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Studies of the interactions between (311) defects and type I and II dislocation loops in Si⁺ implanted silicon

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Abstract

Silicon wafers were implanted with Si⁺ at doses of 2×10^{14} and $1 \times 10^{15}/\text{cm}^2$. Annealing treatments were done at temperatures between 700°C and 1000°C for times between 15 min and 16 h, both with and without an SiO₂ cap. Plan-view TEM micrographs were taken and the density of interstitials trapped in both the (311) defects and the type I and II perfect loops were measured. The results showed that for the $2 \times 10^{14}/\text{cm}^2$ Si⁺ dose, which is below the amorphization threshold, the dominant defect at 700°C is the (311) defect with a much smaller concentration of type I loops. The total trapped interstitial concentration in both kinds of defects was around $7 \times 10^{13}/\text{cm}^2$ for 700°C 1 h anneals. The (311) defects begin dissolving after several hours at 700°C but their dissolution rate is slower than previously reported by Stolk et al. [MRS Symp. Proc. 354 (1995)] for lower dose ($5 \times 10^{13}/\text{cm}^2$) implants. It is not believed that this slower dissolution rate is due to the increased dose. The reduced dissolution rate does not change with capping and may be due to a difference in furnace calibration methods. The type I loops show some growth during the (311) dissolution but quantitatively less than half of the released interstitials appear to be trapped by the type I loops. For the $1 \times 10^{15}/\text{cm}^2$ sample amorphization occurs and both type II (end of range) loops and (311) defects are observed for 700°C anneals. The total number of trapped interstitials for 700°C 1 h anneals is also around $7 \times 10^{13}/\text{cm}^2$. However, the ratio of (311) to loops has switched such that the dominant defect is the type II loop. Upon annealing, the (311) defects again show a reduced dissolution rate and the type II loops are in the growth regime. Increasing the anneal temperature to 800°C results in further growth of the type II loops and all of the (311) defects have either dissolved or unfaulted. The growth of the type II loops appears to be greater than can be quantitatively accounted for by the (311) defects. In addition there is a high level of strain in the lattice that cannot be accounted for by the (311) defects. Both of these results imply there may be an additional source of interstitials besides the (311) defects for amorphizing implants.

1. Introduction

During the ion implantation doping process in semiconductors, various levels of damage can be done to the lattice. In silicon we typically divide these levels of damage into three regimes. The first regime is when the damage is below the threshold dose for formation of a continuous amorphous layer and upon high temperature annealing (e.g. 800–900°C) no dislocation loops are observed to form (i.e. below the type I defect formation threshold). This typically is for doses below $\leq 1 \times 10^{14}/\text{cm}^2$ [2]. The second regime is above the threshold for forming type I dislocation loops but still below the dose necessary for forming a continuous amorphous layer. Typically for Si⁺ implants this dose regime is $\geq 2 \times 10^{14}/\text{cm}^2$ but $< 1 \times 10^{15}/\text{cm}^2$. For B⁺ implants this is for any dose above $2 \times 10^{14}/\text{cm}^2$.

Finally, the third dose regime is for implants where the damage density is sufficient to form a continuous amorphous layer. Upon annealing, type II (end of range) dislocation loops are observed to form.

It has also been known for a number of years that upon annealing transient enhanced diffusion (TED) of the implanted dopant can occur [3,4]. This TED has been attributed to damage from the implant which evolves into a supersaturation of excess interstitials upon annealing. It has been suspected for years that the extended defects which form and dissolve upon annealing could be responsible for the anomalous diffusion behavior. However, in general, the perfect and faulted type I and II dislocation loops dissolve at temperatures much too high ($> 1000^\circ\text{C}$) [5] to explain TED which is over after times as short as 30 min at 800°C. A second form of extended defect that has been observed for a number of years is the so-called (311) defect [6]. This rodlike defect is believed to be a layer of hexagonal silicon [7,8] whose morphology provides a low energy means of accommodating excess interstitials in

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silicon. These defects have been known for years to be very unstable although no quantitative studies of their annealing kinetics were done. Recently, there has been a renewed interest in (311) defects. Eaglesham et al. [9]

and Stolk et al. [1] have shown quantitatively that the dissolution of (311) defects from $5 \times 10^{13}/\text{cm}^2$ implants occurs over the same time/temperature regime as the TED process. This provides very good evidence

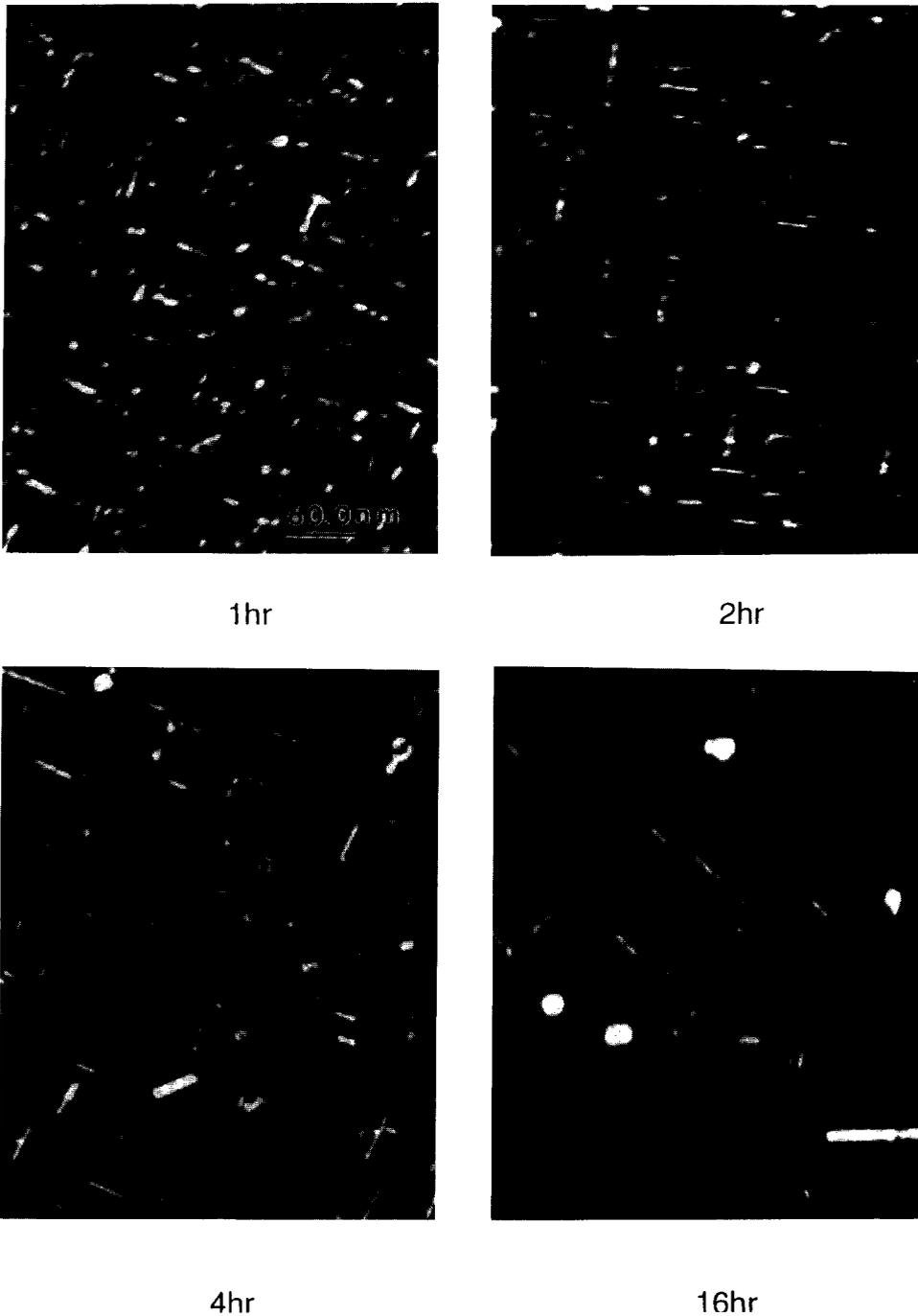


Fig. 1. PTEM images of dislocation loops and (311) defects in 50 keV Si⁺ implanted Si at $2 \times 10^{14}/\text{cm}^2$ and annealed at 700°C for 1, 2, 4 and 16 h, respectively.

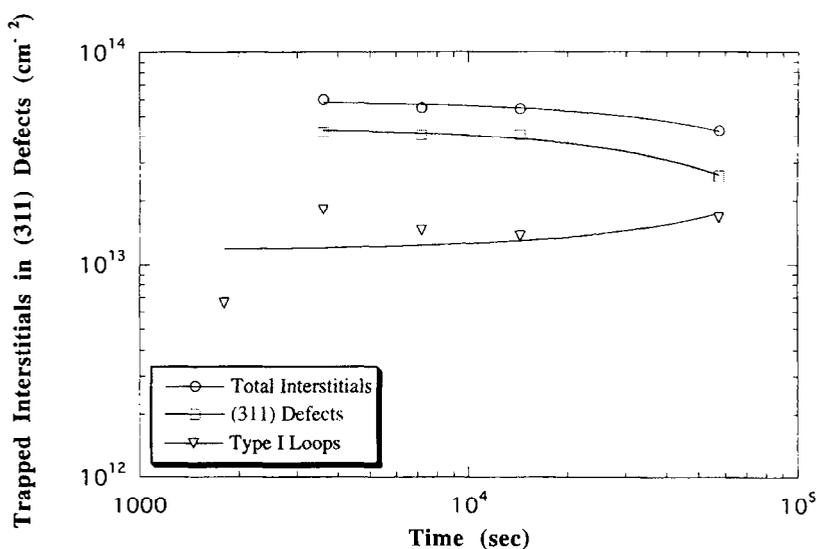


Fig. 2. Trapped interstitials for a $2 \times 10^{14}/\text{cm}^2$ implant.

that the excess interstitials, which are necessary for TED, evolve from nonvisible clusters to (113) defects and these (311) defects then provide the excess interstitials for TED upon their dissolution. These studies have all been intentionally conducted at doses below the critical dose necessary for type I dislocation loops ($\geq 2 \times 10^{14}/\text{cm}^2$) in order to avoid the conflict of having type I or type II dislocation loops. These lower doses are important for modeling the light doped drain regions of devices. However, accurate modeling of diffusion below the heavily doped source/drain contact regions is also critical. These low resistivity contacts require higher doses. In this paper we begin to explore the annealing kinetics of these (311) defects in the higher dose regime where type I and II loops are also evolving.

2. Experimental

Undoped (100) Czochralski grown Si wafers were implanted with 50 keV $^{29}\text{Si}^+$ ions at a dose of $2 \times 10^{14}/\text{cm}^2$ ($4 \mu\text{A}/\text{cm}^2$) or 50 keV $^{28}\text{Si}^+$ ions at a dose of $1 \times 10^{15}/\text{cm}^2$ ($20 \mu\text{A}/\text{cm}^2$) at room temperature using a Wayflow endstation. The wafers were then capped with a CVD SiO_2 layer of 5000 Å to prevent possible oxidation during furnace annealing. The furnace annealing was done between 15 min and 16 h at temperatures between 700 and 1000°C in a forming gas. After annealing, plan-view TEM samples were prepared using the standard jet etching with HF:HNO₃ solution. TEM micrographs using g_{220} weak beam dark field imaging conditions were taken on either a JEOL 200CX or a JEOL 4000FX TEM. The (311) width from

cross-sectional TEM was seen to be independent of annealing time and temperature before unfauling. Thus, a value of 24 interstitials nm of (311) defect (determined from the width and the density of interstitials on the (311) plane) was used. Trapped interstitial concentrations in the (311) defects were determined by multiplying the total length of (311) defects measured, compensating for projection error, by this constant. This is the same method used by Eaglesham et al. [9]. Determination of the trapped interstitials in the perfect loops has been discussed previously [10]. In addition, high resolution X-ray rocking curve measurements of the strain due to the (311) defects and the loops in the samples have also been made for some of the samples [11].

3. Results and discussion

The results if Fig. 1 show that for the $2 \times 10^{14}/\text{cm}^2$ Si^+ dose, which is below the amorphization threshold, the dominant defect at 700°C is the (311) defect with a much smaller concentration of type I loops. The total trapped interstitial concentration in both kinds of defects was around $7 \times 10^{13}/\text{cm}^2$ for 700°C 1 h anneals. The (311) defects begin dissolving after several hours at 700°C (Fig. 2) but their dissolution rate is slower than previously reported by Stolk et al. [1] for lower dose ($5 \times 10^{13}/\text{cm}^2$) implants. The variation in the thickness of the SiO_2 cap between no cap and 5500 Å had no effect on the (311) dissolution rate at 700°C. It is not believed that this slower dissolution rate is due to the increased dose. The reduced dissolution rate does not change with capping and may be due to a difference in

furnace calibration methods. The type I loops show some growth during the (311) dissolution but quantitatively less than half of the released interstitials appear to be trapped by the type I loops. This implies that exceeding the type I loop formation threshold may

have some effect on the amount of transient enhanced diffusion (TED) but will not stop TED.

For the $1 \times 10^{15}/\text{cm}^2$ sample amorphization occurs. Upon annealing, Fig. 3 shows that both type II (end of range) loops and (311) defects are observed for 700°C

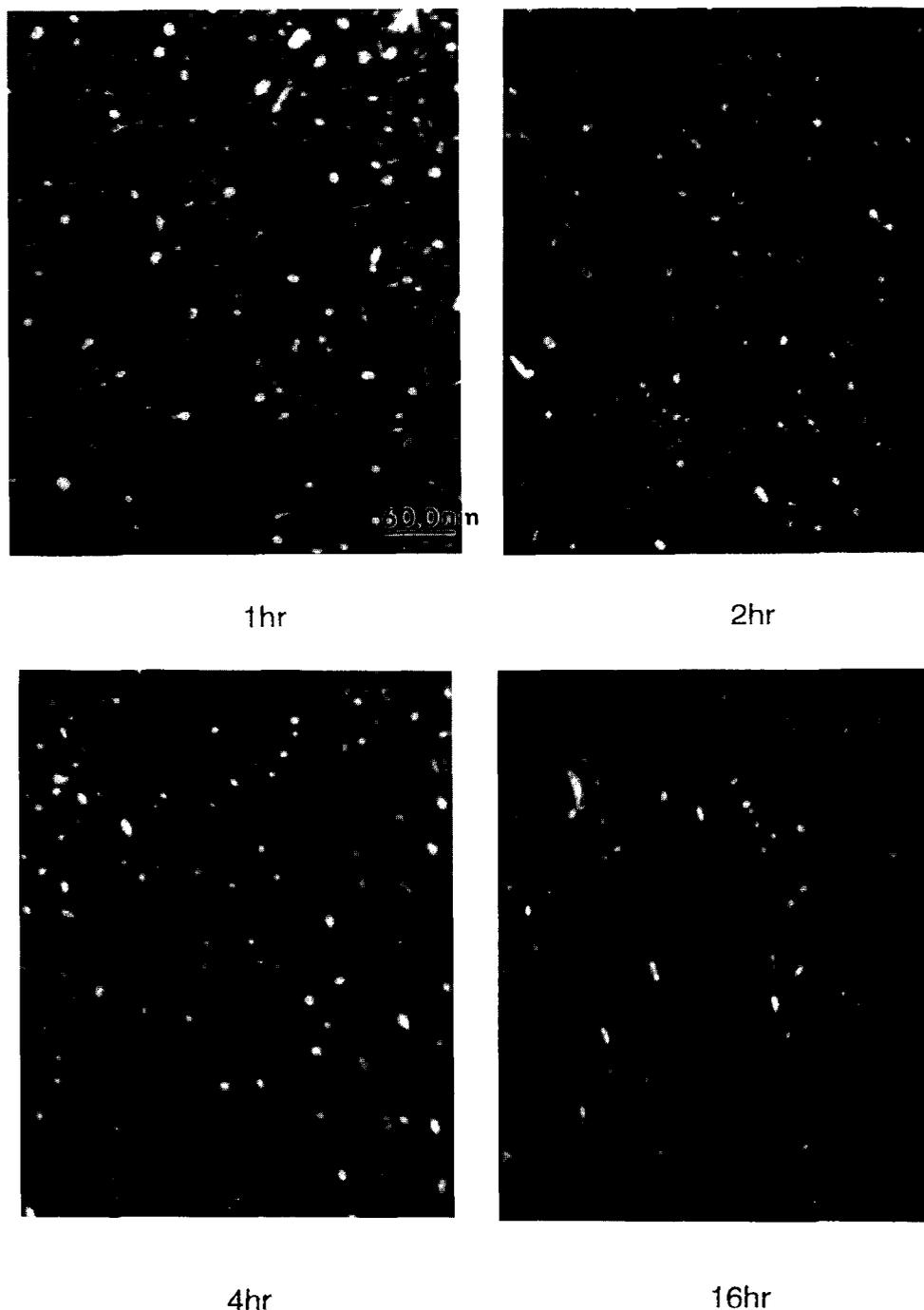


Fig. 3. TEM images of dislocation loops and (311) defects in 50 keV Si⁺ implanted Si at $1 \times 10^{15}/\text{cm}^2$ and annealed at 700°C for 1, 2, 4 and 16 h, respectively.

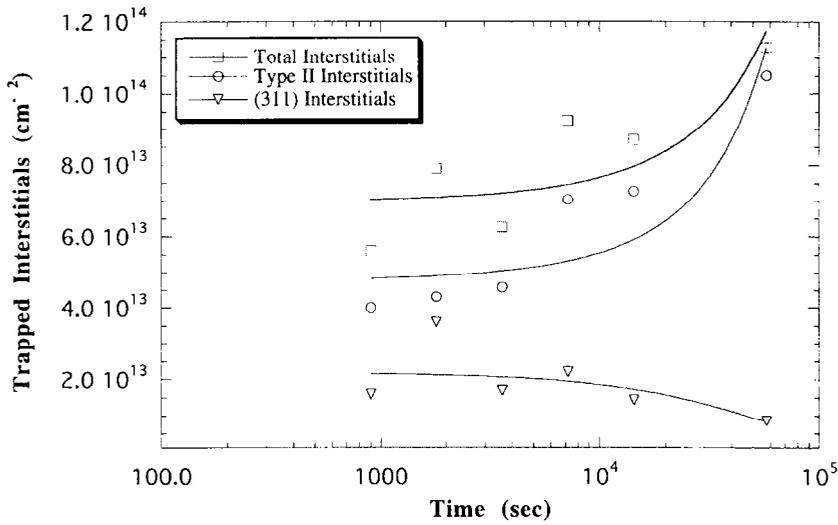


Fig. 4. Trapped interstitials for $1 \times 10^{15}/\text{cm}^2$ implant.

anneals. The total number of trapped interstitials for 700°C 1 h anneals is also around $7 \times 10^{13}/\text{cm}^2$. However the ratio of (311) to loops has switched such that the dominant defect is the type II loop. Upon annealing, Fig. 4 shows that the (311) defects again show a reduced dissolution rate and the type II loops are in the growth regime. Increasing the anneal temperature to 800°C results in further growth of the type II loops and all of the (311) defects have either dissolved or unfaulted. The growth of the type II loops appears to

be greater than can be quantitatively accounted for by the (311) defects.

To further investigate this, high resolution X-ray rocking curve measurements were done of both after 700°C 2 h annealing. It is interesting to note that both samples showed the same trapped interstitial concentration but the ratio of (311) defects to (110) loops was very different as previously mentioned. The HRXRD measurements showed a strain of around 5050 ppm for the (004) reflection for the sample with predominantly

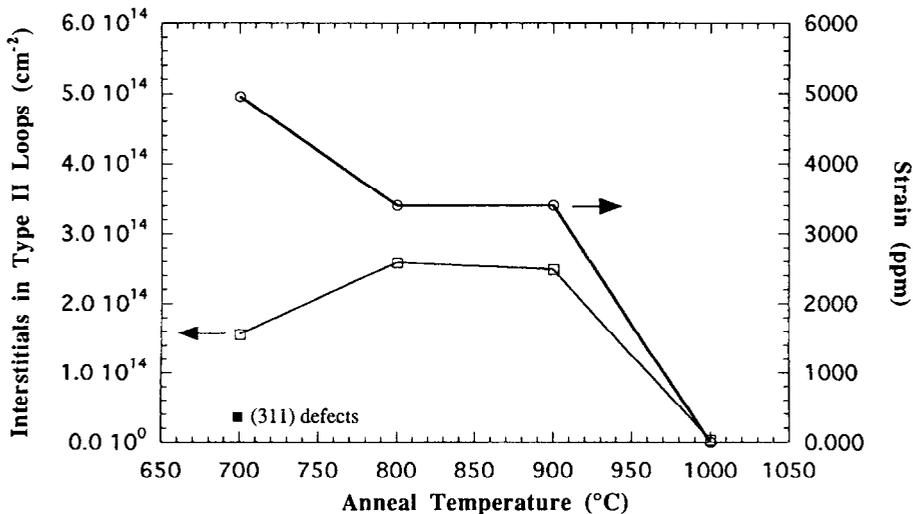


Fig. 5. Strain vs. the number of interstitials in type II loops $1 \times 10^{15}/\text{cm}^2$ Si^+ 50 keV, 2 h anneals.

(110) loops (high dose) and less than 1000 ppm of strain for the sample with predominantly (311) defects (low dose). Fig. 5 shows that upon further annealing at higher temperatures the type II loops continue to grow. This growth cannot be explained quantitatively by the dissolution of (311) defects. In addition, there is a very high strain at 700°C that cannot be accounted for by the interstitials trapped in the type II loops. As previously stated, the (311) defects introduce very little strain per interstitial thus the (311) defects cannot explain the high strain. Both the growth of the type II loops between 700 and 800°C annealing and the unaccounted strain imply there may be an additional source of interstitials. It will be important to account for this damage if accurate modeling of the effect of end of range damage on TED is to be realized.

4. Conclusions

The effect of increasing the dose above the type I and type II formation threshold on the (311) annealing kinetics has been studied. While some variation in the (311) dissolution rate relative to previous reports was observed, these are not believed to be dose related and may in fact be related to differences in furnace calibration methods. The formation of type I loops does not result in complete trapping of the interstitials released by the (311) defects and as such is not expected to completely suppress TED. For the higher dose (amorphized) sample, the type II loops were the dominant defects and not the (311) defects. Upon annealing, the growth of the type II loops is greater than can be

explained by (311) dissolution and the strain measurements imply there may be an additional source of interstitials feeding the type II loop growth.

Acknowledgements

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