performed at a 7° offset to the sample surface (0° rotation) using a beam current ~0.22 μA/cm² in a Wayne tech® end station. In addition, B⁺ and Ge⁺ implanted and nonimplanted controls were prepared without any postanneal treatment. In the B⁺ implanted doped epilayer material, a dose of (1 X 10¹⁴/cm²) was selected to avoid amorphization and the formation of type I dislocation loops during implant/anneal treatments which would only complicate the problem.

Following ion implantation and/or anneal treatment, the boron profiles of the experimental and control samples were measured using SIMS in a Cameca-3f or 4f system. Diffusion enhancement data for each of the three experimental conditions was calculated by numerically diffusing the respective “control” SIMS profile by a factor sufficient to match the boron epilayer profiles arising from each sample type after each of the six different annealing times studied (Fig. 1). All computations were performed using Florida Object Oriented Device and Process Simulator (FLOOPS) assuming the default boron diffusivity (D_B) value (D_B = 1.7 exp[-3.56 x KT]) of Packan and Plummer.15

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FIG. 1. Schematic of the three types of samples studied.
III. RESULTS AND DISCUSSIONS

The first comparison was between the unimplanted control sample and the default value for the B diffusivity in FLOOPS. The control samples diffusivity was typically found to have a time average enhancement of between 6 and 18X above the default diffusivity for three, six anneal periods investigated in this study. This is not unexpected given the low anneal temperature. Previous cross-sectional TEM analysis revealed the as-grown epi material to be free of structural anomalies which could otherwise induce diffusion transients. In addition, the peak boron concentration of the marker layer (6 × 10^{17}/cm^{2}) was intrinsic (at 800 °C) relative to the carrier concentration of the silicon, thus avoiding the added effects of extrinsic diffusion.\(^{16}\) The enhancement stated hereafter is relative to the boron diffusivity values we measured in the control samples not to the FLOOPS default value. The marker layer diffusivity for the B\(^{+}\) implanted material was determined to be enhanced by the average value of 1540X during the initial 2.5 min anneal treatment and declined to intrinsic levels for heat treatments >5 min. When time averaged enhancement values were calculated for the 8 keV (1 × 10^{15}/cm^{2}) B\(^{+}\) implant condition, the enhancement was observed to decrease by a factor of ~0.5 for anneal periods >2.5 min. in a pattern similar to that recently reported by Griffin et al.\(^{13}\) for low dose Si\(^{+}\) implants into silicon containing a boron marker layer. Though their calculated \(D/D^*\) values were higher than those reported in this study, this variation likely reflects the increase damage potential of the heavier Si atom at higher energies relative to that of B implants in this study. A better picture of the interstitial flux as a function of time can be seen by calculating the differential enhancement in the diffusivity, not the time averaged value. The differential enhancement represents the average enhancement for each time interval not for the total anneal time. These values are plotted in Fig. 2. In the boron doped marker layer material implanted with Ge\(^{+}\), Figure 2 shows a similar differential enhancement of ~1540X was measured during the initial 2.5 min anneal. Unlike the B\(^{+}\) implanted material, the enhancement increased to a differential enhancement value of 2500X during the 2.5-5.0 min interval. Thereafter, the boron diffusion enhancement declined sharply to intrinsic levels when anneals were at least 30 min in length. Compared to the B\(^{+}\) implanted epilayer, enhancement values for the Ge\(^{+}\) implanted material ranged from 1-2 orders of magnitude higher for anneals between 2.5 and 30 min.

Because our ultimate goal in this study was to measure those changes in boron epilayer diffusivity arising from the B\(^{+}\) implant damage with and without loops, the contributions of Ge\(^{+}\) implantation and anneal treatments of these changes had to be fully accounted for. Thus by using the results of Fig. 2, the final anneal times could be optimized to insure the completion of Ge\(^{+}\) and B\(^{+}\) implant transients while avoiding the loss of marker layer sensitivity (profile broadening) due to excessive thermal treatment. Based on these results, the following implant/anneal schedule was devised to evaluate what portion of an 8 keV B\(^{+}\) implant damage flux was being captured by the Ge\(^{+}\) loop layer. The as-grown doped marked material layer was annealed at 800 °C/10 min to eliminate any diffusion transient, implanted with Ge\(^{+}\) to a dose of (1 × 10^{15}/cm^{2}) at 75 and 190 keV, respectively and postannealed at 800 °C/30 min in a dry N\(_2\) ambient. The 30 min removed the effects of the loop formation process on the buried marker layer diffusivity. The depth of the type II loop layer (formed from the Ge\(^{+}\) implant/anneal treatment) was measured to be 2200-2300 Å deep using cross-sectional transmission electron microscopy (XTEM) analysis. A portion of this material was postimplanted with 8 keV B\(^{+}\) to a dose of 1 × 10^{14}/cm^{2} and annealed at 800 °C for 2.5 min in a dry N\(_2\) ambient. The specimen was annealed under the identical furnace conditions without the 8 keV B\(^{+}\) implant. The energy and dose of the B implant was chosen such that the entire B profile is contained within the first 2300 Å.

Following the procedure outlined, B\(^{+}\) implanted and nonimplanted samples (fabricated with type II loops and a boron epilayer) were analyzed using SIMS. FLOOPS simulations of this data detected no difference in the boron marker layer profile (boron diffusivity) between the samples containing B\(^{+}\) implant damage and a type II loop layer versus those containing type II loops without B\(^{+}\) implantation. By measuring the size of the loops before and after B implantation, it is possible to determine the flux of interstitials trapped by the loops. Figure 3 shows plan-view TEM micro-

![Graph comparing the differential enhancement of intrinsic, B\(^{+}\) implanted and Ge\(^{+}\) implanted boron doped epi material following anneal treatments from 2.5-120 min at 800 °C.](image)

![Plan-view TEM micrographs of samples of Si after 75 keV Ge\(^{+}\) 1 × 10^{15}/cm^{2} + 190 keV Ge\(^{+}\) 1 × 10^{15}/cm^{2} and annealed at 800 °C 30 min followed by (a) another anneal at 800 °C for 2.5 min, (b) 8 keV B\(^{+}\), 1 × 10^{14}/cm^{2} implant and then annealed at 800 °C for 2.5 min.](image)
graphs of the loop layer after 800 °C 30 min annealing and an additional 800 °C 2.5 min anneal for both the B implanted and unimplanted samples. The net flux of interstitials (from B+ implant damage) trapped by the type II loops was determined to be \( \approx 4 \times 10^{13}/\text{cm}^2 \) using the method described in a previous report.\(^{10}\)

There are several interesting aspects of this experiment. This study is the first of its kind in investigating the enhancement of a boron doped epilayer upon annealing an amorphizing Ge+ implant. The increase in enhancement from 1500X to 2500X during the first 5 min of annealing is believed to arise from the point defect flux dynamics associated with the growth and coarsening of the type II loops and possibly the annealing of (311) defects in the end of range region.\(^{17,18}\)

This is supported by TEM measurements of the same samples that indicate the loops are still growing over the first 5 min time interval. After 5 min, the loops enter a coarsening stage and the enhancement drops down to values similar to the control sample. Despite differences in mass, dose and energy, it is interesting to note that the nonamorphizing, non-loop-forming B+ implants had about the same enhancement as the Ge+ implant over the first 2.5 min of annealing.

Recently, Griffin et al.\(^{13}\) reported less boron marker layer diffusion (for anneal times \( \geq 30 \) min at 750 °C) when amorphizing Si+ implant doses \( (8 \times 10^{14} \text{ and } 2 \times 10^{15}/\text{cm}^2) \) were compared with subamorphizing threshold Si+ doses of \( (1-2 \times 10^{14}/\text{cm}^2) \). This reduction in enhancement (for the amorphizing condition) is attributed to less residual damage being left behind during regrowth and extended defect formation. The observation in the present study that Ge+ implants actually produce a greater enhancement than the B+ implants is most likely due to the decreased energy and dose of our B implants.

As stated, the enhancements associated with the B+ implanted and non-B+ implanted epilayers (both containing type II loops) were found to be nearly identical. Due to broadening of the B profile from the Ge+ implant and anneal, it was concluded (via simulations) that the sensitivity of the delta doped layer was such that only change greater than 400X could be detected. Thus during the first 2.5 min at 800 °C, the presence of the type II loop layer decreased the diffusivity enhancement from 1540 to less than 400X. In addition, the plan-view TEM results in Fig. 3 showed a net increase in the trapped interstitials of \( 3.7 \times 10^{13}/\text{cm}^2 \). This provides rather strong evidence that the majority of the implant damage is being trapped by the loops.

Contrary to our previous results,\(^{10}\) the net flux \( (3.7 \times 10^{13}/\text{cm}^2) \) of interstitials captured by the type II loops was significantly less than the "1 model" would predict for a \( 1 \times 10^{14}/\text{cm}^2 \) B+ dose. From the studies of the boron implant alone, it is apparent that most of the enhancement is complete after the first 2.5 min at 800 °C. So, it is unlikely that the smaller concentration of interstitials is due to insufficient annealing. We have conducted several studies\(^{19}\) on the effect of loop position and determined that when the loops are below the damaged layer as opposed to being at \( R_p \) of the B, the concentration of trapped interstitials decreases from near +0.5 at \( R_p \) to +0.2 at \( R_p +1000 \) Å to +0.15 at \( R_p +2000 \) Å for low-energy (8 keV) \( 2 \times 10^{14}/\text{cm}^2 \) B+ implanted plants. These results are consistent with the reduced interstitial flux measured in this study. Surface recombination may be the reason for the reduced flux measured with increasing loop depth.

The observed decline in marker layer diffusivity from B implantation damage when a loop layer is present is not surprising. Previous studies have reported that a large number of the interstitials generated during the implantation\(^{10}\) and oxidation\(^{14,20}\) of silicon are captured by these extended defects. Roth et al.\(^{12}\) used a similar loop layer and doped epilayer structure to measure the enhanced diffusion behavior associated with wet oxidation of silicon at 900 °C. They reported a significant decline in boron epilayer enhancement when the loop layer was present, corresponding to a 70%-80% reduction in the excess interstitial concentration. Together, these studies of oxidation and implantation have been particularly useful in comparing loop behavior under varying extremes in point defects concentration. However, because the ion implantation typically yields a point defect supersaturation \( (C/C_i ^*) \) two orders of magnitude higher than oxidation conditions, it probably provides a more sensitive method for discerning the rate-limiting mechanism involved during point defect dislocation loop interactions.

IV. CONCLUSION

In conclusion, we have demonstrated that type II loops are effective in capturing most if not all of the point defects generated from a low dose (nonamorphizing) B+ implant. This implies that during annealing of low-energy B implants the interaction between the implantation induced interstitials and a layer of type II loops is predominantly diffusion limited. It also implies that the loops are effective diffusion barriers for anomalous diffusion associated with the annealing of implantation damage. What is still unknown is how the presence of the loops affects to the point defect recombination process during transient enhanced diffusion. Experiments to determine this are in progress.

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